

**Medicating Race:
Heart Disease and Durable Preoccupations with Difference**

by
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Submitted to the Program in Science, Technology and Society
In Partial Fulfillment of the Requirements of the Degree of
Doctor of Philosophy in the History and Social Study of Science and Technology
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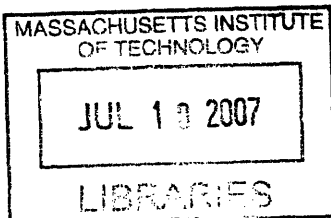
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Abstract

This dissertation is an examination of intersections of race, pharmaceuticals, and heart disease over the course of the 20th century and today. Each of these parts has had a dynamic history, and when they are invoked together they provide a terrain for arguments about interventions in health and in justice in the present.

An enduring aspect of discourses of heart disease over the past century has been articulating connections between characterizations of the modern American way of life and of heart disease. In that process, heart disease research and practice has participated in differentiating Americans, especially by race. This dissertation uses heart disease categories and the drugs prescribed for them as windows into racialized medicine.

The chapters are organized in a way that is roughly chronological, beginning with the emergence of cardiology as a specialty just before World War II and the landmark longitudinal Framingham Heart Study that began shortly thereafter. A central chapter tracks the emergence and mobilization of African American hypertension as a disease category since the 1960s. Two final chapters attend to current racial invocations of two pharmaceuticals: thiazide and BiDil. Using methods from critical historiography of race, anthropology, and science studies, this thesis provides an account of race in medicine with interdisciplinary relevance.

By attending to continuities and discontinuities over the period, this thesis illustrates that race in heart disease research and practice has been a durable preoccupation. Racialized medicine has used epistemologically eclectic notions of race, drawing variously on heterogeneous aspects that are both material and semiotic. This underlying ambiguity is central to the productivity of the recorded category of race. American practices of medicating race have also been mediating it, arbitrating and intervening on new and renewed articulations of inclusion and difference in democratic and racialized American ways of life.

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Chapter 1

Introduction

On November 15, 1915, a front-page obituary in the *New York Times* reported the death of Booker T. Washington, the most prominent black leader of the turn of the 20th century:

Booker T. Washington, foremost teacher and leader of the negro race, died early today at his home here, near the Tuskegee Institute, which he founded and of which he was president. Hardening of the arteries, following a nervous breakdown, caused his death...¹

Yet in the weeks that followed, rumors circulated questioning the diagnosis of arteriosclerosis. One of Washington's physicians publicly stated that his illness and death had in fact been caused in part by "racial characteristics," a phrase that was widely perceived to be insinuating that Washington had died due to syphilis.² Both the indiscretion of his doctor and the facts of the case were contested at the time, and have provided fodder for historical analysis.³

In 2006, a review of Washington's medical records was conducted by a team that included a historian, a hypertension specialist, and an infectious disease specialist, with

¹ "Dr. B. T. Washington, Negro Leader, Dead : Founder of the Tuskegee Institute Expires of Hardening of Arteries After Brief Illness : Taken to His Home To Die," *New York Times*, November 15, 1915, p. 1.

² Louis R. Harlan, *Booker T. Washington: The Wizard of Tuskegee, 1901-1915* (New York: Oxford University Press, 1983), 451-453.

³ Louis R. Harlan and Raymond W. Smock, "Introduction," in *The Booker T. Washington Papers: Volume 13: 1913-1914*, ed. Louis R. Harlan and Raymond W. Smock (Urbana and Chicago: University of Illinois Press 1984), xxv.

the support and cooperation of Washington's descendents.⁴ In the report of their reinvestigation, published in the *Journal of Clinical Hypertension*, the investigators reviewed the facts from Washington's medical files in light of today's methods of diagnosis. Among the highlights included were his history of increasingly severe indigestion, fatigue, and headaches; his physical exam description as "a middle-aged African American man variously described as having the 'medium brown skin of a mulatto...luminous eyes... a rather Irish face...and the odd look of an Italian'"; eye and heart abnormalities; very high blood pressure; and a negative Wasserman (syphilis) test.⁵ They reached the conclusion:

Mr. Washington did not have syphilis. His negative Wasserman test proves that. Rather, as indicated in the hospital record made public for the first time in this conference, he had malignant hypertension, which destroyed his kidneys, damaged his heart, and eventually killed him.⁶

In one sense, this article settles the debate. It uses data to claim a definitive diagnosis of hypertension in place of a suggested one of syphilis. But with regard to the larger question of whether Washington's death was related to "racial characteristics," it renders the debate unsettled:

Were 'racial characteristics...in part responsible'? The data reviewed by Dr. Wright suggest that, indeed, there might be endogenous factors peculiar to

⁴ Jackson T. Wright, Jr., W. Fitzhugh Brundage, and Philip A. Mackowiak, "A 59-Year-Old Man with 'Racial Characteristics,'" *The Journal of Clinical Hypertension* 9 (2007): 128-133. The original case discussion took place at a conference sponsored by the Department of Veterans Affairs Maryland Health Care System and the University of Maryland School of Medicine in Baltimore, MD, on May 5, 2006. More thorough treatment of the investigation is forthcoming in a collection of examinations of mysterious deaths of a dozen prominent historical figures ranging from Alexander the Great to Edgar Allen Poe, Philip A. Mackowiak, *Post-Mortem: Solving History's Great Medical Mysteries* (Philadelphia: American College of Physicians, 2007).

⁵ Other details included in the account included the limited knowledge of his family history; lack of oedema, and lack of abnormalities of external genitalia. Jackson T. Wright, Jr., W. Fitzhugh Brundage, and Philip A. Mackowiak, "A 59-Year-Old Man with 'Racial Characteristics,'" *The Journal of Clinical Hypertension* 9 (2007): 128-9.

⁶ Jackson T. Wright, Jr., W. Fitzhugh Brundage, and Philip A. Mackowiak, "A 59-Year-Old Man with 'Racial Characteristics,'" *The Journal of Clinical Hypertension* 9 (2007): 132.

African Americans that are responsible, at least in part, for the common occurrence of hypertension and its adverse consequences.⁷

Thus, the article both settles and unsettles the relationship between Booker T.

Washington's race and his death, as well as the relationships between African American race and morbidity today. The authors did not reject racialized diagnosis per se, but replaced one outdated and highly-stigmatized racialized diagnosis with a current less stigmatized one.

From this refashioned sense of 'racial characteristics,' the authors considered connections they saw as relevant today: implications for treatment, and the role of genetics. The re-diagnosis was performed by prominent black hypertension specialist Jackson T. Wright, who took the opportunity to both consider the case at hand and expound upon the challenge of African American hypertension generally. Noting that in 1915 there were neither effective treatments for hypertension nor even the awareness of the need for treatment, Wright pointed out that despite our increased knowledge and tools, blood pressure control remains a challenge today, "especially in black populations." Wright offered pragmatic advice for effective treatment through multi-drug therapy, weight loss, exercise, and salt restriction, and offered an array of possible explanations for the disparity, such as socioeconomic status, environmental factors, biomarkers, and "a yet-to-be-characterized genetic susceptibility."⁸ Wright's focus was on the facts of disparities and treatment avenues, remaining agnostic on etiology:

⁷ Jackson T. Wright, Jr., W. Fitzhugh Brundage, and Philip A. Mackowiak, "A 59-Year-Old Man with 'Racial Characteristics,'" *The Journal of Clinical Hypertension* 9 (2007): 132.

⁸ Jackson T. Wright, Jr., W. Fitzhugh Brundage, and Philip A. Mackowiak, "A 59-Year-Old Man with 'Racial Characteristics,'" *The Journal of Clinical Hypertension* 9 (2007): 130.

Hypertension arises more often and at an earlier age, is more severe, and causes more target organ damage in African Americans than whites, for reasons that are nearly as uncertain in 2007 as in 1915.⁹

He concluded: “Hypertension shortened the life of Booker T. Washington, just as it continues to do in poorly treated hypertension patients.”¹⁰

Of all of the possible etiological factors Wright described, his coauthor Mackowiak singled out genetics for further comment, considering and then rejecting it as an explanation for African American hypertension. He noted the poor correlation between racial variation and genetic variation and then elaborated:

Mr. Washington was the product of an African (slave) mother and a white father. If his ‘rather Irish face’ or ‘odd look of an Italian’ were indicative of his father’s heritage, would he not have been as Irish American or Italian American as an African American in terms of his genetic composition (ie, his ‘racial characteristics’)? In view of such ambiguities, it is no wonder we are not much closer today than we were in 1915 to understanding why hypertension singled out this prominent African American or why it continues to be so severe a problem in African Americans.”¹¹

The positioning of “genetic composition” and “racial characteristics” around an “ie” in this analysis is a tantalizing construction of a transhistorical synonym. What holds the place opposite “racial characteristics” has changed: infection, related to hygiene, susceptibility, and immorality, has been displaced by hypertension that is somehow related to genetics.

The time span between Booker T. Washington’s death and its reinvestigation is the same one as this dissertation, and the intervening space serves as my terrain. The changing assessment of his cause of death and its meanings has a similar trajectory to that

⁹ Jackson T. Wright, Jr., W. Fitzhugh Brundage, and Philip A. Mackowiak, “A 59-Year-Old Man with ‘Racial Characteristics,’” *The Journal of Clinical Hypertension* 9 (2007): 130.

¹⁰ Jackson T. Wright, Jr., W. Fitzhugh Brundage, and Philip A. Mackowiak, “A 59-Year-Old Man with ‘Racial Characteristics,’” *The Journal of Clinical Hypertension* 9 (2007): 131.

¹¹ Jackson T. Wright, Jr., W. Fitzhugh Brundage, and Philip A. Mackowiak, “A 59-Year-Old Man with ‘Racial Characteristics,’” *The Journal of Clinical Hypertension* 9 (2007): 132.

of my thesis. The concept of racial characteristics remains durable even as the content of those characteristics has changed, and Booker T. Washington remains “racially characteristic.” In this awkward transformation we can see both the malleability and the durability of preoccupations with race in diagnosis. The diagnosis is durable enough to allow Wright to use it as a basis from which to advocate for treatment in the present, even as it continues to harbor unsettled epistemological quandaries.

* * *

Opening up the temporal and thematic space between the death of Booker T. Washington and its reinvestigation, this dissertation is an examination of the intersections of race, pharmaceuticals, and heart disease over the course of the 20th century and today. Each of these parts has had a dynamic history, and when they are invoked together they provide a terrain for arguments about interventions in health and in justice in the present. As an American story about race, discourses of black/white differences are central. Since my interest is in medicine as both a science that observes the natural world and as a practice that intervenes on it, pharmaceuticals become important objects of analysis. Heart disease serves as my focus because it has become both a major focus of medical practice in the US over the period, especially with regard to pharmaceutical practice, and a central topic for arguments about health disparities. These intersecting and mutually constituting trajectories provide an excellent site to analyze the ambiguity, durability, and mobility of preoccupations with race in medicine.

An enduring aspect of cardiology's project since its inception as a field of research has been theorizing connections between that which is characterized as the modern American way of life and heart disease. Another, related, aspect of cardiology's project has been to differentiate Americans, especially by race. These big questions have not been idle speculations of natural scientists observing the world, but have been enrolled in pragmatic arguments about what should be done in cardiology and beyond. Race in heart disease research and practice is *durable*: it cannot easily be gotten rid of. It is also a *preoccupation*: it cannot easily be let go. New and renewed articulations of inclusion and difference in cardiovascular research and pharmaceutical practices have been part of articulating democratic and racialized American ways of life.

The structure of this dissertation is roughly chronological, beginning early in the 20th century and ending in 2007. After an introductory chapter, Chapter Two considers preoccupations with coronary heart disease, race, and modernity by the founders of cardiology, their contemporaries in the period between 1910 and World War II, and their historians. I read these sources as theorists of a cardiovascularized modernity, in which both differential disease and differential progress were racialized. First, I will attend to aspects of the modernity that they described as connected to heart disease: related to particular strains of mental labor, requiring technologically sophisticated professional medicine, and rising due to the decline in infectious disease. Then I draw those both implicitly and explicitly white arguments about heart disease and modernity into dialogue with arguments about blacks in the period, which posited blacks as not modern in those terms: blacks were represented as not engaged in mental labor, in need of only basic sanitary medicine, and as particularly infectious. Claims of racial difference in heart

disease etiology were present at the founding of heart disease research. The taxonomy of differential etiology by race of the period – that posited whiteness as connected to (non-infectious) coronary disease and blackness with (infectious) rheumatic and especially syphilitic heart disease – maps to enduring distinctions today between white/coronary and black/hypertensive that have been durable in the face of vociferous critique.

The third chapter analyzes the contingent and emergent racialization of the Framingham Heart Study, which was an extremely influential longitudinal study of a Massachusetts town that began just after World War II. The chapter argues that the homogenous whiteness of Framingham arose only as its early investigators both disavowed the imperative to representation and maintained their embrace of extrapolation, and as intra-white ethnic difference was becoming less salient in describing difference among Americans. The chapter also draws the Framingham study into comparative relief with the 2000-present all-black Jackson Heart Study. The latter does not merely repeat Framingham in a new population, but is rather a self-consciously postmodern repetition with a difference. The Jackson Heart Study both supplements and fragments notions of who can stand in as a typical enough American from whom to extrapolate.

Chapter Four grapples with the durability of African American hypertension as a disease category. Hypertension has been a principal site of disparities research, and physicians and many others have used African American hypertension as a way to make opposing claims about the relative importance of the social or the genetic in the etiology of racial difference. The chapter begins by attending to the rise of African American Hypertension in the 1960s at the confluence of the emergence of hypertension as a risk

factor, rearticulation of older racialized infectious-versus-coronary distinctions, and Civil Rights discourses. This provides context for the rest of the chapter, which tracks attempts to place racialized hypertension on one side or the other of the material-semiotic, by connecting it with such heterogeneous etiologies as genetic selection in slavery, socio-economic class, culture, racism and more. My argument is that the durability of African American hypertension, like that of race itself, is irreducibly multifaceted, inseparable from social justice claims, and not vulnerable to critique of any one component in the social or biological. The etiological ambiguity that underlies African American hypertension does not preclude but rather bolsters the durability of that disease category.

The fifth chapter both extends consideration of African American hypertension and moves the focus to pharmaceuticals by attending to thiazide, which is an old generic drug for hypertension that has become linked with the disease category. The chapter starts with a close ethnographic reading of an invocation of thiazide outside of a medical or clinical encounter, in which Henry Louis Gates Jr. responds to an argument at an African American Studies Colloquium that genetic selection under slavery is the cause of African American hypertension by reporting his own consumption of thiazide and identifying himself as a “salt-saving Negro.” The chapter unpacks this moment both to illustrate the heterogeneity of ideas about race and about medicine that can be enrolled in racialized invocations of disease, and to suggest that drugs’ relationships with preoccupations with difference exceeds their role in narrow economic interests or medical data. The chapter then circles back to medical debates over this drug that is at once racialized, proven, old, and cheap, to consider how debates over drugs both within

medicine and beyond it can articulate debates over the nature of inclusion and difference in American ways of life.

Chapter Six finally turns to the drug that has been a focus of renewed interest in race and medicine among STS scholars: BiDil. This chapter tracks the contingent intersection of this branded combination of two generic drugs at the intersection of race, small Pharma, and heart failure as a disease category. In its 2005 Food and Drug Administration approval, BiDil became the first drug ever approved for use in a specific race, bearing the indication for “heart failure in self-identified blacks.” The blatancy of its racialization has attracted considerable scholarly critique, and yet much of the critique has leaned too heavily on old arguments about race and genetics on the one hand, or about blockbuster drugs on the other. Neither of these lines of critique are up to the task of deconstructing a drug that is simultaneously: effective in treating symptoms and delaying death; supported by diverse epistemologically eclectic actors; and commercially unsuccessful. The chapter’s goal is not to render a verdict on BiDil, or indict the racism of its proponents or opponents, but rather to track plural noninnocent discourses at the intersection of race and this particular pharmaceutical. The chapter critiques assumptions that drugs inevitably reach their markets and argues that attempts to purge arguments about this drug’s racialization of either their material or semiotic aspects are unsuccessful. Drugs, like race, are both material and semiotic, and there is irredeemable polyvalence of a “black drug” in this current historical moment.

A brief Epilogue closes the thesis with consideration of the junctures at which debates about race and medicine break down, constructed around a particular encounter in which positions on BiDil were framed as an opposition between those “down with the

people” and those “up in the academy.” I argue that deconstructing the noninnocence of opponents in a debate is part of critical analysis, but does not itself constitute a full analysis. Race and medicine is best characterized as plural noninnocent discourses, rather than as racists versus antiracists. Race is a topic about which neither activists nor academics should feel comfortable, and we need to acknowledge our own noninnocent engagement. Inspired by the prophetic pragmatism of Cornel West, I suggest that although analysis of race and medicine is not a project that can perfect the world, abstention from the messiness is not a solution. There is no place of engagement above the fray, but there is, nevertheless, hope.

Medicating Race: Heart Disease and Durable Preoccupations with Difference

Pharmaceutical-centered medicine is an excellent site for STS critique of race because it intervenes on the boundaries between social and biological, material and semiotic. Race is a dense-transfer point between precisely these boundaries. Thus, analysis of this intersection contributes to theories of race and of pharmaceuticals as well as the history and social studies of medicine. Racial difference in heart disease is material: what leading black cardiologist Richard Allen Williams has called “the death gap” takes place in actual bodies in the world and, no matter what caveats we make about the trickiness of data and etiology, the terrain is necessarily in part about biology. Yet racial difference in heart disease is also semiotic: the data cannot be extricated from

lenses of difference-oriented preoccupations, and arguments about race and disease become ways to articulate difference in other aspects of society and individual identity.

Pharmaceuticals are objects that are very readily recognized as material-semiotic, where it is easy to make the case for the capacity of objects to carry both matter and meaning. Race is also a material-semiotic category, one that takes on a peculiar ontological status at its intersection with heart disease and pharmaceuticals. Racial disparities could conceptually be medicated away, in the sense that differences in such biomarkers of risk as blood pressure could be eliminated with medications. Yet racial difference cannot quite be medicated away, in the sense that preoccupations with racial differences always exceed the data itself. Using disease categories and pharmaceuticals to think through race is productive if we accept that race, too, is irredeemably material-semiotic, and cannot be purged of either aspect.

To open this dissertation, I will consider the terms that frame the project by explicating each of the title's words in turn. All of the concepts are linked and best understood in terms of each other, but prefatory analytic separation might help provide some clarity from which to begin.

Medicating Race: Heart Disease and Diabetes

Preoccupations with Difference

Although this thesis draws from critique of race and science, it also grapples with the distinct status of medicine as a field. Medicine is a heterogeneous field¹² that is both a science and a practice – one that does not merely reify difference but also seeks to act on it in plural ways. Medicine both represents and intervenes on racialized bodies. In medicine's trafficking between the boundaries of biological/social, it is a field that not only medicates but also *mediates* race, in the sense of arbitrating as well as intervening.¹³

This dissertation considers *medicating* in a broad sense, as a verb that refers to actions taken in and about medicine as a field of social relations. Medicating importantly includes the most obvious meaning of the word, the administration of medicines, but it also encompasses much more. In addition to the prescription of drugs, medicating refers to other linked actions that medical practitioners engage in, including setting parameters around diagnostic categories, administering a range of diagnostic tests and treatments, conducting longitudinal studies, and writing clinical guidelines.

¹² As the recent book *Differences in Medicine* compellingly argues, medicine itself is irredeemably heterogeneous. "This is our point of departure: medicine is not a coherent whole. It is not a unity. It is, rather, an amalgam of thoughts, a mixture of habits, an assemblage of techniques. Medicine is a heterogeneous coalition of ways of handling bodies, studying pictures, making numbers, conducting conversations. Wherever you look, in hospitals, in clinics, in laboratories, in general practitioners offices- there is multiplicity. There is multiplicity even in medicine's 'core.'" Marc Berg and Annemarie Mol, Introduction to *Differences in Medicine: Unraveling Practices, Techniques, Bodies, and Bodies* (Durham: Duke University Press, 1998), 3. For more on the heterogeneity within medical practice and patient experience, see Annemarie Mol, *The Body Multiple: Ontology in Medical Practice* (Durham: Duke University Press, 2002).

¹³ In this sense, the attention given to physicians in this dissertation participates in a project that Dumit has called for: that readers "become as aware as possible of the people who interpret, rephrase, and reframe the facts for us (the *mediators*)." Joseph Dumit, *Picturing Personhood: Brain Scans and Biomedical Identity* (Princeton, NJ: Princeton University Press, 2004), 5. Nathan Greenslit has also played with the valences of medicating and mediating. His National Science Foundation dissertation improvement grant (NSF Award ID# 0426130, with Joseph Dumit as co-PI) had the early title of his dissertation as: "Medi(c)ating Illness: An Ethnographic Exploration of Women's Health in the Age of Direct to Consumer Advertising."

Medicating is in the present progressive tense because neither the field itself nor its relationship with race is a ‘thing’ granted by history or science. Medicine and race are emergent processes in motion in those spheres and beyond. Clinical medicine, like natural science that is the subject of much critical scholarship of race, cannot settle the debates about race at the nexus of the biological and the social. Race’s relationship with medical practice remains messy both because medical data and efficacy are complicated notions that are hard to pin down, and because clinical medicine is not independent from other biological and social discourses.

The concept of medicating in this dissertation also carries a valence of *meditating*. When doctors and others argue about how to medicate race, one thing that they are doing is *meditating upon race*, articulating emergent theories of the differentiated world through languages of medical terminology and data. In an elaboration of what Stefan Timmermans has called a “second order scientist,”¹⁴ my project involves second-order meditating: meditating upon the meditations of historical actors to theorize race and medicine.

This dissertation contributes to the emerging field of the STS of pharmaceuticals, in which investigation of psychopharmaceuticals has been particularly rich.¹⁵ A key insight in these studies has been the creative productivity of the intersections of human identities with pharmaceutical practices. Work on gendered diseases is an important

¹⁴ Stefan Timmermans, “Suffering and Hope: A Warrant for STS,” Presented at Presidential Plenary, Society for the Social Studies of Science, Vancouver, BC, November 4, 2006.

¹⁵ Nathan Greenslit, *Pharmaceutical Relationships: Intersections of Illness, Fantasy and Capital in the Age of Direct-to-Consumer Marketing* (PhD diss., MIT, 2007); Joseph Dumit, “Is it me or my brain? Depression and Neuroscientific Facts,” *Journal of the Medical Humanities* 24 (2003): 35-47; David Healy, *The Creation of Psychopharmacology* (Cambridge: Harvard University Press, 2002); Emily Martin, “The Pharmaceutical Person,” *BioSocieties* 1 (2006): 273-287; Jonathan Metzl, “Prozac and the Pharmacokinetics of Narrative Form,” *Signs: Journal of Women in Culture and Society* 27 (2001): 347-380.

model for my thesis because it provides insight into mobilizations of identity-based social movements embracing and resisting disease categories and pharmaceuticals.¹⁶ Historians within STS have attended to the reconfiguring connections between research science, marketing, and distributions of pharmaceuticals for both psychological and somatic disease through the 20th century.¹⁷ I hope to build on these literatures with a unique focus on somatic disease categories and pharmaceuticals that invoke racial difference.

This dissertation also contributes to the scholarship of biological citizenship, which addresses the mediation of inclusion and difference by and through medical discourse and practice. Adriana Petryna has argued that in the Ukraine, enrollment in the post-Chernobyl bureaucratic processes of welfare is part of “biological citizenship” in that context.¹⁸ Andrew Lakoff has studied particularly Argentinean responses to and appropriations of psychopharmaceuticals.¹⁹ João Biehl has interrogated the ways that Brazilian AIDS programs stake out citizenship in terms of globalized pharmaceuticals.²⁰ I am interested in unpacking diverse American responses to and appropriations of pharmaceuticals for heart disease for biological citizenship claims. Participation in medical knowledge and pharmaceutical practice has become part of articulating inclusion

¹⁶ Jennifer Fishman, “Manufacturing desire: The commodification of female sexual dysfunction.” *Social Studies of Science* 34 (2004): 187-218; Nathan Greenslit, “Depression and Consumption: Psychopharmaceuticals, Branding, and New Identity Practices,” *Culture, Medicine and Psychiatry* 29 (December 2005): 477-502; Verta Taylor, *Rock-a-By Baby: Feminism, Self-Help, and Post-Partum Depression* (New York: Routledge, 1996); Laura Mamo and Jennifer R. Fishman, “Potency in All the Right Places: Viagra as a Technology of the Gendered Body,” *Body and Society* 7 (2001): 13-35.

¹⁷ Nicholas Rasmussen, “The Moral Economy of the Drug Company – Medical Scientist Collaboration in Interwar America,” *Social Studies of Science*, Vol. 34, No. 2, 161-185 (2004); Jeremy A. Greene, *Prescribing by Numbers: Drugs and the Definition of Disease*, Baltimore: Johns Hopkins University Press, 2006.

¹⁸ Adriana Petryna, *Life Exposed: Biological Citizens After Chernobyl* (Princeton, NJ: Princeton University Press, 2002).

¹⁹ Andrew Lakoff, “The Anxieties of Globalization: Antidepressant Sales and Economic Crisis in Argentina,” *Social Studies of Science* 34 (2004): 247-269.

²⁰ João Biehl, “The Activist State: Global Pharmaceuticals, AIDS, and Citizenship in Brazil,” *Social Text* 22 (2004): 105-132.

in American ways of life.²¹ Thus we can read differing pharmaceutical grammars as differing articulations of that participation: either cutting edge or accessible to all, differentiated or universal, within reach of present capabilities or emerging only at some future point once the science or the struggle for social justice has proceeded further.

The productivity of medicating race, then, has a reach that exceeds the medical practitioners. Attending to their mediations, meditations, and diverse medication practices is a rich site for race and medicine critique.

Medicating **Race**: Heart Disease and Durable Preoccupations with Difference

The word “race” is a common noun. It appears on census forms and on the medical histories that are so central to medical practice, referring to the distinctions between black, Asian, Native American, and white, with Hispanic as a separate but also important variable. If this seems a rather simplistic definition, it is because I am less interested in how race is defined or ascertained than in how it is mobilized in medicine as a field of social relations.²² I do not put the term in scare quotes, even though I am sympathetic with many theorists who do.²³ Scare quotes, in the words of one linguist, disavow responsibility: “What the writer is doing here is *distancing* himself from the term in

²¹ Inclusion and difference in medical research as inclusion and difference in American citizenship is a topic Steven Epstein has also tracked, “Bodily Differences and Collective Identities: The Politics of Gender and Race in Biomedical Research,” *Body & Society* 10 (2004): 183-203.

²² Do you tick the box or does the box tick you? See Theresa de Lauretis, *Technologies of Gender* (Bloomington, IN: Indiana University Press, 1987).

²³ See for example, Nancy Krieger, “Refiguring ‘race’: epidemiology, racialized biology, and biological expressions of race relations,” *International Journal of Health Services* 30 (2000): 221-2.

quotes. That is, he's saying 'Look, that's what they call it. I'm not responsible for this term.'"²⁴ But my goal in this dissertation is not to create distance from medical invocations of race, but rather to engage with them. The fear of being responsible for race is one better addressed directly than disavowed. As Derrida reminds us, we are guilty insofar as we are responsible.²⁵ I argue that since innocence is not a possible moral stance for anyone participating in discourses of race, we might as well be responsible.²⁶

Moreover, many scholars who use scare quotes around race do so to posit something of a straw man: some other who 'really believes' in the racial categories that anti-racist scholars know better than to believe in.²⁷ However, I have found in my research that division between those with critical notions of race and those with uncritical ones does not correspond with any particular terminology choice, and straddles the medical/social science line. I am interested in tracking both the mobilizations of and retreats from characterizing disparities as *racial*.

I argue that in order to understand the productivity of race in medicine, race needs to be understood as a category that is always a subject of the dual aspects of medicine's mediation – bioscientific arbitration and pragmatic intervention – as well as a site of

²⁴ Larry Trask, "Guide To Punctuation: Scare Quotes," University of Sussex, available at <http://www.cogs.susx.ac.uk/doc/punctuation/node31.html#SECTION00092000000000000000>.

²⁵ Jacques Derrida, *The Gift of Death*, trans. David Wills (Chicago: University of Chicago Press, 1996).

²⁶ "Non-innocence" is a concept that Donna Haraway develops in, among other places, "A Cyborg Manifesto: Science, Technology, and Socialist-Feminism in the Late Twentieth Century," in *Simians, Cyborgs and Women: The Reinvention of Nature* (New York: Routledge, 1991), 149-182, and *Modest_Witness@Second_Millennium.Femaleman@_Meets_Oncomouse™: Feminism and Technoscience*, (New York: Routledge, 1997).

²⁷ For example, when Catherine M. Eagan reviewed *Whiteness of a Different Color* for the *American Quarterly*, she apologized for following the reviewed author's style and not using "undermining quotation marks" around the term race, adding: "But it is worth reiterating his observation that few writers put words like 'Caucasian,' 'white,' or 'black' in quotation marks, perhaps unconsciously taking these words to denote some kind of racial certainty." Catherine M. Eagan, "The Invention of the White Race[s]," review of *Whiteness of a Different Color: European Immigrants and the Alchemy of Race*, by Matthew Frye Jacobson, *American Quarterly* 51 (1999): 921-930. The belief that those who use quotation marks think that race is less certain than those who do not use quotation marks is not an accurate assessment of the field.

meditation about identity. Race in biomedicine does not originate in the science and filter down to the doctors' offices; neither does it simply filter up. It does slightly different work in each sphere, but gains its durability through being a nexus. The task for critical scholars should not be to seek out a true or innocent science, attempting to purge the material-semiotic category of race from biology, but to track plural noninnocent discourses at the dense transfer point of the biological and the social. Critique that rests only on scientific reification without attending to clinical intervention is both a failure to engage, and contributes to the durability of the categories that it ostensibly critiques.

The history of the scientific study of race has been interested in the malleability and fixity of race as a biological as well as a social phenomenon. Attention to cardiovascular disease in these terms is ripe for additional exploration. I will argue that a pharmaceutical solution to African American heart disease both underscores African American biological distinctiveness and undermines it—after all, it is a distinction that both can and cannot be medicated away.

Many critical historians have developed excellent analyses of the power of race as a dynamic and robust concept that does not belong to racists alone but to the terrain of the debate,²⁸ and these arguments have not yet been applied to pharmaceuticals. Doing so will show how tensions between the meanings and uses of race are part of the power of racialized discourses. Using critical historiographic lenses, it becomes clear that the full array of arguments about racialized pharmaceuticals are parts of racial discourses.

²⁸ Ann Laura Stoler, "Racial Histories and Their Regimes of Truth," *Political Power and Social Theory* 11 (1997): 183-206; Wiltraud Ernst and Berard Harris, *Race, science and medicine, 1700-1960* (New York: Routledge 1999); Barbara J. Fields, "Ideology and Race in American History," in *Region, Race and Reconstruction*, ed. Morgan Kousser and James McPhearson (New York: Oxford University Press, 1982), 143-177.

Arguments about race and pharmaceuticals can take diverse forms. For example, pharmaceutical companies or disparities advocates can argue for full integration of African Americans into mainstream American medicine's high levels of medication, or they can argue for race-specific drugs. But they cannot talk about disease categories like coronary disease or hypertension, or drugs like thiazide and BiDil, without talking about race.

Mediating Race: Heart Disease and Durable Preoccupations with Difference

Heart disease is an umbrella disease category that includes heterogeneous phenomena of a few major types that increase the burden on the heart and/or cause it to fail. The primary division within the category of heart disease is between two types: those disease categories that are related to hardening and physical blockages of arteries, which are called coronary heart disease (including arteriosclerosis, coronary thrombosis, and ischemic heart disease); and those that are related to elevated pressure, which are called hypertensive. Before the advent of penicillin, infectious heart diseases were also important. Other types of heart disease are congenital and valvular, but these are less prominent in medical research and are outside the scope of this dissertation.

Heart disease is an apt venue for STS of medicine because it offers an opportunity to critique medicine not at its fringes but as its core. Like all disease, heart disease has been the subject of existential debates, that is, the extent to which it actually exists. For example, there has been debate about whether risk factors such as hypertension should

properly be considered diseases and/or get treated with pharmaceuticals as widely as they do. But, in contrast to psychiatric disease, critiques of medicalization of heart disease do not extend to challenging its very existence, to denying that problems with our hearts can make us sick and die. As well, in contrast to such disease categories as sexual dysfunction, heart disease is also not vulnerable to claims that it belongs only outside medicine. Thus, heart disease poses a worthy challenge. In the words of historian Robert Aronowitz:

A physician colleague of mine, wanting to stress the absurdity of relativist arguments about disease, thought she might deliver the coup de grâce by saying, “Now you’re going to tell me that heart attacks are socially constructed.”²⁹

It is precisely because no one can deny the existence of some biological reality to heart disease that thinking through the nature of social construction here can be particularly productive. Where discussions of psychiatric disease can too often be bogged down in polarized discussions of whether or not they have a biological existence, discussions of heart disease can begin in the nexus of biological and social realities. Where arguments for the social construction of psychiatric disease can be misinterpreted as suggesting their spontaneous invention or unreality, arguments for the social construction of heart disease foreground the real embodied experience of social construction. The challenge Aronowitz accepts, to explore the ways that coronary heart disease “has been as much negotiated as it has been discovered” and the way that its “name, definition, classification, and ultimate meaning...have been contingent on social factors as much as strictly biological ones,” is inspiring to those in the emerging field of history and social science of somatic disease. This dissertation is deeply indebted to Aronowitz’s call. By considering the social

²⁹ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 84.

construction of heart disease together with that of race and of pharmaceuticals, this dissertation elaborates and extends his attentiveness to the contingencies in the social and biological.

Medicating Race: Heart Disease and **Durable**

Preoccupations with Difference

Durability is a concept that I am working through to capture the resilience of race in the face of critique. Something is durable if it resists wear or decay, if it is resilient in the face of stress, age, attack, or changing circumstances. As malleable as race is, its very flexibility helps to make it highly resistant to destruction (yet amenable to deconstruction). Race is always changing and yet lasting and enduring, something that cannot be easily gotten rid of. This durability is the source of great frustration for many critics of race in medicine, who often operate as if race rises or falls on its scientific validity.

A key insight of recent historiography of race has been the simultaneity of the fixity and mutability of race, and it is a literature that this dissertation contributes to. As Ann Laura Stoler has pointed out, ambiguity between phenotype and genotype is not an obstacle to racial thinking, but rather part of what makes it possible, and so it is an error to assume that if we can disprove the scientific concept of genetic race we can dismantle

racism itself.³⁰ The durability of race in medicine does not come from any one source. Acknowledging the epistemological eclecticism that underlies race helps to see why various debates over the etiology of racialized heart disease have not undermined racial reification itself. When debates over the past century have focused on whether the category at hand—whether syphilitic heart disease in the early 20th century, or hypertension since the post-war period, or heart failure since the emergence of BiDil—is due to genetics or the environment, they have not disrupted the reification of black heart disease itself. Diverse actors accommodate considerable epistemological eclecticism underlying what is recorded as status as African-American, and that ambiguity is not a slip in racial discourses but part of what makes their proliferation possible.

Yet, this durability of race need not be a source of hopelessness in a world of injustice. Durability is also flexibility. Recorded difference can be employed both to tell stories about existing inequality and to make demands for medical intervention and social change. Since the force of racial discourse becomes realized not in notions of fixity alone but in combustible combinations of fixity and fluidity, to understand the terrain of racialized pharmaceuticals and racialized medicine as a whole, we should attend to the ways that these debates render race both stable and malleable, to both naturalize it and mobilize it. A conception of race as simultaneously resilient and changing is a better terrain for critique than one that merely decries its resilience, because it opens up possibilities for critical scholars to be part of articulating change.

³⁰ This tendency to treat the genetic science of race as the be all and end all of race is a historiographical error, as Ann Laura Stoler points out: Ann Laura Stoler, “Racial Histories and Their Regimes of Truth,” *Political Power and Social Theory* 11 (1997): 183-206.

Preoccupations with Difference

The concept of *preoccupation* is a way to consider not merely the facts of a disease, but how the mind becomes engrossed, how the attention and the intellect can be commanded in debates over disease categories and identities. Much of this dissertation addresses the ideas of physicians for whom engagement in these practices is part of their daily work, and so preoccupation is also connected with *occupation*, both as a term for a profession and as a way to label the tasks that fill the day. The emergence of cardiology as an occupation and heart disease as a preoccupation have always been intertwined.

At the intersection of a disease category and a social identity, we have the opportunity to think through both the material and the semiotic aspects of our lived experiences. Ideas become our preoccupations when they engross our attention, to the exclusion of other ideas and in a way that is viscerally charged. I argue that a disease becomes a preoccupation when it opens up ways in which we can talk about our identities and our social terrain. Data does not determine preoccupations, and mere prevalence does not make a disease relevant for either professionalization or for cultural or identity practices. For example, as I have argued elsewhere,³¹ breast cancer gives American women at the turn of the 21st century an opportunity to talk about how many children we've had, or haven't had; our sexuality; our beauty; our ethnicity; our mothers; our toxic environments; and more. Breast cancer is not somehow naturally or inevitably available

³¹ Anne Pollock, "A Meditation on the Death of Betty Friedan, Due to Heart Failure," Unpublished Manuscript.

for narration in this way, but came to be so in the wake of feminism's Second Wave.³² Many women have followed the lead of such public figures as Audre Lorde in telling stories of their bodies and their lives through this disease.³³ This dissertation attends to the ways that stories of identity and difference have been told through heart health and heart disease.

A key aspect of preoccupations is that they precede any particular piece of data. That is why when a piece of data is read as contradicting the preoccupation, the piece of data is framed by a rhetoric of surprise. For example, there is a longstanding preoccupation with masculinity and heart disease, and so its status as the number one killer of American women is consistently a surprise. Even among those who know this demographic fact, it is often framed as being contrary to popular perception or common sense. *Look! Women get heart disease, too—isn't that something? Who knew?* Everyone already knew. Yet the framing of the number one disease as an exception is productive. In the insistence of such actors as the founder of the Association of Black Cardiologists that "African Americans get coronary heart disease, too," it is worthwhile to linger on the "too." It points to preoccupation with the implicit whiteness of the category, and articulates the preoccupation even as it contests it.

³² See Barron H. Lerner, *The Breast Cancer Wars: Hope, Fear, and the Pursuit of a Cure in 20th-Century America* (New York: Oxford University Press, 2001). Indeed, breast cancer became a feminist issue much later than reproductive health issues did, Stephanie Hartman, "Reading the Scar in Breast Cancer Poetry," *Feminist Studies* 30: 155-177.

³³ Audre Lorde's influential 1980 book *The Cancer Journals* told her own story, that was both searing social criticism and a personal narrative of a black lesbian feminist with breast cancer, Audre Lorde, *The Cancer Journals* (San Francisco: Aunt Lute Books, 1980).

Preoccupations with **Difference**

One question that is central to this dissertation is how difference comes to make a difference. Two aspects of racial difference are pertinent: that racial difference is amenable to durability because it is *recordable* and that preoccupations with racial difference cannot be understood as separate from lived experiences of racial inequality.

Categories of difference need not be *measurable* to be operationalized in medicine, and many differences that are central to medical practice are recorded rather than measured (such as age, gender, family history, and more). The durability of racial difference comes from its *recordability* – not because of its measurability or elusive lack thereof. Attending to ways that difference is *recordable* (as opposed to measurable) is also a way to grapple with another dual aspect of difference: difference suggests diversity, but codifying difference according to discreet characteristics narrows the scope of the definition of diversity. That is, difference is about othering, but the othering is not so total as to exclude some common ground on which degrees of difference between things can be judged. In the disagreements that I track in this dissertation, I am attentive to what the parameters of difference are imagined to be. One meaning of difference is *distinguishing characteristic*, and various types of heart disease have been held up as corresponding to various types of Americans.

When difference occurs in the context of inequality, difference becomes disparity. This thesis is in dialogue with the interdisciplinary literatures of disparities, including those of historians, health professionals, civil rights activists, and policy makers. David Jones, writing about American Indian epidemics, has pointed out that the meaning of

disparities is not transparent: “Disparities can be seen as proof of natural hierarchy, as products of misbehavior, or as evidence of social injustice.”³⁴ There has been tension in critiques of racialized medicine between the tendency to emphasize severity of racial disparities to call for justice and attempts to find ways to substitute other factors for race to explain the disparities in a less historically—and socially—loaded way, particularly from those who argue for social and environmental causes of disease disparities.

Differences provide fodder for preoccupations when they correspond with situations of dominance. As Catharine MacKinnon has pointed out, any observations of difference are not separable from conditions of dominance.³⁵ Attention to the relationship between difference and dominance helps to see why, for example, though intra-white ethnic difference was recorded early in the Framingham Heart Study, it came to be no longer so. Preoccupations with racial difference in medicine – especially black versus white – have remained durable not because of the compelling statistics about difference but because of the continued social reality of conditions of unequal access to power. This is why this recordable difference becomes not only an opportunity for taxonomy but also a mandate for advocating action against present inequalities.

Departing from the 1915 attempts to diagnose the death of Booker T. Washington and the 2006 reinvestigation, this dissertation will attend to the intervening space to map out new and renewed arguments about racial difference in heart disease and the contested ramifications thereof for medical practitioners and for other social spheres. Medicating

³⁴ David Jones, *Rationalizing Epidemics: Meanings and Uses of Indian Mortality Since 1600* (Cambridge, MA: Harvard University Press, 2004), 3.

³⁵ Catharine MacKinnon, “Difference and Dominance: On Sex Discrimination,” in *Feminism Unmodified* (Cambridge, MA: Harvard University Press, 1988), 32-45.

race in heart disease has remained a compelling site for disparate stakeholders to stake out not only what racial difference is, but also what it should be. By attending to these emergent processes, this dissertation provides an account of race in medicine with political as well as interdisciplinary academic relevance.

Chapter 2

Preoccupations with Racialized Modernity in Early Cardiology

In this chapter, I will consider both early cardiology and its historiography with particular attentiveness to the discourses of race embedded in them. I suggest that existing explanations of cardiology's rise in the period between 1910 and World War II – lifestyle changes in which mental strain replaced physical strain, changing disease demographics away from infection, and the professionalization and standardization of medicine – can be both enriched and problematized by analyzing the ways in which these accounts are imbued with the racial frameworks of the period. I read both the founders of cardiology and their historians as theorists of a cardiovascularized modernity, in which both the cardiovascularity and the modernity are racialized.

Many kinds of problems fall under the category of “heart disease,” and though the conceptualizations of them have changed over time as well as among different practitioners, a few key points about etiological taxonomy are important. Besides congenital malformations, early cardiologists knew that the heart could be damaged by a few major sources: infections such as rheumatic fever and syphilis, malignant hypertension, and inadequate blood supply (which is also called ischemia). Coronary

disease was an umbrella term early cardiologists used for this final category as the field started to become recognized in the interwar period, sometimes also rendered as “coronary heart disease” or “coronary artery disease.” The underlying problem of this category of heart disease was (and is) described as atherosclerosis, or buildup of fatty plaque in the coronary arteries. Therapeutic interest has been in the related phenomena of progressive obstruction and of plaque rupture leading to blood clots. Coronary disease can be asymptomatic, but when it presents clinically it can be angina pectoris (literally “strangling of the chest,” which refers to chest pain associated with lack of blood flow to the heart), or coronary thrombosis (which leads to heart attack, also called myocardial infarction). It was coronary heart disease, as opposed to the congenital, infectious, or hypertensive types, that would come to be the principal preoccupation of cardiology.

I argue that a disease becomes a preoccupation when it opens up ways to talk about identities,³⁶ to talk about self-value and social world in a state of disease, and coronary disease provided this opportunity for the founders of cardiology.³⁷ The concept of *preoccupation* is a way to consider not merely the facts, but how the mind becomes engrossed, how the attention and the intellect can be commanded in these debates. Data does not determine preoccupations. On the contrary, as I will show, data becomes fetishized such that when facts appear that are contrary to what the preoccupation

³⁶ I develop this concept of disease as preoccupation more fully in “A Meditation on the Death of Betty Friedan, do the Heart Failure,” Forthcoming.

³⁷ Unpacking heart disease as a “disease of modernity” worthy of such a preoccupation is connected to consideration of modernity’s other big scourge: cancer. Although many of the discourses of cancer and of heart disease were and are resonant, they are beyond the scope of this thesis. See for example Susan Sontag’s reading of cancer as (in her estimation improperly) articulating anxieties of the age in Susan Sontag, *Illness and Metaphor* (New York: Farrar, Strauss, and Giroux, 1978), and Keith Wiloo’s forthcoming book *How Cancer Crossed the Color Line* (Oxford University Press, Forthcoming).

expects, cardiologists and their interlocutors express surprise, and such facts as black coronary disease remain framed as exceptions.

This chapter is organized around tracking plural senses of the “modern” in the terms of early cardiologists. When these actors used the term “modern” to describe their field and focal disease, it was a concept that had many valences. Unpacking those valences provides an opportunity to reengage with existing explanations of cardiology’s rise. As they worked out the characterization of the modern diseases of the heart and the modern profession of cardiology, they were theorists of I will call cardiovascularized modernity – they narrated theories of race that provide a window into the period and lay some of the ground for what cardiology, cardiologists, and cardiovascular disease itself would become in the post-war periods.

The first valence of the “modern” that I will attend to is the heterogeneous concept of the “strain of modern life” as an etiological factor for coronary disease. The modern white man who suffered from coronary disease was understood to be subject to multifarious conceptions of strain, which I will consider in turn: speed, responsibility, and professional class status. These notions of strain sometimes invoked black difference in order to throw white strain into relief, and were more generally part of preoccupations with the *price of progress*. The founders of cardiology were elite American-born white men of ambition who saw their way of life as epitomizing modern American life, and their clinical study of and personal experiences with coronary disease became emblematic of the price paid for that way of life.

Second, I will consider “diseases of modernity” arguments about the ascendancy of degenerative disease over infectious disease in the period. I argue that one aspect of

early cardiologists' distinction between infectious and degenerative disease, both within heart disease and beyond, was *boundary work* between diseases of the past and of the future, between black diseases and white diseases.³⁸ Physicians, statisticians, and advocates set a terrain of debate that saw connections between differentiated races and differentiated disease. My focus is on how claims of black/white disparities in disease generally and heart disease in particular were used to tell stories about the differentiated nature of American progress. Moreover, vestiges of the preoccupations that were articulated by early cardiologists as racial differences in the vasculature have remained. The distinction early cardiologists made between two types of heart disease as correlates for two racial types – black/infectious versus white/coronary disease – was a taxonomy that would later map onto a distinction between black/hypertensive versus white/coronary disease that will be considered in later chapters of this thesis.

The third sense of the “modern” that I will consider is “modern medicine,” which was a way that these actors characterized medicine that was technologically advanced and engaged in professionalizing and standardizing practices. These modernizing processes set exclusionary boundaries around who could practice specialized medicine in scientific hospitals, and black health movements responded in plural ways. I will analyze

³⁸ The term “boundary work” was initially developed by Thomas Gieryn, “Boundary-Work and the Demarcation of Science From Non-Science: Strains and Interests in Professional Interests of Scientists,” *American Sociological Review* 48 (December 1983): 781-795. Gieryn documents epistemologically diverse methods of boundary work that Victorian scientists engaged in to demarcate science from two of its competitors (religion and mechanics), as well as between some scientists and those they want to exclude as “pseudo-scientists.” Noting that the tactics were different depending on whether the goals are to expand the field, monopolize resources, or protect autonomy, Gieryn notes that these aspects are common to all professions, including medicine. The site of this chapter, early cardiology as *professional, scientific* medicine is a particularly rich site of boundary work, since cardiologists were differentiating themselves from those who were not physicians at all (are not duly certified practitioners of lifesaving treatment) on the one hand and those who treated old diseases with old methods on the other. Boundary work excluded then, non-modern diseases as well as non-modern physicians and patients (including those who were black).

the discursive intervention that African Americans, too, get coronary disease, and thus are appropriate recipients and practitioners of fully modern medicine. This section will provide the ground from which I will argue in later chapters that an aspect of the white modernity of coronary disease and its treatment continues, even as the disease is understood to ‘spread’ to other populations and as cardiologists become more diverse by race, nationality, and sex. A postscript considers former Harvard University President Lawrence Summers’ anxiety about the decline of the white male cardiac surgeon.

The historical examples that I provide are not meant to represent the full range of views in the period, but rather to illustrate the productivity of arguments about racial difference in heart disease and in modernity. I am reading discussions of black-white differences in heart disease not to try to discern the true disease rates, but rather to track what Jones has called the “rationalization of disparities.”³⁹ I make no claims on the validity of data of the disparities themselves, which are unreliable for many reasons. First, those who were not seen by doctors were not recorded by them, and so unequal access to health care had an impact on the data. Second, diagnostic categories were unstable, not yet standardized across the country or across diagnosticians. Third, bias among those making diagnoses led them to see the racially differentiated disease rates that they expected to see. The drive to rationalize these disparities is a symptom of the preoccupation with black-white difference. Later chapters will show that aspects of early cardiologists’ conceptualizations of racial difference and of trajectories of progress will remain durable preoccupations of the field of cardiology for the century to come.

³⁹ As Jones points out, disparities “can be studied without attempting to establish the ‘true’ diagnosis of a past epidemic. The focus is not on diseases themselves, but on beliefs about diseases and disparities.” David S. Jones, *Rationalizing Epidemics: Meanings and Uses of American Indian Mortality Since 1600* (Cambridge, MA: Harvard University Press, 2004), 5.

Modernity as strain

In the history of cardiology and its historiography, explanations for its rise include changes in the disease burden and increased life expectancy of the population, as well as medicine's transformation toward the professional, standardized, and technologically-oriented. There also emerges something in excess of these that is an entrée into theories of race in cardiovascularized modernity: concepts of strain.

Historian of medicine Robert Aronowitz writes of the ways in which the participants in the rise of cardiology connected their field with modernity:

Attributing angina pectoris to the stress and strain of modern life resonated with well-accepted physiological conceptions, in particular the notion that angina pectoris resulted from an imbalance between the levels of oxygen supplied to and demanded by the heart. The demands on the heart were increased by the stress and strain of modern life.⁴⁰

For cardiologists and their historians, coronary disease becomes a way to articulate preoccupations with American ways of life as distinctively and differentially strained.

A dominant feature in the early-twentieth century writings in cardiology is a grappling with notions of the strain of modern life. A 1927 review puts strain into a laundry list of causative factors:

It is true that much has been written of the physical stress and nervous strain of the present-day mode of living as a factor in the production of high blood pressure and hardening of the arteries. Also emphasis has been placed on the role of improper diet, obesity, general toxic conditions, overwork, unsanitary conditions, excesses, and focal and general infections as causes of this form of heart disease. While no preventive methods are at present known for this type of heart disease, people can be taught the right way to live.⁴¹

⁴⁰ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 98.

⁴¹ William C. Munley, "Problems in the Prevention and Relief of Heart Disease," *American Journal of Public Health Nations Health* 18 (September 1928): 1098–1104.

But there were heterogeneous ways to characterize the strain of modern life. Strain is not a unitary category, but rather draws on racialized notions of speed, responsibility, and the unique strains on the professional classes—especially physicians themselves. In the subsections that follow, I will attend to each of these general concepts of the strains of modernity with regard to this specific case. My focus will be on implicit and explicit differentiation of modern whites from premodern blacks in these concepts of modern strain. These set out a terrain in which, like germs, strain as etiology was understood to be connected with the differential disease susceptibility of individuals and groups, and those too were mapped out in terms of race.

Modernity as strain (1): speed

Reading the testimonies of physicians of the period reveals an unease with the pace of modernization, even as they participated in and were part of producing it. These elite white men expressed some distress at the demands of the world they were part of shaping. Paul Dudley White, who is widely considered to be a founding father of cardiology, is a particularly rich witness to the transition because he was already a professor at Harvard Medical School before this transition and remained important throughout it. For him, a key aspect of the strain of modernity was that it was characterized by speed, which has resonances with the Fordist modernity of the period—the modernity of industrialization in which the pace is set by machines rather than on a human scale. White argued in his 1931 textbook that:

Even allowing for missed diagnoses in the past, angina pectoris is evidently increased in frequency, and is encountered more in communities where the strain of life is great and a hurried existence the habit than in leisurely parts of the

world. The situation is appalling and demands some action on our part. Almost certainly the most effective move that we can make is to call a halt on the war of mad rush today.⁴²

White is describing a very particular kind of strain, unique to a life considered to be fast-paced. Through his conflation of speed and strain, it becomes possible for White to articulate preoccupations with the strain caused by the perceived startling loss of leisure experienced by urbanites in prosperous countries.

Modernity as strain (2): responsibility

If for White the strain of modern life comes from the relentlessness of time, others described the strain as coming from increased responsibility.⁴³ Stewart R. Roberts is less-remembered than Paul Dudley White, but was a successful elite physician at the time, professor of clinical medicine at Emory from 1915 to 1941, and widely published on topics in internal medicine including pellagra and heart disease.⁴⁴ He was as leader of professional medicine in his time, serving as president of the Southern Medical Society in 1924-25 and president of the American Heart Association in 1933-34.⁴⁵ He was also part of the leadership of “the New South,” connected to the movement for race reform.⁴⁶

⁴² Paul Dudley White, *Heart Disease* (New York: Macmillan, 1931), 1353. Cited in Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 99.

⁴³ This is connected to more general arguments about the lack of African Americans’ responsibility for their own health, as for example in: H.M. Folkes, “The Negro as a Health Problem,” *Journal of the American Medical Association* 15 (October 8, 1910): 1246-1247.

⁴⁴ Charles Stewart Roberts, *Life and Writings of Stewart R. Roberts, M.D.: Georgia’s First Heart Specialist* (Spartanburg, SC: The Reprint Company Publishers, 1993), xiii.

⁴⁵ Charles Stewart Roberts, *Life and Writings of Stewart R. Roberts, M.D.: Georgia’s First Heart Specialist* (Spartanburg, SC: The Reprint Company Publishers, 1993), xiii. He merited an obituary in the *New York Times*, which characterized him as “a diagnostician and a specialist in internal medicine and heart diseases” (“Dr. Stewart R. Roberts,” *The New York Times*, April 15, 1941, p. 23).

⁴⁶ For example, he participated with other white faculty as well as black faculty in a workshop led by white Christian Southern reformer Willis Duke Weatherford in 1908 to consider the role of college students in

Like Paul Dudley White, Roberts connected increasing heart disease with the civilization of the day: “We have come to the period in which heart disease is the chief cause of death in civilization.”⁴⁷ He writes in particular about the stress of civilization:

Civilization as we know it in Western Europe and America, the ambition, effort, and community state of mind of these areas, the increasing responsibilities that come with age, and an aging circulation, apparently are the foundations for the increasing prevalence of angina.⁴⁸

The case he presents in his 1931 overview of angina pectoris is of a “gentleman of sixty” whose “family had been dominant and influential for a hundred years,” which was “a family of intelligence, efficiency and energy. The trick of power was in the blood and the patient confessed that he had always prided himself in taking the leading part in everything he was in.”⁴⁹ The strain of having gained and lost his fortunes in the economically volatile times was what preceded his angina, and Roberts is both impressed by him and has a prescription for him that suggests taking things more easily.

If in Roberts’ case study, the strain of civilization is implicitly racialized, race is much more explicit in the rest of the piece. The western civilization that Roberts describes is not imagined to encompass African Americans. He writes that angina

improving race relations in ways that would preserve segregation but support Booker T. Washington’s models of racial uplift. That workshop led to a book that had a chapter on health that sensationalized the dangers of unsanitary blacks to Southern whites as well as many invocations of the white man’s obligation to so-called weaker races. W.D. Weatherford, *Negro Life in the South: Present Conditions and Needs*, Revised Edition, (New York: New York Association Press, 1918). Though he was a member of a fraternity that claimed Robert E. Lee as a spiritual leader, Roberts’ sympathies seem to have been not only Southern but also national, for example winning a debating championship with an oration titled “Lincoln, the Typical American.” Charles Stewart Roberts, *Life and Writings of Stewart R. Roberts, M.D.: Georgia’s First Heart Specialist* (Spartanburg, SC: The Reprint Company Publishers, 1993), 12. In 1937, Roberts signed on as “advocating the principle of socialized medicine,” “Signers of the Statement on Socialized Medicine,” *New York Times*, November 7, 1937, p. 40.

⁴⁷ Stewart R. Roberts, “The larger view of heart disease,” *Southern Medical Journal* 18 (1925): 395.

⁴⁸ Stewart R. Roberts, “Nervous and mental influences in angina pectoris,” *American Heart Journal* 7 (1931): 23.

⁴⁹ Stewart R. Roberts, “Nervous and mental influences in angina pectoris,” *American Heart Journal* 7 (1931): 27.

pectoris “occurs usually in the sensitive, nervous type, as the Jew, or in the tense, efficient American, rather than in the dull, happy negro or the calm, accepting Chinaman.”⁵⁰ This is a succinct articulation of the racial archetypes that preoccupied the elite WASPs who claimed for themselves the label “American.” Roberts writes:

The white man, particularly one living a life of stress in urban conditions of competition, work and strain, makes his little plans and lays up cares and riches and takes much thought of the morrow; the negro in the South knows his weekly wage is his fortune, takes each day as it is, takes little or no thought of the morrow, plays and lives in a state of play, hurries none and worries little. What must it be to live unhurried, unworried, superstitious but not ambitious, full of childlike faith, satisfied, helpless, plodding, plain, patient, yet living a life of joy and interest? Even the negro child laughs easily, dances, sings, plays, and that usually in rags and wretchedness⁵¹

In part, Roberts based his racial taxonomy on twenty years of clinical experience, during which time he says he never saw angina in an African American.⁵² But he is epistemologically eclectic in his meditation, and includes insight from his own intimate experience as well, introspectively describing his resentment toward his “Ethiopian maid,” who is not disturbed by the ringing phone nor by his scolding her for not having answered it:

I strain, she lives by the day. She illustrates the advantages of being uneducated, untutored, unambitious, just one who has by nature been born with ‘an internal adjustment—to change her soul into an attitude of acceptance.’ But I meet difficulties every day, and many times a day, and try much of little to change and correct the difficulty externally rather than to adjust myself to it internally.⁵³

⁵⁰ Stewart R. Roberts, “Nervous and mental influences in angina pectoris,” *American Heart Journal* 7 (1931): 23.

⁵¹ Stewart R. Roberts, “Nervous and mental influences in angina pectoris,” *American Heart Journal* 7 (1931): 32.

⁵² Stewart R. Roberts, “Nervous and mental influences in angina pectoris,” *American Heart Journal* 7 (1931): 30.

⁵³ Stewart R. Roberts, “Nervous and mental influences in angina pectoris,” *American Heart Journal* 7 (1931): 33.

In the context of angina's association with such positive traits as responsibility toward oneself and others, ambition and hard work, this disease disparity becomes a way to argue that whites' increased predisposition to coronary disease had a positive valence.⁵⁴

Roberts concludes his piece:

The internist can bear witness to the increase in the number of cases of angina, hypertension, and nervous indigestion among the white races of Western Europe and North America... The psychology of stress, strain and struggle in the white races is in sharp contrast to the humorous carelessness of the musical negro or the placid acceptance of the gentle Chinaman. The white race talks of speed and records, domination and colonization, drives, rallies, sales and sales-resistance, estates, trusts and results, it increases its effort and income to gratify its desires, and then multiplies its desires. We call it progress and western civilization. The Chinese and the negro accept and, perhaps far more than the white man, conquer both their spirits and their nervous systems. In this sense the white man's burden is his nervous system.⁵⁵

Importantly, the "price of progress" that is the "white man's burden" is paid by its *agents*. It is white suffering in the service of their heavy responsibility that is portrayed in Roberts' texts as tragic. The notion of white disease as evidence of responsible white sacrifice for the sake of progress is itself a characterization of the biopolitics of the period. Blacks' deaths, although they were at a higher rate, did not carry such a valence, and rhetorical exclusion of their embodied experience in a modern age points to preoccupation. We might imagine a model in which those who have the higher death rate

⁵⁴ According to Aronowitz, "White persons were generally thought to be more predisposed to vasospasm and angina pectoris, an observation that accorded a positive, if ambivalent, valence to disease predisposition." Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 100.

⁵⁵ Stewart R. Roberts, "Nervous and mental influences in angina pectoris," *American Heart Journal* 7 (1931): 34. Roberts quotes without criticism an even more explicitly racist colleague from New Orleans, Lemann, who elaborates on never having seen angina in a negro this way: "The only probable explanation that comes at once to mind is the fact that negroes seem to be less highly organized nervously. They seem to bear pain better than do the whites. Has it not also occurred to you that suicide is exceedingly rare, almost unheard of, among negroes? In other words, I am suggesting that while the physical basis for angina and coronary disease is certainly present in negroes, the symptom complex is not present because of the less acute perceptions of pain." Roberts quotes an unnamed negro as saying that "the chief difference between the white man and the negro was that the white man knew how to work but he did not know how to play."

in the modern world would be framed as the ones paying the price of progress, but, as Cornel West points out, “the notion of the tragic is bound up with human agency.”⁵⁶ By denying black agency, Roberts also denies blacks the role of heroes in tragic tropes. The ongoing suffering of African Americans in the early 20th century is in these terms not modern tragedy but “mere suffering.”

Here, we can read the distinction between the diseases of whites and those of African Americans in the early 20th century as analogous to Agamben’s distinction between those who can be killed and those who can be sacrificed.⁵⁷ African Americans, for Roberts, remain in the category of what Agamben has understood as “bare life.” At stake is boundary work between (in Agamben’s terms) “the citizen” who is white, and the not yet civilized “bare life” which is black. It is through this maneuver that blacks are not the ones paying the price of progress in Roberts’ framework. African Americans are not full citizens, and only citizens of modernity can be sacrificed to it.

Not all who made these “price of progress” arguments to explain this white disease of modernity made such explicit cases about racial differentiation, yet there is an important implicit racialization in the framing itself. Notions of the strains of modernity were notions of white middle-class masculinity. Who, in the period of the Depression, could be imagined to be living a sedentary lifestyle full of ambition? The “price of progress” model of the cause of increased prevalence of coronary disease assumes that those suffering from it are indeed progressing. The notion of white people moving ahead

⁵⁶ Cornel West, “On Prophetic Pragmatism,” in *The Cornel West Reader* (New York: Basic Books, 1999), 165. West quotes Raymond Williams: “the real key, to the modern separation of tragedy from ‘mere suffering,’ is the separation of ethical control and, more critically, human agency from our understanding of social and political life.” Williams provides examples of “war, famine, work, traffic and politics” as those that are not seen as tragic but are deep in the pattern of American culture. Raymond Williams, *Modern Tragedy* (Stanford, CA: Stanford University Press, 1966), 48.

⁵⁷ Giorgio Agamben, *Homo Sacer: Sovereign Power and Bare Life* (Stanford University Press: 1998).

through history as black people remain in the past or in a static present has resonances that I will attend to again in my analysis of the Framingham and Jackson Heart Studies.

Modernity as strain (3): unique strains on the professional classes – especially doctors

There was, and to some extent still is, a widespread sense that coronary disease was a disease of the professional classes. Aronowitz notes that “the correlation with social class (higher social standing equated with increased incidence of disease), itself more apparent than true, reinforced the connection between angina pectoris and the hurried, busy life.”⁵⁸

Not everyone who noticed this apparent disadvantage for the rich thought that the higher strain born by the prosperous was sufficient to explain their disease experience. Rather, some were attentive to how strain could be simultaneously ubiquitous and differentiated. For example, William Osler was an elite turn of the century physician who believed that “angina pectoris might be caused by diseased arteries, the stress of civilization, and/or nervous exhaustion.”⁵⁹ Noting that Osler believed that the disease afflicted “better classes,” men, Jews, and physicians at a higher rate, Aronowitz states that:

He did not simply attribute this pattern to greater stress, noting that ‘work and worry are the lot and portion of the poor.’ Instead he puzzled over the interaction of constitutional and social factors, arguing that ‘it is as though only a special

⁵⁸ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 98.

⁵⁹ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 89.

strain of tissue reacted *anginally*, so to speak, a type evolved amid special surroundings or which existed in certain families.⁶⁰

Here, we can see a division between the strains that come from deprivation and arduous labor and the strains of professional-class men—and it is a division that is marked onto the vasculature.

Indeed, whether the lives of those who suffered from heart disease were best characterized by ease or by difficulty was an open question. In a 1921 piece that recommended little treatment for heart disease and suggests keeping the diagnosis secret so as to avoid increasing it through inducing nervousness or hysteria, Beverly Robinson described the typical patient who suffers from hypertrophy:

Hypertrophy of the heart with atrophic kidney disease and high blood-pressure, is one of the chronic affections we meet with frequently, especially among men in easy circumstances, who have eaten and drunk without stint, or who have had laborious intellectual lives and taken little exercise, or out-door life.⁶¹

One group of elites emerged as especially vulnerable to anginal strain in this period: the doctors who were studying coronary disease. The content of their occupation became their preoccupation, and they saw themselves as particularly at risk of getting early coronary disease. They were, after all, among the most ambitious of these modern white men they described.⁶²

The connection between the stress of modern living and angina pectoris was reinforced by – and reflected in – reports of increased incidence of angina pectoris

⁶⁰ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 89-90.

⁶¹ Beverly Robinson, *The Treatment of Ordinary Diseases: Notes from the Record Book of an Old Practitioner* (New York: American Medical Publishing Company, 1921), 84-85.

⁶² The masculinity of cardiologists was not accidental, but part of the system design of cardiology. For example, traineeships had specified pay rates as higher for married than unmarried men, with nothing at all specified as a woman's wage. In 1948, the budget of the National Heart Advisory Council included: "\$1 million which presumably could train about 300 men at a salary of \$3,000 for unmarried and \$3,600 for married men per year." Minutes of the National Heart Advisory Council, Volume 1, "Minutes of Meeting December 10-11, 1948" p. 60: National Archives, RG443 Box 21, College Park, MD.

and coronary thrombosis among physicians. Dickinson and Welker argued that heart disease is in reality an occupational hazard of the medical profession. In 1944, the Spens Committee concluded that the “strain of medical practice” resulted in an increase in coronary deaths. The link between physicians’ occupational stress and angina pectoris was strengthened by the many case reports by physicians of their own angina pectoris.⁶³

That doctors saw themselves as at risk is telling because they were agents of modernity. They were its progenitors as well: if there was a wave of modernity, these doctors’ coronary disease marked them as being on the vanguard of it. Coronary disease became the epitome of the thinking man’s disease, and their experience of it promoted an uncanny identification.⁶⁴

Differential susceptibility to strain by race

Some physicians of the period did question whether African Americans really were protected from coronary disease. Writing in 1932, physicians Schwab and Schultze⁶⁵ argued that for each type of heart disease – hypertensive, syphilitic, arteriosclerotic – onset in blacks was earlier and more pathological than in whites. They considered an explanation for the disparity that would focus on social class, but dismissed it by pointing out that their sample had blacks and whites of very similar social and economic circumstances.

⁶³ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 99.

⁶⁴ A relevant sense of the uncanny is that of the blurring of the boundaries between the well-separated ego and the external world. When the subject of investigation becomes the object of investigation, secure boundaries between self and non-self are blurred, provoking the re-recognition of one’s own body after much objectification has framed bodies as external. Sigmund Freud, *The Uncanny*, translated by David McLintock, (London: Penguin, 2003).

⁶⁵ Edward H. Schwab and Victor E. Schultze, “Heart Disease in the American Negro of the South,” *American Heart Journal* 7 (1932): 710-717.

Not only is the mortality rate from heart disease higher in the negro than in the white race, but, likewise, the resulting morbidity is of much greater degree. It has long been a common observation of those who see many negro patients that a diagnosis of organic heart disease in the case of a negro is of much more serious import than a similar diagnosis given a white patient. Such, it is offered, would be anticipated in view of the fact that the economic status and mode of living of the negro would prevent him from caring for himself in a manner that the white patient could and would. However, it should be added that the same difficulties are encountered in the treatment of any individual, regardless of race, who is dependent upon manual labor for sustenance. The majority of white patients with whom we have compared the negro in this regard were of practically the same social stratum and worked under similar conditions. Therefore, logical as the explanation may at first appear, it fails to reconcile the discrepancy in the mortality and morbidity rates from heart disease in the two races.⁶⁶

Having controlled away class to their satisfaction, Schwab and Schultze considered racial characteristics. They explicitly addressed Roberts, yet they posited a very different model of black heart disease than his. The preoccupation with the fundamental difference between white heart disease and black heart disease provides common ground between Schwab & Schultze and Roberts, even though they use opposite facts to support the notion of black inferiority.

Roberts feels that the absence of angina pectoris in the negro is to be accounted for by the fact that nervous and mental strain does not enter into the life of the negro, whereas it is a potent factor in the white races of Western Europe and North America. Certainly, there is a profound dissimilarity in the psyche and sensorium in the two races under consideration. Therefore, it seems logical to assume that the basis for the discrepancy in the occurrence of this syndrome lies in an inherent difference in the sensitivity of the nervous systems in the two races.⁶⁷

That Roberts and Schwab and Schultze have consensus on the meaning of black/white coronary disparity (that it marks blacks as inferior) but disagreement about specific data underscores that the data does not determine the racial preoccupation. Where Roberts

⁶⁶ Edward H. Schwab and Victor E. Schultze, "Heart Disease in the American Negro of the South," *American Heart Journal* 7 (1932): 711. They found angina pectoris rates to be low in their sample in both blacks and whites.

⁶⁷ Edward H. Schwab and Victor E. Schultze, "Heart Disease in the American Negro of the South," *American Heart Journal* 7 (1932): 716.

suggested that blacks' easy-going nature protected them from strain, Schwab and Schultze believe that it is whites who are protected from strain. Schwab and Schultze agree with Roberts that the strain of modern life causes coronary disease, but the difference, for them, is that that whites are better suited to that strain:

In view of the facts that heart disease in the negro compared with the white race is of greater incidence, occurs at a younger age, pursues a more rapid course, and has a higher mortality rate, the opinion is offered that the cardiovascular system of the American negro of the South is inferior to that of the white race, and is more vulnerable to insult whether this be applied as an infection, a degeneration, a toxemia, or in the form of the stress and strain incident to the complexities and modes of modern occidental civilization.⁶⁸

We can see here that claims about differential racial susceptibility do not by themselves determine relationships between disease rates and racial superiority. Arguments can be framed in both ways. For Schwab and Schultze, African American heart disease demonstrated black racial inferiority. For Roberts, white heart disease (especially angina) demonstrated white racial superiority. Differential prevalence of disease does not by itself then force any particular conclusions about the value of the patients suffering from it.⁶⁹ While some diseases stigmatize their sufferers, angina in this period sometimes marked its sufferers as good modern citizens.⁷⁰

⁶⁸ Edward H. Schwab and Victor E. Schultze, "Heart Disease in the American Negro of the South," *American Heart Journal* 7 (1932): 717.

⁶⁹ Indeed, as David Jones points out, when confronted with disparities, "[o]bservers always generate an overabundance of potential explanations. Their choices, which assign responsibility for the disease, have social and political utility. Disparities can be seen as proof of natural hierarchy, as products of misbehavior, or as evidence of social injustice. These assessments motivate or undermine interventions, influencing whether observers prevent an epidemic's spread, treat its victims, or exploit its opportunities." David S. Jones, *Rationalizing Epidemics: Meanings and Uses of American Indian Mortality Since 1600* (Cambridge, MA: Harvard University Press, 2004), 3.

⁷⁰ There are resonances here between angina and the late-19th-century category of "neurasthenia," a category that neurologists described as affecting highly civilized "brainworkers." As Herzeberg notes with reference to neurasthenia: "Diseases have always lived such double lives, with dread epidemics typically stigmatizing poor and nonwhite groups who disproportionately suffer from them. Neurasthenia performed a similar function for the well-to-do, whose unique susceptibility to this newly defined malady provided a natural-seeming proof of their distinctiveness as a class. Only 'advanced,' civilized men and women of

Modernity as transition toward post-infectious disease

In the nascent cardiology of the early 20th century, one way that the field grappled with the modernity of the disease being studied was by making a distinction between longstanding infectious disease and ascendant degenerative disease. Cardiology's founders were among the many of the period talking about the decline of infectious disease and bringing their focus to the degenerative diseases of ageing.

Samuel R. Roberts, quoted above with regard to differential strain by race, characterized the ascendancy of heart disease this way:

The outstanding fruition of the last fifty years of medicine is the increase in the length of the average life from 35 to 58 years...The prevention and cure of acute infectious diseases, the wider range of a scientific surgery and a scientific sanitation are the chief influences. Typhoid fever, yellow fever, and diphtheria are nearly blotted out from civilization, and yet heart disease is on the increase. Organic heart disease is today the greatest cause of death. No more could John Bunyan call tuberculosis the 'Captain of the men of death,' and no more could Osler apply the title to pneumonia.⁷¹

Roberts staked out primary and secondary prevention of heart disease as the next project of the field: "Science and medicine have well attacked the acute infectious diseases. Vaccinate and there is no smallpox. The problem of heart disease is far more complex. The task of properly studying and preventing the cardiac is before us."⁷² Others also characterized tuberculosis as having given way to heart disease – at least for whites.⁷³

high intelligence and complex inner lives had psyches delicate enough to suffer from it; on coarser and less refined souls it would barely register." Herzberg suggests that the new ambitions and popularization of the category led to its decline, but the increasing interest in cardiological models of stress of the elite may have filled that gap. David L. Herzberg, "Designer Consciousness: Psychotropic Medicines and American Culture from Miltown to Prozac," (PhD diss, University of Wisconsin, 2005), 78-79.

⁷¹ Stewart R. Roberts, "The larger view of heart disease," *Southern Medical Journal* 18 (1925): 392.

⁷² Stewart R. Roberts, "The larger view of heart disease," *Southern Medical Journal* 18 (1925): 394.

⁷³ For example, a lecture by Dr. Haven Emerson at the Massachusetts Medical Society in 1922 was reported in the *Boston Globe* this way: "He insisted that heart disease must be attacked as tuberculosis has been for the past 20 years, by creating a public consciousness in regard to the menace and by taking it in

Certainly it is the case that morbidity and mortality from infectious disease generally was declining in the United States in the first decades of the 20th century, leaving degenerative diseases with both an absolute increase in prevalence and a proportionally higher prevalence among an increasingly older population. But the interest in professionalizing around heart disease was not simply a result of changing statistics. Interest in non-infectious diseases of the heart preceded their majority in the caseload, and much thinking through relationships between heart disease and a racialized modernity was done in the boundary work between infectious and non-infectious disease.

Although histories of heart disease assume that something like what we now call coronary disease has always existed in some sense—that, for example, arteriosclerosis has happened in many aged bodies for centuries if not millennia⁷⁴—the historiography of heart disease emphasizes its rise to dominance as a cause of both illness and death. The opening paragraph of a hagiographic book of oral histories of cardiologists is typical:

At the beginning of the 20th century, infectious diseases posed the greatest threat to human health and survival. Tuberculosis alone was the major cause of death in both the United States and Western Europe. However, with the introduction of improved sanitary conditions, public health initiatives such as mass immunizations, and finally the introduction of antibiotics, such threats to human life diminished dramatically, at least in the industrialized parts of the world.⁷⁵

Aronowitz describes the same demographic transition:

the incipient stages...For although there is no panacea or absolute cure for heart disease, it is an easy manner to circumvent it, if taken in time, by a course of treatment or a change of occupation – or both. The white race is more susceptible to heart disease than the colored race.” *Boston Globe*, “Says Heart Disease Can Be Headed Off: Has Taken Place of Tuberculosis in Mortality: Dr. Emerson Lectures Before Massachusetts Medical Society,” June 1, 1921, p. 6.

⁷⁴ Heroic-narrative cardiologist/historian Richard Bing writes of arteriosclerosis that “we know that the disease has been present since ancient times, as it has been discovered in Egyptian mummies dating from 1580 B.C. to A.D. 525.” Richard J. Bing, *Cardiology: The Evolution of the Science and the Art*, 2nd ed. (New Brunswick, N.J.: Rutgers University Press, 1999), 118.

⁷⁵ Allen B. Weisse, *Heart to Heart: The Twentieth Century Battle against Cardiac Disease: An Oral History* (New Brunswick, N.J.: Rutgers University Press, 2002), xxxi.

“[I]ncreased attention to and importance of chronic disease ... resulted largely from the so-called epidemiological transition, that is, the declining morbidity and mortality by acute, infectious disease and the rising morbidity and mortality from chronic disease in industrialized countries in the late nineteenth and early twentieth centuries. The epidemiological transition led to a gradual switch in public health and clinical focus from acute, ‘communicable’ disease to chronic, sporadic disease.”⁷⁶

But statistics of caseloads and incidences were not all that drove increasing attention toward heart disease, as evidenced in part by the fact that cardiology’s founding preceded the transition it saw as axiomatic. The “so-called” in Aronowitz’s assessment may signal his awareness of the ahistorical element of positing that demographic transitions that followed the ascendance of medical focus on degenerative disease were the cause of that focus, or merely that the term “epidemiological transition” emerged in the 1970s. But for my project it is worth highlighting that although prevalence might help a disease to become known, it does not necessarily determine that it will become a preoccupation. Indeed rare diseases are often sources of considerable cultural attention. There is no linear relationship between incidence of disease and anxiety about it. Ideological and cultural resonances of degenerative diseases and coronary disease in particular became ways to think through the American way of life before the diseases themselves dominated morbidity and mortality.

Explaining the rise of cardiology through the epidemiological transition takes for granted that cardiology was, at its inception, defined as attending to non-infectious disease. But rather than our current categorization of coronary versus hypertensive disease, early cardiology’s organizing principal was coronary versus infectious. Hypertension as a chronic state with an etiological relationship with heart disease was not

⁷⁶ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 122.

yet fully a category of its own, though hypertension—especially acute—was often understood as an epiphenomenon of cardiovascular pathology.

The concept of a modern transition from infectious to degenerative disease both enrolled and was enrolled in arguments about race. Heart disease itself was racialized through its subcategorization. As we will see, the infectious versions—in particular syphilitic—would become more associated with blacks, and the degenerative—especially coronary—with whites. This taxonomy of two types of heart disease for two racial types will not simply end once the etiology of heart disease becomes overwhelmingly non-infectious. Rather, we will see in later chapters, it will be renewed in associations between whiteness and coronary disease and blackness and hypertension.

The landmark professionalizing events in the founding of cardiology as a field – the 1915 founding of the Association for the Prevention and Relief of Heart Disease in New York City, and the 1924 founding of the American Heart Association – incorporated efforts to combat infectious forms of heart disease.⁷⁷ One reason that Paul Dudley White took the fateful internship in England to study cardiovascular physiology in 1913, after which he would introduce its tools to the United States, was that his sister had died of rheumatic heart disease.⁷⁸ Heart disease had not yet become a major focus of medicine, and White describes being warned that study of heart disease would relegate his work to the “back few pages of textbooks” in which typhoid fever, pneumonia, diphtheria, and

⁷⁷ These appear on the AHA’s version of its own history: <http://www.americanheart.org/presenter.jhtml?identifier=10860>. The first campaign of the American Heart Association, in 1946, was against rheumatic fever.

⁷⁸ “Several years before, my young sister Dorothy had died of rheumatic heart disease, and since that time I had felt a desire to learn more about heart disease – although heart disease was not yet considered a specialty.” Paul Dudley White, with the assistance of Margaret Parton, *My Life and Medicine: An Autobiographical Memoir* (Boston: Gambit Incorporated, 1971), 14.

tuberculosis were “the infections [that] held the limelight.”⁷⁹ Thus, White narrates the founding of cardiology as preceding the ascendancy of degenerative disease over infectious disease.⁸⁰

In its first decades as a specialty, infectious heart disease would remain an important component of cardiology. A 1928 review describes: “It is fairly well established that there are three common types of heart disease: rheumatic, syphilitic, and arteriosclerotic.”⁸¹ The review describes many local studies, all of which ascribed roughly half the cases of heart diseases to infectious causes.

Even though cardiology at its inception was very engaged in curbing infectious heart disease, it nevertheless started to define itself as a medical specialty that attended to modern diseases, the kind that afflict society after infectious diseases have been brought under control. Narration of cardiology by both its founders and its historians as post-infectious is not an observation but rather a (racialized) strategic claim about which kinds

⁷⁹ Paul Dudley White, with the assistance of Margaret Parton, *My Life and Medicine: An Autobiographical Memoir* (Boston: Gambit Incorporated, 1971), 20.

⁸⁰ “As I returned to the Massachusetts General Hospital with my electrocardiograph, I felt like a lonely adventurer entering an unexplored and unknown country, planning to spend my life in a new and as yet unrecognized specialty limited to the heart and blood vessels, both normal and diseased. This was the decade before a handful of us formed the American Heart Association and two decades before we were permitted by our elders and even by most of our contemporaries to call ourselves cardiologists. It was the dark days B.C. (Before Cardiology), when the great White Plague, tuberculosis, was still the main cause of death, and rheumatic fever was responsible for the majority of heart patients.” Paul Dudley White, with the assistance of Margaret Parton, *My Life and Medicine: An Autobiographical Memoir* (Boston: Gambit Incorporated, 1971), 19.

⁸¹ This is the taxonomy from William C. Munley, “Problems in the Prevention and Relief of Heart Disease,” *American Journal of Public Health Nations Health* 18 (September 1928): 1098–1104. This piece was “Read before clinic and dispensary workers, arranged by the Joint Program Committee of the Associated Out-Patients Clinics of the New York Tuberculosis and Health Association and The North Atlantic District of American Association of Hospital Social Workers, New York, N. Y., February 10, 1927.” The article also describes heart disease as with a recent dramatic increase become the number one cause of death in New York City., and argues that to be able to achieve prevention what is needed is: “first, a comprehensive knowledge of the incidence of heart disease, that is, an understanding of the size of the problem; second, a knowledge of the causes, more particularly the common causes, if any; and third, a knowledge of the natural history of heart disease.” These are among the principal questions Framingham will answer later.

of (heart) disease are diseases of pre-modernity that merely linger in the modern period, versus which kinds of (heart) disease characterize modernity.

Early cardiology did not so much claim that degenerative disease was already dominant, but that it was becoming so. In the 1928 review, we read that although half of heart disease was infectious in etiology, the increasing rate was due to degenerative disease:

It has been suggested that when the statistics are analyzed further it may be discovered that the increasing rate of deaths actually takes place in this group and not in the infectious group in which the possibility of prevention and cure remains open...Practically parallel with a decrease in deaths from the preventable infectious diseases there has been a corresponding rise in the mortality curve of heart disease. This may temper our excitement concerning the increasingly high mortality from heart disease.⁸²

In this declaration not of its dominance but of its ascendance, degenerative disease becomes emblematic of the future. As we will now see, transition from infectious to degenerative was already enrolled in narratives of race, and a site of boundary work inside and outside cardiology.

Blacks as infectious

The period between 1910 and World War II was one of deeply linked transformations in both medical ideologies and racial ideologies.⁸³ The sense that – in contrast to that of

⁸² William C. Munley, "Problems in the Prevention and Relief of Heart Disease," *American Journal of Public Health Nations Health* 18 (September 1928): 1098–1104.

⁸³ In Haraway's helpful taxonomic chart on "Universal Donors in a Vampire Culture: Twentieth-Century U.S. Biological Kinship Categories," she gives the "paradigmatic pathology" of 1900-1930s as "decadence, rotting, infection, tuberculosis," and that of 1940s-1970s as "obsolescence, stress, overload." In this sense, the founding of cardiology can be read as part of the transitional apparatus, starting to address white obsolescence and stress in a period in which blacks remained characterized as infectious and tubercular. It is working in a period of the first decades of the 20th century but thinking through the paradigmatic pathologies of the future. Donna Haraway,

whites – African Americans’ heart or other disease was infectious was one that was of a piece with medical literature of the period and with growing black health movements. As Haraway points out, in the early decades of the 20th century “race—and its venereal infections and ties to sexual hygiene were real, fundamental, and bloody.”⁸⁴ Drawing a line around infectious disease as a category tied up with blackness was in part about marking who conformed to modern standards of hygiene, and who did not. It was within and through these preoccupations that cardiologists and others did their work dividing types of heart disease.

Both medical literature and the social movements around black health in the period were preoccupied with black sickness as infectious, and enrolled slogans like “germs have no color line.”⁸⁵ There was considerable debate over the relative importance of social versus constitutional factors in the high rates of black infectious disease. Tuberculosis, in particular, was to blame for the high death rate, and those advocating for black advancement also considered blackness and TB as connected even as they denied

Modest_Witness@Second_Millennium.Femaleman@Meets_Oncomouse™: Feminism and Technoscience (New York: Routledge, 1997), 222.

⁸⁴ Donna Haraway, *Modest_Witness@Second_Millennium* (New York: Routledge, 1997), 233. As Evelyn Hammonds points out, preoccupations with African Americans (especially women) as passive vectors of infection rather than as patients deserving of treatment in their own right have been renewed in discourses of AIDS (Evelyn Hammonds, “Missing Persons: Black Women and AIDS,” *Radical America* 24 (1990): 7-24). Cindy Patton has discussed the ways that earlier tropes racialized coloniality have also been articulated through discourses of AIDS, with Africa framed as infectious and Asia framed as infectible (Cindy Patton, “Migratory Vices,” in *Queer Diasporas*, ed. Cindy Patton and Benigno Sanchez-Eppler (Durham and London: Duke University Press, 2000), 15-36). Debates over whether infectiousness of oppressed peoples is a characteristic of their pre-modernity or their modernity is one that Charles and Clara Briggs attend to in their analysis of a 1990s cholera outbreak among indigenous peoples in Venezuela, compellingly characterizing the stakes of what they term “sanitary citizenship.” (Charles L. and Clara Mantini-Briggs, *Stories in the Time of Cholera : Racial Profiling During a Medical Nightmare* (Berkeley, CA: University of California Press, 2003).

⁸⁵ Vanessa Northington Gamble, “Introduction,” in *Germs Have No Color Line: Blacks and American Medicine 1900-1940*, ed. Vanessa Northington Gamble, (New York: Garland Publishing, 1989). For fuller discussion of this double-edged campaign slogan used to support the Provident Hospital and National Negro Health Week, see Chapter 2: “Spreading the Gospel of Health” in Susan Smith, *Sick of Being Sick and Tired* (Philadelphia: University of Pennsylvania Press, 1995).

that connection's inevitability.⁸⁶ If there was a transition from infectious to post-infectious, African Americans were not yet generally invoked as part of that. There was heterogeneity of opinion about whether the colorblindness of germs was the case—for environmental as well as constitutional reasons blacks were widely considered to be more susceptible to infectious disease—but the democratic impulse of infectious disease was in any case greater than that of heart disease (especially coronary disease). If germs gathered toward the bottom of social hierarchies, coronary disease collected up.

It is not disease incidence that determines this correlation between black-infectious-consumption/syphilis and white-noninfectious-angina/nervous disorders, especially by the 1930s. Yet the emphasis on infectious disease (especially tuberculosis) as black was a recurring theme in medical literature. An article by Louis I. Dublin, a statistician for Metropolitan Life Insurance, entitled “The problem of Negro Health as Revealed by Vital Statistics,” is typical.

The Negro death rates for practically all diseases in which care and sanitation are of paramount importance are much higher than among whites. It is probably that their higher death rate is due more than anything to ignorance, poverty, and lack of proper medical care. Pulmonary tuberculosis, tuberculosis, typhoid fever, pellagra, malaria, and puerperal conditions are examples of such diseases in which the mortality rates are much affected by unfavorable or insanitary environment—or by low economic status—and all of them have higher death rates among Negroes.⁸⁷

This situation was held to be in contrast with whites, whose diseases were of the mind and nerves, including heart disease with nervous etiology, rather than the lungs:

⁸⁶ Black advocates' efforts to separate out social from constitutional factors in high rates of black disease were longstanding, and particularly important to WEB DuBois. WEB DuBois, “Chapter 10: The Health of Negroes” in *The Philadelphia Negro* (New York: Lippincott, 1899).

⁸⁷ Louis I. Dublin, “The Problem of Negro Health as Revealed by Vital Statistics,” *Journal of Negro Education* 6 (1937): 274.

Army investigators state that the nervous system of Negroes shows fewer cases of instability than that of the whites. Only about one-third as many cases, per 1,000 examined, of neurasthenia and 'constitutional psychopathic state' was found in Negro troops as in the white. There were fewer eye and ear defects, and only half as many cases of functional cardiac disturbances of nervous origin.⁸⁸

Tuberculosis is highlighted in this report as "an outstanding cause of death among Negroes," even though a paragraph mentions that "In 1935, organic heart disease was the leading cause of death among the colored Industrial policyholders of the Metropolitan Life Insurance Company. Their death rate was 208.1 per 100,000, or a little more than one and a half times that for the whites."⁸⁹ Thus there is a gap between what was "outstanding" and what was merely the principal cause of death. Preoccupations with racial difference permeated even insurance, a field that is iconic for its rationalism, such that the principal contributor to high death rates in blacks was still seen as atypical of them.⁹⁰

Dublin's study and others like it were taken up by others interested in articulating racial difference in disease. For example, S.J. Holmes, a zoology professor from the University of California quoted Dublin when he included heart disease in his long list of black illnesses. In an article otherwise dominated by discussion of infectious diseases such as tuberculosis, he includes one paragraph on heart disease. He makes special mention of infectious cases within the heart disease category:

⁸⁸ Louis I. Dublin, "The Problem of Negro Health as Revealed by Vital Statistics," *Journal of Negro Education* 6 (1937): 274. This report also included much discussion of diabetes, to which Negroes were thought to be immune but who turned out not to be. Their death rate from diabetes "now" equaled that of white policy holders, perhaps because of less access to insulin treatment. "Whether or not this be the case, the evidence is that there is little or no racial immunity among the Negroes to diabetes; for, even if the more or less carefree rural Negro is more immune than the white man, it now appears that in urban surroundings the Negro is subject to much sickness and high mortality from this disease."

⁸⁹ Louis I. Dublin, "The Problem of Negro Health as Revealed by Vital Statistics," *Journal of Negro Education* 6 (1937): 270.

⁹⁰ We see this today with women and heart disease, in which the number one cause of death cannot approach the emblematic status of breast cancer and other "women's diseases."

Heart Disease: Negroes are especially prone to diseases of the circulatory system. Mortality from heart disease among Negroes has on the average been running about 50 per cent higher than among the whites, notwithstanding the fact that higher age composition of the whites would favor increased death rate from this cause. A recent summary of twenty years' experience of the Metropolitan Life Insurance Company with diseases of the circulatory system shows that the colored policy holders suffer from a much higher death rate than the whites from organic diseases of the heart and frequently associated disorders of arteriosclerosis, cerebral hemorrhage, and chronic nephritis. According to Dublin, 'it is especially among the colored patients that syphilitic heart disease is prevalent; in this group it accounts for about a third of the cases.' Davidson and Thoroughman, as a result of their studies, find over 25 per cent of positive Wassermanns in Negro heart cases conclude that 'syphilitic heart disease causes the greatest amount of disability next to the arteriosclerotic group.'⁹¹

The phrasing here around syphilis is seductive, but it is peculiar. Holmes' grammar acknowledged that syphilitic heart disease was the *second* biggest portion of heart disease among blacks, "next to" the category that was actually the biggest, and yet did not undermine the suggestion of syphilis' centrality.⁹² Here, the preoccupation with infectious disease and sexual hygiene allowed the numerically greater killer to be simultaneously acknowledged and disregarded.

It was not inevitable that the "outstanding" cause of black deaths would be considered to be infectious tuberculosis rather than genetic sickle cell anemia, another disease that would become a touchstone of racial arguments later in the 20th century.

⁹¹ S.J. Holmes, "The Principal Causes of Death among Negroes: A General Comparative Statement," *Journal of Negro Education*, 6 (1937): 295. He concludes: "If diseases act as selective agents affecting the survival rates of different races, their fatality must depend to a certain extent upon inherited racial peculiarities. If a high mortality rate is the direct result of an anthropological character, such as a relatively small lung capacity, the differential death rate thus produced will remain a permanent handicap, however greatly the mortality from this cause may be reduced. It seems not unlikely that the Negro suffers from some handicaps of this kind; but, on the other hand, he is favored by some partial immunities which will probably constitute a valuable permanent asset. That the Negro is 'constitutionally inferior' to the whites, as was formerly asserted by some writers, is a conclusion devoid of adequate foundation. With the possible exception of his proneness to tuberculosis and acute respiratory infections, he is, on the whole, probably a better animal than the white man."

⁹² Similar grammars are also used around women and heart disease today, such that sentences like "besides heart disease, breast cancer is the number one killer of women [in some category]" function to articulate preoccupations with breast cancer rather than heart disease.

Although discovered in the nineteen-teens (by Herrick, who made ischemia famous), sickle cell anemia would not become either of interest to medical researchers or a rallying point for black social justice movements in this period. Keith Wailoo suggests that this is because:

All the talk of ‘hereditary disease’ rang with fatalistic implications concerning the possibilities for improving black health...Sickle cell anemia would never have appealed as a special cause for those who sought to improve black health in the 1920s and 1930s, for it fit too neatly into the hereditary discourse and reflected too clearly the scientific fatalism they were fighting against.⁹³

For their part, sickle cell did not become a preoccupation among physicians who treated patients with it because they generally considered it in the category of “respiratory disease” to which they in any case considered blacks to be prone.⁹⁴

Diverse political narratives and agendas could draw on this preoccupation with blacks as infectious. Many whites invoked black rates of infection to advocate for white paternalism. For example, William F. Brunner wrote in the *American Journal of Public Health* in 1915 that whites were responsible for the health care of blacks because unless they made sure they lived in sanitary conditions, blacks would not help themselves and would pass infection on to whites.⁹⁵

Yet, the connection in the period between blackness and infection cannot simply be dismissed as a racist invention. Claiming convergence of blackness and infection was both a segregating move and one that could be used to rally and fundraise. One reason that black health was framed as infectious was to appeal to white self-interest in funding

⁹³ Keith Wailoo, *Dying in the City of the Blues: Sickle Cell Anemia and the Politics of Race and Health* (Chapel Hill: UNC Press, 2001), 79.

⁹⁴ Keith Wailoo, *Dying in the City of the Blues: Sickle Cell Anemia and the Politics of Race and Health* (Chapel Hill: UNC Press, 2001), 81.

⁹⁵ William F. Brunner, “The Negro Health Problem in Southern Cities,” *American Journal of Public Health* 5 (March 1915): 183-90.

black public health.⁹⁶ As a trustee at Howard University would point out in 1930: “It would be trite and commonplace to repeat here that disease germs know no color line or that any racial group with a disproportionately high mortality and morbidity rate is a distinctly retarding factor in the progress of any community. Truly the concern of the health of the Negro is the concern of the whole community.”⁹⁷ The National Negro Health Week Movement, which Booker T. Washington had founded shortly before his death in 1915 as the “National Health Improvement Week” and grew through the 1920s into an influential mass movement played up dire assessments of black poor health in order to spur both black mobilization for sanitary uplift and white philanthropic support.⁹⁸

If the threat of African American disease were conceived to be limited to themselves, this would not necessarily be a compelling site for white philanthropy.

Examples of these kinds of invocations appear in advocacy for black hospitals:

Because of the fact that no negro liveth to himself nor dieth to himself the negro health problem is not alone a question of concern to the black man, but is one of equal moment to the white population in communities where the negroes are found in any considerable numbers. Disease germs are the most democratic creatures in the world; they know no distinction of ‘race, color, or previous condition of servitude.’ The white race and the black race will continue to live side by side in the South, and whatever injuriously affects the health of one race is

⁹⁶ See, for example, Lawrence Lee, “The Negro as a Problem in Public Health Charity,” *American Journal of Public Health* 5 (1915): 207-211. “By education of the negro he may be made a better citizen, and a more useful member of the community, and come to live in better homes and more healthy surroundings. Instead of being a burden he may come in time to look after himself. This time will be a long time, generations of time. Until this time comes to pass it is necessary that the whites, out of their own pockets, in order to protect themselves, furnish the negro with public charities and better public charities than exist at present.”

⁹⁷ Peter Marshall Murray, “Hospital Provision for the Negro Race,” *Bulletin of the American Hospital Association* 4 (1930): 37-46.

⁹⁸ Roscoe C. Brown, “The National Negro Health Week Movement,” *Journal of Negro Education* 6 (July 1937): 553-564. This movement was taken over by the Public Health Service in 1930, and continued until 1951. For more about this movement and its context, see Susan Smith, *Sick of Being Sick and Tired* (Philadelphia: University of Pennsylvania Press, 1995), and Vanessa Northington Gamble, *Making a Place for Ourselves: The Black Hospital Movement, 1920-1945* (New York: Oxford University Press, 1995).

deleterious to the other also. Disease among the negroes is a danger to the entire population.⁹⁹

This framing is central to the work of the Rosenwald fund, which would be the philanthropic center for black hospitals. Edwin Embree, of the Rosenwald fund, argued in 1928 that: “Bacteria have a disconcerting fashion of ignoring segregation edicts. Jim Crow laws have never successfully been set up for the germs of tuberculosis, pneumonia, typhoid or malaria.”¹⁰⁰ He concludes: “The facilities for institutional care and health protection are shamefully inadequate and this fact is reflected in unfortunate death rates and in a great amount of sickness and distress, not only among Negroes, but, as a result, among their white neighbors.”¹⁰¹

Playing up notions of blacks as uniquely infectious received ample critique, especially by the 1930s as the pitfalls of organizing around black health were becoming clear. In a criticism that will recur throughout this dissertation, some in public health were concerned that focus on African Americans as a special case would make their diseases seem inherent to them and uniquely intractable. As Bousfield wrote in the *American Journal of Public Health* in 1934:

Perhaps one of the greatest hindrances to public health work among the Negroes is the gradually failing notion that the Negro is biologically different. Often he is thought to be so different, indeed, that the public health worker just stands back and asks the question ‘What shall I do for the Negro?’ and does nothing. It has well been pointed out that while it might be very interesting to prove that there is a biological difference between whites and Negroes, one could really do nothing about it and it would likely have no effect upon control measures.¹⁰²

⁹⁹ L. C. Allen, “The Negro Health Problem,” *American Journal of Public Health*, 5 (1915): 194.

¹⁰⁰ Edwin R. Embree, “Negro Illness and Its Effect Upon the Nation’s Health,” *The Modern Hospital* 30 (April, 1928): 50.

¹⁰¹ Edwin R. Embree, “Negro Illness and Its Effect Upon the Nation’s Health,” *The Modern Hospital* 30 (April, 1928): 54

¹⁰² M.O. Bousfield, “Reaching the Negro Community,” *American Journal of Public Health* 24 (1934): 214.

This question of what shall be done for black health is one that enrolls diverse readings of the meanings of the disparities that public health officials were describing.

Reading disparities: delayed trajectory of progress, or pre-modern people?

When coronary disease is described as a consequence of societal advancement, and infection as evidence of a danger from the past, it is an example of transitions in disease prevalence being used as evidence of transitions in society. What became known in the 1970s as “the epidemiological transition” marks a commonly perceived division between the health characteristics of “less advanced” and “more advanced civilizations.” This division was of great concern to those before and during the early years of cardiology.

If changing burdens of disease are taken as an unproblematic account for the rise of cardiology, the racialized character of the division between infectious and noninfectious disease is obscured. Concepts of progress away from infectious disease preceded the demographic shift itself, in both whites and blacks. Invocations of differential rates of decline in infectious disease became ways of grappling with racial difference.

Early cardiology’s agenda as a field that would investigate post-infectious disease that was characteristic of a modern world is part of an articulation of racialized notions both of progress and of heart disease in the period. The idea that different parts of America and of the world would transition away from infectious disease at different times suggests that not all peoples are existing in the same historical moment. By attending to diverging invocations of the timeline of progress away from infectious

disease, we will see that in this framework there were (at least) two ways to conceptualize the relationship between the pre-modern and the modern.

On the one hand, pre-modern people might be the analogues of modern people's own past.¹⁰³ This reading can foster slightly different responses depending on whether differential progress is seen in relationship to power. If differential power is not considered together with differential progress, it could be assumed that the inevitable passage of time might propel the pre-moderns forth on the same trajectory as the moderns. If differential progress is seen as related to differential power, in that a dominant group is retarding the progress of oppressed groups, then it can lead to calls to alter the relations of power in order to allow history to move forward. On the other hand, it is also possible to imagine that premodern people are on a different path altogether, left behind for extinction or irrelevance (as suggested by Hoffman's influential progressive-era *Race Traits of the Negro*).¹⁰⁴ Both of these attitudes toward the allegedly non-modern African Americans co-existed in early cardiology. While the model chosen for how to read differential progress cannot be settled by science, it does have implications for it.

Some in the first decades of the 20th century considered African-Americans to be pre-modern people in a modern world who were on a different path altogether, and suggested that on this basis modernity would be contraindicated for this group. One proponent of this model was doctor J. Madison Taylor, who wrote in the *Journal of the National Medical Association* in 1915 that

¹⁰³ See Johannes Fabian, *Time and the Other: How Anthropology Makes its Object* (New York: Columbia University Press, 1983).

¹⁰⁴ Late-19th century theorizing about whether black extinction was coming, and if so whether to work against it through a civilizing process or to accept it, had parallels with ideas about Native American extinction in the same period. See "Race to Extinction" in David S. Jones, *Rationalizing Epidemics: Meanings and Uses of American Indian Mortality Since 1600* (Cambridge, MA: Harvard University Press, 2004), 118-144 (esp. 139).

In order that the Afro-American shall survive or even to maintain a fair measure of health, it is imperative that he shall keep out of the big cities and live in the open country. Also that he go and live in the warmer regions, and avoid the cold seasons for which he is not structurally adapted, or he will surely die out in a few more generations, just as other races have done who violated the inexorable laws of race expansion.¹⁰⁵

In this framework, rural warmth was framed as inextricably bound to blackness, and cold cities to whiteness. Relegation of blacks to a stagnant South can be read as a move to exclude them from modernity, albeit for their own good.

Others in the period had a contrary reading of black differential progress toward modernity, and argued that the black trajectory was the same as the white trajectory, but that blacks were merely behind. W.E.B. DuBois began formulating this model to describe health disparities at the end of the 19th century in his landmark treatise on the status of the Philadelphia Negro that included a chapter on health. “What the Negro death rate indicates is how far this race is behind the great vigorous, cultivated race about it. It should then act as a spur for increased effort and sound upbuilding, and not as an excuse for passive indifference, or increased discrimination.”¹⁰⁶ For DuBois, among many other black leaders, closing the disparity would be possible both through black self-help and their greater inclusion in American social institutions.¹⁰⁷

¹⁰⁵ J. Madison Taylor, “Remarks on the Health of the Colored People,” Address to the Colored Convention, Varrick Memorial Temple, February, 1915, printed in *Journal of the National Medical Association* 7 (1915): 160-163. A response appeared in the same issue: Editorial, “On Dr. Taylor of Philadelphia, *Journal of the National Medical Association*, 7 (1915): 208.

¹⁰⁶ W.E.B. DuBois, “Health of the Negroes,” in *The Philadelphia Negro* (New York: Lippincott, 1899): 163.

¹⁰⁷ DuBois would go on to explore this theme in the *Health and Physique of the Philadelphia Negro*: “The undeniable fact is, then, that in certain diseases the Negroes have a much higher rate than the whites, and especially in consumption, pneumonia and infantile diseases. The question is: Is this racial? Mr. Hoffman would lead us to say yes, and to infer that it means that Negroes are inherently inferior in physique to whites. But the difference in Philadelphia can be explained on other grounds than upon race. The high death rate of Philadelphia Negroes is yet lower than the whites of Savannah, Charleston, New Orleans and Atlanta. If the population were divided as to social and economic condition the matter of race would be almost entirely eliminated.” W.E.B. DuBois, *The Health and Physique of the Negro American: Report of a*

Sometimes the natural inferiority of blacks was described together with the suggestion that their progress could proceed along a trajectory analogous to that of whites. The article by Dublin, the statistician for Metropolitan Life Insurance, suggests that excessive deaths for each age and sex group of blacks is related to their poor adaptation to the North American environment, even though he notes that the two decades before had seen a striking decline in black death rates such that “There would have been almost ten thousand more deaths of colored policyholders than actually occurred, in 1935, if the 1911 rate had prevailed.”¹⁰⁸ The statistician also broke down the numbers in this way: “His expectation of life in 1930 was the same as that of the white man about thirty years earlier.”¹⁰⁹

Amid these descriptions of differences between black and white death rates, there was also a critique that is similar to one used today with regard to hypertension, that focuses on comparing American blacks with groups other than their usual comparison group of American whites. For example, the Assistant Surgeon General of the US wrote in 1916 that the difference between the white-black death gap within the US was much smaller than that of blacks versus whites in less developed countries (such as Hungary and “Roumania”), or even specific white populations within the US.¹¹⁰ He also compared the mortality favorably with many white communities-some of the past (Boston 30 years before) and some of the present (such as Lewiston Maine).

Social Study Made Under the Direction of Atlanta University; Together With the Proceedings of the Eleventh Conference for the Study of the Negro Problems (Atlanta, GA: Atlanta University Press 1906).

¹⁰⁸ Louis I. Dublin, “The Problem of Negro Health as Revealed by Vital Statistics,” *Journal of Negro Education* 6 (1937): 273.

¹⁰⁹ Louis I. Dublin, “The Problem of Negro Health as Revealed by Vital Statistics,” *Journal of Negro Education* 6 (1937): 273.

¹¹⁰ John W. Trask, “The Significance of the Mortality Rates of the Colored Population of the United States,” *American Journal of Public Health* 6 (1916): 257.

In conclusion, it is believed that comparison of the mortality rates previously discussed shows: (1) That the colored death-rates of most communities of the United States are not discouragingly high; (2) that they are undoubtedly lower than they have been in the past; (3) that they are as low as many white population groups possessed twenty or thirty years ago; (4) that with the economic and industrial progress of the colored population its death-rate will gradually approach nearer to that of the white population.¹¹¹

In another version of this recurring theme, progress was imagined to start with whites and then (potentially, through the diligence of whites) spread unevenly. At the same time that reactionaries like Taylor thought of modernity as a threat to black health, many were seeing it as the solution to poor black health. If the pre-modern people are on the same trajectory as the moderns, just behind, then the same prescription that brought whites into modernity should be prescribed for blacks as well. In an excellent articulation of efficiency and morality, the two demands of what would become known as the progressive movement, a Southern doctor wrote:

In conclusion, the health of any people is the foundation upon which their happiness and prosperity and usefulness rests. If the individuals of any race yearly diminish in stature and physical strength, that race is doomed. The negro race in America is deteriorating, and at a rapid rate. The death-rate among them from filth related diseases is alarming. The race is headed toward destruction. Unless something is done to arrest the spread of disease among them the race will go as the American Indian went within a few generations. Every influence that helps to increase the negro's efficiency, everything that encourages him to become productive and self-sustaining, and that helps to make of him a better citizen lessens the 'white man's burden.' The fundamental source of disease, as well as vice and crime, among the negroes is shiftlessness, ignorance, and poverty. The remedy is a systematic, *disciplinary* training of his physical, mental and moral powers.¹¹²

¹¹¹ John W. Trask, "The Significance of the Mortality Rates of the Colored Population of the United States," *American Journal of Public Health*, 6 (1916): 259.

¹¹² L.C. Allen, "The Negro Health Problem," *American Journal of Public Health* 5 (1915): 202-203.

White physicians were not alone in their pleas for black discipline. Black physicians saw the imperatives of progress as applying to themselves and their own race as well. As one author in the *Journal of the National Medical Association* in 1924 argued:

The law of 'The survival of the fittest' holds as true today as ever before, and is indeed applicable to the Negro race; for, with the rapid changes in conditions due to the progressive march of civilization, the race will more and more be brought to the test as to its physical, mental, and moral status.¹¹³

He accepted the terms of the progressive march, both for African Americans generally and for black physicians in particular: "But we are not sleepers; we are not stagnant; we are making progress along many lines and can boast of a goodly number of men, any one of whom would be a worthy asset to any group of medical scientists."¹¹⁴

This multiplicity of possible trajectories of black progress toward modernity is connected with the ambiguities of race, and had implications for strategies toward black health. If African Americans were not destined for a trajectory of progress like that of whites, or if their progress on that trajectory was still far away, the focus of their health could focus on immediate matters of hygiene without attending to the diseases of progress. But if their diseases, too, were becoming fully modern this had implications for the kind of medicine they needed. The drive to progress in the terms of modern medicine would be an important one in gaining black access to medical training, which required access to technologically-advanced hospitals.

¹¹³ G.N. Woodward, "Racial Health: Presidential Address," *Journal of the National Medical Association* 16 (1924): 177. (Read at the annual meeting of the John A. Andrew Clinical Society at Tuskegee Institute, Ala., April 1924).

¹¹⁴ G.N. Woodward, "Racial Health: Presidential Address," *Journal of the National Medical Association* 16 (1924): 178. (Read at the annual meeting of the John A. Andrew Clinical Society at Tuskegee Institute, Ala., April 1924).

Modernity as technologically-sophisticated professional medicine

Besides the strain of modern life, a key way that the historiography accounts for the rise of cardiology and of heart disease has been through connecting it to another trend with valences of modernity: the professionalization and standardization of technologically-sophisticated medicine in the period. Bringing attention to race here both enriches and problematizes the account.

As medical historian Christopher Lawrence describes, the divergences of heart disease from disease generally and cardiologists from general physicians were processes that happened together.

At the turn of the century general physicians routinely treated heart and functional disorders. No rigid social boundaries marked off doctors treating heart disease any more than natural boundaries did heart diseases. During the second decade of the century the physicians determined to create an academic medicine and cardiology as a specialty attempted to designate as organic (or at least pathophysiologically demonstrable) a range of cardiac phenomena, notably pulse changes. In doing so, they made finer distinctions between cardiac and noncardiac disorders. This process continued into the 1920s, when more recalcitrant symptoms, for example, angina and breathlessness, were molded into a concrete disease entity, coronary thrombosis. Anything not construable as organic or physiological was no longer cardiological. Lines were drawn around a discipline and the diseases studied. A definite (natural) entity reproduced the material arrangements of men.¹¹⁵

This professionalization and standardization to which Lawrence refers is also part of larger standardization and professionalization movements happening in medicine.

Aronowitz, too, tracks the history of the specialty of cardiology and is particularly attentive to the reorganization of knowledge taking place as diseases and their

¹¹⁵ Christopher Lawrence, “‘Definite and Material’: Coronary Thrombosis and Cardiologists in the 1920s,” in *Framing Disease: Studies in Cultural History*, ed. Charles E. Rosenberg and Janet Lynne Golden (New Brunswick, NJ: Rutgers University Press, 1992), 75.

practitioners become more specific.¹¹⁶ Thus, cardiology provides one example of changes in the nature of medical knowledge and social organization in the early 20th century. The purifying and separating work that early cardiologists were doing, separating themselves and their patients from other specialties, renders their grappings with modernity rich for STS reading.¹¹⁷ If we look at these transformations with keener attention to race, we can see other types of boundary work being done: the professionalization and standardization were deeply racialized transformations as well.

For these physicians, heart disease became simultaneously a way to understand the relationships between their emerging occupation and their preoccupations with modernity. The interwar period was also the age of eugenics – it was a time that, as George Stocking has described, racial theories drew on ethnological, Lamarckian, polygenist, and evolutionist traditions.¹¹⁸ Physicians' claims on subspecialties resonate with their claims on subspecies, and so we should be attentive to the ways that a medical specialty emerging at this time was enrolled in these same racial discourses.

Technology helped define the occupation of cardiology, and the preoccupation of coronary disease. The dissemination of the electrocardiograph (ECG) is a central theme in the historiography of heart disease and of cardiology. Many have pointed out that the ECG made heart disease newly amenable to study as a discreet pathophysiological category, and that it helped make the discipline of cardiology distinct from general

¹¹⁶ Aronowitz's focus is on the decline in earlier holistic notions of disease focused on patient experience, and the ascent of ontological ones focused on pathophysiological processes. (These are all connected to concepts of modernity as standardization, themes that will be particularly important in the next chapter about the Framingham study.)

¹¹⁷ Bruno Latour, *We Have Never Been Modern*, trans. Catherine Porter (Cambridge: Harvard University Press, 1993).

¹¹⁸ George Stocking, Jr., "The Turn of the Century Concept of Race," *Modernism/Modernity* 1 (1994): 4-16.

medicine.¹¹⁹ ECG's early promoters sometimes invoked its particular usefulness in surveilling the previously difficult-to-surveil white diseases of the vasculature and heart.¹²⁰ There seems to be consensus in the historiography that the definition and treatment of heart disease has gone from diffuse to technical,¹²¹ and that the ECG enabled the visual recognition of physiological heart disease and thus its distinction from psychological phenomena.

Both academic historians of medicine and the cardiologists-cum-historians of cardiology of the heroic trope generally reject technological *determinism*, the latter perhaps precisely because they are interested in protecting the heroism of the physicians about whom they write.¹²² One heroic history, for example, calls the ECG “an important,

¹¹⁹ Historians have emphasized the role of the technology in the professionalization of the field, see especially Christopher Lawrence, “‘Definite and Material’: Coronary Thrombosis and Cardiologists in the 1920s,” in *Framing Disease: Studies in Cultural History*, ed. Charles E. Rosenberg and Janet Lynne Golden (New Brunswick, NJ: Rutgers University Press, 1992): 50-82.

¹²⁰ Promoting his use of the electrocardiograph to find previously unknown “cardio-vascular disease” in a cohort of executives, University of Pennsylvania physician C.C. Wolfert told the *New York Times* in 1930: “it is curious that, although degenerative diseases of the heart and blood vessels comprise what is probably the most important problem facing the white race, no dependable information is available as to what percentage of people going about their daily work show evidence of these diseases.” The article notes that “much interest attaches to this group, since the men all carry heavy responsibilities, a factor which is thought to predispose to heart disease.” “Uses New Method in Heart Research : U. of P. Foundation Photographs Pulse Waves and Records Cardiac Disturbances : 150 Executives Studied : Many, Apparently Sound, Are Found To Have Ailments in Early Stages,” *New York Times*, September 29, 1930, p. 17.

¹²¹ Whether that shift is dystopic or utopic depends on the historian. Those invested in critiques of the professionalization of medicine also extend their critique of overwhelmingly technique-oriented care to treatment of heart disease. For example, Stanley Joel Reiser concludes his book critiquing machines like the ECG with what sounds like nostalgia: “Since the nineteenth century, physicians have moved through a series of stages: from direct communication with their patients’ experiences, based upon a verbal technique of information gathering, to direct connection with their patients’ bodies through techniques of physical examination, to indirect communication with both the experiences and bodied of their patients through machines and technical experts. After each move to a new stage and new techniques, skills in using the old techniques have declined, with the resulting sacrifice of the unique insights they provided.” Stanley Joel Reiser, *Medicine and the Reign of Technology* (Cambridge: Cambridge University Press, 1978), 227.

¹²² Historian of cardiology Christopher Lawrence argues against technologically-deterministic arguments from Liebowitz and Herrick which suggest that the ECG led inevitably to the development of the concept of coronary thrombosis. “‘Definite and Material’: Coronary Thrombosis and Cardiologists in the 1920s,” in *Framing Disease: Studies in Cultural History*, ed. Charles E. Rosenberg and Janet Lynne Golden (New Brunswick, NJ: Rutgers University Press, 1992): 50-82. This fine point does not address those who invoke technology as important but not deterministic.

though not essential, part of the mystique of the new specialty” of cardiology.¹²³ In this story, technologically-assisted seeing does not determine the direction of cardiology, but it does work in the service of its progress and lends it an aura of power. Whatever the ontological status of the results of the ECG, it was a machine around which a new specialty could focus.¹²⁴

Cardiology’s use of technology is part of what historian Paul Starr has called “the dream of reason” that would “liberate humanity from scarcity and the caprices of nature, ignorance and superstition, tyranny, and not least of all, the diseases of the body and the spirit.”¹²⁵ But, he laments, the interests of various groups always get in the way of

¹²³ P.R. Fleming, *A Short History of Cardiology* (Amsterdam-Atlanta: Rodopi B.V, 1997), 156. Fleming is, however, of the rising-above-ignorance model described by Lawrence. He suggests (p. ix) that: “During the many centuries when physicians were persuaded by the spurious Hippocratic aphorism, ‘cor aegrotari non potest,’ that the heart was immune to disease, the subject matter of cardiology did not exist.” Similarly, Louis Acierno writes: “The earliest myths concerning the anatomical makeup of the heart and vessels date back to primitive man and rest on beliefs rather than observation. Moreover, these beliefs had a mystical flavor and were religious in origin defying and rejecting, as it were, any attempt at rational analysis. Theocracy rules supreme and when it did not, authoritarianism took its place. It was only much later when the scientific discipline of observation and experimentation became entrenched that authoritarianism was swept away... Against the background of magic, religious beliefs, idolatry and Satanism, the Babylonian practitioners of this epoch had a very conception of cardiovascular anatomy.” L.J. Acierno, *The History of Cardiology* (Pearl River, NY: Parthenon Publishing Group, Inc. 1994), 3-4. In a way that resonates with narratives of racial progress, he argues that “Although Greek culture relied heavily on Egyptian and Babylonian sources, the independent mind of the Greek in time divorced himself from this background and established new practices while clinging to the rituals of old. This was particularly evident in their approach to disease. It meant the gradual establishment of the art of healing that was stripped of sacerdotal monopoly while still paying lip service to pagan deities that were part of the panoply of healing gods.” L.J. Acierno, *The History of Cardiology* (Pearl River, NY: Parthenon Publishing Group, Inc. 1994), 6.

¹²⁴ Whether the electrocardiogram constructed new forms of disease or merely provided clear insight into true phenomena has been the subject of some debate. Medical historian Christopher Lawrence argues that the introduction of the concept of coronary thrombosis (heart attack) was an example of the social construction of a discrete disease entity together with a new medical discipline. As Lawrence shows, heart attacks were not always so obviously discreet, and that discovering the phenomenon was “was not a negative process of removing obstacles but a positive restructuring of clinical and pathological experience,” constructed by physicians as they built a discipline. Christopher Lawrence, “‘Definite and Material’: Coronary Thrombosis and Cardiologists in the 1920s,” in Charles E. Rosenberg and Janet Lynne Golden (ed.), *Framing Disease: Studies in Cultural History (Health and Medicine in American Society)*, New Brunswick, NJ: Rutgers University Press, 1992, pp. 50-82.

¹²⁵ Starr elaborates: “Modern medicine is one of those extraordinary works of reason: an elaborate system of specialized knowledge, technical procedures, and rules of behavior. By no means are these all purely rational: our conceptions of disease and responses to it unquestionably show the imprint of our particular culture, especially its individualist and activist therapeutic mentality. Yet, whatever its biases and probably

perfect rationality. Though not central to Starr's critique, racial injustice is a fundamental obstacle to his dream.

Describing the increased restrictions to entering the profession of medicine that emerged in the first two decades of the 20th century, David Barton Smith has pointed out that although "[s]cientific medicine gave the illusion of elevating the profession above political, class, and racial conflicts," it "became largely an upper-class, white, male profession."¹²⁶ Between 1900 and 1920, the number of medical schools that trained blacks shrank from seven to two. The 1910 Flexner report was a touchstone for the reform of American medicine to make it more professional and scientific. Flexner himself did think that there was a role for black physicians, but that it was not as full members of the modern medical community, but as humanitarian servants of their "own race" and as sentinels against black infection seeping into white communities. He considered their role in the final chapter of his report, "The Medical Education of the Negro," which opened with the statement that:

The medical care of the negro race will never be wholly left to negro physicians. Nevertheless, if the negro can be brought to feel a sharp responsibility for the physical integrity of his own people, the outlook for their medical and moral improvement will be distinctly brightened. The practice of the negro physician will be limited to his own race, which in turn will be cared for better by good negro physicians than by poor white ones. But the physical well-being of the negro is not only of moment to himself...Self-protection not less than humanity offers weighty counsel in this matter; self-interest seconds philanthropy. The negro must be educated not only for his own sake, but for ours. He is, as far as the eye can see, a permanent factor in this nation. He has his rights and due value

because of them, modern science has succeeded in liberating humanity from much of the burden of disease." Paul Starr, *The Social Transformation of American Medicine* (New York: Basic Books, 1982), 3-4.

¹²⁶ David Barton Smith, *Health Care Divided: Race and Healing a Nation* (Ann Arbor: University of Michigan Press, 1999), 14-15.

as an individual; but he has, besides, the tremendous importance that belongs to a potential source of contagion and infection.¹²⁷

Flexner thought black physicians' role would be limited in two key ways: they would serve only blacks, and their mandate was to protect whites from black dangers. David Barton Smith encapsulates Flexner's take this way: "Germs may not have a color line, but medical practice should."¹²⁸ The ideological model Smith describes here is one that will continue in the later periods described in this thesis: white doctors are framed as having access to universal knowledge while black doctors can only have more limited knowledge which is not generalizable.

Flexner's framing of the limited scope of black practice spurred social movement response. A central part of the black hospital movement and nascent civil rights movement was the fight for black access to practicing fully modern medicine in fully modern hospitals. The period of both the Flexner report and the response to it was a time in which black medical professionals both challenged and accommodated segregation. The National Medical Association was founded in 1895, within a year of Booker T. Washington's "Atlanta Compromise" and the *Plessy v. Ferguson* ruling. Early on, the NMA's focus was on getting black hospitals accredited, and it later also came to focus on integrating medical training and practice. The NAACP formed in 1909 and brought integrationist strategies to the fore.¹²⁹ At stake was whether blacks could be included

¹²⁷ Abraham Flexner. "The Medical Education of the Negro," *The Flexner Report on Medical Education in the United States and Canada*, (New York, NY: Carnegie Foundation, 1910), in *Journal of the National Medical Association*, 85 (1993): 152. A point-by-point critique of this Flexner chapter can be found in: Susan Hunt, "The Flexner Report and Black Academic Medicine: An Assignment of Place," *Journal of the National Medical Association*, 85 (1993): 151-155.

¹²⁸ David Barton Smith, *Health Care Divided: Race and Healing a Nation* (Ann Arbor: University of Michigan Press, 1999), 16.

¹²⁹ Integrationism and accommodationism are not necessarily conflicting approaches in a context of dramatic needs. Alondra Nelson captures this: "Efforts to create parallel health institutions for African Americans

among the heroic doctors of the American Hospital System that Rosemary Stevens has described: “The modern doctor, associated with the new hospitals, was – and is – a master engineer, a hero in the American mode, fighting disease with twentieth-century tools.”¹³⁰

In the 1920s, as historian Vanessa Northington Gamble describes, the imperatives of hospital standardization and accreditation threatened the survival of the black hospitals that had both treated many black patients and were central to the professional existence of black physicians.¹³¹ Although some integrated hospitals served black patients, they did not generally train black doctors.¹³² Thus, as Gamble argues, when black physicians mobilized around a black hospital reform movement, they sought to improve black hospitals and training both in service of “the black masses” and to promote “their own professional interests.”¹³³

were complemented by the second significant line of attack employed by black health activists—integrationism.” Alondra Nelson, “Black Power, Biomedicine, and the Politics of Knowledge” (PhD diss., New York University, September 2003), 38-39.

¹³⁰ Rosemary Stevens, *In Sickness and in Wealth: American Hospitals in the Twentieth Century* (New York: Basic Books, 1989), 12. She provides a taxonomy of American medicine in the modern hospital: (1) Segmentation and diversity of hospital ownership (2) the related stratification (3) the money standard of success / pay nexus (4) focus on acute care and technology, surgery in particular (5) built-in tensions between doctors and hospitals (6) authoritative role of university medicine. On professionalization, she points out: “How and why was hospital standardization achieved? I will suggest that this ‘medicalization’ had two distinct aspects and implications. The hospital was to be seen, first, as an institution with standard functions and expectations based on ideas about medical science that were generated in leading U.S. medical schools. But, second, the three major standardization schemes—the American Medical Association’s policies for medical-school affiliations with hospitals, its lists of approved internships, and the standardization of hospitals by the American College of Surgeons—also defined the hospital as the workshop of a responsible profession of practitioners.” Rosemary Stevens, *In Sickness and in Wealth: American Hospitals in the Twentieth Century* (New York: Basic Books, 1989), 52.

¹³¹ Vanessa Northington Gamble, “Introduction,” in *Germs Have No Color Line: Blacks and American Medicine 1900-1940*, ed. Vanessa Northington Gamble, (New York: Garland Publishing, 1989), viii.

¹³² Edwin R. Embree, “Negro Illness and Its Effect Upon the Nation’s Health,” *The Modern Hospital* 30 (1928): 54.

¹³³ Importantly, black physicians of the NMA and NHA often sought to improve black hospitals and training rather than dismantling them into the mass institutions: “Separate institutions were necessary because integration was a slow process and the ill health of the race demanded immediate solutions.” Vanessa Northington Gamble, “Introduction,” in *Germs Have No Color Line: Blacks and American Medicine 1900-1940*, ed. Vanessa Northington Gamble, (New York: Garland Publishing, 1989), ix.

Advocacy for the improvement of black hospitals was a crucial part of the advocacy for blacks' inclusion in modern medicine. In contrast to Flexner's suggestion that black practice should be rather basic, the black hospital movement suggested that the hospitals in which blacks practiced should be "modern hospitals," characterized by teaching and research.¹³⁴ As a Howard University medical professor put it in 1923:

Strictly speaking, the modern hospital should always serve as a post-graduate school from which much can be gained if we approach our work and study with an open mind. The properly organized hospital affords wonderful opportunities for physicians to develop a special knowledge of the several special branches of medicine.¹³⁵

Though there was consensus among black health leaders that black hospitals would eventually be either of the same caliber as white hospitals or indeed be integrated with them, there was disagreement about whether the intermediate step of working in segregated hospitals was necessary. Whether black physicians should be "accommodationist" (and focus on improving the quality of segregated black hospitals) or "integrationist" (and fight for access to privileges at elite white hospitals) was a source of tension.¹³⁶

¹³⁴ See John A. Keanney, "The Negro Hospital Renaissance," *Journal of the National Medical Association* 22 (July-September 1930): 109-112. Keanney argues: "Of particular importance is the stress being placed on teaching and research. The modern hospital cannot fully function unless it has some teaching facilities," and that these new Negro hospitals must be "modernly equipped," (p. 110). He concludes: "Modern medicine requires group practice. Both diagnosis and treatment demand the combined efforts and knowledge of the surgeon, the internist, and the laboratory technician. With great advantage to such a staff will be the addition of the dentist and the pharmacist. To these, depending on the size of the institution should be added various specialists and their individual departments," (p. 112).

¹³⁵ Algernon Brashear Jackson, "Public Health and the Negro," *Journal of the National Medical Association* 15 (1923): 258.

¹³⁶ For example, one physician wrote: "Men who have gone to the front and organized hospital groups, have had to wade through all manner of criticism. They were called enemies of the race, it was said they were 'fostering segregation.' But this is not new. Every time a Negro attempts to stand upon his own two feet and do something for himself, these overzealous persons will raise a hullabaloo about segregation." Dennis A. Bethea, M.D., "Some Significant Negro Movements to Lower Their Mortality," *Journal of the National Medical Association*, Vol. 22 (1930), No. 2, p. 87.

Yet what united both accommodationist and integrationist models was a commitment to modern medicine in terms defined by the elite white medical men creating the new professional and standardized field of medicine. Black physicians' professionalization remained largely segregated in this period, but would nevertheless eventually pave the way for their participation in cardiology, a field that is an exemplar of medical professionalization.

Those who supported immediate full integration of black physicians in the elite ranks of white-dominated technological medicine were critical of the tendency in the black hospital movement to emphasize black infectiousness. Dr. Louis T. Wright, an NAACP leader and physician at the Harlem Hospital, wrote in 1935 that thinking that blacks had special diseases often misled public health efforts to special solutions:

A few years ago, just before the depression, the question of the health of the Negro was a subject of study, intensively pursued, by some foundations and individuals throughout the country. Most of these studies purported to show one thing, namely, that methods and measures necessary to cut down on the mortality and morbidity rates among colored people were different from those that had proved successful in reducing these rates for other peoples. Although all this is patently silly nonsense, these studies have proved to be a troublesome obstacle to the sound solution of our health problems.¹³⁷

Dr. Wright's argument encapsulates a central tension in further race-specific studies and therapeutics that will be considered in this dissertation. He would go on to argue in 1938:

It is hoped at this time that the American people will begin to realize that the health of the American Negro is not a separate racial problem to be met by separate segregated set-ups or dealt with on a dual standard basis, but that it is an American problem that should be adequately and equitably handled by the identical health agencies and meet the identical methods as the health of the remainder of the population.¹³⁸

¹³⁷ Louis T. Wright, "Health Problems of the Negro," *Interracial Review*, 8 (January 1935): 6-8.

¹³⁸ "Interdepartmental Committee to Coordinate Health and Welfare Activities," *Proceedings of the National Health Conference*, July 18, 19, 20, 1938 (quoted in Vanessa Northington Gamble,

As historian Susan Smith describes, Wright's commitment to integration was so absolutist that no amount of urgency with regard to the immediate needs of individual sick African Americans could justify black health programs. Smith attributes this stand to Wright's sense that separate health programs bolstered notions of black inferiority. It is also worth considering how privileged a vantage point Wright is speaking from when he insists that it is better for individuals to suffer short-term than for the people as a whole to suffer long-term, since elite blacks like him were not likely to be the individuals paying with their lives. Smith quotes Wright at a 1938 National Health Conference in Washington DC, expounding what will remain throughout the 20th century an important critique of focusing on health disparities:

There is no such thing as Negro health. Disease draws no color line, but one would never know this from the way in which health services are administered in most places in this country.¹³⁹

There are two things here that are for Wright intrinsically linked, though they need not necessarily be: the existence of distinctively black illness on the one hand, and the goal of black health on the other. Wright rejects the value of both in order to commit completely to integration. As we will see in later chapters of this thesis, the terms that he has laid out for debate among black physicians have continued to resonate.

"Introduction," in *Germs Have No Color Line: Blacks and American Medicine 1900-1940*, ed. Vanessa Northington Gamble, (New York: Garland Publishing, 1989, 87).

¹³⁹ Louis T. Wright, as quoted in Susan Smith, *Sick of Being Sick and Tired* (Philadelphia: University of Pennsylvania Press, 1995), 80. Smith also describes Wright as becoming progressively more absolutist stance by Wright against segregated medicine for any time period or occasion that I be considered in relationship to BiDil and the Jackson Heart Study: "In 1952 Wright repeated this position when he suggested that separate black programs should not be accepted even for humanitarian reasons. 'This is a case where the greater good was served by denying to some of our group the immediate benefit of a segregated set-up, and it represents a casualty in this all-out war on discrimination and segregation,' he explained."

As black physicians sought to expand their scope beyond infectious disease, deemphasizing threats of black infectiousness, one thing that was at stake was whether the medicine that blacks needed was quite as modern as that of whites.

Black coronary disease and fully modern medicine

The foundational narratives of cardiology enrolled coronary disease as simultaneously white/modern/real, and it became important for civil rights oriented physicians to argue that blacks were not, after all, immune to it. Consider the intervention of Richard Allen Williams, founder of the Association of Black Cardiologists (ABC). He read the same cardiologists that I have just considered, and wrote about them in his 1975 *Textbook of Black Related Diseases*. He refuted their suggestion that blacks did not have coronary disease with studies that had come out in the 40s, 50s, and 60s. He attributed earlier underdiagnosis to poor patient-history gathering from black patients. Referring to the 1965 National Health Survey, he noted:

It is interesting to note that the National Health Survey indicated that angina pectoris may actually be *more* common in Blacks than in Whites. In a discussion of differences between Black and White adults in age- and sex-specific rates of coronary disease manifestations, it was stated that “angina pectoris may be more prevalent in the Negro.”¹⁴⁰

Williams described how history changed the ways that blacks were treated; yet he too was surprised that angina was more common in blacks. His use of the word ‘actually’ marks his preoccupation, signaling the embedded idea that blacks have less angina than whites.

¹⁴⁰ Richard Allen Williams, *Textbook of Black-Related Diseases* (New York: McGraw Hill, 1975), 377-378.

Given the unreliability of medical data of the period, there is no way of knowing whether the widely-believed lower incidence of angina in African Americans was an epidemiologic fact of North/South or black/white difference, or an artifact of who was analyzed for what—and who was believed. At a conference of the International Society for Hypertension in Blacks in 2005, I spoke with one physician who said that her father, working in segregated hospitals in the South, used to have to identify patients he believed to have coronary disease as having “indigestion” because the head of the hospital believed that blacks did not get angina, and would not accept blacks under that category of disease. Even if such blatant examples cannot be generalized, they add another layer to the data-oriented history provided by Williams: ‘indigestion’ was a common way to fill in the blank for ‘cause of death’ on death certificates, especially for blacks. But regardless of their truth value, the epidemiological “facts” are mobilized in articulations of racial difference. Data will not solve this problem of racial difference at the center of cardiological taxonomy.

Writing their mammoth history in 2000, physicians Michael Byrd and Linda Clayton mark the 1980s and 1990s as the era when “medical myths about coronary heart disease (CHD) in Blacks were dispelled conclusively for the first time.” They cite a report of the findings of *The Report of the Secretary’s Task Force on Black and Minority Health*:

Instead of CHD being uncommon in Blacks, CHD is the leading cause of death in U.S. Blacks.

Instead of acute myocardial infarction (AMI) being rare in Blacks, AMI hospitalization rates are high in Blacks with higher case fatality than in Whites.

Despite the myth of angina being rare in Blacks, they have high[est?] prevalence rates of angina in the U.S.

Despite the myth of White preponderance of CHD compared to Blacks, U.S. CHD mortality and prevalence rates are similar in Black and White males, with Black females having higher CHD mortality and prevalence rates than White females.

Despite the myth that Blacks are immune to CHD, they proved to be relatively susceptible in the United States.

Moreover, greater percentages of deaths (presumably of cardiac origin) occurred out of hospital for Blacks compared to Whites. And incomplete morbidity data suggest angina pectoris and chronic CHD excesses in Blacks.¹⁴¹

The grammar of “myths” in these protestations is revealing. Byrd and Clayton use “myth” in the sense of falsehood, but their text also carries a valence of “myth” as founding story of a people. As ordinary as they insist that black coronary disease is, they call attention to the whiteness of the diagnosis in the minds of many clinicians. Grammar of exception is a symptom of preoccupation, and reinforces whiteness as the original model of the diagnosis of coronary disease.

The foundational narratives of many of those who would go on to found the Association of Black Cardiologists and the International Society for Hypertension in Blacks involve the historical progression away from the idea that blacks’ (allegedly) pre-modern lifestyle or (allegedly) differing physiology protected them from the disease of civilization. For example, when Richard Allen Williams was presenting on a panel on African American Hypertension in a special session of the American Society for Hypertension, he described being trained by Paul Dudley White at Harvard in the 1960s.¹⁴² According to Williams, when he was a bright young physician in training, his mentor, White, asked what his interest was. When Williams answered that he wanted to

¹⁴¹ W. Michael Byrd and Linda A. Clayton, *An American Health Dilemma : Volume II : Race, Medicine, and Health Care in the United States 1900-2000* (New York: Routledge, 2002), 537.

¹⁴² American Society for Hypertension Annual Meeting, Scientific Sessions, “Special Symposium: Hypertension in the African American Population,” San Francisco, CA, May 14, 2005.

study coronary artery disease in blacks, Paul Dudley White was dismissive, saying that a “full-blooded Negro” never got coronary disease.¹⁴³

In Williams’ commitment to prove White wrong, we can see a dialectical response to the hypotheses of the founding of cardiology. At this intersection of race, cardiovascular disease, and the American Way of Life in the early 20th century, there was a freedom to exclude blacks the attention of cardiology because they didn’t get coronary disease—or if they did, not in the same way as modern whites. As a result, there has been continual contestation ever since – especially by black cardiologists – that blacks *do* get heart disease, and in *both* precisely the same way and in ways of their own. Like the physicians and statisticians writing in the 1930s, for Williams in 1975 and in 2005 the facts of black coronary disease still keep being both important and surprising. This demonstrates the durability of the preoccupation.

And so as I close this chapter on the founding of cardiology and turn attention toward the post-war period, it is important to remember that conceptualizations from this earlier period have not disappeared. The knowledge generated in the postwar period did not *replace* that from early cardiologists like Paul Dudley White—indeed, he himself was an early advisor to the Framingham study, which would take the field in new directions and will be the focus of my next chapter. Rather, new forms of knowledge added to old

¹⁴³ When Williams told this story in 2004, he (and the audience) laughed at the absurdity of the notion and in the comfort of no longer being so misled. This is a frequent narrative device among physicians that I observed at many conferences: laughing at the naiveté of the great thinkers of the past (especially Paul Dudley White), perhaps as a way of demonstrating how far the field has come as a result of efforts of *people like them*. Initially I imagined that this sort of discourse that derides the naiveté of the founding fathers was a *battle* discourse, taking a beef with a more important thinker in order to seem important and stake a claim to the field. However, this is an unsatisfying analysis because the interlocutor being taken on is dead and no one is suggesting that he had the last word. I think that it is probably more likely to be a discourse connected to the simultaneous sense of connection with yet rejection of the founding fathers. There is no getting around the importance of the patriarchs, and yet there is a lot of anxiety about being clear that the field has moved well beyond them.

ones, overlapped with them, expanded them, modified them. Vestiges of the historical moment before survive into what follows. Sometimes, the vestiges come to seem absurd, and cardiologists often joke about White's early ignorance of the danger of hypertension. At other times, the vestiges still 'make sense' to the later generations. Thus, for example, Paul Dudley White is still a founder of something understood to be a continuous development of cardiology. Similarly, racialized notions of progress continue to be enrolled in distinctions within heart disease and between heart disease and other pathologies. The changes and continuities have not turned on data-driven problem solving, but on changes and continuities in our preoccupations. There are an excess of narratives, and choosing and deploying them becomes a complicated endeavor. At the same time, the ambiguity means that every position is vulnerable to both reasonable and unreasonable attack.

PostScript: Whither the white American male cardiologist?

Harvard President Lawrence Summers' comments at the "NBER Conference on Diversifying the Science and Engineering Workforce" became famous for his shrill conjectures about the biological factors that might inhibit women's success in science. A flurry of refutation of his central points followed, and the ample response has lingered on in the ensuing two years. Reading the transcript of the talk, a tangential point he made in response to a question caught my attention.¹⁴⁴ An unidentified audience member

¹⁴⁴ Lawrence H. Summers, "Remarks at NBER Conference on Diversifying the Science & Engineering Workforce," Cambridge, Mass., January 14, 2005, transcript available at <http://www.president.harvard.edu/speeches/2005/nber.html>.

challenged whether Summers' description of focused hard work and drive really characterized white American men at the turn of the millennium. Summers gave an unexpectedly cardiological response:

Q. We saw this morning lots of data showing the drop in white males entering science and engineering, and I'm having trouble squaring that with your model of who wants to work eighty hours a week. It's mostly people coming from other countries that have filled that gap in terms of men versus women.

LHS: I think there are two different things, frankly, actually, is my guess-I'm not an expert. Somebody reported to me that-someone who is knowledgeable-said that it is surprisingly hard to get Americans rather than immigrants or the children of immigrants to be cardiac surgeons. Cardiac surgeon is about prestigious, certain kind of prestige as you can be, fact is that people want control of their lifestyles, people want flexibility, they don't want to do it, and it's disproportionately immigrants that want to do some of the careers that are most demanding in terms of time and most interfering with your lifestyle. So I think that's exactly right and I think it's precisely the package of number of hours' work what it is, that's leading more Americans to choose to have careers of one kind or another in business that are less demanding of passionate thought all the time and that includes white males as well.

Q: That's my point, that social-psychological in nature [unintelligible].

This anxiety about whether the passionate thought and huge time commitment of cardiac surgery is still part of white native-born American masculinity is a revealing one. It situates what Summers has said in the talk that preceded the question in the zone of anachronism. It is no longer clear that elite professional identity is what it means to be a white American man, and he acknowledges that in answer to a question. At stake is not only a demographic detail about who will be operating on our hearts, but also who we are as a nation. Are smart white American men – those with whom Summers clearly identifies – up to the strain of performing cardiac surgery, even if the financial rewards are great? Or is the fact that they have successfully outsourced the price of progress itself evidence of their intelligence?

Summers is not capable, of course, of seeing the historical trajectory into which the paucity of white cardiac surgeons falls. As a February, 2007, Black History Month advertisement for Coca Cola reminds readers of *USA Today*, viewers of the Super Bowl, and attendees of the multiplex, the first person to perform successful open-heart surgery was in 1893 was a black man.¹⁴⁵ He was Daniel Hale Williams, who had founded Chicago's Provident Hospital two years before. In 1913, Williams was inducted into the American College of Surgeons, which was the sole body that conferred specialist status for surgeons at the time. It took another two decades before another black man would be admitted (Louis Wright in 1934). As late as 1945, a would-be applicant named George Thorne received the response "Fellowship in the College is not conferred on members of the Negro race at the present time."¹⁴⁶

The constitution of Summers' "American" male cardiac surgeons was contingent, deliberate, and exclusive. Cardiac surgery, insofar as it became white and male during the ascendance of the fields of both cardiology and surgery, was not so because white men were the only ones ambitious enough to do the hard work. Cardiac surgery was tied up in the historical processes of technology, standardization, professionalization, and tropes of white modernity that have been considered in this chapter.

¹⁴⁵ This advertisement appeared in *USA Today* on January 18, 2007, and was airing in Edwards Cinemas at the same time. It also was shown during the Super Bowl, February 4, 2007.

¹⁴⁶ The role of the American College of Surgeons in enforcing segregation is described by David Barton Smith, *Health Care Divided: Race and Healing a Nation* (Ann Arbor: University of Michigan Press, 1999), 44-46. Though the decision was reversed, a requirement to be a member of the local AMA branch continued to effectively close access for many black physicians in the South. Desegregation of hospitals and medical associations would go on to be an important part of the civil rights movement.

Chapter 3

Constructing and Supplementing Framingham's Normal White Americans: The Framingham and Jackson Heart Studies

Vignette

In June of 2006, the International Society for Hypertension in Blacks (ISHIB) marked its 20th anniversary with a set of lectures during its annual conference. Dr. Charles Curry, the president of ISHIB, introduced the program with genial informal comments about how far the community had come, in terms of understanding how to prevent heart disease through intervening on risk factors. He pointed out that the risk factor was a concept that came from the Framingham Heart Study, before warmly introducing the next speaker,

with a grin: “*Our next speaker is Dr. Herman Taylor from the Jackson Heart Study. I usually call it the black Framingham.*”¹⁴⁷

This introduction was well-received, and warm applause welcomed Dr. Taylor to the stage. And the phrase is evocative. If there is a black Framingham, what does that mean that the original Framingham is? Does the landmark study that would become a touchstone for heart disease research in the second half of the 20th century *lack* race, or is it white? How was its racial identity constructed historically, and how has it been negotiated as the study became invoked as a standard in evidence-based medicine?

To frame this chapter, a note from the field. Here are excerpts from the speech given by the doctor introduced as the head of the “black Framingham:”

Taylor: It's really an unparalleled time in American research and American medicine in terms of developing advances. But it's also an unparalleled time of increasing awareness of the unique challenges represented by the diversity of our population—the growing disparity and awareness of that disparity among medical researchers and providers. What I hope to do today is leave you with a simple message that the appropriate response to unchecked epidemics in particular with this case of cardiovascular disease begins on the local level. The Jackson Heart Study is a local study with global relevance.

I'll briefly remind you all that at the beginning of the 20th century—the century that we were all born in except for Dr. Nesbitt's daughter, I guess—we witnessed across that century an unchecked epidemic of cardiovascular disease that was increasing with each passing decade...Not so obvious at the start because infectious diseases were so prominent. It began to build slowly before the two great world wars and our wake-up call for focusing on cardiovascular disease was probably the end of the second world war and the death of Franklin Delano Roosevelt, the beloved American president who died in office from uncontrolled hypertension. This was America's wake-up call, and what followed was a response from the scientific community. In particular, it formed the Jackson

¹⁴⁷ Taylor, Herman. "The Jackson Heart Study," Part of the Session On "The Evolution of Hypertension and Cardiovascular Risk: A 20-Year Perspective," International Society for Hypertension in Blacks Annual Conference. Atlanta, GA, June 23, 2006.

Heart Study. Sorry, the Framingham study [laughter] also alluded to by Dr. Curry.

The Framingham Heart Study was ingenious. It took a very simple idea and ultimately resulted in a change in our approach to cardiovascular disease. It took a typical American town, mostly at that time it was second-generation immigrants, but a typical American town to look at in a microscopic way, at what might be the underpinnings of this unchecked epidemic. Dr. Curry told you the story, that the concept of the risk factor that we all use commonly every day was actually developed at Framingham ... [the control of which] led to the golden age of cardiology.

... At a glance it looks like a success story. Framingham occurs here and all these other research initiatives happened here, ultimately contributing to this great turnaround in cardiovascular disease mortality that we see in the early 60s and is continuing today. This on the surface is a global success story, but I think all of us here are driven by the awareness that this global success story hides a critical subplot. That is that there are portions of our population that do not share in these tremendous advances[...]. Cardiovascular disease deaths among African Americans in Mississippi seem to be rising as they're falling among others. These are arresting comments. They've become very embarrassing to us as a nation ... This is a local problem that begs a local examination of focused comprehensive evaluation of a population that leads me of course to the Jackson Heart Study...

I always enjoy comparison to the Framingham, because Framingham has done so much for understanding of the cardiovascular disease epidemic. We can only hope to approach that kind of record. The future of the Jackson Heart Study, I believe is in this local and global idea.

In this fragment, Taylor continues the tradition of early cardiologists tracked in the previous chapter: the racialized aspect of progress in America is narrated as isomorphic with advances in cardiology's knowledge. Taylor cites the discourses of racialized modernity from early cardiologists and the researchers at Framingham to invoke concerns about the populations left behind. Framingham becomes invoked as a particular kind of place – a typical one, a white one, an immigrant one, a 1950s one – and a particular kind of knowledge – local with global implications. These are productive invocations that I will return to after delving into the historical racial construction of Framingham.

Introduction

This chapter is spiral in structure. Having opened with a recent invocation of Framingham by the 2000-present Jackson Heart Study's lead investigator, I will now go back chronologically, to where the previous chapter left off in the immediate post-war period. Throwing Framingham itself into comparative relief with this moniker of the "black Framingham," this chapter continues broadly chronologically from the previous one to track intersections of heart disease and racialized American identity in the 1948-present Framingham Heart Study.

This chapter turns particular attention to the continuities and discontinuities of notions of normal American identity since World War II. Part of the story I am telling is a democratizing one: the expansion of the purview of the field of cardiology from elite WASP men in the founding of cardiology, to a larger multi-ethnic white population of men and women in the Framingham Heart Study, and on to a simultaneously fragmented and inclusive notion of the population that includes a diverse black population in the Jackson Heart Study. Through these moments in the history of heart disease research, we can see the expansion and fragmentation of the idea of who lives a modern American way of life. In this changing scope of coronary disease, one thing that has been at stake has been who could stand in as "typical," "normal," and "American" enough from whom to extrapolate a field of research.

Attending to Framingham's early racial frameworks, I will show that much work was put into constructing Framingham's racial and ethnic identity. Its unmarked whiteness was not inevitable or natural. Rather, it was a contingent and emergent aspect shaped by intersections of old racialized diagnoses; old and new notions of whiteness;

and new notions of how to manage difference epidemiological research. As I will show, the investigators did not simply start with an undifferentiated standard white human sample from which to extrapolate, but rather constructed one through two moves: (1) the disavowal of the concept of representation and (2) the use of within-group differences as a way to produce scientifically useful knowledge that could be extrapolated beyond the group. They articulated changing concepts of whiteness in each of those moves, articulating new and re-newed relationships between whiteness and cardiovascularized modernity.

Seeing Framingham in relief with the Jackson Heart Study allows us to see how Framingham and Jackson both posit their populations not necessarily as “typical,” but as “typical enough” – or, as we will see, in the terms of an early Framingham investigator, “not grossly atypical.” At their inception, both were framed as simultaneously racialized and yet sufficiently relevant to provide a basis for broadly applicable knowledge. Both studies rely on the insight that no single population can tell everything, but that any population studied in sufficient detail can tell *something*. Yet I will also attend to the ways in which Jackson is not a simple repetition of the Framingham Study. Jackson’s study design pushes the question of gaps between representation and extrapolation further. Its focus of study is not on a population at “average” risk, but rather a population at higher risk than the rest of America. This selection comes with a promise of simultaneously general knowledge useful for the general population, as well as specific knowledge by and for a population that has been “left behind” by the epidemiological advantages that the rest of the country has gained since Framingham. Jackson has not replaced Framingham, which continues, but is adding to it. It is a self-consciously

postmodern innovation taking off from it, a repetition with a difference. For Jackson, observation is enrolled directly in intervention, and “typical,” “normal,” and “American” are each plural concepts.

Making Framingham “normal”

Background

After World War II, there was a rapid democratization of conceptions of who was at risk for heart disease that were connected to ideas about American solidarity in the postwar period. Without hazarding a causal claim about where the change came from, notions that Americans were “all in it together” were formative of national identity in ways that were both inclusive and exclusive. Histories of whiteness have documented this consolidation of white American identity as simultaneously expanding the category of whiteness and solidifying the line between it and other categories.¹⁴⁸ Heart disease research participated in this construction of inclusive whiteness in the landmark study that would follow the war: The Framingham Study.

The Framingham Heart Study was begun in 1948 under the auspices of the Public Health Service before transferring shortly thereafter to the National Heart Institute (now the NHLBI). It ambitiously sought to follow a large population of residents of Framingham, Massachusetts, for two decades, and has continued well beyond. Originally recruiting 5,209 middle-aged Framingham residents, it expanded in 1971 to add a

¹⁴⁸ See for example: Monica McDermott and Frank L Samson, “White Racial And Ethnic Identity in the United States,” *Annual Review Of Sociology*, 31 (2005): 245-61.

similarly-sized cohort of their children, and is now adding a third generation to the biannual research visits.¹⁴⁹ Its goal has been to find connections between lifestyle (including diet and smoking), test results (including blood pressure and cholesterol), and heart disease incidence. It came to be a landmark in longitudinal research, epidemiology, and concepts of heart disease.

The Framingham Study began just after the sudden death of President Franklin Delano Roosevelt in 1945. That event is the point at which Framingham's current lead investigator Daniel Levy and his coauthor medical writer Susan Brink begin their history of the Framingham Study.¹⁵⁰ The extent to which Roosevelt's stroke was actually seen as connected to heart disease at the time is unclear – it was before the concept of “cardiovascular disease” became robust as a category unified under the valence of the heart.¹⁵¹ But the death of a war-time president who seemed to the public to be in

¹⁴⁹ NHLBI, “Framingham Heart Study: Design, Rationale, and Objectives,” <http://www.nhlbi.nih.gov/about/framingham/design.htm>.

¹⁵⁰ Daniel Levy and Susan Brink, *A Change of Heart: How the Framingham Heart Study Helped Unravel the Mysteries of Cardiovascular Disease* (New York: Random House, 2005).

¹⁵¹ Some popular representations in the period did conflate the cardio-vascular diseases in a way that would make Roosevelt's death relevant to Framingham's project. For example, the *New York Times* explained cerebral hemorrhage due to symptomatic hypertension as the cause of Roosevelt's death article in a way that put it in terms of coronary heart disease from early cardiology that will be Framingham's focus, quoting Irvine Page on the need to control arteriosclerosis to prevent the hypertension-induced cerebral hemorrhage like that of the President. The article laments the lack of evidence on theories such as diet and smoking as causes of this problem, though it reports uncritically: “The nervous system also has something to do with hypertension. Excitement, worry or strain increases our blood pressure. When this happens, there may be a stroke in a hypertensive person. There is no doubt that President Roosevelt, for all his outward equanimity and good humor, was under great strain.” “Causes of Cerebral Hemorrhage,” *New York Times*, April 22, 1945, p. E9. However, promotions for the 1947 “National Heart Week” did not mention the death of FDR as a framing device, and its promotion instead resonated with framings from the previous chapter, as in Howard A. Rusk, M.D., “Nation's Greatest Killer – Heart Disease – Challenged: National Heart Week Drive, Linked to St. Valentine's Day, Opens Tomorrow,” *New York Times*, February 9, 1947, p. 4. When cardiologists who were working through the period narrate the history of presidents and heart disease, the focus is generally not on FDR's stroke but on Eisenhower's heart attack in 1955, to which Paul Dudley White devotes a chapter of his memoir, Paul Dudley White, with the assistance of Margaret Parton, *My Life and Medicine: An Autobiographical Memoir* (Boston: Gambit Incorporated, 1971), 175-194.

excellent health demonstrated that the strong could be struck down by a hidden enemy.¹⁵²

How to prevent this from happening to Americans, and to America? The answer, the founders of the Framingham study hoped, could be found in science.

Between the interwar period and the postwar period, the democratization of attention to heart disease extended from elite whites to a broad if still not fully inclusive American middle class. In this expansion of heart disease, we can see an expansion of the notion of who can participate in the American way of life. The ascendancy of heart disease is also connected to a perceived spread of the shift in the predominant cause of illness and death away from infectious disease and toward degenerative disease, a demographic transition described in the previous chapter. Thomas Royle Dawber, an early principal investigator at Framingham, framed the imperatives of disease research in the post-War period this way:

The major concern of public health workers prior to World War II was the control of infectious diseases that had previously been the major causes of morbidity and mortality. Improved sanitation had greatly decreased the diarrheal diseases. Considerable strides had been made in controlling tuberculosis and pneumococcal pneumonia. With the introduction of penicillin in 1942, a further dramatic change in lessening the prevalence and incidence of diseases took place. At the end of World War II officials in the Public Health Service were confronted with a changing health situation in this country. If further advances were to be made, clearly they would be in the realm of noninfectious disease. Of these, cardiovascular disease and cancer constituted the overwhelming majority, with disorders of the heart and blood vessels approximately twice the rate of cancer.¹⁵³

Here, Dawber is invoking one of the senses of modernity discussed by cardiologists of the first half of the 20th century: diseases of modernity are imagined to be what remain

¹⁵² This narrative trope of heart attack as the hidden enemy of the strong is still culturally present. For example, in an advertisement for Plavix, a strong white professional man much taller than those around him is narrated to have heart disease. The final line of the advertisement tells us: "No matter how formidable you are, you are no match for a dangerous clot," Plavix ad, seen October 26, 2006, on ABC during prime time.

¹⁵³ Thomas Royle Dawber, *The Framingham Study, The Epidemiology of Atherosclerotic Disease* (Cambridge, MA: Harvard University Press, 1980), 11.

after problems of infectious diseases are solved. The other aspects of modernity described in the previous chapter were also reflected in the medical community in Framingham of the period. The practitioners in Framingham were from the newly professionalized, certified, and standardized portion of medical practice that saw its lineage with the elite physicians discussed in the previous chapter. There was only one board-certified cardiologist in the town, but he was part of a group of physicians who Patricia Thomas has described as “Young Turks”:

Dr Cornicelli and his friends weren’t small-town doctors looking for coattails to ride. They were Young Turks who’d come to town in the mid-1930s, rewritten the bylaws of the Framingham Union Hospital so that only board-certified could operate, and set out to attract more board-certified physicians in an era when those credentials were rare.

“They took over a medical community heavy on doctors with skills that now would be called marginal,” says Dr. J. Frederick Harrington, who practiced with Dr. Cornicelli almost 30 years ago. “It was a bit of a coup, an aggressive approach to improving medical care by a bunch of bright guys with a lot of toughness.”¹⁵⁴

The physicians in Framingham defined themselves in part against the pre-scientific local physicians who preceded them. Dawber described the support of these modernizing physicians as crucial:

One of the most potent factors influencing the decision to locate the program in Framingham was the presence of a highly cooperative and well-informed medical profession. A group of these physicians and other interested townspeople agreed to assist in an attempt to involve as many of the residents as possible in the study.¹⁵⁵

The bright, tough approach of the doctors in Framingham was of a piece with the national mood in the wake of World War II. Though heart disease has sometimes been framed

¹⁵⁴ Patricia Thomas, “In the Trenches of Framingham,” *Medical World News*, October 12, 1987, 69-77.

¹⁵⁵ Thomas Royle Dawber, *The Framingham Study, The Epidemiology of Atherosclerotic Disease* (Cambridge, MA: Harvard University Press, 1980), 16.

retrospectively as a disease that struck people from all sectors of society – “paupers and presidents” in the words of Levy and Brink – there had been considerably more attention paid to presidents. Heart disease was on the minds of politicians, and their preoccupation was not incidental to the feasibility of the performance of a study on the scale of Framingham. Aronowitz quotes long-time Framingham investigator Bill Kannel as saying

In those days, funds were not as short as they are now. Congressmen and Senators were getting heart attacks, and they would say, hey, you know, we need to look into this. And we would say, we have this interesting study that is following people to see what is causing these heart attacks, and we are coming up with interesting findings, so we would report that to Congress at the time appropriations bills were going through and they were actually throwing money at us. Do you need more money? Could we give you more help?¹⁵⁶

Normal populations, longitudinal research

The Framingham Study is iconic of an approach that arose in the early 1950s of looking longitudinally and prospectively at “normal” populations.¹⁵⁷ In its attempts to standardize, the study was an extension of the “modern” endeavor of early cardiology, and it provided the rationalizing concept of the risk factor. And yet the study and its risk factor concept would pave the way for the postmodern fracturing of epistemologies of disease.¹⁵⁸ The risk factor concept pushed beyond a focus on etiology, and moved the

¹⁵⁶ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 129. Aronowitz attributes the American interest in risk factors for cardiovascular disease to their optimism, individualism, consumerism, and ambivalence about modern life.

¹⁵⁷ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 120.

¹⁵⁸ A fascinating exploration of epistemological questions in heart disease research can be found in Nicolas Postel-Vinay and Pierre Corvol, *Le Retour Du Dr Knock: Essai Sur Le Risque Cardiovasculaire* (Paris: Editions Odile Jacob, 2000). Postel-Vinay’s treatment of hypertension in particular will be considered in the next chapter.

relationships of study beyond the causal. Along the way, the Framingham investigators innovated in scientific epistemology through multivariate analysis.

The concept of studying a “normal” population turns out to be a complicated and slippery one, and meditation upon it is a principal part of this chapter.¹⁵⁹ Aspects of “normal” at the study’s inception included: healthy, familiar and convenient to investigators, and demographically relevant to the disease in question. Yet once a subject was in the study, a change in status along any of these axes did not disqualify them from the “normal.” Onset of illness, or relocation far from investigators, or shifts in assessment of the subject’s demographic risk categorization – none of these lessened a subject’s status or role. Whether the participants in the Framingham Study were ever normal in the sense of either “representative” or “ideal” would remain unsettled. But once they had been charted according to established characteristics, they would become “normal” in the sense that they could be a standard from which to extrapolate.

Studying healthy populations – rather than ones selected because they already had a particular disease of interest to investigators – was an innovation that reversed the lens of pathology. One part of understanding the kind of knowledge claims made by the Framingham Study is its shift from seeing the body as inherently healthy with need of occasional intervention to restore that natural health, to one always at multiple risk for disease and in need of preventative health care. (The principal biostatistician of the early study would go on to define “free of disease” as a locution of convenience, but a state fundamentally impossible to prove given its reliance on the limitations of the

¹⁵⁹ Theories of the normal and pathological have been important in the history and philosophy of medicine, see especially George Canguilhem, *The Normal and The Pathological* (New York: Zone Books, 1991).

technologies of surveillance.¹⁶⁰) Observation of a healthy population also made the study more time-consuming, since investigators would have to wait longer for events of interest to occur in sufficient numbers to be reportable. And so even at Framingham, subjects who were considered to be at risk for coronary disease were the ones selected: the city was overwhelmingly white, and only the middle-aged were recruited.

A valence of the “normal” that has been in flux has been that of “representation.” Framingham’s popular historiography talks about it not only as normal in the sense of healthy, but also as a *representative* American Town. For example, a 1973 *New York Times* article described it this way:

In the history of the growing awareness that heart attack is associated with certain traits and habits characteristic of affluent civilization, there stands one great scientific landmark – the Framingham study. Framingham, Mass., is a gray factory town surrounded by pleasant suburbs 21 miles from Boston. As a town whose 28,000 residents in 1949 were ethnically and sociologically representative of the American population, it was a logical candidate for the Heart Disease Epidemiology Study of the National Heart Institute.¹⁶¹

Descriptions of post-war Framingham ring familiar because of their conformance with 1950s stereotype. The study’s current principal investigator, Daniel Levy, described the town in which the study began in the recent book he coauthored, *A Change of Heart: How the Framingham Heart Study Helped Unravel the Mysteries of Cardiovascular Disease*, in terms like this: as an independent town with a largely middle class population who had become homeowners through the GI bill, a GM plant, a shopping mall, the “typical” ethnic mix of (Euro-)America, of average weight and diet. There was even a

¹⁶⁰ Felix E. Moore, “Committee on Design and Analysis of Studies,” *American Journal of Public Health* 50 (October, 1960): 10.

¹⁶¹ C.P. Gillmore, “The Real Villain in Heart Disease,” *The New York Times Magazine*, March 25, 1973, 72.

Hostess factory, turning out Twinkies and Wonderbread. Levy treats ethnic representation this way:

The townspeople ate an ordinary diet, earned an average income, worked a typical variety of jobs, lived in run-of-the-mill houses, and fell victim to the usual variety of diseases. Their ethnic mix—including roots in Poland, Ireland, Italy, Greece, French Canada, and England—was a reflection of Euro-American ancestry of the time. Nearly a half century later, the study would recruit minorities to reflect the changing diversity of the town.¹⁶²

The ethnic diversity of Framingham is interesting for those narrating its history. For Levy's purposes, it brings the town to life. In my story, it is worth noting that the study's formulation of who could count as a "normal" American from whence to extrapolate was broad compared to that of early cardiology both because it was full of "new" Americans and included women. Yet, as I will explore further below, the early Framingham investigators disavowed claims that their sample was a representation of America.

The 1950s pan-whiteness that Daniel Levy and Susan Brink narrate in their history is evocative. The way Levy and Brink talk about this post-war environment, with its GM plant and shopping mall, is of a piece with 1950s white Americana. The dietary changes they describe are racially and ethnically specific, about lifestyle changes in response to understandings of risk entailing renunciation of lasagna and butter, not pork and lard. For Levy and Brink, a "middle class" town is "typical"—before the civil rights movement, before the War on Poverty. If there were particular dangers imagined to lurk in the town they did not consist of its inequities, but in its prosperity—as Levy and Brink would have it "ironically, they were about to enter what would become for millions life-

¹⁶² Daniel Levy and Susan Brink, *A Change of Heart: How the Framingham Heart Study Helped Unravel the Mysteries of Cardiovascular Disease* (New York: Random House, 2005), 53.

threatening prosperity.”¹⁶³ These aspects are all key for understanding the continuities of theories of the dangers of modernity and of the price of progress that the founders of cardiology described, as well as their expansion to a broad (white) (middle class) American public in the postwar period.

Levy and Brink describe Framingham as a town “representative of the latter day demographics.” This positioning of Framingham as “representative” is remarkable given the disavowals of those who preceded Levy as Framingham investigators. But it is an assertion that becomes easier to make in retrospect. Even though the Framingham cohort is not representative *now*, Levy seems to suggest, it once was. But by juxtaposing his reading with that of Dawber, we can see the ways in which easy representativeness might be easier to posit in the past than in any given present.

Many have been critical of the notion of Framingham’s status as representative of the United States. For example, psychologist James J. Lynch has described Framingham as representing an ideal of American life that was rare in its time and even rarer now. He writes: “At the time it was a small, rather beautiful, and peaceful town of 28,000 people, some 20 miles or so from downtown Boston. In many ways, it was an ideal town for research. In its ethnic, social, and economic mixes it appeared to be mainstream American—a model city, so to speak, for medical research.”¹⁶⁴ But even though it “seemed average,” Lynch questions whether the characterization of it as normal was in the sense of typical, or, on the other hand, ideal. Lynch describes it as the latter, “hard

¹⁶³ Daniel Levy and Susan Brink, *A Change of Heart: How the Framingham Heart Study Helped Unravel the Mysteries of Cardiovascular Disease* (New York: Random House, 2005), 24.

¹⁶⁴ James J. Lynch, *The Broken Heart: The Medical Consequences of Loneliness* (New York: Basic Books, 1977), 19.

working, mostly white, mostly middle class, and, in 1948, very stable,”¹⁶⁵ and indeed “Framingham...was perhaps one of the most settled communities in the United States in 1948—religious, churchgoing, nondivorcing type people.”¹⁶⁶ Even though according to Lynch today Framingham is “catching up” to the instability that characterizes the lifestyle of much of the rest of America, this stability was both something useful for the researchers and atypical of America.

In the scope of my thesis, it is important that historical descriptions of Framingham become very much descriptions of *America* at a point in time, a place that is considered to be both specific and spreading. Levy and Brink describe the spread of heart disease along with the historical progression of globalization this way, in terms reminiscent of descriptions of the spread of modernity among early cardiologists. They quote a previous Framingham principal investigator: “Despite proselytizing for a heart-healthy gospel, the epidemic of heart disease grows as the American lifestyle spreads...There are countries where heart attacks, strokes, and atherosclerosis kill hardly anybody. Most of the people who live on this earth never get this disease, but they don’t live anywhere near us. They live in Asia and Africa and Latin America. They live largely outside big cities.”¹⁶⁷ Levy and Brink add: “But the minute they start adopting a Western lifestyle, they begin to get heart disease.” With resonances from the writings of early cardiologists, the implication is that the modern/white/American way of life is terribly dangerous for the heart. In Framingham, we can see the continuation of the

¹⁶⁵ James J. Lynch, *The Broken Heart: The Medical Consequences of Loneliness* (New York: Basic Books, 1977), 19.

¹⁶⁶ James J. Lynch, *The Broken Heart: The Medical Consequences of Loneliness* (New York: Basic Books, 1977), 21.

¹⁶⁷ Daniel Levy and Susan Brink, *A Change of Heart: How the Framingham Heart Study Helped Unravel the Mysteries of Cardiovascular Disease* (New York: Random House, 2005).

discourse of doctors seeing heart disease as a way to understand the modern world and themselves, but it is codified to a new level. It was also extending the technological scope of medicine and heart disease.¹⁶⁸ Framingham grappled with how to answer the questions that had been raised in the inter-war period but could not yet be answered, developing new ways to ask and answer questions about the challenges of modern strain, modern lifestyles, and modern medicine.

But this question of representing America at a point in time was not as central for the studies early investigators. The sense of the “normal” that was particularly relevant for early investigators was the sense of familiar and convenient. The selection of the town was made less for representation than for stability and the amenability of the population – as well as easy commuting distance from Harvard. This is not to say that early investigators were completely disinterested in the town’s ethnic makeup. One area in which the Framingham investigators always paid attention to heterogeneity within white America was in what Steven Epstein has called “recruitmentology.”¹⁶⁹ An early manual of operation of the Framingham Study provides evidence that the investigators recognized the importance of understanding the nature of the town for logistical reasons at least:

In order to secure the active participation of people in a research program, certain sociological and psychological factors must be ascertained and given key consideration. What are the peculiar characteristics which exist in a given

¹⁶⁸ Two articles from the Framingham study in the early 60s make the point that is now obvious but at the time marked a transformation: computers are important in biomedical research. T.R. Dawber, W.B. Kannel, G.D. Friedman, “The use of computers in cardiovascular epidemiology,” *Progress in Cardiovascular Diseases* 5 (1963): 406-17; H.A. Kahn, “Use of computers in analyzing Framingham data,” *Circulation Research* 11 (1962): 585-86. Beginning in 1949, the study employed a quantitative sociologist (i.e. Moore) with experience from the census bureau and a talent for applied statistics, Gerald Oppenheimer, “Becoming the Framingham Study,” *American Journal of Public Health* 95 (2005): 607.

¹⁶⁹ Steven Epstein, *Inclusion: The Politics of Difference in Medical Research* (Chicago: University of Chicago Press, 2007).

community? What is the economic status? What nationalities are represented? What is the historical background? What are the traditions? What are the superstitions, What are the habits? What resources contribute to community progress? Who are the leaders. And most important of all—what attitudes exist? How do the people feel about being a part of a research program?¹⁷⁰

The investigators claimed to be concerned about the specifics of the racial and ethnic makeup of the town, but after acknowledging these community aspects they argued that they would not hinder the applicability of their findings to American whites at large.¹⁷¹

They were making a conceptual division between representation, which they disavowed, and extrapolation, which they embraced.

This distinction between representation and extrapolation would become critical in epidemiological research. The Framingham sample was never representative even of the town in which it was situated,¹⁷² much less the whole country or world. Nevertheless, by creating an epistemological model that contrasted cases and controls according to set characteristics, the risk factors identified at Framingham have turned out to hold up very

¹⁷⁰ “General Statement of Plans,” November 11, 1949, in the Heart Disease Epidemiology: Manuals of Operation Folder, Papers of the NHLBI, Bethesda, MD. In print, the investigators put it this way: As a start, a health educator was placed in the Health Department with the assignment of studying the community. This meant not only learning about the history, resources, and government of the town but, more important, getting to know the people—their national origins, economic conditions, and lines of social stratification; their religious, fraternal, and civic organizations; and their recognized and potential leaders.” Thomas R. Dawber, M.D., Gilcin F. Meadors, M.D., and Felix Moore, Jr., “Epidemiological Approaches to the Heart Disease: The Framingham Study,” *American Journal of Public Health* 41 (March 1951): 282.

¹⁷¹ As Gerald Oppenheimer points out (Personal Communication, April 2, 2007), part of what was going on in this transition from interest in knowing the community in order to gain its support to focusing on the cold hard facts of the differential distribution of risk factors might be contrast between the first investigator, Meadors, and the second, Dawber. Of course, the second already had the advantage of the successful recruitment by the first.

¹⁷² The sick and uncooperative were underrepresented in the sample. As Dawber pointed out: “The population for study was selected with the thought that data on prevalence and incidence of the various clinical entities would be applicable to much larger populations, including that of the United States as a whole. Yet because of incomplete participation of the selected sample, the Framingham investigators were hesitant afterward to publish reports on their findings of the prevalence of cardiovascular disorders...[undercounting sick, who were already in care]...Unless a comprehensive house-to-house survey of nonparticipants had been conducted, reliable data on prevalence could not have been obtained. However, as part of the public relations approach in the community, it had been agreed that no personal visits to households would be conducted...” Thomas Royle Dawber, *The Framingham Study, The Epidemiology of Atherosclerotic Disease* (Cambridge, MA: Harvard University Press, 1980), 59-60.

well in studies in other populations around the world. Thus the population became “normal” in a crucial sense: after they had been standardized into cases and controls along specific characteristics, participants in the Framingham study became a standardized norm from which to extrapolate.

The founding director of the Framingham Study was Gilcin Meadors.¹⁷³

Meadors’ 1947 proposal is still recognizable as what the project would become: “This project is designed to study the expression of coronary artery disease in a normal or unselected population and determine the factors predisposing to the development of the disease through laboratory experimentation and long-term followup of such a group”¹⁷⁴

The terms “normal” and “unselected” probably refer to the status of the population as “free of disease,” but there is a slippage that gives a valence to the community itself as normal. In an early undated draft of a description of the Heart Disease Epidemiology Study (which would become Framingham), Meadors wrote:

There is of course no ideal or perfect sample of the population in this country. Such a population would have the same distribution of such characteristics as race, sex, occupation, economic status, rural and urban location, and age as the general population. It is of less importance to have such a sample than to record rather accurately the characteristics of the group studied. To simplify the interpretation of results, however, a community with fair representation of the main characteristics would be desirable.¹⁷⁵

What are the bounds around concepts like “fair representation” and “main characteristics?” Who is eligible to be “normal?” Here, we can see a slippage between the population being studied as being normal and unselected in terms of being free of

¹⁷³ For an excellent description of his role in the early Framingham study and the interstices of the early history of the project, see Gerald Oppenheimer, “Becoming the Framingham Study,” *American Journal of Public Health* 95 (2005): 602-610.

¹⁷⁴ Gilcin Meadors Correspondence to Surgeon General Bert R. Boone, July 19, 1947, Papers of the NHLBI, Bethesda, Maryland, Epidemiology Correspondence Folder, 1947.

¹⁷⁵ Gilcin Meadors, Undated Draft, “Descriptions of the H.D.E.S. 1947-1949” Folder, Papers of the NHLBI, Bethesda, Maryland.

disease, and being normal and unselected in terms of being a “fair representation.”¹⁷⁶

Distinctions between representation and extrapolation continued to be articulated under the leadership of the new head of the study, Thomas Royle Dawber. In a published report in 1951, the investigators suggested that while it might be optimal to do the study in many cities and across groups, it would be too expensive. Writing in the *American Journal of Public Health*, they pointed out:

Ideally, perhaps, epidemiological investigations of cardiovascular disease should be set up in a number of widely separated areas simultaneously, so that various racial and ethnic groups will be represented, and a variety of geographic, socio-economic, and other environmental factors can be considered.¹⁷⁷

The two words that frame this sentence – “ideally” and “perhaps” – are both worth meditating on, because it is in the space between them that the Framingham study takes place. These words acknowledged the ideal of an infinitely expansive notion of sample population that was circulating in the period. And yet, at the same time, they questioned the assumption that such a sample would be ideal, independent of whether it was realistic. They suggested that even if it were possible to have a purely representative sample, the data produced would not necessarily be better. In this piece, the investigators defended their limited scope on practical grounds, but their scientific grounds for defense were not quite as well developed as they would become. They left open the possibility that until their findings were verified elsewhere they would not be generalizable:

It was concluded, therefore, that the study should be set up in a single area, and that coverage would have to be limited to approximately 6,000 persons in a limited age range, who would be observed for a period up to 20 years. A town of

¹⁷⁶ Although Meadors was from Mississippi, he nevertheless thought that such representation could be found in New England. That is, although he was Southern, he still understood a northern, Rockwell-type town as a fair representation for the population of the country.

¹⁷⁷ Thomas R. Dawber, Gilcin F. Meadors, and Felix Moore, Jr., “Epidemiological Approaches to the Heart Disease: The Framingham Study,” *American Journal of Public Health* 41 (March 1951): 281.

25,000 to 50,000 population will supply this number of adults, and it was felt that a town of this size would be more desirable than a larger city for the type of community approach required to secure full cooperation and coverage. This limitation in geographic coverage clearly limits the generality of conclusions which can be reached.¹⁷⁸

Yet, in the very same 1951 article, the Framingham researchers suggested that their findings might be capable of being extrapolated after all—but only as far as “the white race.” The investigators used the term “community” to describe Framingham rather than the more scientific-sounding “population” or “sample” used earlier by Meadors, but made a statistical argument for extrapolatability to all those of diverse European ancestries included in the post-war definition of “white:”

There is, however, reasonable basis for the belief that the distribution of arteriosclerosis and hypertension **in the white race** in the United States is such that within-community variance is greater than between-community variance, and a wide range of type-situations influencing development of these diseases may be found in any community.¹⁷⁹ [emphasis added]

After an article that had considered difference along many possible axes—regional, socioeconomic, “other environmental”—the only axis of difference that remained worthy of mention was whiteness. The fact that they were using an argument that is familiar in form to arguments about inter-racial and intra-racial difference today should alert us that it is not that mode of argument itself that is effective or not in closing the debate, but that its effectiveness depends on its alignment with other modes of defining racial difference. Yet as I will show, this homogenous whiteness of the Framingham study was not a given at the outset of the study, but a conceptualization of the population that emerged and became stable over the 1950s and 60s.

¹⁷⁸ Thomas R. Dawber., Gilcin F. Meadors, and Felix Moore, Jr., “Epidemiological Approaches to the Heart Disease: The Framingham Study,” *American Journal of Public Health* 41 (March 1951): 281.

¹⁷⁹ Thomas R. Dawber, Gilcin F. Meadors, and Felix Moore, Jr., “Epidemiological Approaches to the Heart Disease: The Framingham Study,” *American Journal of Public Health* 41 (March 1951): 281.

It is notable here that although race was routinely mentioned as a characteristic that might matter, the study being all white would not be understood as an important limitation. This is connected to the racialization of coronary disease discussed in the previous chapter. Infectious causes of heart disease were included in the scope of Framingham, and hypertension as etiology was included as one of many hypotheses, but the focus was squarely on the heart disease category understood as white: coronary artery disease. This would not by itself preclude investigators from studying blacks or others imagined to be at lower risk for coronary artery disease – after all, they included women in part to see what it was that might be protecting them. Yet it does set the scope of the relevant subjects of study.

As the descriptions of the study evolved, and leadership of the study transferred, the Framingham sample became framed less as “representative” and more as “not grossly atypical.” This description from an interim report by Dawber and Moore described the sample selection characteristically:

The town of Framingham cannot itself, of course, be regarded as a sample. It was picked with the advice of Dr. Vlado A. Getting, Health Commissioner of the Commonwealth of Massachusetts and Dr. David D. Rutstein, Professor of Preventative Medicine, Harvard University Medical School, because it was of the desired size to yield the required number of persons; it was close to a medical center; it contained both industrial and rural areas, and some assurance had been received that the townspeople, both lay and medical, would be willing to cooperate. In short, it was a place where such a study could be done, and it was not grossly atypical in any respect that appeared relevant.¹⁸⁰

¹⁸⁰ Thomas R. Dawber and Felix E. Moore, “Longitudinal Study of Heart Disease in Framingham, Massachusetts: An Interim Report,” *Research in Public Health: 1951 Annual Conference of the Milbank Memorial Fund, 1952*. Published by the Milbank Memorial Fund, 40 Wall Street, New York. More on its selection from the Epidemiology Correspondence Folder, 1947: September 8, 1947 letter from Gilcin Meadors to Bert Boone “Dr Rutstein mentioned Framingham to me as a possible site some time ago. If it is not too small (around 35,000 will be the lower limit) it appears to be otherwise suitable. I understand it is on a main highway 20 miles from Boston, and that it is a community and not a suburb of Boston.” Also, a September 5, 1947 letter from Lewis Robbins to Gilcin Meadors “Concerning the site, he [Getting] said

This concept of *not grossly atypical* was an innovation in understanding the applicability of biomedical knowledge, and can be read as a precursor for what Steven Epstein has described as the “creation of the standard human.”¹⁸¹ My project provides an example of the phenomenon Epstein observes, as well as extending it to consider how much work it takes to get people to look “not grossly atypical” before they can plausibly stand in as “standard.”

Dawber would continue to caution throughout his career against over-concern about the ways in which a sample population is not representative of a whole, including in his 1980 book which he meant to be the definitive summary of the study. He emphasized that a representative sample was both impossible and unnecessary. He argued that using scientific approaches to make comparisons within the group by well-defined characteristics would render random sampling superfluous:

Many epidemiologists become overly concerned about the degree to which a sample is representative of a larger population. In many ways, efforts to obtain complete participation of the entire selected sample were not justified. The objectives of the Framingham Study did not include determination of the absolute incidence of the various manifestations of atherosclerotic disease. Yet the determination had only limited applicability, since the population of this New England town was in itself not totally representative of the United States. There were virtually no blacks or Orientals, and the composition of the white population was not necessarily that of white populations elsewhere. Random sampling is not

that there were three likely places: Beverly, Peabody, and Framingham. Beverly is where the Children’s Bureau may set up a rheumatic fever program. Peabody is too far from Harvard. So the most likely place is Framingham, (Rutstein pulled for Framingham.) Gettings put in a call for the Superintendent of the hospital there for a bad [sic] for space in the event we should decide on this location. The Superintendent made a date to discuss this. Framingham has about 25,000 population, has been the site of former studies and is not one of the bed rooms of Boston. It is closer to the residences of Boston cardiologists, which he seemed to think would make a difference in their attendance.” “Rutstein was of great moral help in all this. He secured permission of the dean of Harvard Medical School to put us on the faculty, and assign us a room!”

¹⁸¹ Steven Epstein, “Bodily Differences and Collective Identities: The Politics of Gender and Race in Biomedical Research in the United States,” *Body and Society* 10 (2004): 191.

essential if the purpose of an epidemiologic study is to compare subgroups of the population determined by specific characteristics.¹⁸²

Later in the same text, Dawber went further. He suggested that not only could risk factors found through scientific comparison between groups of Framingham participants be imagined to apply to Americans generally, but even their incidence of heart disorders could be extrapolated:

In spite of these uncertainties, there appears to be good reason to accept the Framingham findings as a reasonable estimate of the actual incidence of the various disorders, with some obvious exceptions: there were too few black residents of Framingham to provide sufficient incidence data; the makeup of the white population was not completely representative of the U.S. white population: and there were more participants of Italian extraction than would be found in most communities in this country. However, unless national origin plays an important role (which apparently it does not), the data may be considered reasonably representative of the North American white population.”¹⁸³

In this quote, it went without saying that blacks (among other nonwhites) were not within the scope of extrapolation. And yet there is also something tantalizing in the brief mention of white national origin. When Dawber mentioned that national origin “apparently does not” play an important role, he glossed over the emergent processes that made that white functional homogeneity “apparent.”

Opening Framingham’s ethnic black box

In the invocations of connections between heart disease and race in the first few decades of the 20th century tracked in the previous chapter, it seems clear that heart disease

¹⁸² Thomas Royle Dawber, *The Framingham Study, The Epidemiology of Atherosclerotic Disease* (Cambridge, MA: Harvard University Press, 1980), 21-22.

¹⁸³ Thomas Royle Dawber, *The Framingham Study, The Epidemiology of Atherosclerotic Disease* (Cambridge, MA: Harvard University Press, 1980), 59-60.

investigators thought they knew who they were talking about. But by the time of the founding of the Framingham Study, heart disease investigators were not quite as sure. There was a fracturing of whiteness in their categorization, as well as an embrace of its inclusivity. Both fracturing and inclusivity are part of articulating the intersections of race and heart disease in the period. It mattered that the Framingham sample was white in the sense that it is a study of coronary disease, but at its inception the question of whether it mattered which kinds of whites they were remained open.

This section will attend to the actual racial imaginary from early in the Framingham study, as encoded in its early intake forms. It will then put them into context. This process will show that the homogeneity of the whiteness of the sample was not simply a given but was rather constructed iteratively in particular moments in time.

Between 1947 and 1951, the racial and ethnic coding on the subject intake forms was not stable. An early form marked “Set used April 1949 - June 1950 for volunteers to clinic” had questions about both race and national origin.¹⁸⁴ Options for race had two checkboxes and a blank: “W, N, or OTHER (Specify).” Options for national origin had a simple fill-in-the-blank. One set of proposed codings for the race question looks familiar to today’s racial categories: “Race: 1-White, 2-Negro, 3-Other, 4-Unknown.”¹⁸⁵ On the other hand, another proposed coding sheet in the same file has extraordinarily complex combinations the race and national origin questions:

¹⁸⁴ “Brief Cardiovascular History for Survey Screening,” PHS-1030(SR) 9-48, from the file labeled *Coding – First Examination*, at the Framingham Heart Study, Framingham, MA.

¹⁸⁵ “Proposed detail coding of Exam I, not used. Only Card 9 (the Summary card) of this series was actually punched,” from the file labeled *Coding – First Examination*, at the Framingham Heart Study, Framingham, MA.

- Col. 5-6 Race and sex:*
- | | | |
|------------------------|------------------------|-------------------------|
| <i>01 White male</i> | <i>02 White female</i> | <i>03 Negro male</i> |
| <i>04 Negro Female</i> | <i>05 Yellow male</i> | <i>06 Yellow female</i> |
- Col 11-12 National Origin*
- 01 All U.S.*
 - 02 U.S. and Gt. Britain*
 - 03 U.S. and Ireland*
 - 04 U.S. and Italy*
 - 05 U.S. and Scandinavia*
 - 06 U.S. and Germany or Holland*
 - 07 U.S. and France*
 - 08 U.S. and other*
 - 09 All Gt. Britain or Ireland*
 - 10 All Italy*
 - 12 Gt. Britain or Ireland, and Italy*
 - 13 British Isles and Scandinavia*
 - 14 British Isles and Germany*
 - 15 British Isles and France*
 - 16*
- Col. 13 Place of Birth of father*
- 0 U.S.*
 - 1 Canada*
 - 2 Gt. Britain*
 - 3 Ireland*
 - 4 Italy*
 - 5 Scandinavia*
 - 6 Germany or Holland*
 - 7 Belgium, France, Spain or Portugal*
 - 8 Other European*
 - 9 Other*
- Col. 14 Place of Birth of mother*
- 0*
 - ...*
 - 9*
- Col. 15 Place of birth of father's father*
- Col 16 Place of birth of father's mother*
- Col 17 Place of birth of mother's father*
- Col 18 Place of birth of mother's mother*

These sorts of coding practices seem never to have been actually implemented. These elaborate attempts suggest that it was not obvious how to manage fitting something as complex as race and national origin into new modes of data recording. At a time when many medical publications were based on a few cases or a few hundred, it was not yet

clear how to manage complex information on thousands of subjects. The iterative process of charting and re-charting coding options can be read as part of an emergent methodology.

A coherent order was arrived at by 1951. This coherence was presumably a result of the addition of the census-bureau veteran Felix E. Moore to the Framingham team in 1949.¹⁸⁶ Yet Moore did not use census categories. On the new forms, there was no longer a question on race,¹⁸⁷ and categories of National Origin were much less expansive and elaborated than those on the census forms of the same period.¹⁸⁸ The new forms would survive the 1950s and be coded onto punch-cards for the multivariate analysis that would become so important in how Framingham would make its claims.¹⁸⁹ Race may have dropped out because the whiteness of the study population had already been established. With data processing as onerous as it was, there was some disincentive against including variables not expected to vary much. There was only one question

¹⁸⁶ According to Daniel Levy, questions about applicability and representation came up in 1949 when the study was transferred to the National Heart Institute, and Moore was particularly sensitive. Daniel Levy, personal communication, October 30, 2006.

¹⁸⁷ “Code Sheet Framingham Heart Study Abstract,” PHS-1583, National Heart Institute Biometrics 1-15-51. From the file marked “Cohort Code Sheets 1-12,” Papers of the NHLBI, Bethesda, MD.

¹⁸⁸ The 1950 Census, for example, had 98 categories of “Foreign Country or Outlying Area” that could be listed as place of birth, including such specifics as Ireland separate from northern Ireland, as well as (this categorization is mine:) each European, Latin American, and Middle Eastern, Pacific country separately, though the whole continent of Africa was in one category. U.S. Dept. of Commerce Bureau of the Census, *Census of the Population, 1950 [United States]: Public Use Data Microsample*, “Appendix A3: Foreign Country and Outlying Area Codes.” Accessed through the Inter-University Consortium for Political and Social Research.

¹⁸⁹ The absence of a line for race would be source of a consternation later, when the researchers were asked about the racial makeup of their samples. In 1954, Philip Person wrote to McElholm: “After talking with various members of the staff I have compiled the list of case numbers shown below which are remembered to be negroes. It is unlikely that there are more than two or three others. However we will be glad to search the full list of case numbers if it is considered necessary.” He goes on to list “case numbers of known negroes,” and there are handwritten notes on the paper that say “another noted 9/02/57,” “another noted 3/16/60,” “another noted 6/29/60 (Portuguese + Negro).” Correspondence from Philip Person to D M McElholm, August 26, 1954. From the file marked “Coding – First Examination” at Framingham, MA.

about national origin, and it allowed for combinations to be punched in a much less unwieldy way than the one proposed above. The categories coded were:¹⁹⁰

National Origin (multiple punch)

This column is multiple-punch to include info on subject, parents, and grandparents—without specification as to which is which.

- 1 - United States and Alaska
- 2 - Great Britain, England, Scotland and Wales
- 3 - Ireland
- 4 - Italy
- 5 - Scandinavia (includes Finland, Denmark, Norway, Sweden and Iceland)
- 6 - Germany
- 7 - France
- 8 - Canada (includes Nova Scotia, Prince Edward Island and Newfoundland)
- 9 - Other

As implied in the notation “multiple punch,” “National Origin” was a question designed to accommodate more than one answer. First, the interviewer entered a number for the subject’s place of birth. For most of the participants, that meant entering a “1” in the box, which indicated United States and Alaskan origin, because the question was understood to be one not of indigeneity but of nativity. Then, if ancestors were from a different country from the subject, a number correlated with that place was entered as well. There was no limit on how many numbers could be checked, so mixed ancestry could be accounted for.

There were no numbers assigned here for Eastern Europeans or, remarkably, given their visibility in the community, for Jews.¹⁹¹ In early coding, it was clear that the

¹⁹⁰ “Abstract Card,” from the file marked “Cohort Code Sheets 1-12,” Papers of the NHLBI, Bethesda, MD. The same coding survives in “Deck 05 Coding Manual,” from *Tape Coding Manual Book 2 (of 3), File 06*. From the Papers of the NHLBI, Bethesda, MD.

¹⁹¹ Though the exclusion of religion as a category is logical enough in the immediate post-war period, I have not been able to guess at why Eastern European categories were not included. Perhaps Eastern European was in this community too close a proxy for religion. Though some Jews might have been coded as “Germany,” most were presumably marked simply “other.” Another possibility is that the categories at Framingham were simply taken from somewhere else with a different demographic range.

main interest was in “Native U.S.” versus “Foreign Born,” which is how an early spreadsheet filled in by hand breaks down the data.¹⁹² Even though early coding showed that the group was heavily British (1315 with British ancestry + 182 British born) and Irish (859 ancestry + 100 born), interest was strongest in Italian (400 ancestry +235 born). I would speculate that interest in Italians was disproportionate to their representation in the sample because a relatively large portion was foreign born compared with other subgroups as well as with the US at large.¹⁹³

In the early 1960s, there were some comments about the Italian-ness of Framingham’s sample in publications from the study. For example, a report about Nutrition noted: “The foods called ‘Pasta’ collectively are important in the Framingham Study because about 20 percent of the population is of Italian ethnic origin.”¹⁹⁴ There is something absurd about this note, since that table also features an important American pasta-based food staple: macaroni and cheese. It suggests that the Italians of Framingham still seemed a bit exotic to these researchers.

In 1967, in response to questions about the “Environmental Factors in Hypertension,” principal investigator Dawber talked about the Italians in the study with a most generous assessment of their numbers, even as he discounted the difference between them and the rest of the Framingham population:

¹⁹² “National Heart Institute Office of Biometric Research Framingham Epidemiological Study,” from the file marked *Coding Manuals*, Papers of the NHLBI, Bethesda, MD.

¹⁹³ According to a 1958 letter from the director of the Biometrics Research Branch of the NHLBI, 7.8% of the Framingham cohort was born in Italy and 20.2% had at least a grandparent born in Italy, as opposed to the white population of the US of which only 1.4% was born in Italy and 4.5% had at least one parent born in Italy. Correspondence from Tavia Gordon to Dr Kagan, June 4, 1958. “General Correspondence File: Jan 1, 1958 – June 3, 1958,” Papers of the NHLBI, Bethesda, Maryland.

¹⁹⁴ Thomas R. Dawber, Georgiana Pearson, Patricia Anderson, George V Mann, William Kannel, Dewey Shurtleff and Patricia McNamara, “Dietary Assessment in the Epidemiologic Study of Coronary Heart Disease: The Framingham Study,” *American Journal of Clinical Nutrition* 11 (September 1962): 224.

Q: Thomas: Have you compared the ethnic background and the number of generations in America between your college people and the high school and grade school people? In view of the waves of different nationalities arriving in America, I should think there might be differences in ethnic origin which would be worth looking at.

A: Dawber: About 40 per cent of the population in Framingham is of Italian extraction, mostly from Southern Italy. The educational level in these two precincts where they live is somewhat lower than in some of the other precincts. We could not get any clear relationship of national origin to coronary heart disease. I have not directly checked the relationship of national origin to blood pressure. My guess is that probably it too would be nil.¹⁹⁵

The exaggeration of the number of Italians may reflect the simultaneous foreignness and prominence of Italians in Framingham in the period. On the one hand, a high proportion of the Italians were foreign born compared to other nationalities. On the other hand, Italians were prominent in the city. Before the study began, the only certified cardiologist in Framingham had an Italian surname—Cornicelli.¹⁹⁶

National Origin did not remain a parameter in the findings of the Framingham Study. One reason that the National Origin question became less important was explicitly technological. Because it was a “multiple punch” question, data from it was lost when the NHLBI moved the data from punchcards to tapes in 1969.¹⁹⁷ To some degree, the shape that this large medical study would take has been deeply linked with its

¹⁹⁵ Dawber et al, “Environmental Factors in Hypertension,” in *The Epidemiology of Hypertension. Proceedings of an International Symposium sponsored by the Chicago Heart Association and the American Heart Association, Chicago, IL, February 3-7, 1964*, edited by J Stamler, R Stamler and TN Pullman (New York: Grune & Stratton, 1967), 284.

¹⁹⁶ Gerald Oppenheimer, “Becoming the Framingham Study,” *American Journal of Public Health* 95 (2005): 605.

¹⁹⁷ Paul Sorlie, Personal Communication, October 19, 2006. In 1969, according to Sorlie, certain types of multiple punch responses could not be easily transferred to data tapes, and so as a result the ancestry codes were left blank. Instructions for coding ancestry in all the files instructing coders in data conversion are crossed off and “Blank” is written in the corner, “Deck 06 Coding Manual,” from the *Tape Coding Manual Book 2 (of 3)*, from the Papers of the NHLBI, Bethesda, MD.

modes of data storage.¹⁹⁸ But the limitations of data recording and storage don't exactly preclude any given questions from being asked or pursued. These limitations only create an uneven terrain, in which some questions become easier and others more difficult to ask. This requires that the threshold of motivation be higher to make the harder-to-ask questions worth asking.

Looking at earlier publications from the Framingham Heart Study, we can see a transition in preoccupations with national origin around 1960. Two different publications had the subtitle "Six Years Follow-up Experience." The 1959 version, "Some Factors Associated with the Development of Coronary Heart Disease,"¹⁹⁹ described national origin as "not statistically significant." In the 1961 version, "Factors of Risk in the Development of Coronary Heart Disease,"²⁰⁰ national origin as a category of analysis had disappeared. A negative result on national origin was originally reported as a result nonetheless, but its declining relevance rather than its statistical power contributed to its disappearance.

The disappearance of this intra-white ethnic parameter from the Framingham data set was not inevitable. Two things might have led investigators to preserve the data on the National Origin questions. First, it might have remained of interest to investigators if early analysis had found more striking differences in cardiovascular disease by National

¹⁹⁸ This is a key STS point, and in this particular case the link between institutions of health data and computing are today physically striking, as the offices of the NHLBI and a branch of IBM are in facing buildings that share ground in suburban Maryland

¹⁹⁹ Thomas R Dawber, William B. Kannel, Nicholas Revotskie, Joseph Stokes, III, Abraham Kagan and Tavia Gordon, "Some Factors Associated With the Development of Coronary Heart Disease: Six Years' Follow-up Experience in the Framingham Study," *American Journal of Public Health* 49 (October 1959): 1349-1356.

²⁰⁰ William B. Kannel, Thomas R. Dawber, Abraham Kagan, Nicholas Revotskie and Joseph Stokes, III, "Factors of Risk in the Development of Coronary Heart Disease – Six-Year Follow-up Experience," *Annals of Internal Medicine* 55 (July 1961): 33-50.

Origin. Alternatively, if differences by National Origin, matter how statistically insignificant, had remained something that seemed to resonate with post-1960s preoccupations with differentiated American identities, the investigators might well have transferred more National Origin questions to working decks and thus preserved the data. A couple of questions related to ethnicity do survive in the data set because they had been put into binary working decks before the data transition: whether foreign born or not, and whether Italian or not. This may provide insight into the preoccupations with nativity and particular subgroups of whiteness that remained longest. Other questions along these lines could have been maintained/preserved in the later data had they remained salient to the researchers. But the specifics of National Origin have not remained durable in the way that black/white racial differences have; they have faded into the haze of 1950s pan-whiteness.

Framingham beyond white America

Over the 1960s, 70s, and 80s, the Framingham Study has provided the concept of the “risk factor” and evidence about some of the most foundational ones such as smoking, diet, hypertension and lipids. It has had an impact on evidence-based medicine and conceptualizations of risk categories of heart disease that is larger than can easily be estimated. Although this study was made by and about the implicitly white middle class and can be understood to tell a story about American health and life in the middle of the

20th century, it has also provided tools to tell stories of other categories and communities.²⁰¹

Early Framingham investigators did their research in an all-white population, but they participated in larger conversations about black/white differences, too. The Framingham investigators themselves participated in the simultaneous constructions of hypertension and African American hypertension in the 1960s, an era that saw the ascendance both of hypertension as a risk factor and of the Civil Rights Movement. Their own study's lack of inclusion of African Americans did not preclude their participation in arguments about racial differences in hypertension. Addressing "Environmental Factors in Hypertension" in a 1967 publication, the investigators wrote:

The principal population groups among whom blood pressures have been reported to be lower than among Americans and Europeans are various primitive peoples. The sample size has usually been small, especially in the older ages, and conclusions about age trends are complicated both by this fact, and by the fact that it is often not possible to accurately determine the age of the subjects. Among those population groups studied adequately, the following may be said:

Blood pressure distributions are similar among such diverse groups as: Caucasians living in Europe, the United States, and the West Indies; among Chinese living in Taiwan, and among Japanese in Japan.

Negro populations have higher blood pressures than whites living in the same areas and studied by the same investigators, particularly among females and in the

²⁰¹ It was also presented in the media as diagnosing the dangers of modern American life. For example, a 1963 *New York Times* article described: "The evidence now seems to indicate that cardiovascular diseases are diseases of civilization, of industrialization. Fifty years ago, for example, coronary heart disease was virtually unknown in the United States. Now it is a common cause of death. It is rare in underdeveloped areas of the world... The prevalence of cardiovascular disease in industrialized nations suggests that it is a social disease, that can be brought under control by manipulation of society. This means a change in the way of life. But what change? That is the question physicians are striving to answer. The growth picture of the type of person who is now believed to be most prone to cardiovascular diseases was given at the session by Dr. William B. Kannel of the Framingham (Mass.) Heart Disease Epidemiology Study. The susceptible person Dr. Kannel said, is an older male with an elevated cholesterol blood level, elevated blood pressure, and an abnormal electrocardiogram. He is a heavy smoker, he may be diabetic, and he has a family history of heart trouble. He does not get much exercise, he drives himself, and he is overweight." Robert K. Plum, "Social Clue Cited in Heart Disease: High Incidence in Industrial Nations Studied by Experts," *New York Times*, October 26, 1963, p. 10.

older age groups. Distributions of blood pressures among Negro populations living in the United States and in the West Indies, whether rural or urban, high or low salt eaters, are similar. Their blood pressures are higher than those of Negroes in Liberia, a principal source of Negro migration to the Western Hemisphere. Admixture of the Negro races in the Western Hemisphere makes the interpretation of this data difficult. It is in this general background of unencouraging experience that the study of particular environmental factors, which could conceivably affect the blood pressure level, must be approached.²⁰²

I will return to the question of African American Hypertension as a disease category in Chapter 4, but for now attend to other aspects of this quote. Here, we can see the distance between direct evidence or argument and the invocation of a common sense of racialization of cardiovascular disease. Although their phrasing evokes neutral grammars of data, there are no citations or evidence for these assertions about “Negro populations,” suggesting that the authors conceive of these statements less as arguments than as reflecting the consensus of the field. Unable to grapple with the embodied admixture that is not merely biological but also historical and cultural, much history is paved over in word choices such as “migration” to describe the slave trade and “admixture” to describe oppressive sexual relations under slavery.

Paucity of data is not actually the problem. The investigators make an odd claim about the cause of the difficulty of research into environmental causes of racial disease disparities: that “admixture” gets in the way of interpretation. Logically, assimilation would be the kind of mixing that would pose a problem for separating out environmental causes of disease by race, but the investigators lacked a language for cultural, in addition to biological admixture. The peculiarity of the investigators’ framing should alert us both

²⁰² Dawber et al, “Environmental Factors in Hypertension,” in *The Epidemiology of Hypertension. Proceedings of an International Symposium sponsored by the Chicago Heart Association and the American Heart Association, Chicago, IL, February 3-7, 1964*, edited by J Stamler, R Stamler and TN Pullman (New York: Grune & Stratton, 1967), 256.

to the fact of racialized hypertension's existence at the nexus of the biological and the environmental, and that Framingham is telling both a white story and a universal one.

Framingham's racial framework since the 1990s

The tremendously influential 1993 NIH Revitalization Act, which required that women and minorities be included in research studies,²⁰³ has had an impact on the Framingham study. Like other research studies, Framingham has had to address the requirement to include "diverse" populations in its sample. Though the study had always included women, it did not include a population that was *ethnically* diverse in the terms of the 1990s. In the process of complying with the act, Framingham has obscured the diversity of its own original cohort. Here is a snippet from a pamphlet celebrating Framingham's 50th anniversary:

On Oct. 22, 1994, during a visit to Framingham High School where the Elementary and Secondary Education Act was signed into law, President Clinton told students and other supporters that "our diversity in America is a goldmine of opportunity."

Framingham had a population of 64,989 then, and 11,464 were minorities. The Latino population was highest among those, with 5,291 residents. Once a homogenous, white middle-class suburb, this town had become more like a city, with an ethnic patchwork that included African Americans and new immigrants from Latin America and Asia.

Early the following year, the Framingham Heart Study began recruiting its minority population for the "Omni Study."

When 5,209 people were chosen for the Study in 1948, a random sampling was taken. Because few minorities were living in the town at the time, no conclusive information about specific groups could be culled.

²⁰³ See Steven Epstein, "Bodily Differences and Collective Identities: The Politics of Gender and Race in Biomedical Research in the United States," *Body and Society* 10 (2004): 183-203.

The Omni Study, with its narrower focus, would help health officials understand how and if heart, lung, and blood diseases exist disproportionately in some groups, see if risk factors associated with these diseases are the same or different for minorities than among the other cohorts, and find out if the so-called Americanization of some minorities had become detrimental to their health.

“It is important that the Study reflect the diversity of Framingham,” says Levy. This diversity gives us a chance to observe, and compare, the risk factor levels and unfolding of certain diseases in various groups.²⁰⁴

Having to comply with the new requirements was not an easy fit for a long-standing study never designed to be representative. It also revealed the entrenchment of Framingham’s racialization. Participation in the Framingham study has not since the first generation of participants been determined by residence in Framingham. Rather, only descendents of the original participants have been eligible to join the new cohorts of the main study. Citizenship in the Framingham study is thus hereditary. Unlike a model of citizenship determined spatially, kinship is a mode of relatedness that is presumed to necessarily preserve races of the past in the present. Therefore, the Framingham Study is particularly unsuited for this historical moment that Epstein has characterized as under an inclusion/difference paradigm.²⁰⁵

The Framingham Study has complied with new NIH guidelines by adding the Omni study, which looks at minorities in Framingham itself. However, even the investigators suggest that the numbers are too small to make any claims about racial/ethnic comparisons – there are only a few hundred participants, from all minority groups, chosen without any attempt at randomization. In the public relations pamphlet quoted above, the reason to include minorities is conceptualized in a very narrow way –

²⁰⁴ *You Changed America’s Heart: A 50th Anniversary Tribute to the Participants in the Framingham Heart Study*, NHLBI and Boston University, Pamphlet, 1998, pp. 14-15.

²⁰⁵ Steven Epstein, *Inclusion: The Politics of Difference in Medical Research* (Chicago: University of Chicago Press, 2007).

for the purposes of comparison against whites – rather than reconceptualizing community in a way that includes minorities as primary members. And indeed attempts to include the whole of the community have been half-hearted. Portuguese speakers, though highly visible in Framingham through the signs on the storefronts in downtown, are not included because the study staff lacks the language capabilities to include them.²⁰⁶

In discussions of the addition of the Omni cohort, funding considerations appear widely. O'Connor and Levy wrote in a memo in 1994 that included a line that would continue to appear in many more memos over the later 1990s: "To assure that the Framingham Study population will be representative of the U.S. population, and to be competitive for future NIH funding, it is essential that the Omni Cohort become an integral part of the Framingham Study. An Omni Cohort even larger than 300 will undoubtedly be needed."²⁰⁷ In 1998, a memo from an investigator to others read: "The feedback on my grant was very direct and critical about the lack of minorities. Whether it is scientifically sound, as both of you have pointed out, recruiting minorities will be critical for funding of ancillary studies."²⁰⁸ The recruitment of a random sample from amongst various diverse populations is not considered, but rather recruitment focus is on churches and hair salons. Problems of data management in the new cohort were not understood to be similar to those faced in the original study, but unique to "diverse"

²⁰⁶ As geographers Eugene Turner and James P. Allen have pointed out, the number of Brazilians in the Boston "Primary Metropolitan Statistical Area" has quadrupled over the 1990s, and they are now the highest foreign-born percentage of any group. Moreover, "Framingham has become the strongest Brazilian enclave, with many immigrants starting businesses that have helped rejuvenate the formerly declining Framingham Center." James P. Allen and Eugene Turner, "Boston's Emerging Ethnic Quilt: A Geographic Perspective," Paper Presented at the Population Association of America Annual Meeting, Boston, April 1, 2004. Available at their website: <http://www.csun.edu/~hfgeg005/eturner/gallery/Bostonatlas/Bpaper.pdf>.

²⁰⁷ Memo from George O'Connor and Daniel Levy to All FHS Staff, 6/14/94, from the OMNI Overview Binder, Framingham, MA.

²⁰⁸ Email from Emelia Benjamin to Phil Wold and Raph (?), April 25, 1996. From the OMNI Overview Binder, Framingham, MA.

populations. For example, cardiovascular disease events occurring “in non-English-speaking countries” are described as hard to verify.

Thus, the investigators have added the Omni Study to comply with new funding regimes, but do not incorporate the minority study comprehensively on the scale of the Framingham study itself. However, this does not suggest lack of commitment to the principle of including diverse populations in longitudinal research. Rather, the investigators have focused their energies on providing support for comprehensive study of diverse populations elsewhere. The Framingham investigators have been invaluable consultants on the most ambitious longitudinal research project to date that focuses on minorities, and does so with sufficient resources and attention to make it valuable: the Jackson Heart Study.

The Jackson Heart Study: The “black Framingham”

The founder of the Association of Black Cardiologists, Richard Allen Williams, cited the Framingham study many times in the cardiology chapter in his 1975 *Textbook of Black Related Diseases*, even though he was skeptical of its full applicability to black patients.

He made a plea for a Framingham-type study of a black population:

Numerous efforts have been made to draw a picture of the coronary profile of the American population. The Framingham study stands out as the best longitudinal investigation in this regard, and there is no doubt about the general usefulness of the information gathered. However, as mentioned above, the study was conducted mainly on Whites. The data collected in predominantly White Framingham may not be applicable to predominantly Black Watts, Hough, Roxbury, Newark, or Washington.

In order to know exactly what the characteristics of Black coronary-prone persons are, it should be obvious that Black persons at risk must be tested. This has not

been done on a prospective basis. Ideally, a longitudinal Framingham-type study should be structured for a community consisting of Blacks, Whites, and other racial and ethnic groups...

The use of an approach such as this would certainly help to establish the true incidence and nature of CAD in the Black population. No firm conclusions can be drawn regarding these matters by utilizing the data now in existence. It is hoped that such a study will be assembled in the near future. No better application of epidemiological principles to cardiology can be conceived.²⁰⁹

The Jackson Heart Study would be, in part, an answer to Williams' plea.

Whereas Framingham defined a white northeastern town as "an American town," the Jackson Heart Study, begun in 2000, has defined the large black population of a southern city as its foundational population. The parameters have changed in the fifty years between the two research designs, and the discourse of "the black Framingham" does interesting work with regard to concepts of American typicality, normality, and representation. I argue that the Jackson Heart Study can be read as a double post-modernity. First, both its population and research design are beyond modern/typical. Second, its epistemological framework is beyond observation to an emergent epistemology that interrogates the ways of knowing of both the investigators and the participants in ways that shape the science.

As mentioned at the opening of this chapter, the Jackson Heart Study's principal investigator Herman Taylor made a presentation on the occasion of the 20th anniversary of the International Society for Hypertension in Blacks.²¹⁰ Both he and his introducer spoke in laudatory terms about the Framingham Study (even if, amusingly, Taylor did accidentally refer to it as the Jackson Heart Study). In general, Taylor's version of the

²⁰⁹ Richard Allen Williams, "Cardiology," in *Textbook of Black Related Diseases* (New York: McGraw Hill, 1975), 381-2.

²¹⁰ Herman Taylor, "The Jackson Heart Study," Part of the Session on "The Evolution of Hypertension and Cardiovascular Risk: A 20-Year Perspective," International Society for Hypertension in Blacks Annual Conference. Atlanta, GA, 2006.

history of Framingham mapped very well onto that of Levy and Brink. He described it as a “typical American town, second generation immigrants.” Like the Framingham investigators writing their own history, he talked about that iconic death of FDR. Taylor and the authors of the Framingham study do not disagree on those things that frame the study: Framingham as typical, important. Taylor did not suggest that the people of Framingham were too white to be representative. On the contrary, he suggested that looking at any group “microscopically” would help to understand all people.

In this vein, the Jackson Heart Study does not necessarily aim to show the distinctiveness of African Americans. On the contrary, many of the findings have emphasized the similarities between blacks and whites. Principal investigator Taylor mentions, for example, that there are higher rates of coronary disease among blacks than many physicians think, since physicians have the erroneous idea in their “memory banks that blacks have only hypertension.”²¹¹

It is not, then, only (or even primarily) a belief about the radical difference of their population that drives the researchers of the Jackson Heart Study, but rather a belief that they, too, can represent “humanity.” They believe that looking closely enough on the local level can help to shed some light on the global level. They make the same disavowal of representation of an American whole that Dawber made. Like him, they stake a claim that attentiveness to intra-group difference will be extrapolatable to that group and beyond.

²¹¹ This coronary versus hypertensive divide is one I described in the previous chapter, and will delve into more fully in the next chapter.

History of the Jackson Heart Study

The Jackson Heart Study emerged out of the multi-community study called Atherosclerosis in Communities (ARIC), which began in 1987, seven years before the NIH guidelines requiring minority participation in research and yet clearly part of the trend that would lead to those guidelines. Jackson was selected as a African-American sample:

Of the four communities, Jackson, MS has been the site of the only all African-American cohort. African-Americans have a higher cardiovascular morbidity and mortality than Caucasians and Mississippi African-Americans exceed the national average. Focusing on an all African-American cohort, the JHS hopes to extend the ARIC focus on atherosclerosis and provide answers for the excessive cardiovascular disease burden of this ethnic group. There is a need to determine the manifestations, natural history and risk factors for African-Americans.²¹²

In an effort to reach decent levels of African American participation for the four-site ARIC study overall, which was mostly taking place in white-majority areas, Jackson was chosen as an all-black sample. The decision to make Jackson an all-black study, rather than merely black in proportion to its population, was made in the context of concern about the difficulty of recruitment and retention. It was hypothesized that recruiting and retaining a high enough level of black participants might be easier to do if the study were completely focused on that population. Key here, as with Framingham, is that the capacity to do the study well is more important than the sample's representation of some whole. When the time came to evaluate the ARIC study, it was decided to expand its Jackson site. Thus emerged the terrain for the Jackson Heart Study.

²¹² Wyatt et. al, *Community-Driven Model of Recruitment: The Jackson Heart Study: A Final Report of the Jackson Heart Study Participant Recruitment and Retention Study*, presented to the NHLBI, November 30, 1999.

Although the phrasing of Jackson as “the Black Framingham” does not capture it comprehensively and is one that some investigators are trying to “get beyond” for the Jackson Heart study to come into its own,²¹³ it is productive to take comparison between the Framingham and Jackson studies seriously. Why were these populations chosen? For Framingham, emphasis was on (1) stable but varied population that could be tracked for a long time, (2) proximity to investigators, and (3) amenability of the population to the research. Efforts to make each of those true shaped the design of the study, for example by including whole families as units rather than individuals, and having a role for volunteers as well as randomly selected subjects. A sense of civic duty fostered by identification with the small city was both sought out and bolstered by the investigators, and investigators cultivated a sense that the study “belongs to” the community of Framingham, in the service of the world.²¹⁴ Similarly, in Jackson, the choice was made to make it an all black study because it was felt that the community would be more likely to rally around it if it were all black rather than merely reflective of the demographics of the city (in the manner of the other ARIC populations).²¹⁵ Making the study all black was a way to help the community identify with it and take ownership of it, thus making the study itself feasible.

When I asked Bill Kannel, a former head of the Framingham Study and longtime investigator, what he thought about the Jackson Heart Study’s moniker as “the black Framingham,” he responded that “everybody tends to want to understand their own

²¹³ Francis Henderson, personal communication, November 13, 2006.

²¹⁴ Gerald Oppenheimer, “Becoming the Framingham Study,” *American Journal of Public Health* 95 (2005): 606; and Daniel Levy and Susan Brink, *A Change of Heart: How the Framingham Heart Study Helped Unravel the Mysteries of Cardiovascular Disease* (New York: Random House, 2005).

²¹⁵ Paul Sorlie, Personal Communication, October 19, 2006.

group.”²¹⁶ He explained that Framingham investigators had done an all white study of this kind, and suggested that it was logical that blacks would also want to do an all-black study of this kind. But there is both identification and elision in this concept of one’s “own” group. Most of the Framingham investigators were not, of course, from Framingham either. Kannel himself was from New York City, Meadors was Southern, Dawber was the Canadian-born child of English immigrants raised on Massachusetts’ South Shore. They need not identify with its population in any demographic or other sense in order to find the New England town as an appropriate object of study. Moreover, the town came to be their “own” as they developed the real, concrete connections of extended engagement with the community.

At Jackson, too, it is both true and a misrepresentation to say that the investigators are studying “their own” people. As in the Framingham study, there is some slippage in whether the community being studied is “the same” as the investigators on the one hand and some general population on the other. It is important that it is black investigators studying black patients, and they frame their particular expertise and mission in these terms. The relationship between the Jackson investigators and the population is between likeness and difference. The investigators are overwhelmingly black, and indeed an important part of the process has been increasing the pipeline of black health professionals and building connections between historically black institutions (Jackson State University and Tougaloo College) and the medical campus of the main state university (University of Mississippi Medical Center).²¹⁷ At the same time, most of the

²¹⁶ Bill Kannel, personal communication, September 29, 2006.

²¹⁷ NHLBI, “Jackson Heart Study: Design, Rationale, and Objectives,” <http://www.nhlbi.nih.gov/about/jackson/2ndpg.htm>.

investigators are not from Jackson, have been trained elsewhere, and the investigators are very conscientious about the gaps between themselves and their research participants.

Jackson's research design

Jackson's scientific goals include further study of known heart disease risk factors with a focus on hypertension-related disease, as well as sociocultural risk factors such as stress, racism, and coping strategies. It also hopes to study intra-racial genetic factors and identify novel risk factors in CVD.

The racial and ethnic categorizations in ARIC, like NIH-funded studies generally, use the current census categories and look very different from the multiple-punch Framingham model. It was the interviewers filling out the forms who chose how to categorize the patients by race, and the possibilities were limited to white, black, Native, or Asian:

23. Race: Record the participant's race as White, Black, American Indian or Alaskan Indian, or Asian or Pacific Islander. This may require asking the question verbally if it is not obvious.²¹⁸

Questions that were considered “too sensitive” to ask in Framingham included those about class and psychological state. In contrast, in Jackson those questions are at the fore. There is attention not only to socioeconomic status (SES) generally, but to variations in SES over the life course (which is a particularly American way to conceptualize class—not as a fixed category, but as a state that is simultaneously real and

²¹⁸ FORM A-113, FASTING / TRACKING FORM INSTRUCTIONS, from the “Cohort Component Procedures,” Manual 2, Appendix 9, p. 4, available at http://www.csc.unc.edu/aric/visit/Appendix.1_2_9.pdf.

in flux). There are also careful measures of stress, focused on stressors of racism and of daily life.

One question that was not considered “too sensitive” in Framingham but was considered “too sensitive” in Jackson was skin color. Where Framingham asked investigators to include their assessment of “skin color” as “normal,” “pale,” “ruddy,” “sallow,” or “jaundiced,” as well as degree and location of freckling,²¹⁹ in Jackson “[i]ncluding a direct or indirect measure of skin color was given substantial consideration and ultimately abandoned for reasons of cultural sensitivity.”²²⁰ On the other hand, while questions about religion were specifically excluded from Framingham, “organizational and private religiousness; spiritual experiences; religious coping” are all inquired about as measures of the construct of religion as a potentially important psychosocial resource and specifically included in the Jackson data.²²¹

There are a couple of key differences between the way that Framingham was conceptualized and the way that Jackson has been. Where Framingham’s observation-only approach made no claims to benefit the participants in the study themselves or take their questions and concerns to heart in the study design,²²² that narrow framing is no

²¹⁹ “Cardiovascular Examination Code Sheet,” Federal Security Agency Public Health Service, PHS-1446-4(NIH) 8-50. From the file marked “Cohort Code Sheets 1-12,” with handwritten note at top stating “used for Exam I; beginning case #3375.” Papers of the NHLBI, Bethesda, MD.

²²⁰ Thomas J. Payne, et. al., “Sociocultural Methods in the Jackson Heart Study: Conceptual and Descriptive Overview, Ethnicity & Disease,” *Ethnicity and Disease* 15 (Autumn 2005).

²²¹ Thomas J. Payne, et. al., “Sociocultural Methods in the Jackson Heart Study: Conceptual and Descriptive Overview, Ethnicity & Disease,” *Ethnicity and Disease* 15 (Autumn 2005).

²²² Which is not to say that the Framingham participants were not treated, they were imagined to have their own doctors treating them, and that treatment was imagined to be facilitated by the Framingham investigators rather than inhibited by them. As Dawber describes, “At the time the Framingham Study began, therapy for hypertension was in its infancy. For the most part it consisted in minimal barbiturate sedation and weight control. The difficulties of the rice/fruit diet and other sodium-free regimens made such treatment impossible except for a very few (usually those who had advanced hypertension, often with severe complications such as congestive failure). During the course of the study the various agents used today to treat elevated blood pressure were gradually introduced. Reports of the physical status of the

longer acceptable. Now there is a need to help the actual community of study, while studying it and beyond, and incorporate community concerns in a way that reaches beyond “recruitmentology” in Epstein’s terms and changes the way the science is done.²²³ Relatedly, there is an explicit aspect of *capacity building* built into the purpose of the Jackson Heart Study. Part of the purpose of the study is not only its own findings, but building a cadre of future minority investigators. Including two historically black institutions among the three running the study is a way of putting African Americans into the pipeline of the major economic engine of biomedical research.

Jackson as a model city, post-typical

If the presence of the “Shoppers’ World” mall in Framingham was part of what made that town emblematic of the age in which the study arose, the Jackson Heart Study’s location attached to “the medical mall” is also fitting. The Jackson Heart Study is attached to a facility called “the medical mall,” because it was a mall that was defunct for some time

Framingham subjects, including blood-pressure values, were supplied to family physicians with attention directed to any change of status; it would therefore be expected that the participants would not only be placed on antihypertensive treatment but would very likely receive much more therapy than a similar group not so carefully evaluated.” Thomas Royle Dawber, *The Framingham Study, The Epidemiology of Atherosclerotic Disease* (Cambridge, MA: Harvard University Press, 1980), 86-87.

²²³ A summary of the design and methods of the Jackson Heart Study summarizes the aspects of recruitment this way: “1) the need to integrate scientific data gathering with serving community and family aspirations; 2) the importance of continuous involvement in the community, rather than sporadic involvement primarily serving investigator priorities; 3) the importance of community involvement in selecting and training recruiters and clinic staff and in defining clinic procedures; and 4) the value of community members in helping to define the language and description of the study and in seeking health community assent to proceed.” Herman A. Taylor Jr. et. al., “Toward resolution of cardiovascular health disparities in African Americans: design and methods of the Jackson Heart Study,” *Ethnicity and Disease* 15 (Autumn 2005) (4 Suppl 6): S6-6.

and then became a clinic and medical center of sorts.²²⁴ It is in one of Jackson's black neighborhoods, and was neglected before being reclaimed as "one stop shopping" for nonprofit medical care. This edifice of the shopping-mall-turned-medical-area captures a postmodern moment that is postindustrial in its organization of labor and of consumption.²²⁵

Jackson, like Framingham, disavows representation and yet still operates on valences of a "typical American town." If America of the 1950s was plausibly a New England small town, it might today be Southern sprawl. When the Framingham investigators found something "not grossly atypical" in 1948 it was a New England town with second-generation European immigrants adding to the old white mix. In an atypicality that is no longer afraid to be blatant, the Jackson investigators found their model population in 2000 in a Sun Belt city in which it is African Americans who are added to the old white mix, but considered separately. In terms of immigration, Jackson is not "typical" – indeed, ironically, if Framingham were to be studied in a fully inclusive way it would be more emblematic of the current historical moment in terms of immigration. The retro sensibility evoked in descriptions of the 1950s' small town white middle class described by Levy and Brink among others²²⁶ is in striking contrast with pessimistic and even alarmist media invocations of recent immigration to Framingham

²²⁴ The Jackson Medical Mall is technically separate from the Jackson Heart Study, though spatially they are indistinct. The Jackson Medical Mall includes a few retail shops and eating facilities as well as Cancer Care and Kidney Care centers and clinics under the auspices of the University of Mississippi Medical Center. See <http://www.jacksonmedicalmall.org/home.html>

²²⁵ See David Charles Sloane and Beverlie Conant Sloane, *Medicine Moves to the Mall* (Baltimore: Johns Hopkins University Press, 2003); Allan M. Brandt and David C. Sloane, "Of Beds and Benches: Building the Modern American Hospital," in *The Architecture of Science*, ed. Peter Gallison and Emily Thompson (Cambridge, MA: MIT Press, 1999), 281-308.

²²⁶ Joel Millman critiques alarmism about immigrants and crime in his hometown of Framingham in "Going Nativist: How the Press Paints a False Picture of the Effects of Immigration," *Columbia Journalism Review*, January/February 1999, available at <http://archives.cjr.org/year/99/1/nativist.asp>.

from Brazil and elsewhere. And yet Jackson is pulling on some considerable valences of this historical moment in America: post-industrial, sprawling, sun belt, diffuse. In the words of it's principal investigator: "The scientific questions and the population on which the JHS focuses make it a study for our time."²²⁷

The Jackson Heart Study does not pretend to represent its city's entire population; it is nonwhite by design. Unlike in Massachusetts, where the OMNI study included whites who were involved in minority communities because of concerns that they would feel excluded,²²⁸ there do not seem to have been concerns from whites in Jackson about lack of "inclusion."²²⁹ Some physicians who serve blacks in the Mississippi Delta have complained that they should have been included, because their problems are the most acute and the study should have been done there rather than in the city of Jackson, but no whites have complained.²³⁰

In the context of studies that claim to represent the "whole" while routinely failing to represent the African American part of that whole, claims to be able to study a whole no longer seem tenable in quite the same way. Similar to but more convincingly than Framingham, Jackson articulates its project as a valuable contribution to a field of which it is a stellar member rather than as a lone study that will answer all questions. In

²²⁷ Herman A. Taylor, "The Jackson Heart Study: An Overview," *Ethnicity and Disease*, 15 (Autumn 2005): S6-1.

²²⁸ "The 6 non-Hispanic subjects were recruited as courtesy because of their association with Framingham's minority community. They were assigned a different ID type "A" instead of "7". Their data was not used for analysis and their information was deleted from the roster." Paulina Drummond, "Omni Cohort of the Framingham Heart Study," from the Omni Overview Binder, Framingham, MA.

²²⁹ Frances Henderson, personal communication, November 13, 2006.

²³⁰ I asked one of the Jackson investigators whether it might be a regional thing. She said that for better or for worse there is an understanding that blacks and whites are separate. The rural/urban divide is another wrinkle, one that could raise questions about how representative can Jackson be of the state of Blacks in the country? Blacks are to Mississippi in some way that is different from the way that Mississippi is to Blacks.

the context of huge amounts of data and what appear to be particular gaps, selecting the African Americans alone comes to make sense.

Analogously to the Framingham Heart Study population compared with the general white population, the population studied at the Jackson Heart Study is more middle class, more educated, living a better lifestyle than the African American average. But this is part of how it becomes “normal.” It is a norm in the normative sense of the word, not in the sense of an average. Poverty is explicitly represented in the Jackson Heart Study population, but so is the college-educated black middle class, with the disproportion to the latter, and this atypicality is invoked to suggest that the study is uniquely comprehensive.

Emergent epistemology

In understanding Jackson as a post-modern project, I am using a framing that is in alignment with that of the investigators themselves. The researchers consider themselves to be making contributions to interpretive phenomenology.

One epistemological challenge of which the Jackson investigators are well aware is the influence that the questions asked have on the data. In their report *Community-Driven Model of Recruitment*, they cite Heidegger and interpret him to expound:

The research paradigms of modern physics and health-related research are similar. Whether researchers are attempting to model complex interactions between subatomic particles, physiological control systems or constructs like hostility and cardiovascular disease the basic conceptual tools of measurement and inference are similar to those outlined above. The question to be asked here is: How do we bring to the process of questioning the phenomenon we seek to study in terms of

setting it up to respond in certain ways? What is closed down, invisible or outside the gaze of the researcher when a particular question is tested?²³¹.

Moreover, they emphasize that not only their own framings but also those their research subjects bring to the encounter shape the data:

It is possible to contrast basic sciences with social sciences based on the extent to which meanings and interpretations for both participants and investigators hold sway for the phenomenon being studied. The questions asked by basic science generally lead to experiments that can be replicated by trained individuals without concern for cultural or group context. This is not to say that the questions being asked do not still have a dominant cultural context but simply that the phenomena being studied are not particularly sensitive to this effect. In contrast, it is possible for interviewers from different ethnic backgrounds to obtain different responses on surveys than those whose ethnicity matches the participants. Instruments developed for measuring psychosocial dimensions in one group may fail or be meaningless for another group because of differences in the shared languages, meanings and practices between the groups. Certainly the importance of cultural understanding, shared meaning and trust to African-American participation in research highlighted in the previous literature review is particularly relevant here and points to the potential for nonnumeric questions to be asked of a phenomenon of interest.²³²

They see their contributions as not limited to science in the sense of data, but also interpretation:

Interpretive Methodology: Interpretive phenomenology, specifically Heideggerian hermeneutic phenomenology, was the background for the interpretive interview component of the study. Hermeneutics, or interpretation, as an approach to research acknowledges the situated temporal nature of both the researcher and the participants. The hermeneutic method works to uncover how humans are 'always already' given as time. That is to say, humans are always already in their own worlds, living in particular spaces, times and ways. Hermeneutics has no beginning or end that can be concretely defined, but is a continuing experience for all who participate. The work of the interpretive phenomenologist moves beyond traditional logical structures in order to reveal and explicate otherwise hidden

²³¹ Wyatt et. al. *Community-Driven Model of Recruitment: The Jackson Heart Study A Final Report of the Jackson Heart Study Participant Recruitment and Retention Study*, presented to the NHLBI, November 30, 1999, pp. 26.

²³² Wyatt et. al. *Community-Driven Model of Recruitment: The Jackson Heart Study A Final Report of the Jackson Heart Study Participant Recruitment and Retention Study*, presented to the NHLBI, November 30, 1999, pp. 26.

relationships. The descriptions of what *is*, are interpreted to show what *could be*.²³³

They quote Cornel West, suggesting that “vigilance is necessary to guard against systems of domination and control in cultural studies where research practices may serve to ‘highlight notions of difference, marginality, and otherness in such a way that it further marginalizes actual people of difference and otherness.’” And they explicitly set out to do a different kind of science. Jackson’s interest in the lives of its subjects exceeds that of Framingham’s focus on recruitment. Jackson’s approach combines this interest in recruitment and retention with a sense that the value of the research is at stake. It is not a mere repetition of Framingham, but a repetition with a difference.

Jackson in the shadow of Framingham

As described at the chapter’s opening, the Jackson Heart Study’s principal investigator asserts that the study is necessary because this population has been left out of the “golden age of cardiology” that was ushered in by Framingham and related therapeutic advances. While other populations have benefited greatly and seen a reverse in the increasing CVD mortality they had been experiencing in the first half of the 20th century, there has been a plateau among African Americans. This sense of being left out of a progressive narrative of history is an interesting one in the context of the history of cardiology.

Even though the investigators hope that Jackson can tell us something not only about black people in Mississippi but about people in general, like Framingham did, that

²³³ Wyatt et. al. *Community-Driven Model of Recruitment: The Jackson Heart Study A Final Report of the Jackson Heart Study Participant Recruitment and Retention Study*, presented to the NHLBI, November 30, 1999, pp. 26-27.

is not a seamless proposition. It is an open question whether the reception of Jackson will be read to suggest anything for “America,” or “people.” Its results may be raced in such a way as to be exempted from the broadly applicable. This is the sort of question with which I will reengage with in discussions of pharmaceuticals, in particular the African American Heart Failure Trial (AHeFT), which was the trial that led to the approval of BiDil for “heart failure in blacks.” There has been frustration among some black cardiologists that the Food and Drug Administration could not see that AHeFT showed that BiDil works in people including blacks, not just in people who are blacks. At stake is who can speak to universality.

Questions of branding are paramount in this sort of grammar of the [Black] [Framingham], which we might map grammatically as the [new kind of] [known thing]. To point out that associative branding work is being done is not necessarily to criticize it. When Daniel Levy describes the Jackson study’s reference to Framingham in terms of a “brand name that physicians know,” it is without derision.²³⁴ Indeed, brand extension does not diminish the value of the original brand, but raises its status as referent. The emergence of Jackson does not reduce the funding or other resources available for Framingham, but rather increases the size of the field of its type of large scale longitudinal research. And Levy, himself, together with others involved in Framingham, are proud of the work they have done supporting the Jackson Heart Study.

Anxieties of each age of the 20th century are expressed through the disease categories that become the focus of cardiological research. In the previous chapter, we saw that coronary disease was considered in terms of the racialized modern life of the

²³⁴ Daniel Levy, personal communication, October 30, 2006.

elite. This chapter has explored two more periods, each with attendant racialized cardiovascular disease. Framingham has attended first and foremost to the coronary artery disease from combined risk factors of the white middle class. Jackson attends to the burden of morbidity and mortality on people of color who were left behind by America's post-war medical advances. Medical knowledge about the diseases of the heart comes to include an expanding notion of who counts as American: from WASP, to pan-white, to racially fractured, and in parallel from high SES to middle class and then also low SES.

In some senses, Framingham is a fulfillment of the modernizing aspirations of the founding of cardiology. Framingham reaches the standardization of research and diagnosis identified as goals in the earlier period and does so in a larger community understood as modern and American. Framingham stakes out a space between racial representation and racial normality as it grapples with how to answer questions about normal pathologies of normal life. Jackson is a postmodern *repetition with a difference*. The Jackson Heart Study, I have argued, is a postmodern innovation that both elaborates and fractures the enterprise of early cardiology and the Framingham study. It simultaneously represents innovations in and limits of the concepts of systematization and of model population.

“The black Framingham” is not Framingham with merely a tonal difference. The differences in its parameters due to its racialization are not separable from its historical location a half-century after the former began. It is also the socio-culturally-aware Framingham, the genomically-savvy Framingham, the Southern Framingham, the urban-

sprawl Framingham. Each of these differences are intertwined with its racial difference, as are its double post-modernities of post-typical and post-observational.

Chapter 4

The Durability of African American Hypertension as a Disease Category

Vignette

On May 14, 2005, the American Society of Hypertension (ASH) had its 20th Annual Meeting at the San Francisco Marriott.²³⁵ As a new and special feature, the proceedings dedicated the first day to “special symposiums” that addressed “special populations.” Parallel 6-hour-long programs addressed special concerns of treating hypertension in African American, Hispanic, and Japanese populations.

The Symposium on “Hypertension in the African American Population” consisted of a series of presentations that included speeches, panels, and a debate. The first presenter was genetic epidemiologist Richard Cooper who spoke about race and genetics, arguing that “continental race” – whether ancestry is, say, African or European – is *not* useful in understanding disparities. He pointed out that, contrary to popular belief, American blacks have lower blood pressures than some European populations, notably

²³⁵ American Society for Hypertension Annual Meeting, Scientific Sessions, “Special Symposium: Hypertension in the African American Population,” San Francisco, CA, May 14, 2005.

Finns. Richard Allen Williams, founder of the Association of Black Cardiologists spoke next, and explicitly declined to refute what Cooper had said about race lacking a genetic basis. He said that his concern was not whether race was real, but whether racial disparities are: the answer was yes, as is especially evident in the death gap. After talking about the history of race and medicine, from 19th century predictions of African American extinction to examples of abuse in experiments and unequal access to treatment, he showed a cartoon of a black patient saying to his white doctor: “Doc, studies show if I were white you’d be rushing me to catheterization,” and the doctor responding “I thought Roloids would do.” The audience laughed warmly. He described his training by Paul Dudley White, who told him that “the full-blooded Negro never has coronary artery disease,” which got another laugh. In a juxtaposition, he closed with a photo of three hearts, which he described as black, white, and yellow, pointing out that they all look the same and that indeed “we are all the same.”

Some sessions dealing with clinical trials and drug regimes followed. A stand-in for the African American Heart Failure Trial (A-HeFT) investigator Anne Taylor spoke next, summarizing the trial that would lead to the imminent approval of BiDil. Seeming defensive in the context of the anti-genetics discussions that preceded it, the bottom line of the presentation was that “we need to do studies of subgroups not because of genetics, but because people bring a lot to the table we don’t control for.” Then came International Society for Hypertension in Blacks’ Janice Douglas, who explained the rationale for the ISHIB guidelines which urge lower blood pressure goals for African Americans. She justified the aggressive goals by pointing out the premature onset, greater severity, nocturnal non-dipping, and greater target organ injury in that population. The next

session was a debate between Kenneth Jamerson and ALLHAT's²³⁶ Jackson Wright about whether ACEs and ARBs should be included in first-line therapy for African Americans. While Wright argued that African Americans' relatively poor response to those expensive new drugs was a suggestive lack of evidence for their effectiveness in that group, Jamerson suggested that it was an "artifact of data overanalysis" without physiological basis. Animated questions followed.

Then there was a change of tone. Looking precisely the elder statesman that he is in the community, ASH charter member and ISHIB founder Elijah Saunders took the stage and talked about the practical implications of African American hypertension. He spoke with an authority that projected an above-the-fray sensibility, saying that there is a lot we don't know about mechanisms, but we do know that blacks are dying and we need to save lives. Rather than worrying about genetics, or who is really black, or what have you – OK, research that, he said. "But we are talking about people who look like this picture," he said, showing a picture of an older African American couple, which brought a warm laugh. He said that for people like those in the picture, we know that the dietary modification works, and that no drug class should be eliminated from their treatment.

The panel continued, but I want to pause at this moment. Saunders' discursive intervention here is an instance of a tactic I have seen often in my investigation of the field of race and medicine: a call to clinical pragmatics²³⁷ after expositions of natural science. There are two rhetorical moves happening here. First, there is a call to open up

²³⁶ ALLHAT refers to the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial, which was the largest anti-hypertensive trial ever conducted and will be important in the next chapter.

²³⁷ The pragmatism of these clinicians has resonances with that theorized by Cornel West because it accepts the impossibility of the perfectibility of the world without abdicating the moral imperative to strive toward justice. Cornel West, "On Prophetic Pragmatism," in *The Cornel West Reader* (New York: Basic Books, 1999), 149-173.

fundamental questions about race. Second, there is a call to close the conversation to allow implementation of operational answers. These are not actually opposed rhetorical moves, as we can tell by the fact that they are made by the very same people. Elijah Saunders himself had participated in the organization of the symposium. Indeed, he saw the event as something of a vindication of his long advocacy for more attention within ASH to African Americans as a high-risk group.²³⁸ Thus, both in his career and in this particular symposium, Saunders participates in both opening the debate on the definition and role of race in hypertension and moving to close it, at least sufficient to mobilize response to African American hypertension. The panel he had pulled together had used an array of approaches to try to define the complexities of race. They invoked genetics (albeit to reject them), history, humanistic moral claims, pathophysiology, subgroup analysis, and focused clinical trials. And yet Saunders made the rhetorical move here of saying that the epistemological debates and cutting edge research are beside the point: we know who we are talking about and that they are not getting well-established good care.

Introduction

How can we understand the simultaneous drive to further research, and the assertion that sufficient therapeutic intervention is already possible? We can gain insight into a format of presentation that alternates between opening up the status of African American Hypertension as a disease category and then moving to close and operationalize it by

²³⁸ Interview with Elijah Saunders, San Juan, PR, July 18, 2005.

seeing this alternation as central to the productivity of medicine as a field that is both scientific and therapeutic.

Medicine is an excellent sphere within which to interrogate race at the intersection of the social and the biological because medicine's research and practice lie at precisely this intersection. Medicine's emphasis on *intervention* on the social and the biological makes medicine a better site for STS analysis of race than the one usually explored - genetics. If the Human Genome Diversity Project, for example, fixes (reifies) race in order to put it in a museum,²³⁹ a conceptually different process is at work when practitioners fix (reify) race in order to act on it. The mobilization of race in medical practice as distinct from natural science is a theme I will pursue further in subsequent chapters on race and therapeutics.

In this chapter, I argue that in order to understand the productivity of race in medicine, its status as category needs to be recognized as *simultaneously* one of bioscientific inquiry and one of practical clinical taxonomy. The task for STS scholars should not be to seek out a true or innocent science that makes false promises to purge the material-semiotic category of race from biology, but to track plural noninnocent discourses at the nexus of the biological and the social. I argue that critiques that address only scientific reification without attending to clinical intervention both fail to engage, and contribute to the durability of the categories that they ostensibly assail. Race in biomedicine does not originate in the science and filter down to the doctors' offices; neither does it simply filter up. It does slightly different work in each sphere, but gains its durability at the nexus.

²³⁹ See Jenny Reardon, *Race to the Finish: Identity and Governance in an Age of Genomics* (Princeton, NJ: Princeton University Press, 2004).

This chapter has three parts. The first section outlines the historical emergence of “African American Hypertension”²⁴⁰ as a disease category at the confluence of the emergence of hypertension as a risk factor, professionalization and civil rights articulations around it, and the transposition of older ideas about the differences between black and white heart disease (from coronary/white and infectious/black to coronary/white and hypertensive/black). The racialization of this disease category has proven to be durable. The second section closely reads a nature/nurture debate about African American Hypertension about what is called “the slavery hypothesis” to understand the consensus that the debate rests on and reinforces. The durability of race in the debate does not come only from its connection with genetics, but also from far more epistemologically eclectic interventionist medical practice. The third section considers attempts to dislodge race amid current invocations of the disease. I will show that race is durable because it can stand in for so many other variables that are harder to record (genetics, socio-economic status, culture, racism, and more) and give purchase to critiques of persistent inequality. Diverse actors seeking to mobilize around African American Hypertension need not agree on what underlies it in order to operationalize it in action.²⁴¹ We will see that race’s productivity in medical data and practice relies not on

²⁴⁰ As with so many linguistic constructions of race, the term has changed over time. Writing now, I use the current phrase, leaving references to “Hypertension in the Negro” or “Black Hypertension” unchanged when quoting. This is not necessarily an ahistorical move, since my interest is precisely how these earlier categories come to be understood in the present as genealogies of current disease discourses.

²⁴¹ This approach has resonances with the concept of the “boundary object,” which Star and Griesemer have theorized as “an analytic concept of those scientific objects which both inhabit several intersecting social worlds *and* satisfy the informational requirements of each of them. Boundary objects are objects which are both plastic enough to adapt to local needs and the constraints of the several parties employing them, yet robust enough to maintain a common identity across sites.” Susan Leigh Star and James R. Griesemer, “Institutional Ecology, ‘Translations’ and Boundary Objects: Amateurs and Professionals in Berkeley’s Museum of Vertebrate Zoology, 1907-1939,” *Social Studies of Science* 19 (Aug 1989): 387-420. However, my focus is less on how this object of “African American hypertension” represents an incomplete intersection of diverse social worlds imagined to be autonomous on different sides of some

its measurability, but rather on its *recordability*. The flexible gap between immeasurable race and recordable race is precisely part of its power.

Notes on the historical emergence of “African American hypertension”

The ASH symposium that I have described as an introduction and will use as a framing device represents a particular moment in the status of African American Hypertension as a disease category. How did we get to this point, such that it makes sense to set aside a day of a hypertension conference for “special populations,” African Americans among them? How did this disease category come to have these tensions in which arguments about the social and biological are raised without quite intersecting, and bioscientific arguments remain somewhat parallel to clinical medical practice?

Three trajectories intersected from the 1960s on to make African American Hypertension a durable disease category: (1) the hypertension that had long been observed to be high in African Americans emerged as a key modifiable etiological risk factor for heart disease, (2) black and other Civil Rights oriented physicians mobilized and professionalized around non-epidemic disease, especially heart disease; (3) generative racialized distinctions between coronary and infectious heart disease that had been important in the founding of cardiology mapped onto distinctions between coronary and hypertensive heart disease.

disciplinary divide (say, science versus medicine), than on the productive tension of the boundary-crossing heterogeneity of race *within* the sphere of clinical medicine itself and among those who claim standing to speak on questions that cross the divides.

It has “always” been known that African Americans had high rates of hypertension²⁴²—even back when hypertension was only conceptualized as a rare and immediately malignant condition. It has also “always” been known that blacks have higher blood pressures than whites, even in nonpathologic ranges. According to epidemiologists Kaufman and Hall,²⁴³ the first instance of racial comparison of blood pressure was in a 1932 report in the *American Journal of the Medical Sciences*. That piece considered blood pressure as one of the “physical differences” between races considered in a separate section from the differential morbidity the article would go on to catalogue:

An experience of 11 years in an industry employing 5074 men between the ages of 18 and 65, approximately one third of whom were colored and the remainder white, showed marked differences in physical characteristics and morbidity statistics in the two races. The most striking difference noted on physical examination was blood pressure.²⁴⁴

The morbidity tracked in the article, however, had nothing to do with blood pressure, but rather spanned the gamut of infectious diseases (tuberculosis, influenza, malaria, venereal disease), appendicitis, home injuries, and degenerative diseases. Blood pressure was a physical marker without – yet – clear connections to morbidity.

²⁴² “Always” is the word many physicians with whom I discussed the arguments over hypertensive versus ischemic disease in African Americans used. They suggested that while ischemic disease among African Americans may have increased or been misreported, high prevalence of hypertension among African Americans has “always” had consensus.

²⁴³ Jay S. Kaufman and Susan A. Hall, “The Slavery Hypertension Hypothesis: Dissemination and Appeal of a Modern Race Theory,” *Epidemiology* 14 (January 2003): 111-118.

²⁴⁴ J.M. Adams, “Some Racial Differences in Blood Pressure and Morbidity in Groups of White and Colored Workmen,” *American Journal of the Medical Science* 184 (1932): 342. This is cited by Kaufman and Hall as the first racial comparison of blood pressures. In addition to the data on comparative blood pressure, the focus of the article is on comparative incidence of diseases, which were overall the same, and “the recuperative powers of the colored,” which were argued to be less. Whites were argued to be more susceptible to respiratory and gastrointestinal diseases, while colored more susceptible to rheumatic and degenerative diseases. Greater exposure and less prophylaxis was blamed for greater prevalence of malaria and venereal diseases among the colored.

Indeed in the first half of the 20th century elevated blood pressure was rarely considered to be a problem on its own, but rather an epiphenomenon of other problems. As prominent early hypertension researcher Irvine Page noted in his memoir: “As late as 1945, there appeared to be only a handful of true hypertensive individuals, and to most people, “hypertensive” was an adjective meaning “high-strung.”²⁴⁵ Writing of early hypertension researchers, Cushing and Crile, he notes: “I suspect that if they had been asked, ‘What was the problem of hypertension?’ most would have answered ‘What problem?’ The association of hypertension with stroke, heart failure, and renal failure was still nebulous.”²⁴⁶ Luminaries such as Paul Dudley White were skeptics on the danger of hypertension, suggesting in this period that it was either not worth treating for itself or potentially dangerous to treat because lowering it might interfere with a necessary compensatory mechanism.²⁴⁷ This made hypertension very different from the category of coronary disease so central to early cardiology discussed in Chapter 2 (and so connected with whites).

Although academic medicine was not focused on hypertension, it was already emerging as a correlate of risk in the sphere of insurance. Postel-Vinay among others has argued that although “experimental and pathophysiological preoccupations” contributed

²⁴⁵ Irvine H. Page, *Hypertension Research: A Memoir 1920-1960* (New York: Pergamon Press, 1988), vii. The association between hypertensive and high strung has been incompletely replaced with hypertension as a risk factor.

²⁴⁶ Irvine H. Page, *Hypertension Research: A Memoir 1920-1960* (New York: Pergamon Press, 1988), p. 6.

²⁴⁷ Page mentions White by name as reluctant to recognize hypertension as an important aspect of heart disease, and suggests: “According to the gospel of many of our sainted medical ancestors, decreasing arterial pressure would further reduce the blood supply to the tissues of hypertensive individuals. This doctrine was convenient, because no one knew how to decrease pressure effectively for long periods. Even in the early 1930s this fact still held true for most physicians. Hypertension simply had no “class” as a disease. I have already mentioned that many astute physicians still did not regard it as a disease, so why treat it?” Irvine H. Page, *Hypertension Research: A Memoir 1920-1960* (New York: Pergamon Press, 1988), p. 126.

little to the development of hypertension as a risk factor, physicians for life insurance in the first decades of the 20th century were important because of their attention to non-etiological correlative work.²⁴⁸ It was not until after World War II that hypertension emerged as a risk factor that could be understood as physiological and etiological.²⁴⁹

Hypertension as a disease category in the sense used today – a generally asymptomatic pathology of risk for cardiovascular disease – and African American Hypertension as a disease category emerged contemporaneously, in the 1960s.²⁵⁰ The demonstration of hypertension as a risk factor by the Framingham study, and the

²⁴⁸ Postel-Vinay uses the term “risk factor” (a term that would only come much later, after Framingham,) to capture the non-etiological correlative work that was being done: Physicians working for life insurance companies were the first to suspect, and then to demonstrate that hypertension was a major cardiovascular risk factor. This essential contribution was not due to chance. It was the result of almost a century of work calculating the life expectancy of individuals. It is noteworthy that the emergence of the concept of hypertension as a risk factor had little, if anything, to do with experimental and pathophysiological preoccupations. Thus, it was neither the inventors of the instruments nor the clinicians who reaped the first practical rewards of the early work on hypertension, but physicians working for life insurance companies.” “In 1939, the ‘Blood Pressure Study,’ based on statistical data from 15 or so insurance companies (totally over 1,309,000 insurance policies!) confirmed the epidemic proportions of this new ‘disease.’ It is fair to say that physicians working for insurance companies made a much greater contribution than their colleagues to the identification of this major risk factor. It took much longer for clinicians, cardiologists, and general practitioners to recognize the importance of this finding.” Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 31.

²⁴⁹ As Postel-Vinay describes: “While it might be true to say that hypertension is a consequence of modern medicine, it is equally true that it is one of its inventions. Discovered as a result of the commercial pursuits of life insurance companies, defined arbitrarily and situated beyond the usual anatomico-clinical boundaries, hypertension acquired its current status of a major public health problem just after the end of the Second World War.” Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 6-7.

²⁵⁰ Although the emergence of this connection is recent, like other aspects of race it is often presented as trans-historical. For example, disparities-oriented physicians Byrd and Clayton include hypertension in their chapter on “Black Health in the Republican Era, 1713-1812,” writing: “Other clinically obvious conditions that were more prevalent in African American slaves were polydactyly (extra fingers), umbilical hernias, and lactose intolerance (wherein the ingestion of cows’ milk causes gastrointestinal upset, cramping, and diarrhea). Since milk was very seldom included in slave diets, the latter condition probably rarely manifested itself clinically. Hypertension, another disease more commonly afflicting Blacks, is usually clinically asymptomatic until end organ complications such as renal failure, heart failure, or stroke occur. Black slave life spans were very short. Thus, they seldom lived long enough to manifest these clinical symptoms. Other biological, anatomical, and medical ‘differences’ presented by eighteenth- and nineteenth-century physicians and natural scientists have not withstood the rigors of scientific scrutiny, deteriorating into the category of scientific racism in the process.” W. Michael Byrd and Linda A. Clayton, *An American Health Dilemma: Vol. 1: A Medical History of African Americans and the Problem of Race: Beginnings to 1900* (New York: Routledge, 2000), 239.

availability of drugs like thiazides that had a low toxicity profile,²⁵¹ turned hypertension into a focus of medical attention. At the same time, the Civil Rights and Black Power movements made illnesses of blacks a focus for physicians, community organizers, the government, and society as a whole.

Framings of African American hypertension in the period were resonant with the larger civil rights indictments of poor African American health.²⁵² As a foundation of a social movement, hypertension had some appealing qualities that sickle cell—another focus—did not. In particular, hypertension had the possibility of quick and effective intervention. Where sickle cell had captured imaginations of a generation, promises for treatment through molecular medicine were always in the future and ultimately unfulfilled.²⁵³ In contrast, African American hypertension *could* be treated, with mechanisms that were at hand if only they could be made properly available. There was good treatment upon which to improve, a welcome reason for optimism amid the hopelessness that came to characterize sickle cell. Moreover, as risk factors rather than

²⁵¹ See Jeremy Greene, “Releasing the Flood Waters: Diuril and the Reshaping of Hypertension,” *Bulletin of the History of Medicine*, 79 (2005), 749-794.

²⁵² For example, one Washington Post article from 1971 had the headline: “Victims of High Blood Pressure In Inner City Sought by Doctor,” said “no one knows why it occurs four or five times more often in blacks than whites, but hypertension (high blood pressure) is a mass killer in Washington’s inner city... [The expert, Dr. Frank Finnerty from Georgetown] estimates that hypertensive diseases each year kill more than 1,000 Washingtonians and about 85% of them are black. In addition, hypertension often brings about vascular diseases, which he believes are the cause of more than half of all inner-city deaths. ‘Only about 10 per cent of the general population has high blood pressure, but about 45 per cent of the inner city population has it,’ he says. ‘It isn’t known why this is so,’ he adds, ‘but there are three good possibilities.’ High blood pressure may be hereditary, it may be due to ‘socio-economic stress’ and other pressures that are especially severe on persons growing up in the slums and, lastly, it may be due to excessively salty diets common in low income areas.” Harry Aubin, “Victims of High Blood Pressure In Inner City Sought By Doctor,” *Washington Post*, July 19, 1971, p. C1.

²⁵³ See Keith Wailoo, *Dying in the City of the Blues: Sickle Cell Anemia and the Politics of Race and Health* (Chapel Hill: UNC Press, 2001).

Mendellian diseases came to characterize paradigms of cutting edge research,²⁵⁴ African American Hypertension captured the interest of new and renewed civil rights and medical communities. And yet, perhaps because of its status as asymptomatic, African American Hypertension has been more compelling to clinicians than to broader social movements. It has been enrolled in Civil Rights arguments, but demands around the category have not become as associated with the movement as have those around sickle cell.

The Framingham investigators themselves, discussed in the previous chapter, also participated in the simultaneous constructions of hypertension and African American hypertension in the 1960s. Their own study's lack of inclusion of African Americans did not preclude their engagement with larger debates about racial differences in hypertension. Addressing "Environmental Factors in Hypertension" in 1967, the investigators referred without citation to the existence of evidence of higher blood pressures among black populations in the US and West Indies than among whites in those areas or blacks in Africa.²⁵⁵ Their lack of citation suggests that they were reporting the consensus of the field. When they lamented that "admixture of the Negro races in the Western Hemisphere makes the interpretation of this data difficult," they testify to a durable notion of black heart disease that is both new and re-newed.²⁵⁶ That is, they speak of black heart disease in terms of the new risk factor of hypertension, but in a way

²⁵⁴ As Nicholas Postel-Vinay points out: "When physicians working for insurance companies began to look for asymptomatic disorders, then called 'obscure diseases,' they were making an important step because they were, unknowingly, inventing a new approach to medicine." Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996).

²⁵⁵ Dawber et al, "Environmental Factors in Hypertension," in *The Epidemiology of Hypertension. Proceedings of an International Symposium sponsored by the Chicago Heart Association and the American Heart Association, Chicago, IL, February 3-7, 1964*, edited by J Stamler, R Stamler and TN Pullman (New York: Grune & Stratton, 1967), 256.

²⁵⁶ Ann Laura Stoler points out that in each historical moment, race is always both "new and re-newed." "Racial Histories and Their Regimes of Truth," *Political Power and Social Theory* 11 (1997): 183-206.

that continues the older preoccupation with black heart disease as distinct from white heart disease. Black heart disease was becoming characterized as hypertensive rather than coronary in a way analogous to previous notions of black heart disease as infectious rather than coronary. There is something peculiar about the Framingham investigators' suggestion that admixture presents problems for research into environmental causes of disease—wouldn't assimilation be what would pose a problem of distinction? How might we theorize questions of cultural, rather than biological, admixture?²⁵⁷ But that peculiarity should alert us to hypertension's racialized existence at the nexus of the biological and the environmental.

But while the genetics of African American hypertension remained indeterminate, the social justice elements of the intersection of black consciousness and the disease category were important and salient. In the 1970s, black physicians among others were at the forefront of trying to focus attention on the patient populations they served. When black physicians wrote medical textbooks, they posited the patients they served as both subjects of “black-related diseases” and as representatives of humanity, of “the typical.” African American hypertension was portrayed as both a physical fact and a result of social structures of inequality. For example, from Richard Allen Williams' *Textbook of Black-Related Diseases*:

A typical patient is a middle-aged Black male with a history of hypertension for several years. His high blood pressure is in the moderate range, i.e. 170/110, a level which has persisted although he has been on various forms of treatment over

²⁵⁷ Michael Montoya is theorizing this question in interesting ways, using the concept of “bioethnic conscription” to mark how the “social identities and life conditions of DNA donors are grafted onto biological explanations of disease causality.” Montoya points out that “Race and ethnicity are ideological in Althusser's sense of the term, operating as a system of ideas and representations that – as shown here – work within and are sustained by both scientific and sociopolitical registers” (p. 101). .Michael Montoya, “Bio-Ethnic Conscription: Genes, Race, and Mexicano/a Ethnicity in Diabetes Research,” *Cultural Anthropology* 22: 94-128.

the years. He confesses to not taking his medication faithfully because of undesirable side effects. He is “under care” at the local hospital, where he is seen every 6 months and where an intern or resident checks his blood pressure, notes that there is no change, and indifferently reorders the same medication which the patient has been taking, in the same dosages. The doctor does not bother to question the patient about new symptoms, but if he had done so, the patient might have revealed the recent onset of dyspnea, and the need to sleep on two pillows. Had the physician taken the time to do a physical examination, he may have found engorged, distended neck veins and a very forceful cardiac apex impulse. A grade 3/6 apical pansystolic murmur radiating to the left axilla would be heard on auscultation, as well as an S₂ gallop at the lower left sternal border. An electrocardiogram might reveal the previously existing changes of left ventricular hypertrophy, but some new features might also be seen: increased ST-segment depression and T-wave inversion, and frequent premature ventricular contractions. A chest x-ray would show some slight increase in the size of the cardiac silhouette since the previous study, but comparison with other earlier x-rays would demonstrate a gradual increase in the cardiothoracic ration. In addition, the new appearance of Kerley B lines in the lungfields might be noted.

If the physician is concerned and is astute enough to look for them, the changes presented in this oversimplified example should alert him to the fact that the patient has developed congestive heart failure secondary to his hypertension. Immediate action at that point such as the institution of therapy with diuretics and cardiac glycosides may forestall the rapid progression of the heart disease. However, as is frequently the case, the doctor does not investigate the patient beyond the point of recording his blood pressure, and thus the opportunity to avert further rapid advances of this morbid event is missed.²⁵⁸

Williams’ representation mixes indictment of poor medical treatment of African Americans with technical medical language. The passage both laments the near-inevitability of poor treatment and documents the situation to advocate for change. The way he describes this patient is not as an ideal case but as a way of narrating the intersection of individual biology and long-standing poor medical care. In the process, he invokes a very different notion of “typical” than the one we saw in the previous chapter. It is like the non-technical, mocking remark: “that’s typical.”

²⁵⁸ Richard Allen Williams, “Cardiology,” in Richard Allen Williams, ed., *Textbook of Black-Related Diseases* (New York: McGraw Hill, 1975), 363.

African-American physicians such as Williams who integrated their health research with their civil rights advocacy often claimed (and for that matter continue to claim) more than one genealogy and source of authority. On the one hand they claim mainstream medical expertise, and on the other, connection to the history and community of African Americans. Particularly in the 1970s, they sought Black roots for medicine. For example, Richard Allen Williams often refers to Imhotep, the Egyptian god of medicine, first physician known by name, as an ancestor in a genealogy distinct from the Greeks, as “someone who would be recognized as Black if he walked down the street in Oakland today.”²⁵⁹ He puts a photo of a statue of him in the opening of his *Textbook of Black Related Diseases*, with the caption:

Imhotep, the son of Ptah, was born in Egypt about 3,000 B.C. during the Third Dynast. During his life he was renowned as a philosopher, sage, scribe, poet, astronomer, chief lector priest, magician and architect (he designed and constructed the Step Pyramid at Sakkara, the world’s first large man-made stone structure). However, he was most famed for his skill as a physician. Imhotep was probably responsible for the production of the monumental Ebers payrus, in which the treatment of over 1,000 diseases was detailed. Imhotep and other Egyptian physicians knew of the heart as the source of blood supply thousands of years before William Harvey rediscovered this principle. They practiced surgery, knew auscultation, understood the relationship between the pulse and the heart beat, and extracted medicine from plants. Imhotep’s medical excellence led to his deification about 2850 BC. Thus, the great African physician became acknowledged as the god of medicine almost 2,500 years before Aesculapius laid claim to the same title in Greece.²⁶⁰

In the invocation of pan-African sensibility, Williams is connected to much broader cultural movements in the period. My dissertation focuses primarily on the medical professionals engaged in the construction of African American hypertension as a disease

²⁵⁹ Helen Hammer, Richard Allen Williams, and Deborah Gould.; Kaiser Audio Visual Center (Calif.); Permanente Medical Group (Oakland, Calif., Staff Education, *African American Health Issues* [videorecording] / a co-production of the Regional Audio Visual Center and Physician Education & Development. Oakland, CA : Kaiser Foundation Health Plan, c1995.

²⁶⁰ Richard Allen Williams, ed., *Textbook of Black-Related Diseases* (New York: McGraw Hill, 1975).

category, though it is worth noting that the range of thinkers in racial theory who have weighed in on black hypertension has been vast. For example, afrocentrist psychiatrist Frances Cress Welsing considers high blood pressure as part of her overall theory of melanin and white supremacy.²⁶¹ She first published on the connection between melanin and hypertension in 1975. She attributes higher rates of hypertension in blacks (as well as, to a lesser degree, Mexicans) than in whites to caused by the receptivity of skin melanocytes in non-white peoples, which gives them a “highly sensitive and refined nervous system apparatus” but systemically vulnerable to the negative energy of living in a white supremacist society.²⁶² It is amid these and other black power movements that the physicians’ organizing is taking place.

Williams, joined by many of the other physicians described here, would found the Association of Black Cardiologists (ABC) in 1974. This instance of professional black organizing around hypertension and related diseases was happening in a context of black health and black power²⁶³: the Black Panther Party included high blood pressure screening, along with sickle cell and STD screening, in their People’s Medical Centers.²⁶⁴ ABC was simultaneously a professional organization and one interested in community

²⁶¹ See Frances Cress Welsing, *The Isis Papers: The Keys to the Colors* (Chicago: Third World Press, 1990).

²⁶² Frances Cress Welsing, “Blacks, hypertension, and the active skin melanocyte,” *Urban Health* 4 (1975): 64-72.

²⁶³ Close links between the NAACP and NMA suggest that their current campaigns and framings may also come from this period. As is characteristic of the pro-civil rights invocations of black hypertension as a disease category, there is both an statement of the severity of the problem and a reassurance of the ease of solution. For example, from the NAACP Detroit Chapter’s website: “Anyone can develop high blood pressure, also called hypertension. African Americans are at higher risk for this serious disease than any other race or ethnic group. High blood pressure tends to be more common, happens at an earlier age, and is more severe for many African Americans. The good news is that high blood pressure can be controlled and better yet, it can be prevented!” <http://www.detroitnaacp.org/health/high.asp>.

²⁶⁴ Mumia Abu-Jamal, *We Want Freedom: A Life in the Black Panther Party* (Boston: South End Press, 2004). For a thorough analysis of the role of black health organizing in the black power movement, see Alondra Nelson, *Body and Soul: The Black Panther Party and the Politics of Race and Health*, Forthcoming.

organizing: in the late 1970s it was through ABC that Elijah Saunders organized churches as high blood pressure control centers.

Larger public health initiatives in the 1970s were both informed by the physicians' focus on black hypertension as a health civil rights issue and helped to feed it. For example, from the inception of the National High Blood Pressure Education Program in 1972 as "a cooperative effort among professional and voluntary health agencies, state health departments, and community groups,"²⁶⁵ the organization had a special focus on trying to get its message to a "general public" imagined as racialized. For example, from a 1973 pamphlet:

The Task Force has adopted the premise that the "general public" is not an adequate description of the audience for a health message. Because this Task Force considers high blood pressure to be a public health problem as well as an individual medical problem, it has elected to develop its planning on the basis of the questions, "What groups within the population have the greatest numbers of persons at risk?"²⁶⁶

The task force answers the question about who is at elevated risk as those in three categories: patients known to have hypertension, those from minority groups, and those from medically defined high-risk groups (who are pregnant, have diabetes or gout, have high cholesterol, family history and the like). It's articulation of the role of minority status has hedging that often characterizing such articulations:

A second large category of potential patients may be identified from certain minority groups who (not because of the minority per se) have a much higher

²⁶⁵ NHLBI, "National High Blood Pressure Education Program: Program Description," http://www.nhlbi.nih.gov/about/nhbpep/nhbp_pd.htm

²⁶⁶ US Department of Health, Education, and Welfare, Public Health Service, *National High Blood Pressure Education Program : Report to the Hypertension Information and Education Advisory Committee : Task Force III Community Education*, September 1, 1973, National Library of Medicine, Bethesda, MD, p. 22.

prevalence rate of hypertension. These potential patients include blacks, Spanish-speaking groups, and American Indians.²⁶⁷

There is a tension in this pamphlet between seeing blacks as part of the general population or in opposition to it: “The problem of hypertension among the American Indian population is greater than among the general (Caucasian) population. Indeed, the prevalence of high blood pressure falls intermediate between black and white populations.”²⁶⁸ In this quote, the general population is first specified as “Caucasian” (rather than inclusive of all races). Yet just thereafter, the durability of the notion of black/white difference is illustrated when whiteness is separated out as a contrasting pole for blackness in a continuum that includes all races. Incidence of hypertension is mapped out as directly proportional to distance from whiteness.

Polls suggest that physicians got the message that blackness was associated with high risk of hypertension. According to the US Department of Health, Education and Welfare’s 1979 survey, a strong majority of physicians thought that “being black” “was associated with the likelihood of a patient having hypertension.” Of all the possible factors listed, only family history of hypertension received such a strong majority believing that it was related to a “definite increase in likelihood of hypertension” (Family History: 69%, Being black: 61%, Obesity: 54%, High salt diet: 50%, Diabetes: 39%, Increasing Age: 33%, Environmental stress: 32%, Nervous personality: 25%, High cholesterol diet: 15%, Lack of exercise: 9%, Low income: 6%). Moreover, younger

²⁶⁷ US Department of Health, Education, and Welfare, Public Health Service, *National High Blood Pressure Education Program : Report to the Hypertension Information and Education Advisory Committee : Task Force III Community Education*, September 1, 1973, National Library of Medicine, Bethesda, MD, p. 22.

²⁶⁸ US Department of Health, Education, and Welfare, Public Health Service, *National High Blood Pressure Education Program : Report to the Hypertension Information and Education Advisory Committee : Task Force III Community Education*, September 1, 1973, National Library of Medicine, Bethesda, MD, p. 24.

physicians were more convinced of most of the proposed hypertension risk factors, including African American as risk. Among physicians under 35, belief that “Being Black” “definitely increase the likelihood of hypertension” was 73%. (For this group, the full statistics are: Family history: 74%, Being black 73%, Obesity: 58%, High salt diet: 46%, Diabetes: 44%, Increasing age: 51%, Environmental stress: 25%, Nervous personality: 15%, High cholesterol diet: 10%, Lack of exercise: 6%, Low income: 5%).²⁶⁹ These numbers suggest that the new generation of physicians was more credulous of this racial disease relationship than the generation of physicians that they were replacing. These young physicians were using an epistemology that emerged in Framingham by collapsing two kinds of risk factors –correlative and etiological – to solidify the racialization of this disease category.

As the 1970s drew to a close, professionalization around the categories of both hypertension and African American hypertension increased. The first issue of the American Heart Association’s journal of its newly incorporated Council for High Blood Pressure Research came out in 1979, and included an article that would come to be widely cited on “Pathophysiology of Hypertension in Blacks and Whites: A Review and Basis of Blood Pressure Differences.”²⁷⁰ It was written by Richard F. Gillum, who had

²⁶⁹ US Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. (NIH) 79-1056, *Diagnosis and Management of Hypertension: A Nationwide Survey of Physicians’ Knowledge, Attitudes, and Reported Behavior: Conducted for the Food and Drug Administration and the National High Blood Pressure Education Program, National Heart, Lung, and Blood Institute, 1979*, pp 10-11. National Library of Medicine, Bethesda, MD.

²⁷⁰ Richard F. Gillum, “Pathophysiology of Hypertension in Blacks and Whites: A Review and Basis of Blood Pressure Differences,” *Hypertension* 1 (1979): 468-475. The summary demonstrates simultaneous assertion of consensus around the existence of black hypertension and the agnosticism about etiology: “Differences in blood pressure between blacks and whites in the United States are now well documented. The causes of these differences remain speculative. Genetic factors, personal characteristics, renal physiology, endocrine factors, autonomic nervous system function, cardiac function and various environmental factors are examined in the present review as potential determinants of racial blood pressure differences. Racial differences in renal physiology and environmental influences such as socioeconomic

been one of the founding members of the Association of Black Cardiologists. Two separate professional organizations were soon to come: American Society for Hypertension (ASH) in 1985 and the International Society for Hypertension in Blacks (ISHIB) in 1986. The organizations were founded by overlapping groups of people with overlapping goals.

Technically, the founding of ASH preceded that of ISHIB by a year. However, the two organizations' founding can actually be understood as the same moment, as has been described by Elijah Saunders, who was a founder of both.²⁷¹ He had discussed the potential of a special focus on blacks in ASH with his colleague who was then the president of that organization. The (white) then-president of ASH responded that he didn't think that the issue could be adequately addressed by ASH, and that a separate organization might better address the special needs of that community. Saunders, together with two white physicians whose practices focused on hypertension in blacks, had a meeting to write a book on the topic,²⁷² and out of that book project the organization emerged.

ISHIB was not only distinct from ASH, but also had two characteristics that made it distinct from two other organizations with overlapping membership: focus on patient population and international sensibility. Unlike the National Medical Association and the Association of Black Cardiologists (which started holding scientific sessions at the same

status seem to be likely candidates for important contributions to blood pressure differences. Further research aimed specifically at black-white blood pressure differences is of great importance to a fundamental understanding of the etiology and prevention of essential hypertension."

²⁷¹ Interview with Elijah Saunders, July 18, 2005; "A 20 Year ISHIB Perspective," Part of the Session On "The Evolution of Hypertension and Cardiovascular Risk: A 20-Year Perspective," International Society for Hypertension in Blacks Annual Conference. Atlanta, GA, June 23, 2006.

²⁷² W. Dallas Hall, Elijah Saunders, and Neil Shulman, *Hypertension in Blacks: Epidemiology, Pathophysiology, and Treatment* (Chicago: Year Book Medical Publishers, 1985).

time, in 1987), the defining factor in membership was not the identity of the practitioner but the identity of the patient population that the practitioner served. And unlike those organizations, it had an explicitly international focus, interested from the beginning in connecting these practitioners with those of Africa and its diaspora. After its first few meetings in Atlanta, the group started to reach out geographically and had its meetings outside North America every other year after from 1989 in Nairobi, Kenya, until 2003 (when concerns about security led the planned Ghana meeting to be canceled). The symposium that I am using to frame this chapter was at the ASH meeting, but was in fact co-organized and co-hosted by ASH and ISHIB together.

Interest in African American hypertension both as a disease category and as a site for critique of racialized scientific medicine continued in the 1990s. The ASH Symposium emerges in that context. This moment both connects with and exceeds general interest in race and biology. The three factors colluding in the increasing focus on racial difference at the level of biology were outlined by Lee, Mountain, and Koenig²⁷³: the US government's health disparities initiative; the success of the Human Genome Project; and the increasing body of genetic research focused on variation among populations. These trends are all part of a spreading interest in African American hypertension. Much of the recent data about disparities in hypertension comes as a result of the 1993 change in NIH policy requiring inclusion of women and minorities in studies and recording of data along those lines.²⁷⁴

²⁷³ S. S-J. Lee, J. Mountain, and B. Koenig, "The Meanings of Race in the New Genomics: Implications for Health Disparities Research," *Yale Journal of Health, Policy, Law, and Ethics* 1 (Spring 2001): 36.

²⁷⁴ See Steven Epstein, "Bodily Differences and Collective Identities: The Politics of Gender and Race in Biomedical Research in the United States," *Body and Society* 10 (2004): 183-203.

But interest in African American Hypertension as a disease category comes from another three-part confluence that exceeds interest in genetics per se. From a pharmaceutical company's perspective, its status as a chronic condition might have particular draw because of its potential market of lifetime pharmaceutical consumers.²⁷⁵ From a clinical researcher's perspective, its impact on the morbidity and mortality of populations served might contribute to making it a focal point. From a community organizer's perspective, the potential for interventions through health care reform and community education might make it an appealing campaign focus.

African American Hypertension continues to receive attention from diverse constituencies, including mainstream medical communities, African-American medical communities, mainstream media, and African-American health activists. As I will now explore, explanations rooted in notions of genetic specificity have coexisted with explanations based on the social experiences of African-Americans living in a racist society. What does it mean to select as genetically heterogeneous a group as African Americans to understand the genetics of hypertension? What does it mean to put African-American status into "risk factor" discourse? Continuing to reflect on the ASH symposium, in the next section, I will explore further the tensions that exist between social and biological explanations of racial disparities, and how those have become productive in creating a durable notion of "African American Hypertension."

²⁷⁵ Many have written of this shift of interest toward lifelong medication, and among the most interesting is Joseph Dumit, *A Pharmaceutical Grammar: Drugs for Life and Direct-to-Consumer Advertising in an Era of Surplus Health*, Forthcoming, Duke University Press.

The durability of African American hypertension in debates over the slavery hypothesis

Although no advocates of it spoke up at the ASH Symposium, Richard Cooper mentioned the Slavery Hypothesis in order to argue against it. The Slavery Hypothesis is an etiological hypothesis that purports to explain why African Americans suffer more from hypertension. It has garnered considerable historical critique, and will be important in the next chapter of this dissertation as I discuss racialized invocations of thiazide. Here, I will attend to the theory and especially the critique of it to highlight the points of consensus that underlie the arguments of both the proponents and the opponents of the theory.

A representative example of the narration of the debate can be found in Parisian physician and medical historian Nicolas Postel-Vinay's *Century of Arterial Hypertension*, in a section called "Hypertension in Blacks: A Questionable Entity."²⁷⁶ Although his heading promises to throw the existence of African American hypertension into question, the segment that follows does not in fact question whether hypertension in blacks exists as an entity. Rather, it frames the debate as whether its cause is social or genetic, and to conclude that: "hypertension in black subjects is the best example of a population in which prevalence of a disease is low in the country of origin, high in the country of

²⁷⁶ Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 148-149.

adoption, and for which it is impossible, despite a considerable mass of work, to separate out genetic and environmental factors.”²⁷⁷

Postel-Vinay posits “the black population of the United States” as both a “an example of a transplanted population group whose pressure is higher than that of subjects remaining in their original homeland (sub-Saharan Africa) and that of their adopted homeland” and one that suffers from “psychosocial stress” from “the plight of an underprivileged minority.”²⁷⁸ Here is the way he describes the slavery hypothesis, connecting it to questions of sodium regulation:

Black Americans, originally from Africa, were transferred to the American continent during the slave trade. They traveled under appalling conditions of hygiene where malnutrition, diarrhea and vomiting took an extremely heavy toll. Blackburn and Prineas suggest the hypothesis that subjects who best retained sodium, for genetic reasons, were probably the ones who best survived this terrible ordeal. According to the hypothesis, this resulted in a sort of genetic selection whereby the individuals who arrived in the New World were those who best retained sodium. If this is true, it would be logical to observe a high incidence of hypertension in their descendants.²⁷⁹

Postel-Vinay does not portray the hypothesis as in any way definitive, noting immediately that the hypothesis has been “widely challenged.” He characterizes the opposing evidence as low rates of hypertension in much of Africa and in sickle cell, that “there is no statistical correlation between blood pressure level and skin colour,” and that environmental causes can explain differences. He suggests that the controversy is interesting because “[n]ot only does it raise the problem of innate and acquired

²⁷⁷ Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 149.

²⁷⁸ Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 148. Interestingly, he not only describes the slavery hypothesis but also the theory that preceded it that it purported to replace: that higher sodium retention and lower renin of presumably genetic origin would have been useful in tropical climates but not in temperate ones.

²⁷⁹ Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 148.

hypertension, but it also highlights methodological problems of epidemiology.”²⁸⁰ Yet Postel-Vinay settles into a consensus that poses etiological but not existential questions about this allegedly “questionable entity:”

The special features of this form of hypertension (frequency of complications, low renin and sensitivity to diuretics) are well known to clinicians and do indeed to support the idea of a homogenous group. Specific responses to various classes of antihypertensive drugs have been demonstrated in a number of studies.²⁸¹

All the caveats do not actually throw the existence of the entity itself into question, merely its etiology. I argue that the debate over whether the cause is genetic or social serves to solidify medical consensus on the existence of African American Hypertension. Both “black hypertension is genetic” and “black hypertension is social” affirm the notion that “black hypertension is.”

Competing origin stories of the slavery hypothesis

The Slavery Hypothesis is simultaneously marginal and mainstream. On the one hand, little research has been done on it by very few people. On the other, it is widely known and referred to in venues like medical conferences and textbooks, albeit with caveats of its lack of proof. It is appealing for history of science critique because it is a dramatic narrative combined with odd Darwinian claims brought down to a small time scale (in evolutionary terms). Discussion of it takes place in the major journals in the field, but without earning anything close to consensus acceptance.

²⁸⁰ Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 148.

²⁸¹ Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 148-9.

The hypothesis, as made in its fullest form by TW Wilson and C Grim in *Hypertension* in 1991,²⁸² is that the high mortality in slavery due to salt-wasting diseases led to selection pressure for those genetically pre-disposed to preserve salt. This theory argues that these selection pressures in Atlantic slavery predispose African Americans to salt retention, leading to hypertension and thus to cardiovascular disease.²⁸³ The suggestion is that those Africans who were genetically predisposed to conserve salt were the ones to better survive the diarrheal diseases of the journey to slavery, and so as a result their descendents are salt-conserving. According to this argument, while a tendency to conserve salt might have been protective against old hazards like cholera, it is dangerous today because it leads to hypertension and cardiovascular disease. And so, according to the theory, this difference in salt sensitivity explains the difference in cardiovascular disease and the excessive morbidity and mortality of African Americans today. This hypothesis has been critiqued vociferously, for many reasons.²⁸⁴

Both proponents and opponents of the thesis reify African American Hypertension, and it is more productive to look at the ways that race becomes a dense

²⁸² TW Wilson and CE Grim, "Biohistory of Slavery and Blood Pressure Differences in Blacks Today: A Hypothesis," *Hypertension* 17 (1991): Suppl I122-8.

²⁸³ The argument has been well-described and critiqued by epidemiologist Jay S. Kaufman, both in "The Anatomy of a Medical Myth," *Social Sciences Research Council Forum Is Race Real?*, <http://raceandgenomics.ssrc.org/Kaufman/>, accessed September 1, 2005, and in Jay S. Kaufman and Susan A. Hall, "The Slavery Hypertension Hypothesis: Dissemination and Appeal of a Modern Race Theory," *Epidemiology* 14 (January 2003): 111-118. He argues that the theory has been widely accepted by medical professionals and the media despite the lack of any evidence for it because of the normalcy of racial genetic essentialism in medicine. He points out how the theory fails on the bases of "population genetics, details of the physiology of hypertension, and basic evolutionary biology." He considers Philip Curtin's historical arguments to be the most scathing against it. There have also been debates on the theory in venues such as *Transforming Anthropology: Journal of the Association of Black Anthropologists*, between Fatimah Jackson (2005) who argues that the problem with the theory is dilution and George J. Armelagos (2005, 2006) who argues that the genetic bottleneck never existed to be diluted.

²⁸⁴ See, for example, one exchange: Joel E. Dimsdale, "Stalked by the Past: The Influence of Ethnicity on Health," *Psychosomatic Medicine* 62 (2000): 161-170, Jay S. Kaufman, "No More "Slavery Hypothesis Yarns"," *Psychosomatic Medicine* 63 (2001): 324.

transfer point of discourses between the social and the biological than to argue about the racism of the theory's proponents. Here, I will present two competing origin stories of the hypothesis, one from its principal advocate and one from its principal critic. Both the theory and the criticism of it have considerable consensus with regard to the realness of African American hypertension and urgency of response. Debate over the role of genetics in the theory does not undermine African American Hypertension as a disease category because its durability does not come from genetics alone.

To call the debaters "interlocutors" would be to exaggerate their direct engagement. Both sides complain that the other refuses to respond seriously to their arguments. Here, I will bring them into dialogue. The principal critique I will address is one by University of North Carolina epidemiologist Jay Kaufman, and his student Susan A. Hall, published in *Epidemiology* in 2003. (Kaufman was a student of Grim's longtime nemesis Loyola University epidemiologist Richard Cooper, whose critique of the genetic basis of racial disparity was part of the ASH symposium that opened this chapter.)

Second, I will discuss an alternative origin story of the hypothesis by the theory's main proponent, Clarence Grim, as he told it to me in 2005 at the annual meeting of the International Society for Hypertension in Blacks that year. Grim is a professor of medicine at the Medical College of Wisconsin and director the High Blood Pressure Diagnosis and Treatment Center at St. Michael's Hospital in Milwaukee. I suggest that the disconnect in the debate comes from the would-be interlocutors differing perspectives on the relationships between (1) science and practice, and (2) biology and determinism. Yet I will also attend to the consensus that underlies the debate.

The most extensive review and critique of the slavery hypothesis was written by Kaufman and Hall and appeared in *Epidemiology* in 2003.²⁸⁵ Here is the origin story of the hypothesis that they provide. Kaufman and Hall suggest that the hypothesis emerged in dialectical response to earlier medical literature that posited that high rates of African American hypertension were the result of high rates of hypertension in Africans. However, Africans were shown to in fact have low rates of hypertension. And so, according to Kaufman and Hall, the slavery hypothesis was a way for physicians who were committed to genetic explanations for racial disparities to continue to understand African American hypertension as genetic, and yet account for genetic difference between Africans and African Americans. The language that Kaufman and Hall use is highly deterministic. They describe the researchers as “locked in a paradigm that favored genetic explanations for the black-white disparity.”²⁸⁶

Here is the origin story of the hypothesis provided by Grim.²⁸⁷ He described his interest as coming from two places simultaneously: clinical experience, and the science of salt in physiology and history.

On the one hand, Grim has had formative and longstanding clinical experiences with successfully treating severely hypertensive black patients. This included a case of an extreme form of treatable hypertension (now called Conn Syndrome) he saw in a black woman when he was a medical student in Missouri in the early 1960s, many black patients with renal failure he saw during his residency at Duke, work on the Evan’s

²⁸⁵ Jay S. Kaufman and Susan A. Hall, “The Slavery Hypertension Hypothesis: Dissemination and Appeal of a Modern Race Theory,” *Epidemiology* 14 (January 2003): 111-118.

²⁸⁶ Jay S. Kaufman and Susan A. Hall, “The Slavery Hypertension Hypothesis: Dissemination and Appeal of a Modern Race Theory,” *Epidemiology* 14 (January 2003): 112.

²⁸⁷ Interview with Clarence Grim, July 15, 2005.

County Study in Georgia,²⁸⁸ a fellowship at the Martin Luther King Hospital in Los Angeles in the 1980s—where he started reading black history at the urging of fellow clinicians there—and his longstanding practice in Milwaukee.

On the other hand, Grim became convinced of the importance of salt metabolism in his training by two key advisors: one an interventionist doctor (Kempner) and one a basic scientist (Denton). Dr. Kempner at Duke, where Grim did his residency, became famous for his “rice diet” that was designed to treat kidney disease but which also dramatically decreased blood pressure – in the rich white patients who could afford to stay for in his care for six months. In the early 80s, Grim spent a year with Derek Denton, an Australian basic scientist who in a popular book called *The Hunger for Salt* explained more or less the whole of human evolution and diversity through appetite for and endocrine response to salt.²⁸⁹

Grim described these two aspects of his training and experience as starting to come together when he read descriptions of the stench of slave ships in James Michener’s book on Hawaii. The more he read about the nature of black mortality in the Atlantic slave trade, the more he became convinced of the connections between salt, black history, and the hypertension he treated in his patients. Grim attributes his skepticism of the alternative explanation for black hypertension—psychosocial stress of living in a racist society—to the fact that blacks in the Caribbean have high rates of hypertension even though they don’t deal with racism on a day to day basis. Grim characterizes his

²⁸⁸ This study is a touchstone for race and cardiovascular disease, and purported to show that blacks have more hypertension while whites have more ischemia.

²⁸⁹ For simplicity, this chronology skips a few steps – including time with Dr. Helmer in Indiana, who found that “blacks are more salt-sensitive than whites, reasons unknown. Using the Helmer sort of hypothesis that since they’d evolved in an environment where it was necessary to be good salt conservers,” and Dighton in Mississippi who emphasized the role of salt in kidney function and hypertension.

argument as a minority one, one that has always led people to attack him for being racist, but that he perseveres with because of his dedication to the validity of the theory. He has done research in the basic science: twin studies in Barbados to show that salt sensitivity is genetic in whites as well as blacks, for example, and there are many more he'd like to do, as well. He described with disdain the previous hypothesis for black hypertension—that African Americans hadn't evolved enough for the modern world—and sees his theory as a nonracist alternative that fits well with clinical practice.

What is at stake in these differing origin stories? For Kaufman and Hall, what is important about the slavery hypothesis is that clinicians refer to it at all in spite of its poor evidentiary basis. They are particularly critical of its proponents' scant explication in peer-reviewed journals and their failure to respond to critiques of the theory. For Kaufman and Hall, the fact that the theory nevertheless has widespread knowledge in the community of hypertension researchers demonstrates the continuation of a pernicious racialization that has long characterized unequal medicine. What they are not attentive to, however, is where these conversations are taking place, and what precisely underlies them. In most of the citations of the theory, including the section from the history discussed above, reference to the theory is markedly agnostic. The textbooks are not "locked" into genetic ideas alone, but rather intrigued by genetics as they tell a story of race that draws on a messy complex of nature and nurture.

For Grim, what is important in this hypothesis is that it brings together biology and history with practical medical interventions. Throughout his scientific research, Grim has used the model that "all humans are salt-sensitive, the question is the level," and that responsiveness to diuretics is a measure of salt-sensitivity. All the while, he has

been a practitioner advocating careful measurement of blood pressure, and salt restriction and diuretics as preferred treatments for everyone with hypertension, especially for blacks. I asked Grim whether it was scientific interest or clinical interest that primarily drove him, and he said “It’s all together. The ultimate experimental animal is humans. I don’t care about those rats. I don’t treat any rats. So everything that drives my interest [is] in trying to control high blood pressure and find out the causes of high blood pressure.”²⁹⁰

If I sound like something of an apologist for Grim, this is not to discount the speciousness of his theory. Rather, I want to highlight that Kaufman and Grim *both* participate in racial discourses, and it is more productive to track those than to hunt for racists. I am guided here by Ann Laura Stoler, who has pointed out: “a common historiographic assumption is that racial discourse is a discourse of those with power (or those trying to maintain their hold) rather than a ‘dense transfer point’ of it.”²⁹¹ By assuming that the fundamental thing done by Grim’s theory is to prop up racism in natural science, Kaufman and Hall miss the clinical and other arenas at stake. For Kaufman and Hall, science precedes practice, but attending to clinical practice helps us to see that – whatever his other failings – Grim is right that, in medicine, science and practice are immanent.

When Kaufman and Hall describe the implications of the theory, they write as if Grim and others sympathetic to the theory were merely basic scientists observing natural states, not also clinicians intervening in them.

²⁹⁰ Interview with Clarence Grim, July 15, 2005.

²⁹¹ Ann Laura Stoler, “Racial Histories and Their Regimes of Truth,” *Political Power and Social Theory* 11 (1997): 192.

Finally, the seemingly irresistible siren song of the Slavery Hypothesis may derive in part from its implications for scientific conceptions of race and identity. Once Grim had devised the evolutionary hypothesis of rapid genetic selection during the period of slavery, it provided a basis for exceptionalism in discussion of African American hypertension, a basis for treating blacks as a group that had been uniquely and intractably transformed, genetically mutilated. The essential “defect” or “abnormality” in this group therefore achieved not only rational basis, but became something innately pathologic, thereby reinforcing blacks’ essential physical inferiority in the modern world. Given the common African origins of all humanity, therefore, the Slavery Hypothesis may serve not merely to provide an explanation for the biological distinction between racial/ethnic groups, but to judge that distinction as a deformity rather than a mere divergence.²⁹²

In this critique, there is a failure to engage with Grim as a subject rather than a caricature.

For Grim, who was trained by a physician renowned for the belief in radical dietary change, and who is a proponent of low-salt diets and aggressive diuretic treatment for hypertension, difference in blood pressure is far from “intractable.” Indeed, as I will discuss in the next chapter on thiazide, responsiveness to salt restriction and treatment through diuretics is part of how black hypertension is characterized. The language of “inferiority” that Kaufman and Hall use would be abhorrent to Grim himself, and for him the salt aspect of both the cause and intervention for hypertension is as important as the genetics aspect. Moreover, Kaufman and Hall themselves note that medical beliefs in African American inferiority, including ones that used hypertension as evidence, preceded Grim by a good deal, and those beliefs do not rise or fall on the truth value of the theory. This is a common error Stoler documents in antiracist historiography: the fantasy “that if we can disprove the credibility of race as a scientific concept, we can

²⁹² Jay S. Kaufman and Susan A. Hall, “The Slavery Hypertension Hypothesis: Dissemination and Appeal of a Modern Race Theory,” *Epidemiology* 14 (January 2003): 116.

dismantle the power of racism itself—that racisms rise and fall on the scientific credibility of the concept of race.”²⁹³

In their critique of the slavery hypothesis, Kaufman and Hall acknowledge the identity practices around seeing black hypertension in these terms, but counter by suggesting the danger of discrimination that would result from calling black hypertension genetic with references to the problems of discrimination based on sickle cell:

As in the case of sickle cell anemia, the presence of a biological trait that is understood to derive directly from Africa or from the initial passage from Africa may [...] become an authenticating characteristic. The myth of genetic determinism cuts both ways, however, for although it absolves the individual of responsibility, it also absolves society at large.

They cite Dorothy Nelkin to suggest:

Deterministic biological explanations (‘it’s in my genes’) – much like theological explanations (‘the devil made me do it’) – locate problems, (and, therefore solutions,) within individuals.²⁹⁴

However, in its context of cheap and effective treatment, “black hypertension” is radically different from “black sickle cell,” much less demonic possession. Kaufman and Hall’s suggestion that genetic determinism absolves both the individual and society of responsibility is in brazen disregard for how Grim actually uses the theory in practice (like, as we will see in the next chapter, other sympathizers). On the contrary, when the theory is combined with interventionist medicine, it becomes a way to locate blame in the social processes of a horrific history *without* abdicating responsibility for changing an unacceptable present. Grim conceptualizes change as possible through clinical care and social distribution of drugs. When disease definition and therapeutic promises are

²⁹³ Ann Laura Stoler, “Racial Histories and Their Regimes of Truth,” *Political Power and Social Theory*, 11 (1997): 196-197.

²⁹⁴ Jay S. Kaufman and Susan A. Hall, “The Slavery Hypertension Hypothesis: Dissemination and Appeal of a Modern Race Theory,” *Epidemiology* 14 (January 2003): 116.

immanent, the stakes of biological arguments change, and critiquing genetic determinism is not up to the task.

Kaufman's critique of the genetic framing of African American hypertension extends that of his advisor, Richard Cooper, who argued at the symposium for social and environmental causes of disease disparities, as he has for decades. However, in his critique, like Kaufman and Hall's, there is a remarkable failure to engage with what is at stake for his opponents in the debate. For example, writing in 1986 in the *Journal of Health Politics, Policy and Law*, Cooper and his colleague Richard David suggest that the danger of assuming that genetic factors are more important than social ones, and that a sophisticated analysis of class is unnecessary, is that important public health interventions are ignored and the black rates are imagined to be immutable.²⁹⁵ They connect conceptualizations of black hypertension as genetic to the history of "drapetomania" and syphilis and more, cases in public health in which "the theory of race serves to justify racial inequality."²⁹⁶ Yet Cooper does not challenge the existence of the disparity itself, and opens a 1997 article coauthored with Rotimi on "Hypertension in Blacks" with the unequivocal claim that: "The excess of hypertension among blacks has

²⁹⁵ "Although it is widely assumed that blacks are genetically predisposed to hypertension, an environmental hypothesis is equally tenable. In the HDEP data, BP differences between black and white women disappear with control for education and obesity. The same is not true for black men (black men being no more obese than white), yet a clear social class gradient—as estimated by education—exists, which will sizably reduce the black-white gap for males as well. Rates of hypertension for blacks with a college education were similar to those for whites who did not finish high school. Earning capacity of black college graduates is almost identical to that of white high school graduates." Richard Cooper and Richard David, "The Biological Concept of Race and Its Application to Public Health and Epidemiology," *Journal of Health Politics, Policy and Law* 11 (Spring 1986): 97.

²⁹⁶ Richard Cooper and Richard David, "The Biological Concept of Race and Its Application to Public Health and Epidemiology," *Journal of Health Politics, Policy and Law* 11 (Spring 1986): 113.

been recognized since early in this century and explains a substantial portion of the black health disadvantage.”²⁹⁷

Kaufman and Cooper’s “race is not genetic” mantra, while true, is not a threat to the clinicians doing racially reifying work. Clinicians are perfectly capable of using a wide variety of modes of understanding race that do not rely on genetics alone. We can get a hint of this disconnect between Cooper’s critique and those he is criticizing in the responses to Cooper at the ASH symposium. The event is set up as a debate, and Cooper opened up his anti-race-as-genetic comments saying that he would be provocative. But neither Williams nor the A-HeFT speaker were interested in disputing Cooper’s assessment, because they are perfectly capable of using a wide variety of modes of understanding race that do not rely on genetics alone. Moreover, for both sides in each of the debates (Cooper versus Grim, or Cooper versus Williams, or Cooper versus A-HeFT), what is important is that action can be taken. Neither side of any of these thinks that disparities, whether natural or social, are inevitable and should be accepted.

More unites the two sides than Kaufman and Hall let on, in particular, belief that African American Hypertension is a legitimate topic of research and intervention. Kaufman and Hall suggest that a genetic theory precludes intervention, but Grim actually participates in both medical and social change. Grim is a white physician participating in white-dominated discussions of basic-science oriented arguments about racial difference, and this is the aspect that Kaufman and Hall address. Yet Grim is also a clinician treating hypertension in blacks. He is intervening, not just observing, and changing the biology that Kaufman and Hall assume is fixed.

²⁹⁷ R. Cooper and C. Rotimi, “Hypertension in Blacks,” *American Journal of Hypertension* 10 (July 1997): 804-12.

In this sense, we might read Grim as less a practitioner of genetic determinism of an old school than part of what Skinner has called “new biologism.” Skinner argues:

Dystopian critics associate biological arguments with determinism, essentialism and the naturalization of difference but new biological knowledge can and does take contrary forms that challenge conformist assumptions and existing divisions with an account of material heterogeneity and contingency...

This raises the possibility that critics, like generals, are often caught fighting the battles of a past war. Perhaps excessive genetic fatalism was the primary problem of an earlier era, but an excessive sense of the malleability of genes might present its own, quite different risks in an era where simplistic understanding of genes is being transformed into visions of readily available genetically-based products.²⁹⁸

Grim is not a passive natural scientist; he participates in organizing with diverse health care providers committed to improving the health of African Americans. As important as the scientific argument between him and his nemesis genetic epidemiologist Richard Cooper is, it is also important that they are both members of the International Society for Hypertension in Blacks and both participating in practical efforts at amelioration of disparities. Thus, neither the theory’s proponents nor its opponents really think that no action can be taken. When Cooper and Grim debate the hypothesis in venues like the International Society for Hypertension in Blacks, neither argues that disparities are inevitable and should be accepted. Kaufman and Cooper are far less clinically-oriented than Grim, so it is not surprising that they see the social as malleable and the biological as fixed. However, it is important not to take this dichotomy for granted. Grim has made his career not only by describing but also by altering patients’ biologies.

²⁹⁸ David Skinner, Manuscript of Paper Presented at the Society for the Social Study of Science, Paris, France, 2004.

Consensus underlying the debate

Each time that the debate over the slavery hypothesis is re-enacted, it takes for granted the existence of African American Hypertension as an entity. For example, one recent debate about the slavery hypothesis took place in the journal *Psychosomatic Medicine* in 2000. A leader of the journal, Joel Dimsdale, wrote of the theory sympathetically in the context of a larger piece that describes diverse etiologies for persistent ethnic disparities in health in which hypertension was a major focus. After defining phenotype as “a composite of culture, genotype, and the physical environment”²⁹⁹ in which all elements come into play, considering the shaky definitions involved in self-reported ethnicity, and remarking at the nonetheless powerful influence ethnicity has on health, Dimsdale wrote about relationships between ethnicity and memory in an epistemologically eclectic way:

At its heart, ethnicity involves the legacy of the past, and that legacy comes from many sources of memory. There is memory in our genes. William Least Heat-Moon recognizes this legacy by speaking "of genetic inclinations whereby ancestors seem to stalk our blood". Memory also comes, of course, from our personal life experiences and our families' experiences. As Faulkner points out "the past is not dead; the past is not even past." Thus, there are reverberating levels of history and ethnicity that influence health on multiple levels.³⁰⁰

Dimsdale devoted roughly equal space to consideration of the possible roles of “salt and slavery,” “stress and physiology,” and smaller sections on “emotional expression” and “letting go.” He concluded with his quirky ideas about memory as well:

I have tried to summarize many of the approaches my colleagues and I have used in our studies of ethnicity and health. Low social class, invidious life treatment, and differences in physiological vulnerability all determine risk trajectories across ethnic groups. Every one of us carries legacies (bequests and burdens), which are encoded in our genes, immune system, our group's experience, and our own

²⁹⁹ Joel E.Dimsdale, “Stalked by the Past: The Influence of Ethnicity on Health,” *Psychosomatic Medicine* 62 (2000): 162.

³⁰⁰ Joel E.Dimsdale, “Stalked by the Past: The Influence of Ethnicity on Health,” *Psychosomatic Medicine* 62 (2000): 162-3.

individual life experiences. These "memories" must be considered in understanding different susceptibilities to illness.³⁰¹

Kaufman himself wrote a letter in response, which suggested that the rest of the article was “thoughtful and illuminating,” and focused singularly on refuting “Dr. Grim’s fairytale” and warns that “The essay in *Psychosomatic Medicine* has unwittingly perpetuated this old pseudoscientific canard, which plays into the hands of racial essentialists and biological determinists.”³⁰² Kaufman concludes, “We may wonder why, despite an absolute lack of supporting evidence, in fact a great weight of contradictory evidence, otherwise smart and reasonable people continue to rehash this fantasy as though it were sensible and respectable science.”³⁰³ Yet it is precisely Kaufman’s drive to purge genetics from other larger racial arguments that leads Kaufman to fail to understand what is at stake for his interlocutors. Kaufman does not suggest that Dimsdale is himself a reductionist biological determinist (and such a claim would be an odd one since Dimsdale is an elite psychiatrist who is a leader in the field of the relationships between stress and physiology and cultural factors in illness). Instead, Kaufman suggests without basis that Dimsdale’s article feeds into the hands of the elusive and dangerous biological determinists out there.

Yet if we set aside any search for the racists, we can benefit from seeing ways in which the debate over the details of this particular hypothesis happens in a context of much more fundamental ontological questions uninterrogated. For example, Dimsdale acknowledged debate on that hypothesis itself, but states a point of consensus: “most

³⁰¹ Joel E. Dimsdale, “Stalked by the Past: The Influence of Ethnicity on Health,” *Psychosomatic Medicine* 62 (2000): 169.

³⁰² Jay S. Kaufman, “No More ‘Slavery Hypothesis’ Yarns,” *Psychosomatic Medicine*, Letter to the Editor, 63 (2001): 324.

³⁰³ Jay S. Kaufman, “No More ‘Slavery Hypothesis’ Yarns,” *Psychosomatic Medicine*, Letter to the Editor, 63 (2001): 324.

readers of this literature would agree that black individuals hyper-respond to stressors.”³⁰⁴

Kaufman does not address this point, which is not surprising given that there does indeed seem to be consensus on this in the literature.

But the non-controversial claim – that African Americans hyper-respond to stressors – is itself worth unpacking for my project. It is striking that the sentence conforms to white stereotypes of black personalities. That is, if stressors in the world are constant, white people respond to them in a standard way while black people respond to them with overreaction. That overreaction, in turn, is (at least in part) the cause of the increased harm experienced by black people. If this narrative is oversimplified, it does resonate with mainstream racism. Dimsdale’s arguments about the “withheld anger” of African Americans and their “hyper-response” to stressors are not any less enrolled in racial discourse than his points about the slavery hypothesis, and yet excessive focus on genetics leaves them under-addressed in the critique.

The category of African American Hypertension is not proposed by racists and critiqued by anti-racists. That it is a racial category does not by itself determine how precisely it will be mobilized. As Stoler points out, “a discourse is racial not because it displays shared political interests but rather because it delineates a field and set of conditions in which it becomes impossible to talk about sexuality, class membership, morality, and childrearing without talking about race.”³⁰⁵ As we have seen in the history of cardiology and of African American Hypertension, we cannot talk about hypertension in America over the past forty years without talking about race.

³⁰⁴ Joel E. Dimsdale, “Stalked by the Past: The Influence of Ethnicity on Health,” *Psychosomatic Medicine* 62 (2000): 161.

³⁰⁵ Ann Laura Stoler, “Racial Histories and Their Regimes of Truth,” *Political Power and Social Theory* 11 (1997): 194.

As Jonathan Metzl has argued with regard to schizophrenia, when considering race in medicine we need to consider not only the race of the physician and the race of the patient, but also the race of the diagnosis. Hypertension is not an exclusively black diagnosis, of course, but it is racialized as having a special belonging with this ‘special population.’ Hypertension as a combination of accessible root words seems to be evocative: *hyper* indicates excess, *tension* indicates strain, and there is an easy isomorphism between medical and popular ideas of African Americans as an over-reactive, highly-strained population, and one sick in these particular ways. Suggesting that the tension’s elevation comes from the social rather than the biological does not take it out of the bodies of African Americans. Critique of this category must not lean too heavily on critiques of genes and medicine, because the category does not rest on genes alone but rather on race as a dense transfer point of a wide range of factors that can be understood to be part of race.

As much invocation of the slavery hypothesis as there is, little of the theorizing of African American Hypertension purports to actually rely on it. Most theories of black hypertension do not seem to need an origin story, but simply begin with an assumption that we know who African Americans are and can measure their vasoresponse to various stimuli in comparison with white populations. Many studies do not specify how they knew who was black and who was not. For example, one with the title “Reduced Potassium Reversibly Enhances Vasopressor Response to Stress in African Americans,”³⁰⁶ which argued that blacks consume “from childhood” a diet higher in salt and lower in potassium than whites and are at the same time more sensitive to and are

³⁰⁶ Krishnankutty Sudhir et al., "Reduced Dietary Potassium Reversibly Enhances Vasopressor Response to Stress in African Americans," *Hypertension* 29 (May 1997): 1083-90.

more susceptible to vasopressor response to those minerals in excess or lack. But the authors do not define who counts as black for their own study, and certainly are not clear about the boundaries around black hypertension they cite, (and even a quick review of the footnotes shows that one of the studies cited to support their claims about blacks was specifically about biracial individuals).

The literature in this vein, endless measuring and comparisons of vasoresponse in response to various stimuli across ethnic groups portrayed as stable, is vast. On the one hand, this discussion is distinct from the debates between Grim and Cooper/Kaufman. Indeed, both Cooper and Grim participate in this research. But in both nature versus nurture debates and in research that assumes no need to answer that question before proceeding to document differences, conceptions of distinguishable racial differences in vasoresponse are solidified.

Cooper is far less clinically-oriented than his interlocutors at the ASH symposium, so it is not surprising that he sees social as malleable and biology as not. Kaufman and Hall make the same division of social=malleable, biology=fixed. However, it is important not to take this dichotomy for granted. While Williams is not only a clinician but also a social activist, both Grim and Williams make their careers not only by observing but also by altering their patients' biologies.

Debate about whether African American hypertension is social or biological does not undue the durability of the racialized disease category. Debate over whether African American hypertension is caused by genes or by environment does not throw into any question the reification of Black hypertension itself. Indeed, it provides content to the symposia and journals that can be devoted to it. There can be eclecticism in etiologies

that helps interest in it to proliferate. As Ann Laura Stoler has argued, “the ambiguity of those sets of relationships between the somatic and the inner self, the phenotype and the genotype, pigment shade and psychological sensibility are not slips in, or obstacles to, racial thinking but rather conditions for its proliferation and possibility.”³⁰⁷ Stoler points out that “the force of racial discourse is precisely in the double-vision it allows, in the fact that it combines notions of fixity and fluidity in ways that are basic to its dynamic.”³⁰⁸

At stake, then, on all sides of this debate is not whether “race is real” generally or whether African American Hypertension is “real.” It is impossible to talk about hypertension in American medical science and practice without talking about race. Both explanations of African American Hypertension – social and genetic – are racial discourses, and clinicians are perfectly capable of being epistemologically eclectic. Both sides of the debate contribute to the durability of the category of African American Hypertension, because both Grim’s claim that “African American Hypertension is genetic” and Cooper, Kaufman, and Hall’s counter that “African American Hypertension is not genetic” presuppose that “African American Hypertension *is*.”

³⁰⁷ Ann Laura Stoler, “Racial Histories and Their Regimes of Truth,” *Political Power and Social Theory* 11 (1997): 187.

³⁰⁸ Ann Laura Stoler, “Racial Histories and Their Regimes of Truth,” *Political Power and Social Theory* 11 (1997): 198.

Durability of race in hypertension in the face of attempts at displacement

In discourses of African American Hypertension, there are often rhetorical moves to displace race onto some other explanatory mechanism. These include class, discrimination, segregation, and more. And yet the displacement is not successful; the category remains uniquely attached to race. Whenever we are told that X is “just” Y, we should be alerted to a deception. The “just” is never true. Its iteration is evidence that there is an argument, an urge to subsume. Arguments that racial differences are “just” class ones – or psychosocial stress ones, or genetic ones, or insert causal agent – miss the important combinatorial work that the category’s very ambiguity and multivalence provides.

We might imagine race in medicine as a multi-headed hydra,³⁰⁹ with each of the specters of genetics, SES, stress, and culture as one of its heads. Each of these are resilient components that are both hard to impede individually and independent enough to take over if another component is temporarily weakened. In Greek mythology, the hydra was many headed creature that was difficult to kill because the heads had the capacity to re-grow if severed. Heracles and his nephew were only able to kill the hydra after they went beyond merely cutting off individual heads and used fire to cauterize each of the

³⁰⁹ Invoking the hydra as a way to describe race is not uncommon, but using the term has not lead to the rethinking of strategy that it should. For example, historian of science Garland A. Allen wrote in *Genewatch* about genetic determinism generally with some consideration of race in an article entitled “The Hydra and its Many Heads.” After noting that genetic arguments keep re-appearing after being vanquished, his counterargument does not consider what new strategies might be necessary to counter such a resilient problem, and merely walks through the history of the dangers of genetic determinism. Gerald E. Allen, “The Hydra and its Many Heads,” *Genewatch* 19 (September-October 2006). available at <http://www.gene-watch.org/genewatch/articles/19-5Allen.html>.

wounds to prevent regeneration. (The last head of the hydra was immortal, but after a bludgeoning Heracles buried it under a rock where it was no longer a threat.) There is no technology for cauterizing the hydra heads of race in medicine. Without that capacity, when arguments are leveled at any individual head, that head can be battered or removed momentarily but not destroyed, and the others remain unfettered. When Kaufman and other critics keep anti-racist focus so resolutely on genetics, they refuse both to grapple with the constant regeneration of race and genetics and to acknowledge the larger beast behind that particular head. Continually holding up the severed head race and genetics, they seem uncomprehending when others are not convinced that the monster of race and medicine is dead.

The assault on any hydra is a difficult venture, and in the case of race and medicine it is not necessarily the right fight. Attempts to kill race in medicine have left it with undead uncanny power.³¹⁰ Critique of race in medicine can better proceed by engaging with the hydra for as long as it remains vital. If it dies, it will die not from force of argument against its individual heads, but from a shift in the preoccupations of race in society. Weakening of generativity of race in medicine can only follow its irrelevance to our preoccupations. We saw in the case of intra-white difference described in the previous chapter that the concept did not die because of data or logical argument but

³¹⁰ This is connected with Duster's surgical metaphor for why we should critically embrace race in science rather than aspiring to purge it: "I propose a framework that makes sense of how scientific studies deploy the concept of race: not a radical surgical removal, not an uncritical acceptance of old taxonomies, but an acknowledgement and recognition of complex feedback loops." Against those pronouncing the 'death of race,' Duster argues: "I am not trying to resurrect race here as a social construct (with no biological meaning), any more than I am trying to resurrect race as a biological construct with no social meaning. Rather, when race is used as a stratifying practice (which can be apprehended empirically and systematically), there is often a reciprocal interplay of biological outcomes that makes it impossible to disentangle the biological from the social." Troy Duster, "Buried Alive: The Concept of Race in Science," in *Genetic Nature/Culture*, ed. Alan H. Goodman, Deborah Health, and M. Susan Lindee (Berkeley: University of California Press, 2003), 258-277.

because it became a less salient way for Americans to talk about racial difference. Interest in such distinctions as Italian versus Irish withered away rather than dying in an assault. Vestiges of such distinctions (perhaps an analogue to the hydra's immortal head buried under a rock) remain in old books and old data but have lost their power to inspire or terrorize. As long as black/white difference remains a preoccupation in America, we cannot have a fire or ice capable of cauterizing discourses of race and medicine, to once and for all stop the productivity of connections between race and culture, race and genetics, and the others. Even if we did, cauterizing is a technology with plural implications: since cauterizing not only prevents regeneration, but also deadens feeling, it is not an appropriate tool for those who want to remain morally engaged.

Efforts to explain away race in hypertension through appeals to other social characteristics don't free us from the task of addressing race because race is durable and has been encoded into the disease category itself. In the ineffectiveness of critiquing race claim by claim, we can see that displacing race does not damage it. As Waltraud Ernst argues in the introduction to the wide-ranging volume he edited with Bernard Harris *Race, Science and Medicine, 1700-1960*, idiosyncratic and contradictory discourses about race do not weaken them but rather are part of what makes them "work":

Racial discourses work well not despite their logical inconsistencies, ambiguities, and mixing up of premises, but *because* of them. They are destructively all-pervasive precisely because they are overdetermined and multivariant, creating the possibility for different arguments or perspectives (moral, biological, cultural, etc.) to be accentuated within different contexts and depending on the aims pursued.³¹¹

³¹¹ Waltraud Ernst and Bernard Harris, eds., *Race, Science and Medicine, 1700-1960*, (London ; New York: Routledge, 1999), 7.

The ambiguities and plural registers, then, are generative. And so it should be no surprise that discourses of connections between racialized bodies and disease are not always coherent, or that pieces of them are sometimes mobilized in unpredictable ways. By considering the attempts to displace race onto one of its components, and then either slay that component or try to offer it in the place of the hydra entire, we can see that displacement does not close down race but rather is productive in multiplying it.

Alternative 1: African American status as a marker of low socio-economic status

Many suggest that socio-economic status (SES) is actually what causes hypertension disparities, and perhaps should be used as a variable instead of race. But attempts to use SES to explain away racial disparity have met with very limited success, and SES is not necessarily a less messy variable for study.

SES as an alternative causative factor for disease may not seem particularly odd—it is after all well established in the history of medicine that poverty puts one at risk for disease—but is actually new in relationship to heart disease. As discussed in Chapter 2, from the early days of professionalization of cardiology, heart disease was understood as a disease *not* of poverty, but of affluence, of both societies and individuals. Although historians of heart disease assume that in some sense what we now know by that term has always existed, its historiography emphasizes its rise to dominance as both a cause of illness and death and as a paradigm of thinking about health and medicine in prosperous industrialized societies in the late 19th and early 20th centuries as their morbidity and mortality became less due to infectious and more due to chronic disease. A connection

between *low* SES and cardiovascular disease is naturalized if we forget its recent development. Invisible in the turn to SES as solution is the peculiarity of a disease of high socio-economic status becoming also a disease of low socio-economic status. Fantasies of replacing messy race with clean class are not possible when all class attributes can bestow risk.

Moreover, the idea that SES could be separable from race in the experiences of any individuals seems to be an idea that emerges from epidemiology but does not necessarily intersect with lived experience in a racially stratified society. Janet Shim describes the “lived intersections of race and class” for people with CVD. When she asked lay people with CVD about their experiences related to race (such as lack of ability to build wealth due to segregation or attenuation through middle class status), they included in their answers experiences related to class, and vice versa. This was frustrating for epidemiologists, who wanted to eliminate the technical conundrum of confounding race and class and make them separate and independent variables. But, following Shim, we should question the desire to isolate race from class, rather than look at how the two intersect and interact.³¹²

³¹² Janet K. Shim, “Constructing ‘Race’ Across the Science-Lay Divide: Racial Formation in the Epidemiology and Experience of Cardiovascular Disease,” *Social Studies of Science* 35 (June 2005), 411-412. In a sense, their origin stories for their own cardiovascular disease are like Grim’s origin story for the slavery hypothesis for hypertension, in that they mix race, health, class, history, and intervention.

Alternative 2: African American status as a marker of stress

As discussed in Chapter 2, stress has a longstanding relationship with the category of cardiovascular disease. And yet, it is tricky to use as a risk factor. One problem with trying to let the argument rest on “stress” is that blacks do not have a monopoly on stress. Indeed, much theorizing about stress in cardiovascular disease has been focused on the unique stressors elite white men of ambition described in the founding of cardiology (See Chapter 2) and through the concept of Type A personality. Aronowitz argues that Type A Personality – a notion I would mark as elite white male risk – ultimately failed as a clinically tested indicator because it was not quantifiable in a way that could be compressed into risk factor discourse.³¹³ It is important to qualify its failure a little. The concept of Type A has certainly lived on in the public imagination along with an image of a white male executive who dies too young of a heart attack—few in that demographic would imagine that they are *not* at risk.

However, if previous attention to the stress of white men was predicated on a contrast with imagined care-free African Americans, some more recent discussions of stress have come to center on African Americans themselves. Without challenging the existence of white stress, clinical and epidemiological studies began in the 1990s to consider the stress of racism in quantifiable ways, sometimes operationalized as discreet things like “race-based discrimination at work,” other times in a more generalized way as a catch-all to explain black stress. Often the stress of racism is conceptualized as part of

³¹³ Robert A. Aronowitz, “The Rise and Fall of the Type A Hypothesis,” in *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 145-165.

the category of risk factor “residual compounding,” explaining in a structural way all the remaining disparity between races after factoring out things like class and lifestyle.

In one widely-cited critique of the use of race as a variable in medical research, Osborne and Feit provide a guideline for the use of race in research: they disapprove of it as a variable itself, but approve of it when it is presented in a way that can allow a critical reader to identify what race is standing in for. They cite a hypertension study in which the authors argue that darker skinned African Americans suffer more from hypertension. Though the first assumption might be that this would suggest a relationship between dark skin and gene prevalences, the way in which the pool was described allowed it to be explained another way. Dark skin was only predictive of hypertension in the case of lower SES, which gave the authors an alternative explanation: namely, perhaps those of lower SES have a harder time dealing with the stresses of discrimination based on color.³¹⁴ In their framing, race is standing in for class and stress of discrimination in a way that promises to replace race itself as a variable. Yet because race is for them a peculiar combinatorial variable, irreducible to either of its components, their disavowal of race as variable in hypertension does not quite undo the racialization of hypertension or the reification of African American Hypertension as a disease category.

Biologist and STS scholar Anne Fausto-Sterling, too, posits the stress of racism as the underlying cause of black hypertension. She uses a thermostat metaphor to get at the problems of homeostasis as a model for understanding the regulation of hypertension for those under chronic stress:

³¹⁴ M.J. Klag, P.K. Whelton, and J. Coresh et al, "The Association of Skin Color with Blood Pressure in Us Blacks with Low Socioeconomic Status," *JAMA* 265 (Feb 6 1991): 599-602; Newton G. Osborne and Marvin D. Feit, "The Use of Race in Medical Research," *JAMA* 267 (1992): 275-279.

[A]n increasingly accepted model of human physiology that suggests that so-called essential hypertension, the health scourge of African Americans, is in some sense a natural physiological response to the deprivations and stresses of being a person of color in America. In other words, it is not that different biological processes underlie disease formation in different races, but that different life experience activates physiological processes common to all, but less provoked in some.³¹⁵

Fausto-Sterling suggests that allostasis (a basal state that anticipates future demands) rather than homeostasis (a basal state that maintains a constant 'normal' pressure) best helps to understand why elevated African American blood pressures do not abate. She argues:

Hypertension is an orchestrated response to a predicted need to remain vigilant to a variety of insults and danger—be they racial hostility, enraging acts of discrimination, or living in the shadow of violence. Over time, all of the components that regulate blood pressure adapt to life under stress.³¹⁶

For Fausto-Sterling, this model suggests that hypertension should be better treated with emotional and lifestyle interventions than through drugs that would treat only late manifestations rather than the source. That is, altering later manifestations resulting from allostasis rather than the conditions that induce the state – by treating hypertension rather than decreasing stress – can exacerbate the problem by inducing the body to do even more to achieve its own norm of a heightened equilibrium. This model allows her to account for biological processes that originate not from genetic determinism but from embodied responses to stressful lived experience.

The stress of racism is also a way to tell stories about connections between ways of life and experiences of disease, no less now than when physicians at the beginning of

³¹⁵ Anne Fausto-Sterling, "Refashioning Race: DNA and the Politics of Health Care," *Differences: A Journal of Feminist Cultural Studies* 15 (2004): 26.

³¹⁶ Anne Fausto-Sterling, "Refashioning Race: DNA and the Politics of Health Care," *Differences: A Journal of Feminist Cultural Studies* 15 (2004): 27-28.

the 20th century were diagnosing their own heart disease as a “disease of modernity.” One provocative example of this that explicitly engaged many of the health professionals and academics who have critiqued race as genetic was the documentary film *The Angry Heart: The Impact of Racism on Heart Disease among African Americans*.³¹⁷ Nancy Krieger, Cornel West, and Camara Jones all participate as commentators on the compelling documentary of a particular Roxbury man’s struggle with heart disease and its interventions. Stress-induced heart disease remains a compelling site to negotiate understandings of race and class, now for African Americans as well as whites.

Alternative 3: African American status is a marker of culture

As Shim has pointed out, it is more common for epidemiologists to explain racial differences as ‘culture’ than as ‘genetics,’³¹⁸ but these cultural explanations are also a reductive. The assumption that everyone who identifies as or is identified as black shares a common diet, lifestyle, and healthcare-seeking behavior obscures the fact that cultural practices are not precisely bounded by racial identity. Moreover, cultural explanations hide the power that precedes the presumably neutral cultural practices. In contrast to epidemiologists, clinicians do not necessarily imagine the cultural differences as fixed, and sometimes have interesting interventions post-difference—recommending using

³¹⁷ *The Angry Heart: The Impact of Racism on Heart Disease among African Americans*, Directed by Jay Fedigan, 57 minutes, © 2001. More information on the film is available at <http://www.theangryheart.com>. I saw it at the Boston Social Forum, University of Massachusetts-Boston, July 25, 2004.

³¹⁸ Janet K. Shim, “Constructing ‘Race’ Across the Science-Lay Divide: Racial Formation in the Epidemiology and Experience of Cardiovascular Disease,” *Social Studies of Science* 35 (June 2005): 405-436.

elastic fitness bands to exercise where walking isn't safe as well as urging malls to host walkers, and both modifying the DASH diet to make it "Soul-Food-friendly" and critiquing the valorization of the oppression of continuing to eat a diet with historical roots in "slave food."

In an overall illuminating article about the complexity of biology beyond genetics, Lundy Braun points out that culture and genetics can be equally pernicious:

Disease prevention policies differ dramatically depending on whether genetic factors or environmental and social conditions are invoked to explain racial inequities in causes and the natural history of disease. If racial discrimination is an important factor in the etiology of hypertension, reduction of the burden of hypertension in the black community requires addressing racism and how racism works, rather than focusing so exclusively on lifestyle factors or perceived cultural differences, all of which are loaded with blame and direct responsibility for change to the individual rather than the society.³¹⁹

Although she challenges the assumption that social in the sense of lifestyle causes will lead to more just solutions, Braun assumes without demonstration that identification of social injustice as the problem would necessarily lead to social justice solutions.

Alternative 4: African American status is a marker of victim of racism

One of the most interesting models of understanding African American hypertension is through marking how *racism*, rather than race, accounts for the disparity. This concept, as developed by Krieger and C Jones among others, includes institutional racism (e.g. lack of access to medical care); personally-mediated racism (e.g. racism of medical

³¹⁹ Lundy Braun, "Race, Ethnicity, and Health: Can Genetics Explain Disparities?" *Perspectives in Biology and Medicine* 45 (Spring 2002): 170

providers), and through internalized racism (e.g. accepting stereotypes as true). The second two of these are deeply related to notions of stress.

For Nancy Krieger racism as risk is both intertwined with and exceeds the psychosocial stress of living in a racist society. She highlights the difference between focusing on “biological expressions of race relations” and “racialized expressions of biology.” “The former draws attention to how harmful physical and psychosocial exposures due to racism adversely affect our biology, in ways that ultimately are embodied and manifested in racial/ethnic disparities in health. The latter refers to how arbitrary biological traits are erroneously construed as markers of innate ‘racial’ distinctions.”³²⁰ Physicians, after all, are one group that mediate the space where Krieger posits the location of the health consequences of discrimination: “the investigation of intimate connections between our social and biological existence.”³²¹

However, not all those that indict racism as cause of African American Hypertension indict drugs as solution. In fact, racism can also refer to deprivation of drugs. For disparities-focused physicians like those at the International Society for Hypertension in Blacks, racism is also a way to label physician resistance to aggressively treating African Americans. In part because many physicians (like Fausto-Sterling) believe that African American hypertension is poorly controlled with drugs, they are less likely to aggressively medicate it. Many of the physicians in ISHIB are investigators on huge diverse clinical trials that have set the gold standard of hypertension and CVD

³²⁰ Nancy Krieger, “Does Racism Harm Health? Did Child Abuse Exist Before 1962? On Explicit Questions, Critical Science, and Current Controversies: An Ecosocial Perspective,” *American Journal of Public Health*, 93 (2003): 195.

³²¹ Nancy Krieger, Quoted in Gabrielle Amersbach, “Through the Lens of Race: Unequal Health Care in America,” *Harvard Public Health Review*, Winter 2002

treatment for all populations, and are frustrated that physicians are not necessarily following this gold standard, in particular for their African American patients.

And so it is worth considering the stakes in displacing race onto one of these alternatives, be it socioeconomic, stress, culture, or racism. The often stated goal is that a more precise understanding of the cause of health disparities will lead to a more appropriately focused solution. Yet this is difficult to put into practice, because of the status of race as a difference that is both particularly recordable and particularly intertwined with dominance.

Why is race so much more durable than its alternatives?

Race is recordable, and rouses calls to action

We could trouble as historically inspire contingent not just the contents but also the parameters of a whole host of things subsumed by the category “race.”³²² All of the alternatives to race as risk described above are ways of telling stories about heart disease that are racialized but not exclusive to any one race. Part of the advantage to using race as a category rather than any one of these is that it captures what anti-racist epidemiologists have described as “accumulated insults” of living in a racist society.³²³

³²² As Lundy Braun points out, “Observed differences are generally explained through two dominant frames: one emphasizes genetic susceptibility to disease, while the other focuses on cultural practices. In both cases, it is minority communities—their inheritance or their culture and behaviors—that are seen as problematic. Rarely is the relationship among social conditions, power relations, and health the focus of study.” Lundy Braun, “Race, Ethnicity, and Health: Can Genetics Explain Disparities?” *Perspectives in Biology and Medicine* 45 (Spring 2002), 160.

³²³ Nancy Krieger, Quoted in Gabrielle Amersbach, “Through the Lens of Race: Unequal Health Care in America,” *Harvard Public Health Review*, Winter 2002

Despite these and other arguments that something besides race would better characterize disparities, race in medicine generally and African American Hypertension in particular is resilient. The debate over what underlies it does not destabilize it, but rather renders African American hypertension as a disease category that is overdetermined (in Althusser's terms). Black hypertension exists in a *contextual* relationship with genes, discrimination, SES, but need not be seen as being in *causal* relationship with any of these.

One thing that unites all of the aspects of what race might point to as risk is that none of them are as consistently recorded as race is. Telling physicians to base therapeutic decisions on whether someone is of low socioeconomic status, stressed out by racism, living a lifestyle typical of highly segregated postindustrial urban areas, or even some kind of genetic test given the expense and time that that approach still involves—all of these are less efficient than telling physicians to use a particular drug regimen for a population that is already reified and recorded, by race. So defining race as a risk factor in and of itself is a way to add it to things that are recorded and serve as indicators for prescriptions. The recorded fact *demand*s explanation, both for its existence and for its divergence.

An evocative phrase used by ISHIB founder Elijah Saunders among others is that African American hypertension is a risk factor “like diabetes.”³²⁴ Status as African American might be “like diabetes” because it is in fact diabetes—African Americans are likely to have more conditions that compound the risks of hypertension and heart disease generally. It also may be “like diabetes” in that it is correlated with higher morbidity and

³²⁴ Elijah Saunders, International Society for Hypertension in Blacks Conference, Detroit, MI, June 2004.

mortality from heart disease. But more importantly, Saunders is arguing that status as African American is “like diabetes” in terms of prescription guidelines. Anything that underlies the need for a lower blood-pressure goal is a risk factor “like diabetes.”

Saunders argues that, like those with diabetes, African Americans’ blood pressure goals should be lowered and thus their drug regimens more aggressive.

Two ideological trends are coming together to make the status as African American into a risk factor for various constituencies, including African American physicians’ organizations and pharmaceutical companies: First, the totalization of the “risk factor” such that everyone has at least one, which means both that labeling any particular thing a “risk factor” doesn’t mean that someone without that it is not at risk and that to be labeled with a risk factor loses its power of stigma. Second, hegemony of pharmaceutical thinking, in which drugs are almost always better than no drugs (and two drugs are better than one drug and so on...). In this logic, the way to get that intrinsic good of maximized medication is through liberal risk factor doling. From the perspectives both of the pharmaceutical companies and the African American physicians advocating the categorization of “African American” as a risk factor, there is no downside, only an upside: increased prescriptions of drugs (yielding increased profit for the one and improved patient health for the other).

Robert Aronowitz credits the utility of the concept of the risk factor to two of its characteristics: its ambiguity—it can “denote association with, cause of, predisposition to, or responsibility for disease”—and its utilitarian criteria—it can command treatment

like a disease.³²⁵ Aronowitz notes that for the designers of the Framingham Study: “Anything you could measure that became associated with a higher rate of heart attack or stroke later in life became a risk factor.”³²⁶ I would argue that the notion of the “risk factor” is in fact even more expansive than that suggests: it doesn’t even have to be measurable. It only has to be *recordable*.

In this recordability, we can see a moment of *both* action and of labeling. Once the category has been recorded, no matter how messy what underlies it might be, action can be framed around it. This is the move that Saunders called for in the symposium – let the messiness behind the category be even as we take action to help the “people who look like those in this picture.” Because of existing preoccupations with race, more than the alternatives, this move almost always works. Doctors look for patients’ race and almost always find it, and Saunders is trying to intervene in what they do next.

The debate between whether the realness of race comes from the social or from the biological does nothing to stop the reification of race, or of African American hypertension and heart disease. If race is social and economic, many would argue, it is mutable in a way it isn’t if it is biological. But, in the context of interventionist medicine, is this labeling of some things as “controllable” and others as “uncontrollable” only telling a story of the immutability of races?

³²⁵ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 144.

³²⁶ William Castelli and William Kannel, quoted in Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 144.

Trying to get control of race

The American Heart Association, on its website explaining hypertension to a lay audience, divides risk factors into “controllable” and “uncontrollable.” This might at first appear to be a taxonomy of mutability and immutability, but it also plays on other medical registers of ‘control’:³²⁷

Controllable risk factors

Obesity — People with a body mass index (BMI) of 30.0 or higher are more likely to develop high blood pressure.

Eating too much salt — A high sodium intake increases blood pressure in some people.

Drinking too much alcohol — Heavy and regular use of alcohol can increase blood pressure dramatically.

Lack of physical activity — An inactive lifestyle makes it easier to become overweight and increases the chance of high blood pressure.

Stress — This is often mentioned as a risk factor, but stress levels are hard to measure, and responses to stress vary from person to person.

Uncontrollable risk factors

Race — African Americans develop high blood pressure more often than whites, and it tends to occur earlier and be more severe.

Heredity — If your parents or other close blood relatives have high blood pressure, you're more likely to develop it.

Age — In general, the older you get, the greater your chance of developing high blood pressure. It occurs most often in people over age 35. Men seem to develop it most often between age 35 and 55. Women are more likely to develop it after menopause.³²⁸

³²⁷ This is connected with Metzl’s work on schizophrenia becoming uncontrollable when it became black. Jonathan Metzl, “Race, Stigma, and Schizophrenia,” presented at *Race, Pharmaceuticals and Medical Technology*, MIT, Cambridge, MA, April 8, 2006.

³²⁸ American Heart Association, “Am I at Risk?” (2004 [cited May 15 2004]); available from <http://www.americanheart.org/presenter.jhtml?identifier=2142>.

The division between “controllable” and “uncontrollable” does participate in the naturalization of race. It is presented in a way that makes race itself seem to explain disparities, and to exist within bodies of racialized people. It frames the response to disparity as enrolling them in treatment for the disease rather than address any social or environmental causes of disease disparities.

But at the same time, the chart works to collapse controllable and uncontrollable. This is because within medical practice things on both sides of the un/controllable division are treated in the same way. It is worth noting that many of the things that we might imagine to occupy the underlying risk in the category of African American are included in the “controllable” list – salt consumption, obesity, stress, and sedentary lifestyle – even as race is taken out, thus considered on both sides. Moreover, when race is imagined to be “uncontrollable,” it is often in connection with its heritability. What work is done by separating those out?

Despite the protests of those who argue for social causes and social solutions for disease, there is no evidence that medical researchers could easily be moved away from pharmaceutical solutions no matter how the problem is defined. The risk factors are not really imagined to be “uncontrollable” once they are incorporated into practical medical (and in particular pharmaceutical) logic.³²⁹ Although race is uncontrollable in this chart, its effects on the cardiovascular system can be undone by the right pills. In effect, the

³²⁹ Indeed, if we take seriously Karrin Garrety’s argument that the cholesterol hypothesis became stable not because of evidence shifts but because many medical, activist, and food industry social worlds saw the opportunity to “do something” if it was accepted, we can see a connection here between the durability of African American as a risk factor and the durability of cholesterol or any other risk factor. Karin Garrety, “Social Worlds, Actor-Networks and Controversy: The Case of Cholesterol, Dietary Fat, and Heart Disease,” *Social Studies of Science* 27 (1997): 727-73.

“controllable” and “uncontrollable” risk factors are all treated the same way: principally through drugs.

For the turn toward medical and in particular pharmaceutical response, it is not necessary to argue that race is genetic.³³⁰ That we find many of the underlying aspects of the so-called “uncontrollable” category of race within the “controllable” is worth unpacking, but is at the same time beside the point for the pragmatic racist or antiracist physician. STS critiques’ focus should be thus not only on the marking of bodies in today’s medicalization, which in any case is less interesting to the physician on the ground than “what now” questions, but on the processes in which racialized medicine plays one part: drugs into bodies, bringing African Americans more fully into the pharmaceutical grammar Dumit has termed *Drugs For Life*.³³¹

Pharmaceutical companies pushing both race-specific drugs and full integration of African Americans into gold-standard hyper-medication are both parts of racial discourses. To understand the terrain, we should attend to the ways that their debates render race both stable and malleable. To complement attention to the ways that pharmaceutical grammars naturalize race, we must attend to the ways that they seek to mobilize it.

The essentialist move is only part of the story of how race becomes durable. The power of the notion of “immutability” never lies in “immutability” itself, but rather its

³³⁰ Social reasoning does not necessarily lead to social responses. When Osborne and Feit critique research on genetic correlations between racial disease differences, they point out that “Constant attention on African Americans and Hispanics as being disproportionately affected by certain diseases often leads to a belief that one of the best efforts to reduce illness is to concentrate on more health programs for these groups. In this context, other, more virulent societal problems that predispose to disease, such as underemployment, poor management, and adverse public attitudes, need not be addressed.” Newton G. Osborne and Marvin D. Feit, “The Use of Race in Medical Research,” *JAMA* 267 (1992): 278.

³³¹ Joseph Dumit, *A Pharmaceutical Grammar: Drugs for Life and Direct-to-Consumer Advertising in an Era of Surplus Health*, Duke University Press, forthcoming.

tension with malleability. Stoler points out that “The notion of ‘immutability’ may play off deep cognitive beliefs about what constitutes human kinds, but, ironically, it only becomes a potent political principle when the attributes designated as immutable are pliable and plastic....”³³² She argues that contamination, the racialized discourse that she focuses on, is “a cultural and political notion that simultaneously does two kinds of work: it both confirms *and* calls into question the discreteness of human kinds.” As I will explore in the two chapters that follow, something similar could be said of race and pharmaceuticals: a pharmaceutical solution to African American heart disease both underscores African American biological distinctiveness and undermines it—after all, it is a distinction that both can and cannot be medicated away.

³³² Ann Laura Stoler, “Racial Histories and Their Regimes of Truth,” *Political Power and Social Theory* 11 (1997): p. 99.

Chapter 5

Thiazide and Racialization of a Generic Drug

In the previous chapters, we have seen the importance of differentiating Americans in constructing cardiovascular disease knowledge and practice within medicine through tracking some ways that this has been done over the course of the twentieth century. In this chapter and the one that follows, the focus turns toward particular contemporary racialized pharmaceuticals, and includes investigations of these drugs as they move outside the scope of medical conversations. In the process, we will be attentive to the role of pharmaceutical practices in articulating racialized American identities.

Thiazides, or “thiazide-type diuretics,” are a class of drugs introduced in the 1950s (and now long generic) that treat hypertension by reducing the amount of fluid in the body. Decreasing volume decreases blood pressure, and thus the work the heart has to do to, and thiazide’s impact on morbidity and mortality seems to exceed this effect as well. Thiazide is often combined with other antihypertensives to increase its antihypertensive effect while mitigating its side effects such as increased blood sugar and lowered potassium. Thiazide also (relatedly) decreases the sodium level in the body. When debates about thiazide intersect with debates about race, this relationship with salt

is important. The previous chapter showed that African American Hypertension as a disease category is generally characterized as “salt-sensitive,” and this chapter will attend to ways in which thiazide becomes linked to that racialized category.

This chapter will analyze a few of the ways that thiazide is invoked and contested in identity, in particular with regard to race. On the one hand, thiazide’s efficacy in African Americans has been employed in the clinical definition of African American Hypertension as a disease category characterized by salt sensitivity, a characteristic attributed by some inside and outside medicine to the selection pressures of slavery. That line suggests that thiazide could be a key to solving racial morbidity and mortality disparities. At the same time, it has been touted by the National Institutes of Health as the best antihypertensive medicine for everyone – and especially for blacks. This guideline has not been universally applied, as doctors generally prefer to prescribe the newer more expensive antihypertensives they have become accustomed to (and there has been some skepticism about whether the government is a completely disinterested party in touting an old cheap drug). It is in the nexus of these associations – racialized, proven, old, cheap – that I will map out not a position but a process, unpacking just some of the plural productions of coherence and incoherence at the intersections of race and this particular pharmaceutical.

In the science studies of pharmaceuticals, there has been rich analysis of the ways in which doctors, patients, and society as a whole both incorporate and contest the language of drugs’ marketing into their descriptions of disease experience and identity.³³³

³³³ Joseph Dumit, *Drugs for Life: Managing Health and Identity Through Facts and Pharmaceuticals*, Duke University Press, forthcoming; Nathan Greenslit, “Pharmaceutical Branding: Identity, Individuality, and Illness,” *Molecular Interventions*, 2:342-345 (2002).

But how can we approach generics? If we want to understand the allure of pharmaceuticals, we need to look beyond both medical efficacy and profit motives. The success (or failure) of a drug depends not only on these, but also on how it mobilizes prior conceptions of identity. The extent to which a drug is taken – or talked about – is related to commodity properties that exceed the physiological and the economic.

In this chapter, I will explore how the links between race and pharmaceuticals can be both unstable and generative even when the drug in question is old and generic. By first attending closely to an encounter around generic thiazide *outside* a medical or marketing context, I will show that although pharmaceuticals can seem to rely on scientific data and marketing for their power, they are in fact also subject to claims on many more registers that cannot quite subsume or refute each other. I will then circle around to consider how the drug has been important in the debates within medicine about race, disease categories, and drugs in ways that are also connected to aspects of commodity fetish.

An exchange at an African American Studies colloquium and identity practices around thiazide

Take, as an entry point, one particular encounter outside medicine or marketing:

The setting was a midday colloquium at the WEB DuBois Institute for African American Studies, featuring rising young Harvard economist Roland Fryer (who later became more generally known as the result of front page profile of him in the *New York*

*Times Magazine*³³⁴). The title of the talk was ambitious: “Understanding the Racial Difference in Life Expectancy.”³³⁵ Fryer’s goal in his captivating presentation was to explain the persistent differential in African American versus white morbidity and mortality. After controlling away such explanations as education, income, diet, geographic region, and access to health care—the explanations that come to mind most readily for economists or for that matter social scientists as a whole—Fryer turned outside our disciplines’ traditional line of argument.

Fryer cited the theory that Dr. Clarence Grim became famous for developing twenty years ago in medical journals and conferences, which I described in the previous chapter.³³⁶ This theory argued that selection pressures in Atlantic slavery predispose African Americans to salt retention, and thus to cardiovascular disease.³³⁷ The suggestion is that Africans who were genetically predisposed to conserve salt were the ones to better survive the diarrheic diseases of the journey to slavery, and so as a result their descendents are salt-conserving. According to this argument, while a tendency to conserve salt might have been protective against old hazards like cholera, it is dangerous today because it leads to hypertension and cardiovascular disease. And so, Fryer posited, this difference in salt sensitivity explains the difference in cardiovascular disease and the excessive morbidity and mortality of African Americans today.

³³⁴ Stephen J. Dubner, “Toward A Unified Theory of Black America,” *New York Times Magazine*, March 20, 2005, p. 54.

³³⁵ Roland G. Fryer, “Understanding the Racial Difference in Life Expectancy,” WEB Dubois Institute for African American Research Colloquium, October 20, 2004, noon-2pm.

³³⁶ See previous chapter for analysis of this theory, with particular attention to competing narrations of its origins and motivations.

³³⁷ As analyzed in the previous chapter, the hypothesis has been extensively described and critiqued by epidemiologist Jay S. Kaufman, both in “The Anatomy of a Medical Myth,” Social Sciences Research Council Forum Is Race Real?, <http://raceandgenomics.ssrc.org/Kaufman/>, accessed September 1, 2005, and in Jay S. Kaufman and Susan A. Hall, “The Slavery Hypertension Hypothesis: Dissemination and Appeal of a Modern Race Theory,” *Epidemiology* 14 (January 2003): 111-118..

The audience response at this particular Colloquium was high energy. Here was a black scholar reactivating a racist theory that most academics have rejected, yet the response was overwhelmingly positive.³³⁸ When an elderly white geneticist in the audience protested, asking “doesn’t making the cause of disparities genetic mean that there is nothing to be done, it’s unchangeable?”, Fryer responded “Oh no, there’s a drug for that!” Which drug?, the audience wanted to know. Fryer drew a momentary blank, and uttered some fragments: Chloro... Hydro... A voice shot up from the front row — it was the chair of the Harvard department of Afro-American Studies, renowned literary theorist-cum-public intellectual Henry Louis Gates Jr. “Hydrochlorothiazide!” he exclaimed. “I’m on that! I’m a salt-saving Negro!”

This statement was dramatic, and it was jarring, given that discussion in the history and social studies of science generally operates on the consensus that race is a social construct and not a genetic one. Evelyn Hammonds, an African American historian of medicine in the audience, was at a loss to address the situation, beyond pointing out that Philip Curtin, the prominent historian of slavery, had roundly refuted the salt-saving hypothesis. After a brief question about the nature of Curtin’s critique, Gates and the audience returned to the thesis with enthusiasm. The historian’s critique missed the emotive point at hand, which did not actually rest on the scientific and historical facts underlying the theory.

This vignette is presented as an opportunity to open up emergent processes for analysis. The moment I am describing comes neither at the end of thiazide’s story, nor at

³³⁸ Work Fryer did with his advisor, Steven Levitt, on *Freakonomics* – which also involves economists reaching well out of bounds of their own discipline to find surprising answers to social problems from other fields – has also found broad appeal.

the beginning, so I hope to mobilize it less as ground than as a node from which to begin unpacking meanings that circulate around this drug in an effort to provide insight into how to analyze pharmaceuticals more generally. The meaning-making capacity of pharmaceuticals does not necessarily have marketing at its base. Marketing is rather drawing on something prior, which I argue has to do with pharmaceuticals' nature as objects, as commodities, whether or not they have high-budget advertising campaigns. Analyzing the commodity fetishism of generics is an opportunity to get conceptually closer to the processes of fetishization of pharmaceuticals generally. And so I will review the concept of the commodity in terms of medicine, and then examine a variety of lenses through which to read this exchange, each of which will provide partial perspectives on it. My goal is to leave the terrain unsettled, to show that the reification of race in contestation over this drug is always incomplete even as it is always being done.

The pharmaceutical as an object can be understood as a commodity. According to Karl Marx, a commodity is an external object that satisfies human needs of whatever kind and has a dual nature of both a use value and a bearer of value. Though all commodities are expressions of human labor, and so are purely social, they come to be understood outside of social relations and to take on a magical quality. The commodity reflects the social characteristics of human labor in objective form, and through this substitution human labor becomes a sensuous thing that is simultaneously supra-sensible. Marx makes an analogy with religion to explore the peculiar agency commodities come to take on. Like gods, commodities are "products of the human brain [that] appear as

autonomous figures endowed with a life of their own, which enter into social relations both with each other and with the human race.”³³⁹

As soon as objects become commodities, they become amenable to this fetishism, in which it is possible to fantasize that the objects of our own creation have mastery over us. This fantasy is itself structural, and cannot be punctured by simply naming it as such. Pharmaceuticals, as objects that can be understood to have an embodied as well as ideological mastery over us, are a peculiar commodity. Like all commodities, they come to have a life of their own that exceeds their design and distribution.

In this moment of the Colloquium, Fryer and Gates are participating in the meaning-making of thiazide. For each of them, in different ways, thiazide becomes a thing to think with as they invoke it to talk about their own economic research and racial identities respectively. Thiazide becomes an *evocative object* that can be encountered on many levels: one-on-one as a physical object (the pill itself); as a gateway to health (its promise of protection from sickness and death); and as an artifact with which we can relate to define our own sense of self (something “extra”).³⁴⁰ Thiazide offers something physiologically: to lower blood pressure by 10 millimeters of mercury. It also makes a larger promise of health: to provide protection from cardiovascular disease economically and effectively.³⁴¹ It was on these relatively narrow promises that Gates originally took the drug. But for Gates and Fryer, invoking this commodity also invokes something

³³⁹ Karl Marx, *Capital: Volume 1*, trans. Ben Fowkes, (London: Penguin 1990), 165.

³⁴⁰ This taxonomy is analogous to that of Sherry Turkle regarding the computer. “Whither Psychoanalysis in Computer Culture?” *Psychoanalytic Psychology* 21 (Winter 2004): 16-30. The evocative object is a concept she began to theorize through and with computers in *The Second Self: Computers and the Human Spirit* (New York: Simon and Schuster, 1984).

³⁴¹ These two aspects may seem to be the same thing, but in fact only after medical knowledge on an individual level becomes biomedical knowledge on a large scale can the intervention into asymptomatic states of risk be understood to have an impact on something we can understand as “health.”

“extra,” which reaches a scale that exceeds the mundane or the individual and touches on the projects that frame not only their life times but also their respective life’s work. Thiazide makes two new promises after it has already been consumed: it promises to connect Gates to his kin, and for Fryer it promises to solve one part of racial inequality in America. The magical potential of this commodity is underscored when it is revealed to be doing more work than it had been advertised to do.

Indeed, the notion of what a drug is advertised to do is important here, and connects to the well-developed STS critique of processes of interpellation in the pharmaceutical advertisement.³⁴² For Louis Althusser, the subject is interpellated in an encounter with the police who yell, “hey, you!”³⁴³ The subject is the one who responds with “It’s me!” Donna Haraway has noted that the process of interpellation extends to technoscience, through whom we all—not just scientists and engineers—are interpellated with its “hey you!”³⁴⁴ Joseph Dumit has extended this to explore the ways in which the pharmaceutical advertisement interpellates particular kinds of subjects, in a way that is simultaneously partial and effective.³⁴⁵

Now, in this exchange between Fryer and Gates before an audience, we can see something similar happening, but without the intervention of physician-scientists or pharmaceutical marketers. We can see Gates’ subjectivity interpellated by invocations of

³⁴² See especially Joseph Dumit, *Drugs for Life: Managing Health and Identity Through Facts and Pharmaceuticals*, Duke University Press, Forthcoming; Nathan Greenslit, “Pharmaceutical Branding: Identity, Individuality, and Illness,” *Molecular Interventions*, 2 (2002):342-345.

³⁴³ Louis Althusser, “Ideology and State Apparatuses,” in *Lenin and Philosophy and Other Essays*, trans. Ben Brewster, (New York: Monthly Review Press, 1971), 171.

³⁴⁴ Donna Haraway, *Modest_Witness@Second_Millennium.FemaleMan©_Meets_OncoMouse™*, (New York: Routledge, 1997).

³⁴⁵ Joseph Dumit, *Drugs for Life: Managing Health and Identity Through Facts and Pharmaceuticals*, forthcoming, Duke University Press. The interpellation is successful precisely because it is partial—you can fill in the blank.

a drug outside of its explicit marketing or a medical encounter. This suggests that the interpellation of the subject by a pharmaceutical is not, at base, marketing, but rather that marketers are using the pharmaceuticals' prior interpellative potential. But in this case as in Dumit's, if the interpellation is successful and the drug is taken it makes the patient him-or-herself into a particular kind of subject, one who is the object of scientific knowledge, in this case, a Salt-Saving Negro.

We can tell from his entire oeuvre that Henry Louis Gates Jr. is not naïve of the volatile power of inscribing the body with race. He is one to put the word "race" in quotes in many a title, and is always careful to connect it to a mode of historical experience and not a static biological given.³⁴⁶ Gates appeals to his use of the drug to describe a kinship with his ancestors, but not in a simply naïve way. Reification is something that Gates knows is a dangerous path, and he does it in the form of a half-joke with the odd logism "salt -saving Negro." But to point out that reification is a dangerous path does not refute the work that it is doing.

Indeed, the locution "salt-saving Negro" may be read as part of Gates' African American literary criticism, as being in the voice of the trickster, and meant to be figurative and ambiguous.³⁴⁷ When Gates makes this interjection, he is not only making a claim, but also playing a prank. He is disobeying normal rules of behavior for the academic setting of a colloquium in which ideas and not individuals are the subject of discourse, and there are no Negroes, only African Americans. In doing this, he is

³⁴⁶ For example Henry Louis Gates, Jr., ed., *"Race," Writing, and Difference* (Chicago: University of Chicago Press, 1986); and Henry Louis Gates, Jr., *Figures in Black: words, signs, and the "racial" self* (New York: Oxford University Press, 1987).

³⁴⁷ Gates himself can be understood to be signifying, the narrative process he describes in *The Signifying Monkey: A Theory of Afro-American Literary Criticism*, (New York: Oxford University Press, 1988).

displaying many of the characteristics he ascribes to the trope of the trickster in African and African American literature: individuality, satire, parody, irony, magic, indeterminacy, open-endedness, ambiguity, disruption and reconciliation, betrayal and loyalty, closure and disclosure, encasement and rupture. And he is not only making a polemical claim based on the literal ingestion of a drug, but making the kind of double-voiced statement he characterizes as fundamental to African-American vernacular tradition: inciting action and debate through playing on rhetorical principles.

So what is at stake is not whether Gates — or the audience of the Colloquium, or the reader of this chapter — “really believes” in either the risk category of the Salt Saving Negro or in the efficacy of thiazide as the solution. If we limit our critique of the ideology of pharmaceuticals to the machinations of their sellers or the naiveté of their consumers, we only understand the most straightforward of situations. But there is more than one way to “buy in.” Some consumers may indeed be ignorant, but many are perfectly capable of simultaneously being skeptical of and participating in pharmaceutical grammars,³⁴⁸ even trying to shape them a bit along the way.

And so, I would suggest, it is possible for the subject interpellated by this concept of the Salt-Saving Negro on thiazide to be read not necessarily as a naïve believer in

³⁴⁸ This term is from Dumit, *Drugs for Life: Managing Health and Identity Through Facts and Pharmaceuticals*, forthcoming Duke U Press. Much literature about social movements have described this shaping process, such as Steven Epstein, *Impure Science: AIDS, Activism, and the Politics of Knowledge* (Berkeley, CA: University of California Press, 1996); Nathan Greenslit, “Depression and Consumption: Psychopharmaceuticals, Branding, and New Identity Practices,” *Culture, Medicine and Psychiatry*, 29 (December 2005), 477-502. Andrew Lakoff has described the considerable “interpretive flexibility” in pharmaceuticals, in a context in which acceptance of a biological model of depression was not a prerequisite for antidepressants, which could become a way to understand a social situation of crisis: Andrew Lakoff, “The Anxieties of Globalization: Antidepressant Sales and Economic Crisis in Argentina,” *Social Studies of Science* 34 (2004): 247-269.

specious science but as a cynical subject, as described by Slavoj Zizek.³⁴⁹ Like Marx, Zizek is framing his argument about ideology and commodities with a comparison to religious thinking. Zizek argues that one mark of modernity is that religion becomes something separate from a way of life, and modern subjects assume one of two roles: either the Christian who says “I really believe!” or the skeptic who relies on the figure of the Other who “really believes.” Zizek argues that we cynical subjects cannot understand what (or even that) we really believe, because we always insert this distance. Pharmaceuticals can be successful in interpellating both kinds of modern subjects, both the Christian and the skeptic. If we critique the ideology of pharmaceuticals on the basis of false consciousness of their pushers or their consumers—they know not what they do, so they do X—we miss the insight from Zizek that the cynical subject is perfectly capable of knowing what they are doing and doing it anyway.

Not every claim we make that invokes pharmaceuticals is literal, and so the claims cannot necessarily be refuted on the basis of scientific evidence. If we understand the connections we see between our identities and these pharmaceuticals as commodity fetishes, we can understand both the identity and the distance invoked. In Donna Haraway’s work on gene fetishism, she has unpacked the fetish in this way: “The fetishist is not psychotic, he ‘knows’ that the surrogate is just that. Yet he is uniquely invested in this power-object. The fetishist, aware that he has a substitute, still believes in—and experiences—its potency, he is captivated by the reality effect produced by the image.”³⁵⁰ The reality effect she refers to, and which is also provoked in this Fryer/Gates encounter,

³⁴⁹ Slavoj Zizek, *The Puppet and the Dwarf: The Perverse Core of Christianity* (Cambridge, MA: MIT Press, 2003).

³⁵⁰ Donna Haraway, *Modest_Witness@Second_Millennium.Femaleman©_Meets_Oncomouse™: Feminism and Technoscience* (New York: Routledge, 1997), 144.

is that of the Barthesian realistic novel in which the story is infused with verisimilitude by attending to details that appear to have no reason to be there besides their correspondence with historical reality.³⁵¹ In this encounter, the presence of a cheap generic drug in Gates' medicine cabinet provokes a reality effect. It had seemed irrelevant, but it was now part of the evidence for Fryer's story of uniquely black hypertension. It is convincing in the same way that a novel is, not in the way of a scientific argument. That is why refutation based on citing a prominent historian did not undo the captivation of the audience with this theory. The exchange produced the reality effect not only in spite of but also because of the contingency and coincidence of Gates' own case.

The resistance of this reality effect to contestation based on the limitations of the scientific data is also captured in the work of Alondra Nelson.³⁵² She has collected narratives of African Americans who have used mail-away genetics test to discover what part of Africa their ancestors came from. She describes the ways that they use the tests, however inconclusive, in objective self fashioning of their identities. As she demonstrates, this process is not exactly undone by pointing out the limits of the science on which it is based. Narratives of connection preceded the procurement of the tests, and have power that does not rise or fall on the results.

In the media response to Fryer, which is still emerging, much has been made of the fact that he is black and pushing this theory of biological race that can be medicated.

³⁵¹ Roland Barthes, *Mythologies*, trans. Annette Levers (New York: Hill and Wang, 1972).

³⁵² Alondra Nelson, "Genealogical Branches, Genetic Roots, and the Pursuit of African American Ancestry," presented at MIT STS Colloquium, March 13, 2006.

There has been tut-tutting about his naiveté.³⁵³ If he were touting a branded pharmaceutical, it would be pretty easy to let the explanation rest with an argument about economic motivations.³⁵⁴ But instead, Fryer is touting a cheap generic drug. His lack of financial stakes in the drug illustrates an important way in which the commodity fetishism of a pharmaceutical exceeds that of its marketing and moves in unpredictable ways.

We might turn again to Roland Barthes for another mode of reading this encounter: mythologies and identification. We can read the images of Fryer as a proponent for pharmaceuticalized race the same way that Barthes reads the image of the Negro soldier before the French tricolor.³⁵⁵ Barthes reads that image three ways: as an *example* of French coloniality, and as an *alibi* for it, and as simply the *presence* of it. The analogy between French coloniality and American pharmaceutical hegemony is dramatically imperfect and yet evocative: Fryer as example of, and an alibi for, and demonstration of the presence of a particular model of racial disparity that is simultaneously biologically real and capable of being medicated away.

It is worth highlighting that Fryer's model of race here is not exactly the same as Gates', because his ground is different. Fryer responds to the concern raised by the

³⁵³ Patricia Williams, "Salt in the Wound," *The Nation*, June 6, 2005.

³⁵⁴ Indeed, as we will see in the next chapter, groups with high standing have had their credibility on their support of BiDil suffer because of financial interests. The NAACP received \$1.5 million from NitroMed when they agreed to a strategic partnership, "NAACP and NitroMed Announce Partnership to Narrow Disparities in Cardiovascular Healthcare," *Business Wire*, December 14, 2005. Many mentioned the money in the case of the Association of Black Cardiologists, in grammar like this: "Nitromed did what other pharmaceutical companies have always done. It gave money to people who later gave its medication the thumbs up. The Association of Black Cardiologists co-sponsored the clinical trials for Bidil, received \$200,000 from Nitromed, and enthusiastically supported the drug's approval." Margaret Kimberly, "Rx for Black Hearts," at [BlackCommentator.com](http://www.blackcommentator.com)

http://www.blackcommentator.com/143/143_freedom_rider_rx.html

³⁵⁵ Roland Barthes, *Mythologies*, trans. Annette Levers (New York: Hill and Wang, 1972).

geneticist in the audience about “fixing race” in the sense of making it unchangeable with an answer that promises to “fix race” in the sense of easy repair: “there’s a pill for that!” He uses a racialized invocation of that pill to hold out the promise of putting an end to the only racial difference that he is paying attention to: mortality disparity. Gates, on the other hand, is invoking his use of the pill to connect him to the racial differences that he is most interested in: the role of the common history of slavery in shaping the African American experience, and a mode of narration. Fryer has a model of racial disparity that is simultaneously biologically real and capable of being medicated away, but Gates has a model of race that is socially real yet capable of being medicated in. For each of them, the drug’s capacity to traffic between the material and the semiotic helps to move race across that boundary, and yet the traffic is in differing directions. And so we can see the limits of an analogy to Barthes, who promised to reveal *the* cultural logic of the Negro before the tricolor. These identifications and mythologies are unstable, diverse, ambiguous, even among African American Harvard professors, let alone as they move across other venues.

Use value and exchange value in medical debates on thiazide

Using thiazide as a way to mark African American Hypertension as distinct is not only done on this one occasion. Gates and Fryer are not the only ones to use thiazide as a nexus through which to talk about ideologies of race. Indeed, for Clarence Grim, the proponent of the slavery hypothesis, as well as clinicians broadly “responsiveness to diuretics” is part of how African American Hypertension is defined as a disease category.

The drug's particular effectiveness in managing salt sensitive hypertension is a recurring theme in the definition of the category of African American Hypertension (as discussed in the previous chapter). I want to expand my scope to add two more commodity aspects of thiazide that are prevalent in medical debates about this drug: efficacy and price.

Debates about the differential effects of various classes of antihypertensives have been central in the arguments about whether there are racial differences in the efficacy of drugs more generally. Kahn has pointed out in the recent debate about the role of racial difference in drug response in *Nature Genetics* that of the 13 drugs that have been suggested to have evidence for physiological differences between races, all are antihypertensives.³⁵⁶

Thiazide has been of renewed interest in clinical practice because of a landmark trial known as ALLHAT.³⁵⁷ This trial, which ended in 2002, is the largest antihypertensive trial ever conducted, with over forty-two thousand participants. It was conducted by the National Institutes of Health to compare three classes of newer more expensive antihypertensives with a thiazide diuretic. It began in 1994, after the NIH Revitalization Act that required inclusion of women and minorities in trials, and about half its participants were women, and a third were African American. According to this landmark study, the newer more expensive antihypertensives don't work any better than the old cheap diuretics like thiazide, and that is particularly true in African Americans. A slide from the trial itself summarizes its implications:

³⁵⁶ Jonathan Kahn, "Misreading Race And Genomics After BiDil," Letter, *Nature Genetics* 37 (July 2005), 655.

³⁵⁷ The Antihypertensive and Lipid Lowering Treatment to Prevent Heart Attack Trial, <http://www.nhlbi.nih.gov/health/allhat/facts.htm>

(1) diuretics should be the drug of choice for initial therapy of hypertension. The evidence for this recommendation is even stronger for Black hypertensive patients;

(2) For the patient who cannot take a diuretic (which should be an unusual circumstance), CCB's and ACEI's may be considered. However, in Black hypertensive patients, ACEI's should be considered second-line therapy.

(3) Most hypertensive patients require more than one drug. Diuretics should generally be part of the antihypertensive regimen. Lifestyle advice should also be provided.³⁵⁸

By attending to ways that physicians talk about ALLHAT, we can circle back and see the ways in which clinical discussions are working through some of the same questions as Fryer and Gates.

For example, when Jason van Steenburgh writes in the American College of Physicians about ALLHAT implications, he mentions that Blacks' salt sensitivity makes them particularly good candidates for thiazide:

There is one group that researchers say you should definitely consider switching from ACE inhibitors to diuretics: black Americans. ALLHAT found diuretics lowered systolic blood pressure 4 mm Hg more than ACE inhibitors, probably because black Americans' salt sensitivity makes them especially responsive to diuretics.³⁵⁹

³⁵⁸ The Antihypertensive and Lipid Lowering Treatment to Prevent Heart Attack Trial slide deck, <http://allhat.uth.tmc.edu/Slides/RaceSexAge.ppt#18>.

³⁵⁹ Jason van Steenburgh, "Diuretics for Hypertension Get a Big Boost, But Will Data Change Prescribing Patterns?" *American College of Physicians Observer*, April 2003, available at <http://www.acponline.org/journals/news/apr03/diuretics.htm>. It continues in a vein that suggests that including blacks in America may throw off statistics for efficacy, in ways that resonate with the questions raised in Chapter 3 about who can stand in as normal America: "Even more significantly, the study found that black Americans taking ACE inhibitors experienced a stroke 40% more often than their counterparts taking diuretics. (Black Americans typically require high levels of ACE inhibitors to control their blood pressure.) Black Americans responded so well to diuretics in the ALLHAT study that some physicians questioned whether the high percentage of blacks in the study (35%) skewed the overall results against ACE inhibitors."

But the elaboration in this article acknowledges that there is something unsettling about the implication that expensive drugs are inappropriate for black people.³⁶⁰ Part of the skepticism of the cheap drug is related to its quality as a commodity, and the piece notes that diuretics cost just five to ten per cent what newer drugs do.³⁶¹ The piece suggests that because "...[b]ecause diuretics cost less and are much older than ACE inhibitors and calcium channel blockers, patients switched to diuretics from more high-profile drugs may think they are getting substandard care." Seeking to "dispel that notion" a Yale clinical professor of medicine is quoted saying:

"We aren't advocating good medicine for people who can afford it and bad medicine for people who can't," he said. "The data are clear: The less expensive medication is at least as good."³⁶²

This quote illustrates even as it contests slippage between exchange value and use value of the drug. These are two things that complicate the simple assessment of drugs' relative natural properties: the role of price in a commodity's fetish, and the difficulty in assessing the use value of drugs – especially for asymptomatic conditions.

First, drugs are subject to the aspect of commodity fetishism in which their different equivalences are imagined to be inherent to them. We might imagine that because drugs have scientifically demonstrated 'natural properties,' they are somehow exempt from the strange phenomenon of 'cool' that attends all commodities. Yet we

³⁶⁰ At a conference for the International Society for Hypertension in Blacks that I attended, an investigator on ALLHAT was criticized as biased on account of he was funded by the NIH, which already has an incentive to recommend cheap old drugs and perhaps can more easily get away with doing so on disadvantaged populations.

³⁶¹ The piece references prices at drugstore.com: "At drugstore.com, diuretics cost \$22 to \$36 per year, ACE inhibitors range from \$282 to \$532, and calcium channel blockers cost between \$584 and \$679 per year." Jason van Steenburgh, "Diuretics for Hypertension Get a Big Boost, But Will Data Change Prescribing Patterns?" *American College of Physicians Observer*, April 2003, available at <http://www.acponline.org/journals/news/apr03/diuretics.htm>.

³⁶² Jason van Steenburgh, "Diuretics for Hypertension Get a Big Boost, But Will Data Change Prescribing Patterns?" *American College of Physicians Observer*, April 2003, available at <http://www.acponline.org/journals/news/apr03/diuretics.htm>.

should remember that Marx's key example,³⁶³ a coat, has physical aspects too, and its value as something useful and its value for monetary trade are often easy to conflate. But if we do so, Marx argues that we are making an error: its ability to keep us warm is physical, but its price is purely social. When we say that random controlled trials show that generic drugs have "the same" efficacy as brand name new ones, we ignore the real and continuing power of the magical thinking of the commodity's fetish that can never quite divide exchange value from use value.

Second, the clarity of the data turn out to be tricky to establish. Unlike exchange value, use value is hard to pin down. Even determining what utility means is complicated for a drug used for an asymptomatic condition such as hypertension. When choosing a commodity like Marx's coat, we know that we choose not only on the basis of its temperature control. We might choose one that is class-marked, regionally-marked, racially-marked. We choose one that makes us "cool," or comfortable, or is associated with any combination of characteristics that transcend simple functional utility maximization. In an age of market-driven Pharma, we should attend to these diverse identity forms with regard to pharmaceuticals as well.

Moreover, we also choose a coat or a drug based not only on maximizing utilities but also, perhaps predominantly, on minimizing disutilities. Consider the coat/drug analogy a little further, to understand utility versus disutility. For example, one problem with coats is that they can cause sweating. We could consider the problem of sweating being caused by something that is designed to protect from cold to be analogous to a side effect of a drug. We might choose a coat then based not only on its ability to keep us

³⁶³ Karl Marx, *Capital: Volume 1*, B. Fowkes, trans., London: Penguin, 1990 pp. 125-163.

warm, but also on its ability to ventilate and prevent sweat: these effects need to be weighed against each other. Weighing utilities and disutilities of drugs that do not treat symptoms is analogous to but much more challenging than that involved in coat selection. Unlike the purchase of a coat – or drugs that have a transparent utility to the patient because they cure disease or treat symptoms – selection of antihypertensive drugs are from the patients’ perspective mostly about minimizing disutilities. The utility of something as far-off and abstract as a statistically significant reduction in cardiovascular events must be weighed against short and long term disutilities. While the short-term side effects of thiazides are generally mild, they can be important to patients’ experiences: hassle of taking a drug, some cost, increased urination (which should subside after the first few days but often is experienced as continuing), and more rarely impotence³⁶⁴ and gout. And, as we will now see, the long-term side effects are the subject of much debate on the differential value of thiazides versus the new expensive drugs. Establishing that the efficacy of thiazides is “the same” or “better” turns out to be a very complicated task, and has been widely challenged.

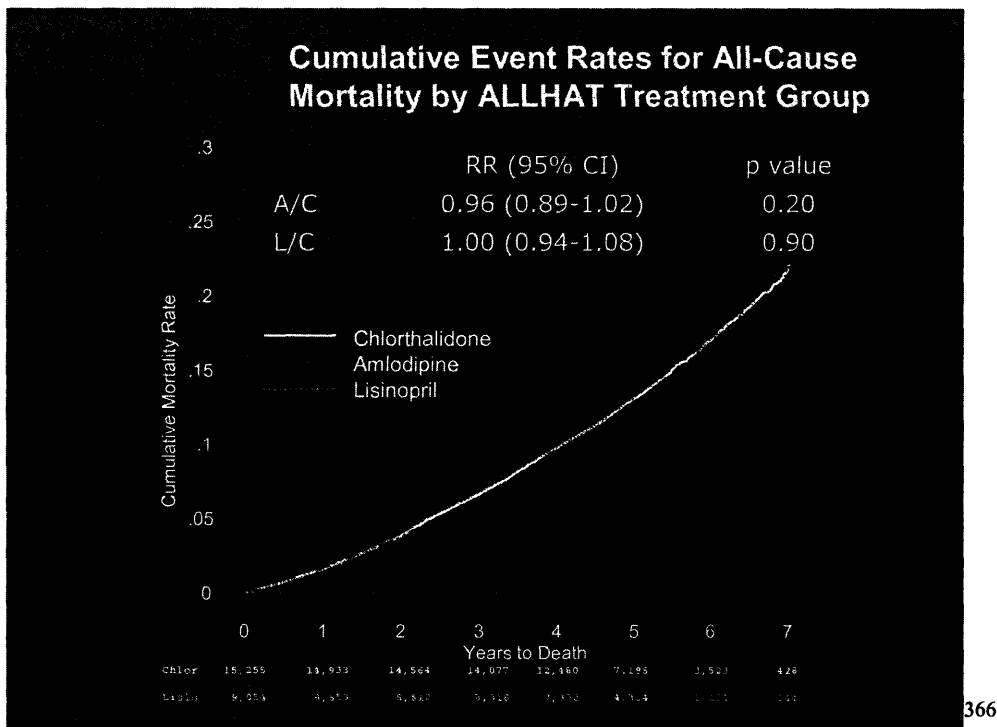
In 2006, there was a debate on the topic “Diuretics are over-emphasized as anti-hypertensive therapy” at the International Society for Hypertension in Blacks meeting.³⁶⁵ The one arguing for that case was University of Michigan professor Kenneth Jamerson, and the one arguing against it was Case Western professor and NHLBI researcher Jackson Wright. Both showed the same slides from ALLHAT, and both agreed that

³⁶⁴ Fascinating discussion of the role of racialized hypertension in the marketing of Viagra can be found in Laura Mamo and Jennifer R. Fishman’s “Potency in All the Right Places: Viagra as a Technology of the Gendered Body,” *Body and Society* 7 (2001): 13-55. They point out that it is not hypertension but the drugs that treat it that can cause impotence, and thus Viagra allows prescription drug use to promote more prescription drug use, which is important in expanding biomedicalization in such a pharmaceutical-saturated environment (p. 27).

³⁶⁵ International Society for Hypertension in Blacks Annual Meeting, June 23, 2006.

multi-drug therapy was ideal. However, they disagreed about whether the data suggested that thiazide was ‘best.’

Jamerson started out his presentation by emphasizing first that ALLHAT did not show that diuretics were ‘best.’ Showing a powerpoint slide from ALLHAT with the lines on a chart comparing rates of blood pressure reduction over time for each of the three drug classes tested head to head – in which the lines representing each drug almost completely overlap – he said with sarcastic derision that ‘clearly’ diuretics are best:



Since these lines were too similar to settle anything, Jamerson suggested that consideration should include something key that was missing from the slide: metabolic effects. Diuretics have low side-effect profiles, but that is not the same thing as having

³⁶⁶ ALLHAT slide deck, <http://allhat.uth.tmc.edu/Slides/Results.ppt#31>.

no side effect profile at all. From physicians' perspective, the biggest risk besides rare instances of gout is increased blood sugar levels.³⁶⁷ The risk of inducing diabetes is a serious one, since it is both a serious medical condition in its own right and a potent risk factor for cardiovascular disease. Averting this long-term risk, Jamerson suggested, was a reason to choose newer drugs even if they didn't lower blood pressure any more than diuretics or improve morbidity and mortality within the timeframe of the trial.

The con argument – that diuretics are not over-emphasized in treatment of hypertension – was made by ALLHAT investigator Jackson Wright. He suggested that Jamerson's concern was merely speculative while the data was definitive. He showed data that although there was an increase in blood sugar of those on diuretics as expected, there was not an increase in adverse events. At stake here is which kind of counting is more compelling: that of blood sugar levels or that of strokes, heart failures, heart attacks. If it seems clear to Wright that the latter are obviously more compelling, their conclusiveness is diminished by the time limit of the trial. Wright had no answer for Jamerson's central point: What metabolic dangers lurk in the timeframe beyond the trial's scope? For his part, Jamerson could not quantify those, either.

Wright blamed physicians' lack of enthusiasm for diuretics – despite what he saw as ALLHAT's conclusive evidence – on diuretics' lack of pharmaceutical marketing, that no pharmaceutical representative ever took him out to dinner to promote these old cheap generics. He was frustrated that although ALLHAT was conclusive in the safety and efficacy of diuretics, its results have not been widely followed. Far from

³⁶⁷ The risk of new-onset diabetes on diuretics has been the subject of much literature in the vein of redeeming the new expensive drugs in the wake of ALLHAT. See for example, Katsuyuki Ando and Toshiro Fujita, "Anti-diabetic effect of blockade of the renin-angiotensin system," *Diabetes, Obesity and Metabolism* 8 (July 2006): 396.

overemphasizing diuretics, Wright said, physicians have continued their prescription practices of focusing on the expensive drugs. He made numerous references to his lack of reliance on pharmaceutical marketing, but how could an audience filled to the brim with pharmaceutical marketing take this in?

In this debate, there is a peculiar invocation of narrow economic interests that is rare from the podiums of medical conferences. Pharmaceutical companies' support is always recognized by name before presenting data, yet there is rarely the sort of base accusation of bias that attends government support of ALLHAT. When dinners are provided by pharmaceutical companies at conferences or outside them, there is of course a sense that the companies are doing this in the hope of persuading physicians to prescribe their wares. But there is an understanding that the speakers are not merely representing the pharmaceutical companies. The system of continuing medical education of which this conference is part operates on the assumption that sponsorship and science are separable. The knowledge being presented – while paid for – is not simply bought. When ALLHAT's investigator Jackson Wright suggests base economic motives of his opponents, he rankles the sensibilities of the many physicians, who accept gifts from the pharmaceutical companies without thinking that it affects their own professional judgment. But neither the government nor pharmaceutical companies are disinterested parties in this debate. There is no innocent place from which to judge.

Indeed, there has been ample criticism of the governmental endorsement of the old cheap drugs. In his scathing indictment of the design of ALLHAT, its application beyond its primary endpoints, and the hastiness of its peer review, Jay Meltzer argued in the *American Journal of Hypertension* that contrary to what it deserves "ALLHAT has

had widespread acclaim in part due to a massive government sponsored public relations campaign.”³⁶⁸ Part of the disconnect between Meltzer and Wright is in the uncharted space Meltzer identifies as “public relations” and that we might situate between “marketing” and “publicity.”³⁶⁹ Like punditry, publicity both has “two sides” and conceals considerable differences between the interests of the sides.

Getting at the pure biomedical knowledge amid competing biases of government and pharmaceutical companies turns out to be a goal of considerable difficulty, but nevertheless the stakes are high in continuing the pursuit. At stake is the integrity of the science and art of medicine. Although he was writing his history of hypertension before the ALLHAT results, the comments of Nicolas Postal-Vinay on treatment options are illuminating. He challenges the recommendation of diuretics first because of their cost under the heading of “hypertension and politics:” Complaining about the fifth Joint National Committee recommendation that cheaper agents be used before new expensive ones unless the latter can be shown to be advantageous, he quotes physician Menard: “*The recommendation to prescribe the cheapest drug is a denial of both scientific practice and medical art.*”³⁷⁰

Nostalgia for the artistry of medicine is resonant with that for pre-capitalist modes of production, and this in turn raises a question about whether generics, stripped as they

³⁶⁸ Jay I. Meltzer, “A specialist in clinical hypertension critiques ALLHAT,” *American Journal of Hypertension*, 16 (May 2003): 416-420.

³⁶⁹ For explication of the various forms of public relations including these, see Kevin Moloney, *Rethinking Public Relations: PR, Propaganda, and Democracy* (New York: Routledge, 2000).

³⁷⁰ “Indeed, there is evidence to suggest that angiotensin converting enzyme inhibitors do have advantages, not only in terms of patient acceptability and quality of life, but probably also in terms of cardiac, vascular, and renal protection. Will we have to deny patients these benefits for years while we wait for the results of hypothetical mortality studies? Are such recommendations not in contrast to the prospects for “personalizing treatment?”” Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 164.

are of authorship and innovation, can play a role in art. But there is something even more peculiar about the ways in which art versus lowest-common-denominator science are played off each other here:

In the midst of this debate, how can general practitioners, the main prescribers of antihypertensive drugs, find their way? They are trapped between the pressure of pharmaceutical advertising which constantly reminds them that increasingly effective drugs are available, and official bodies which encourage them to be more economical in their prescribing. Both parties refer to the conclusions of their 'experts' which overwhelm the modest practitioners trying to do their best for their patients. The confusion of doctors faced with these recent developments is a totally new facet of the impact of contemporary society on hypertension and its treatment.³⁷¹

In this quote, pharmaceutical companies are read as telling the truth about the newest things being best, but alas the government steps in and pressures doctors to consider price as well. This reversed-suspicion (when compared to that of mass media, more skeptical of pharmaceutical companies than government guidelines) is one that we see in Meltzer's reference to PR above, and something that I have heard often among physicians at conferences. The coincidence of the cheap drug and the drug for blacks is thus particularly irksome for those like Jamerson who support blacks' integration into the science and art of cutting-edge medicine.

Certainly, the government is hardly a disinterested player in the treatments given to patients. But in a context otherwise saturated with privately-funded drug research, the character of the skepticism of ALLHAT is conspicuous. Even if we accept ALLHAT's basic assessment of the identical efficacy of cheap old thiazide to the new expensive drugs, commodity arguments don't rest there—even if now and in this one case, the NIH is correct in recommending the cheap old drug first. Many clinicians are not content to

³⁷¹ Nicholas Postel-Vinay, *A Century of Arterial Hypertension: 1896-1996* (West Sussex, England: Wiley, 1996), 164-5.

celebrate the efficacy of a cheap old drug on its own, and have come to emphasize the aspect of ALLHAT that illustrates the importance of *combining* a diuretic with a new expensive drug in a multidrug “gold standard” treatment regimen.³⁷²

Are diuretics particularly good for blacks?

In the reports about ALLHAT, there has been a repetitive promotion of diuretics not only but especially for blacks: “While the improved outcomes with [the thiazide] chlorthalidone were more pronounced for some outcomes in blacks than in nonblacks, thiazide-type diuretics remain the drugs of choice for initial therapy of hypertension in both black and nonblack hypertensive patients.”³⁷³ This aspect makes it a particularly salient issue to debate at the International Society for Hypertension in Blacks as described above, but also in considering the role of race in providing evidence based care.

³⁷² The NHLBI guidelines for hypertension in minorities are a hodgepodge: “BP control rates vary in minority populations and are lowest in Mexican Americans and Native Americans. In general, the treatment of hypertension is similar for all demographic groups, but socioeconomic factors and lifestyle may be important barriers to BP control in some minority patients. The prevalence, severity, and impact of hypertension are increased in African Americans, who also demonstrate somewhat reduced BP responses to monotherapy with BBs, ACEIs, or ARBs compared to diuretics or CCBs. These differential responses are largely eliminated by drug combinations that include adequate doses of a diuretic. ACEI-induced angioedema occurs 2–4 times more frequently in African American patients with hypertension than in other groups.” National High Blood Pressure Education Program, *The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7)*, December, 2003, <http://www.nhlbi.nih.gov/guidelines/hypertension/express.pdf>. ISHIB’s full guidelines also recommend diet and lifestyle changes, as well as the inclusion of every class of drug in the treatment of African Americans not only for blood pressure lowering but also protection from end-organ damage. Douglas et al, “Management of High Blood Pressure in African Americans: Consensus Statement of the Hypertension in African Americans Working Group of the International Society of Hypertension in Blacks,” *Archives of Internal Medicine* 163 (Mar 10, 2003), 525-541, available at http://ishib.org/supportfiles/Mgt_of_Hypertension_in_African_Americans.pdf.

³⁷³ Jackson T. Wright, Jr, MD, PhD; J. Kay Dunn, PhD; Jeffrey A. Cutler, MD; Barry R. Davis, MD, PhD; William C. Cushman, MD; Charles E. Ford, PhD; L. Julian Haywood, MD; Frans H. H. Leenen, MD, PhD; Karen L. Margolis, MD, MPH; Vasilios Papademetriou, MD; Jeffrey L. Probstfield, MD; Paul K. Whelton, MD; Gabriel B. Habib, MD; for the ALLHAT Collaborative Research Group, “Outcomes in Hypertensive Black and Nonblack Patients Treated With Chlorthalidone, Amlodipine, and Lisinopril,” *JAMA* 293 (April 6, 2005): 1595-1608.

ALLHAT is a study that was remarkably inclusive of African Americans, and so we should not be surprised that the data has been broken down to draw contrasts between racial groups. Its data has been invoked in physicians' arguments for using race to determine which treatments to select, in turn spurring critique of simplistic racial profiling in health care.

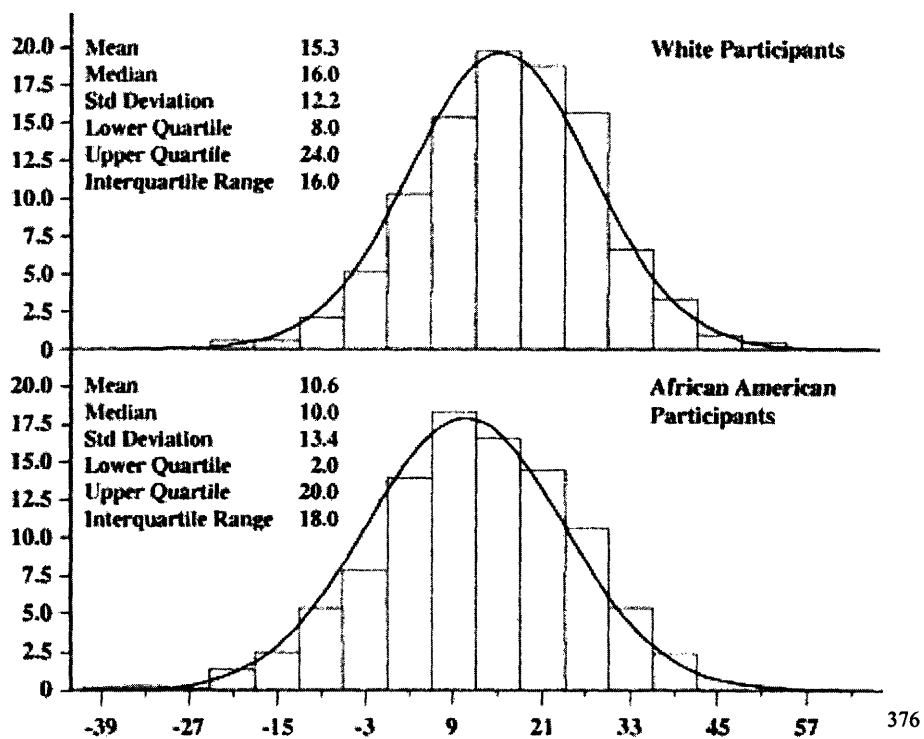
As mentioned above, the American College of Physicians suggests that blacks are particularly good candidates for diuretics. Yet, they go further, suggesting that such a high rate of inclusion of blacks may throw off statistics for efficacy of drugs overall, in ways that resonate with the questions raised in Chapter 3 about who can stand in as normal America: "Even more significantly, the study found that black Americans taking ACE inhibitors experienced a stroke 40% more often than their counterparts taking diuretics. (Black Americans typically require high levels of ACE inhibitors to control their blood pressure.) Black Americans responded so well to diuretics in the ALLHAT study that some physicians questioned whether the high percentage of blacks in the study (35%) skewed the overall results against ACE inhibitors."³⁷⁴ Yet is race a good basis upon which to choose which class of drug to prescribe?

If it turns out not to be the case that African Americans are uniquely good candidates for diuretics, that could call into question the category of African American Hypertension as a disease category, because African American Hypertension has been defined by its salt-sensitive receptivity to diuretics. Mokwe et. al. observe that "Race has been considered an important factor in determining blood pressure response to treatment

³⁷⁴ Jason van Steenburgh, "Diuretics for Hypertension Get a Big Boost, But Will Data Change Prescribing Patterns?" *American College of Physicians Observer*, April 2003, available at <http://www.acponline.org/journals/news/apr03/diuretics.htm>.

and selection of antihypertensive drug therapy.”³⁷⁵ However, they provide data from a trial of an expensive class of drugs often described as ineffective in blacks (ACE inhibitors) that shows that intra-racial variation is much larger than inter-racial variation, rendering race a very poor indicator of drug response.

As we can see in Mokwe et, al.’s charts, there was a slight difference in blood pressure response in whites compared to blacks, but “the response distributions largely overlapped.”



³⁷⁵ Evan Mokwe; Suzanne E. Ohmit; Samar A. Nasser; Tariq Shafi; Elijah Saunders; Errol Crook; Amanda Dudley; John M. Flack, “Determinants of Blood Pressure Response to Quinapril in Black and White Hypertensive Patients,” *Hypertension* 43 (2004): 1202.

³⁷⁶ Evan Mokwe; Suzanne E. Ohmit; Samar A. Nasser; Tariq Shafi; Elijah Saunders; Errol Crook; Amanda Dudley; John M. Flack, “Determinants of Blood Pressure Response to Quinapril in Black and White Hypertensive Patients,” *Hypertension*, 43 (2004): 1202.

For Mokwe et al, this data provides compelling evidence that race should not be used as a guideline of which drug to prescribe. “In these models, participant characteristics, including age, gender, body size, and pretreatment blood pressure severity, significantly predicted either attenuated or enhanced blood pressure response to treatment. Our findings demonstrate that a large source of variability of blood pressure response to treatment is within, not between, racial groups, and that factors that vary at the level of the individual contribute to apparent racial differences in response to treatment.” Since the patient white or black before the physician might fall anywhere on their respective curves, small mean differences are of paltry clinical value.

And yet these bell-curves as evidence of similarity rather than dissimilarity are not necessarily convincing given the dominant scientific and practical bases of decision making. That is, the epistemological framework of evidence based medicine is extraordinarily focused on statistically significant difference, and at the same time clinicians are pragmatically oriented toward clinical rules of thumb that can be applied to aspects of patients that seem most readily observable.³⁷⁷ The difference between the bell curves is slight, and means that which curve you are on matters less than where you are on either curve. But it is difficult for many clinicians to accept that that means that the difference should be ignored.

Materson, a proponent of considering race when deciding which drug to prescribe wrote an editorial response in the same journal in which Mokwe et al presented their

³⁷⁷ As we saw in the previous chapter, the relevant aspects of patients need not be measurable, they need only be recordable, and race is particularly robust as a factor upon which to base treatment guidelines.

data.³⁷⁸ He acknowledged that there are not accurate means of “identifying the individuals who comprise these group response distribution curves who respond quite well to drugs that interfere with the renin-angiotensin-aldosterone system.” And yet, he justifies race-as-proxy even as he holds out the hope of progress in the future: “Pharmacogenomics offers that promise, but if and when that will be realized is unknown. In the meantime, the age by race construct is simple, cheap, and reasonably accurate for those patients with stage 1 hypertension who will receive a single antihypertensive drug.” He posits race as a proxy of a proxy.

Materson acknowledges caveats that seem to bolster case that Mokwe et al are making against knee-jerk diuretic monotherapy for African Americans: “If more than one drug is used, as is now so often recommended given lower goal blood pressures, the issue becomes irrelevant because the racial difference disappears. Under no circumstances should a patient be denied a drug on the basis of race for conditions concomitant with hypertension such as diabetes or chronic kidney disease.”

And yet, Materson does not want to give up that dear proxy. Curiously, given that Materson is editorializing while Mokwe et al are providing data, Materson accuses those who would abandon the use of race as a guide to first antihypertensive drug as “idealistic”:

In summary, Mokwe et al have provided a clear demonstration of the variability of blood pressure response to single-drug therapy, have confirmed the previously known group differences in response, and have reiterated that race alone is not sufficient to predict response to monotherapy. That is the idealistic position. The pragmatic point is that the clinical practitioner still must be aware that in contrast to the uncertainty of random initial drug selection, the age by race construct is a

³⁷⁸ Barry J. Materson, “Variability in Response to Anti-hypertensive Drug Treatment,” *Hypertension*.43 (2004):1166.

simple, cost-free, and relatively accurate way of guiding the choice of single-drug therapy for patients with stage 1 hypertension.

This declaration of the “pragmatism” of race-based treatment design in the face of evidence that race is not a successful way to predict which treatment will work is an oddly defined pragmatism. It is efficient for the doctors to sort patients in this way, but the unrefuted evidence suggests that it does not actually help in sorting patients into the appropriate drug classes for them. Thus, it is the racially profiling position that might be more accurately characterized as the “idealistic” position.³⁷⁹ If such editorials decry evidence-based arguments as “idealistic,” we should come to realize that in this intra-medical argument, as in the extra-medical conversation at the Colloquium that opened this chapter, the argument does not only rest on the evidence.

Nevertheless, the drive to present evidence against the practical value of racial profiling in medicine continues. In a tactic that complements that of Mokwe et al, nephrologist Ashwini Sehgal has done a meta-analysis of differential drug response to emphasize not the divergence but the *overlap* in drug response between races.³⁸⁰ Sehgal questions the practical value of the tiny “average” differences in drug response by race often highlighted in reviews and practice guidelines because they are dwarfed by overwhelming overlap. Rather than asking how the races differ in response, he looks at how often they respond the same.

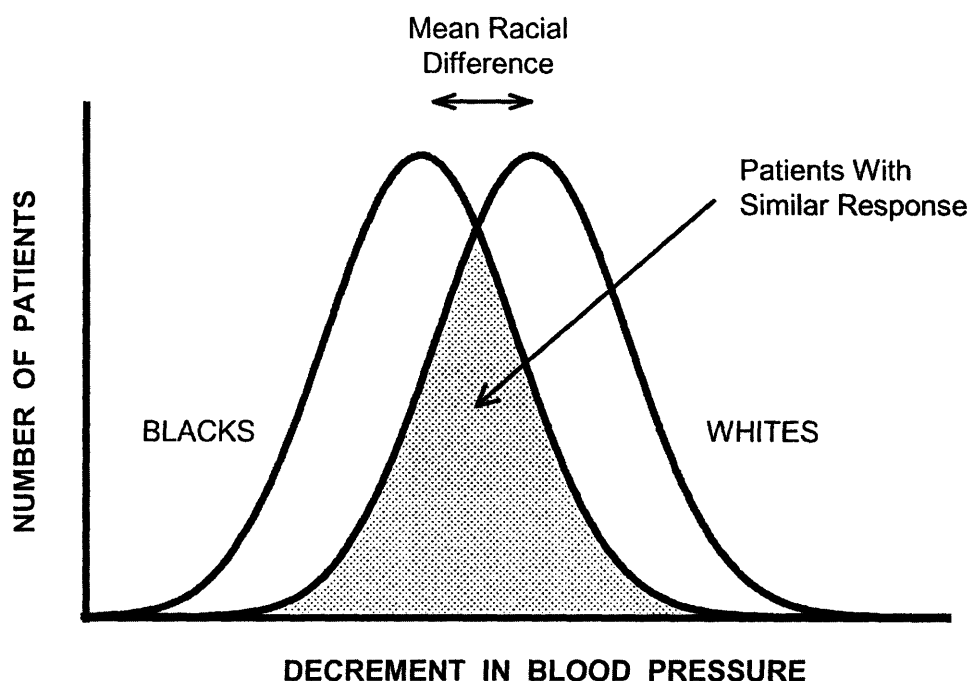
The percentage of whites and blacks with similar drug-associated changes in diastolic blood pressure was 90% for diuretics, 90% for β -blockers, 95% for

³⁷⁹ This move is resonant with many of the return-to-race ideologues, such as the *New York Times* editorial by Armand Marie Leroi that spurred the SSRC forum “Is Race Real?”, <http://raceandgenomics.ssrc.org/>

³⁸⁰ Ashwini R. Sehgal, “Overlap Between Whites and Blacks in Response to Antihypertensive Drugs,” *Hypertension* 43 (2004): 566-572.

calcium channel blockers, and 81% for angiotensin converting enzyme inhibitors....In conclusion, the majority of whites and blacks have similar responses to commonly used antihypertensive drugs. Clinical decisions to use a specific drug should be based on other considerations such as efficacy in individual patients, compelling indications, and cost.

It is another instantiation of the bell curve as a device for illustrating similarity, which has counterpoints with the overtly racist *Bell Curve* and raises questions about how to consider the individual before the bell curve, Sehgal provides this chart:

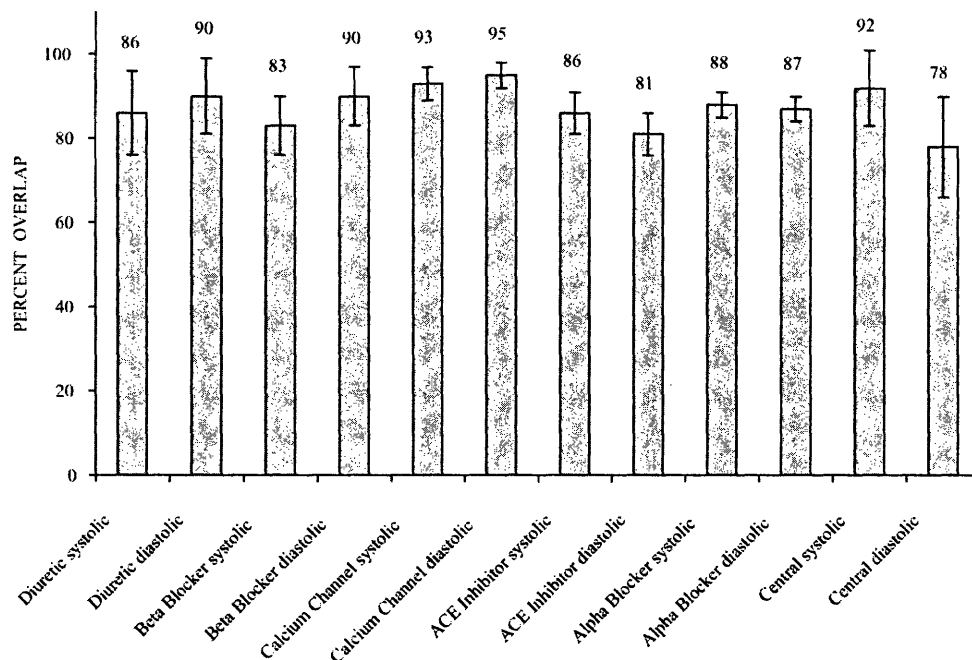


This chart actually drastically under-represent the level of overlap, since much less than 90% of the area looks shaded.³⁸¹ Yet the representational mode of comparative bell curves poses an epistemological quandary: how to approach a glass 90% full. The box-plot graphs more commonly used in the presentation of racially differentiated group

³⁸¹ Thus this is an error in visualization in Tufte's terms: E. R. Tufte, *Visual and Statistical Thinking: Displays of Evidence for Decision Making*. Cheshire, CT: Graphics Press, 1997.

response put all the focus on the difference in means, obscuring similarities within groups. But even comparative bell curves leave the question open: given that not all the area is shaded common ground, how shall we consider the separation of the curves' peaks in practice?

Sehgal's other graph seems to better represent how much inter-group similarity overwhelms difference in response to treatments:



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Sehgal argues that what is at stake is not only the selection of drugs for hypertension, but also the role of racial differences in racial treatment guidelines. He suggests a method for deciding whether race is a good way to choose drug treatments, driven by two questions: “(1) what are the most important genetic and environmental determinants of drug response and (2) is race a good proxy for such genetic and environmental determinants?”

³⁸² Ashwini R. Sehgal, “Overlap Between Whites and Blacks in Response to Antihypertensive Drugs,” *Hypertension* 2004 43: 566-572.

According to Sehgal, “If race is a good proxy for relevant genetic and/or environmental determinants of drug response, then clinicians should consider race in selecting drugs for individual patients. However, this meta-analysis found that race has little value in predicting antihypertensive drug response, because whites and blacks overlap greatly in their response to all categories of drugs.” There are potentially infinite ways that differentiated bell curves of drug response could be drawn: by insurance status, by region of the country, or by any clinical attribute. Sehgal is making a call to search for something more predictive than race, rather than putting so much weight on differences in a small minority of responses.

In suggesting the implications of this finding, Sehgal makes a move characteristic of the anti-racist tactics we have seen in the previous chapter on African American Hypertension as a disease category and will return to in the next chapter on BiDiI: reiterating that race is not a genetic category. He writes: “These findings are consistent with other work demonstrating that most genetic diversity exists within and not between races and that race is a poor predictor of drug-metabolizing enzymes (which in turn influence drug response).” This is striking given that he has left open that the environment as well as genes might affect drug response. Sehgal’s conclusions, by tying back only to the genetic rather than to the multiple levels of the social, are more modest than they should be. The work done in dividing out effects of genetics and environment in determining drug response will be returned to in the next chapter.

The durability of race in this debate over antihypertensive classes suggests that the data itself cannot close the discussion. Thiazide increases contradictions, and preoccupation with fetish of race intrudes on cold data. As noted in Chapter 2, on

Framingham, the sorts of arguments about intra-group difference versus inter-group difference were effective there in closing comparisons between categories Italians and Irish, foreign born versus native born. That they are unable to do so with regard to racial response suggests that the discursive strategy itself is neither flawed nor effective, but simply incommensurate with the levels at which race is durable in clinical practice.

In these debates about whether diuretics are best overall or best for blacks, we can see that the debate within medicine is not settled even for this old generic drug class. Moreover, there is no consensus on the grounds on which arguments could be refuted. Questions keep emerging from within ALLHAT and from without, and from opposing readings of the clinical relevance of small average racial differences. Instability in the medical meaning of a drug does not result, then, from lack of evidence or from its novelty, but in epistemological and practical invocations of it on diverse registers.

Conclusions: Thiazide as terrain for participation in American ways of life

Leaving aside the messiness of the clinical data here, I'll close with some points about how in both of these contexts – the African American Studies Colloquium and in clinical medicine – and others, championing thiazide has been an appealing story for an array of actors. Each of them can be read as using thiazide to frame participation in American ways of life. For example, Levy and Brink write in their history of the Framingham Study that:

Today, there are dozens of drugs available to treat high blood pressure, and eleven new ones are being developed, according to the Pharmaceutical Research and

Manufacturers of America. But, as the saying goes, what goes around, comes around. Despite the many new drugs available, and \$15.5 billion spent annually on medications to lower high blood pressure, the ALLHAT study proved that diuretics, the drug class that was the cornerstone of Freis's clinical trial nearly four decades ago, were superior in blood pressure control and in preventing some of the most serious adverse consequences of cardiovascular disease than newer and much more costly medications. Using thiazide diuretics in the treatment of the 50 million people with hypertension in the United States would save billions of dollars each year. That money could be better spent on further research into the causes of heart disease and stroke, or applied to additional clinical steps to prevent heart disease in those at greatest risk.³⁸³

The optimism here in the affordable quick fix thiazide could provide is resonant with Fryer's optimism that opened this chapter, as well as the 1970s and 1980s mobilization around African American Hypertension as a disease category that was described in the previous chapter. Whether thiazide is the boon for affordable disease prevention for all Americans or a boon for an available means to help African Americans catch up demographically, promoting it can be part of a democratic impulse.³⁸⁴ Promoting access to diuretics does more than advocate an even more complete saturation of American publics with drugs. Arguments about thiazide can be understood as framing that participation in American pharmaceutical grammars. If, as I have argued throughout this dissertation, participation in cardiovascular knowledge and pharmaceutical practice has become a mode of articulating citizenship demands, framings of these pharmaceutical practices as either cutting edge or accessible to all, differentiated or universal, become part of articulating participation in American ways of life.

³⁸³ Levy and Brink, *A Change of Heart: How the People of Framingham, Massachusetts, Helped Unravel the Mysteries of Cardiovascular Disease*, p. 148.

³⁸⁴ As David Jones points out (personal communication) the "quick fix" offered by thiazide brings, like other drugs as quick fixes, attendant anxieties. Critiques of drugs in psychiatry as well as cardiology include that they are quick fixes for social problems, paving over depression, trauma, bad diets, lack of exercise, etc. Drugs as quick fixes for health disparities. Drugs as quick fixes for identity, etc. Insofar as they are effective, there is a fear that they will replace other modes of answering these questions, that would be better more moral ways to solve the problems.

Thiazide's historian, Jeremy Greene, has written the history of thiazide as tragedy, ending his narration at a historical moment in which thiazide was outshined by newer drugs even as ALLHAT "proved" it was superior. Greene's fascinating history describes how when the first thiazide, Diuril, was launched as a brand-name revolutionary drug in 1958, it was a triumphant interplay of marketing and research; in fact the first 'blockbuster' drug.³⁸⁵ Its low toxicity profile made it possible to expand the definition of hypertension at a moment in which it was becoming an identifiable 'prepathological' state of risk for future cardiovascular disease. After a history that demonstrates the fluid boundaries between the knowledge of thiazide and of disease, Greene is suddenly declarative, as if the "real" value of diuretics can now be ascertained:

Ironically, the recent publication of the National Institutes of Health's Antihypertensive and Lipid Lowering Therapy to Reduce Heart Attacks Trial (ALLHAT) places us at a moment when thiazide diuretics have recently been demonstrated to be the most efficacious and appropriate first-line antihypertensive therapy for most patients, though their actual prescription rates now lag far behind those of newer, more heavily advertised drugs.³⁸⁶

Greene is more pessimistic about thiazide's future than Fryer or Levy. Writing as if thiazide's story is at its end, Greene laments that the blockbuster drug model thiazide ushered in became part of its ultimate decline, as ever newer blockbuster drugs come to dominate the moment. Greene writes that: "examination of Diuril's career, then, leaves us with some unsatisfying ambivalence toward the drug-promotion process: while we might applaud the fact that Diuril was launched into the world so effectively, it is clear that the same efficient machine of promotion was instrumental in the subsequent decline

³⁸⁵ Jeremy A. Greene, "Releasing the Flood Waters: Diuril and the Reshaping of Hypertension," *Bulletin of the History of Medicine* 79 (Winter 2005): 749-794.

³⁸⁶ Jeremy A. Greene, "Releasing the Flood Waters: Diuril and the Reshaping of Hypertension," *Bulletin of the History of Medicine* 79 (Winter 2005): 791-792.

and neglect of the thiazide diuretics once they ceased to be a financial priority for the industry. Grasping this irony is essential to understanding the dual nature of pharmaceutical promotion as a process rooted in both education and salesmanship, a process that is now fundamental to the circulation of the knowledge and changing practice of American medicine.”³⁸⁷ And yet by both opening the black box around ALLHAT and by attending to invocations of thiazide outside medicine, we can see that its story is not yet over.

Back when thiazide was brand-name cutting edge Diuril, as Greene has described, it was enrolled in tropes of greatness and revolutionary medicine. It is now enrolled in tropes of mundane mediocrity and stalwartness, aspects I have suggested are inseparable from its familiarity and decreasing equivalent value. As it lost its patent protection and its novelty, it has lost its edge. But, as we will see in the next chapter, on BiDil, both novelty and stalwartness have their positive and negative valences. At stake is a tradeoff between risk and reliability, between being in on “the latest” and being at the mercy of fads. Drugs, like fashion, are amenable to retro-chic. When Fryer and Gates style thiazide’s meaning in markedly different ways from those that its marketers celebrated early on, or those of the present experts in ALLHAT, they are participating in the meaning-making of pharmaceuticals that exceeds that of marketers or medical experts.

The efficacy of thiazide as demonstrated in ALLHAT seems to provide a refreshing counterpoint to pharmaceutical narratives focused on the private, new, and

³⁸⁷ Jeremy A. Greene, “Releasing the Flood Waters: Diuril and the Reshaping of Hypertension,” *Bulletin of the History of Medicine* 79 (Winter 2005): 792.

expensive.³⁸⁸ It has been especially appealing for those interested in affordable health care for all and who believe that the government has a role in providing it. Promoting thiazide can be part of an egalitarian impulse. Thiazide's status as generic gives it a different valence than other drugs, and promoting it makes two moves. On the one hand, endorsing thiazide advocates an even more complete saturation of American publics with drugs. On the other, it frames participation in American pharmaceutical grammars in some ways rather than others.

If, as I have argued throughout my dissertation, participation in cardiovascular disease knowledge and pharmaceutical practice has become part of articulating inclusion in American ways of life, we can read differing pharmaceutical grammars as differing articulations of that participation: either as cutting edge or accessible to all, differentiated or universal, within the reach of present capabilities or emerging only at some future point once the science or struggle for social justice has proceeded further.

In this sense, we can see that the biological citizenship theorized by (among others) Rose and Novas is not necessarily characteristic only of genomic technologies. Though they talk about "contemporary biological citizenship" in terms of "genetic prudence" in managing an as-yet scantily modifiable genome, the phenomenon they

³⁸⁸ This makes thiazide very different from the pharmacogenomic drugs that Anne Fausto-Sterling, among others, have criticized as being a diversion from focus on the possibilities of social progress against the effects of racism on disease states such as African American hypertension. Thiazide fits more into the model of "public health" that she champions rather than pharmacogenomics she criticizes when she writes: "If ... we already know how to solve a lot of the public health problems before us, including those that exact an excess toll from various minority communities, then why aren't we just doing it? And why are geneticists spending so much time on huge, multinational snp and haplotype mapping projects, promising that their new definitions of race will help members of the newly defined racial groupings to improve their health? An analysis of our health care system's love of high-tech solutions for low-tech problems, of our national unwillingness to fund and carry out well designed public health measures, and of the force of the pharmaceutical industry as it drives for pill-based solutions to socially produced ills is much more than I can accomplish in this paper. Not here, not now, but . . ." Anne Fausto-Sterling, "Refashioning Race: DNA and the Politics of Health Care," *Differences: A Journal of Feminist Cultural Studies*, 15 (2004): 30.

describe is already happening in relationship to pharmaceuticals, including thiazide. Thiazide operates in the same “political economy of hope” as genomic technologies Rose and Novas describe, in which “Biology is no longer blind destiny, or even foreseen but implacable fate. It is knowable, mutable, improvable, eminently manipulable.”³⁸⁹ But yet the hope is, depending on ones point of view, either insufficient or tantalizingly (if frustratingly) right here right now.

The increasing prominence of pharmaceuticals has been part of a change in the center of gravity of medicine as a field. There has been a move away from the centrality of the unmasked human labor of the physician, toward a medical model centered on objects whose social relations are more hidden. As Greenslit has pointed out, this is illustrated even by the definition of the word “medicine:” for most people, the first definition of the word would be not a field of social relations with physicians and others but rather a commodity, a pharmaceutical.³⁹⁰ And it is into this terrain of medicine – a field of social relations that comes to be understood outside of social relations – that I see both this encounter at the DuBois Institute colloquium and the medical debates about thiazide. Interrogation of generics shows that our analysis of pharmaceuticals must

³⁸⁹ Nikolas Rose and Carlos Novas, “Biological Citizenship,” for Aihwa Ong and Stephen Collier, eds., *Global Anthropology*, Blackwell, 2003. Accessed at: <http://www.lse.ac.uk/collections/sociology/pdf/RoseandNovasBiologicalCitizenship2002.pdf>, p. 5. They elaborate: “Of course, on the other side of hope is undoubtedly anxiety, fear, even dread at what one’s biological future, or those that one cares for, might hold. But whilst this may engender despair or fortitude, it also frequently generates a moral economy of hope, in which ignorance, resignation, and hopelessness in the face of the future is deprecated.” They conclude: We have argued that, while citizenship has long had a biological dimension, a new kind of biological citizenship is taking shape in the age of rapid biological discovery, genomics, biotechnological fabrication and biomedicine. New subjectivities, new politics and new ethics are shaping today’s biological citizens. As aspects of life once placed on the side of fate become subjects of deliberation and decision, a new space of hope and fear is being established around genetic and somatic individuality. In the nations of the West – Europe, Australia and the United States, this is not taking the form of fatalism and passivity, and nor are we seeing a revival of genetic or biological determinism.”

³⁹⁰ Nathan Greenslit, unpublished material shared in personal communication, November 8, 2004.

attend not only to postwar innovations in marketing or their elaboration at the turn-of-the-21st-century, but also to age old commodity fetishisms. This is an important foundation from which to go on to the next chapter, on the more blatantly racialized and aggressively marketed drug BiDil. Interrogating thiazide's intersections with race shows that to understand durable preoccupations with race and biology, we must attend not only to genetics but also into the pharmaceutical interventions into biology.

Through tracking conversations in thiazide's middle age, I hope that I have shown that no amount of clinical data has been able to settle either the drug itself nor its relationships with race. I suggest, further, that data cannot do so. Meanings of both thiazide and race also draw from beyond the clinic as thiazide becomes a site of exchange among pharmaceutical-centered medicine, NIH politics, and emerging rearticulations of black identities.³⁹¹ Through analysis of both a vignette among Harvard professors and plural medical debates in which thiazide is invoked, I have begun to show how the meanings of both this generic old drug and of race are multiple and still in motion.

³⁹¹ In this, I am inspired by Keith Wailoo has described sickle cell as a commodity, exchanged through the 20th century between three groups of stakeholders: networks of emerging molecular medicine in the New South, political liberalism, and articulations of civil rights and black identities. Keith Wailoo, *Dying in the City of the Blues: Sickle Cell Anemia and the Politics of Race and Health* (Chapel Hill: University of North Carolina Press, 2001). There are at least two key differences between sickle cell and HTN/thiazide. First, the possibility of treatment reduces stigma. Second, these thiazide stakeholders are not yet fully in dialogue with each other, but as Fryer's piece is published in a medical journal shortly, it will be a fascinating debate to continue to track.

Chapter 6

BiDil: Medi©ating the Intersection of Race and Heart Failure

Vignette

The scene was a two-day conference on “Race, Pharmaceuticals and Medical Technology,” put on by the Center for the Study of Diversity in Science, Technology and Medicine, that I had been part of organizing at MIT.³⁹² The conference was just months after the launch of BiDil, a combination of two generic drugs that was the first pill to be FDA-approved with a racial indication – for “heart failure in blacks” – and had spurred considerable interest among social scientists and historians of race.

African-American studies scholar Dorothy Roberts had just presented her paper on the diverse responses of African Americans to racial therapeutics such as BiDil. She explicated ways in which both conservative colorblindness and identity politics can lend support to racial therapeutics, while anti-race absolutism is less inclined to do so.

³⁹² “Race, Pharmaceuticals, and Medical Technology,” Massachusetts Institute of Technology Center for the Study of Diversity in Science, Technology and Medicine, April 7-8, 2006.

After the talk, one questioner stood up. He identified himself as Juan Cofield, the president of the NAACP New England Area Conference, and then declared: “There is consensus in the black community that this drug is good for black people.” Roberts, after a beat, said: “There isn’t consensus among the black people *in this room*.”

It was an exchange that would resonate through the rest of the conference. It would seem in a formal logical sense that the disagreement among the African Americans who spoke would be enough to prove the lack of African American consensus. But, as the chair of the sessions, David Jones, would note in his closing remarks, it turned out to be more complicated than that.

Cofield’s intervention provides an encapsulated opportunity to consider a few common rhetorical moves in the BiDil debates. First, he represented his moral stakes as exceeding those of himself as an individual conference participant. This rendered his intervention both more important and more explosive. In this move, he claimed an authority that both included (some of the) conference participants and exceeded them, opening questions of accountability both of himself and of us all. Second, his intervention denied the polyvalence of BiDil. Cofield’s framing of BiDil as a pure salve spoke on a different register from the suggestion made by many of the conference participants that the drug’s race-specific approval and marketing had racist potential. Third, he moved – unsuccessfully – to close the debate.

Claiming consensus within or without medicine is never a practice of observation, but always a rhetorical strategy. If there were consensus, the tense conference would have been a bland affair. As theorist Chantal Mouffe highlights in the politics of deconstruction, it is precisely the impossibility of consensus that renders democratic

debate lively.³⁹³ The chaos that underlies a provisional stabilization of consensus provides, in Derrida's terms, both a risk and a chance.³⁹⁴ Cofield was responding to the risk: he was worried that any doubts about BiDil might hinder its distribution. But there was also a chance here in this debate, to get specific about questions of *cui bono*, make interlocutors' stakes in debates in race and medicine more explicit, and come to more democratic deliberation.

Underlying both BiDil and race, there are sets of what Derrida would characterize as undecidables, most pressingly: is this drug fundamentally beneficent or maleficent? Cofield wants to shove that undecidability to the side, render it secondary to the consensus about its clinical effectiveness. There is not consensus, either in the black community or in medicine, about whether BiDil is a good thing. The project for critical scholars is different: not to traverse or overcome undecidability, but to track race at its intersection with this drug, and to develop modes of engagement with unclosable undecidabilities.

Introduction

In this chapter, like in the previous one, the focus turns toward a particular contemporary racialized pharmaceutical, and follows this drug as it moves outside the scope of medical conversations. In this case, the process will be attentive to pharmaceutical practices

³⁹³ Chantal Mouffe, "Deconstruction, Pragmatism, and the Politics of Democracy," in *Deconstruction and Pragmatism*, ed. Chantal Mouffe (New York: Routledge, 1997), 9.

³⁹⁴ Jacques Derrida, "Remarks on Deconstruction and Pragmatism," in *Deconstruction and Pragmatism*, ed. Chantal Mouffe, (New York: Routledge, 1997), 77-88.

around a branded drug that is more explicitly racialized than thiazide. Yet the terrain is similar: debates around BiDil can be read as articulating inclusion and difference in American biological citizenship.

BiDil is a pill that combines two drugs (hydralazine and isosorbide dinitrate) that cause dilation of the veins and arteries, easing the work that the heart has to do.³⁹⁵ BiDil was approved in June 2005 by the US Food and Drug Administration for “heart failure in self-identified black patients.” It was approved after a study known as the African American Heart Failure Trial (A-HeFT), which enrolled 1050 African Americans with heart failure and, though designed to run longer, was stopped in 2004 after two years because the “standard of care plus treatment” arm had just over half as many deaths and two-thirds as many hospitalizations as the arm treated with “standard of care plus placebo.”

This chapter will analyze a few of the ways that BiDil is invoked and contested with regard to race inside and outside the field of medicine. On the one hand, it’s been described as a boon by the NAACP leader quoted above, and some from the Association of Black Cardiologists and the FDA. How could anyone seek to deny the value of a salve for ailing African Americans, so long denied access to the drugs that can save their lives and lessen their suffering? On the other hand, although granting that it should be available, many involved in debates about race and science within history, social science, and medicine say that its racial indication is a travesty. How could anyone deny the

³⁹⁵ Taylor AL, Ziesche S, Yancy C, Carson P, D’Agostino R Jr, Ferdinand K, Taylor M, Adams K, Sabolinski M, Worcel M, Cohn JN; the African-American Heart Failure Trial Investigators. “Combination of isosorbide dinitrate and hydralazine in blacks with heart failure,” *New England Journal of Medicine* 351 (2004): 2049-2057. The combination also increases bioavailability of nitrous oxide, which the authors of the study that led to its approval theorize could be another mechanism of action.

danger of racialized medicine, fraught as it is with naturalization of disparities and untrustworthy medical and government officials?

Critiques of BiDil have emerged from two conversations that are both central to this thesis: race and pharmaceuticals. What can we gain by starting at this intersection? What challenges does the case of BiDil present for these discussions, and how can it test their limits? When BiDil medicates race, it *mediates* race - in the sense of arbitrating as well as intervening. Meditating on BiDil opens up opportunities to meditate upon the meanings of race for an array of stakeholders that includes civil rights activists, physicians, and scholars.

First, pharmaceuticals. This chapter will begin by tracking the trajectory of the drug, with special attention to the contingency of that trajectory. After discussing the promise and limits of “heart failure” as an indication for prescription, this chapter will track the circuitous route that the combination of drugs that would become BiDil took, through government trials and biotech companies, to reach this indication. Then it will consider the constellation of pharmaceutical brands to which BiDil has been compared. BiDil is very different from the Big-Pharma blockbuster drugs for the worried well that have been the subject of most STS and popular critiques. BiDil is a product of the smallest of Pharma - a partnership between a doctor with an idea and a small biotech company - and is for very ill patients. Pharma as an industry is a constant experiment, and the historical juncture of BiDil’s emergence as a raced drug promoted by a biotech company was part of a particular experimental moment. BiDil emerged under that name in the late 1990s, and epitomizes that period of American exuberance about the possibilities of startups and of high profits from highly segmented markets. Its medical

and marketing model turned out not to be a paradigm shift as predicted, but a fleeting moment that now seems out of step with today's dominant pharmaceutical frameworks.

BiDil has been remarkably unsuccessful in a commercial sense so far, reaching only a few percent of its target market. And so an overriding theme of this chapter is trying to understand why this drug with a story that so many diverse groups found compelling – white conservatives, stock investors, the NAACP – has not been able to “penetrate” the pharmaceutical practices of African Americans with heart failure. The poor economic performance of BiDil has been explained away by some through citation of structural/market aspects (availability of generic versions, resistance from formularies, too small a marketing force,) as well as tactical errors (price, too little marketing to hospitals). If we let these explanations rest, we miss an opportunity to use BiDil to extend not only STS scholarship of race but also STS scholarship of pharmaceuticals. Exploring the case of BiDil, then, is an opportunity to open up the contingency of pharmaceuticals more generally. Drugs do not always reach their markets, and we can use analysis of BiDil to de-stabilize notions of the inevitability of drugs' capacity for infinite expansion. Where much scholarship has attended to the expansive potential of drugs, this chapter seeks to understand what sites of resistance impede complete saturation of American publics with drugs.

The second part of the chapter attends to race. I will consider both the allure and the impossibility of this drug settling debates about race in arenas of medicine or science. The critiques about race that have been mobilized around BiDil have drawn heavily from older arguments about race and genetics, which focused on the danger of suggestions of immutability. These arguments are not quite suited for mobilization against something

that is capable of being an agent of change in the immediate term, a drug that promises to intervene on differentiated biologies to help African Americans who are suffering right now to live longer, better lives.

Tracking the peculiar commodity aspects of BiDil reveals the intersection of race and this pharmaceutical to be an irredeemably polyvalent one. Tending to the *pharmakon* and the ontological status of race in turn, I suggest that each of these arenas are the terrain of irreducible contradictions. The *pharmakon* is both remedy and poison, and race is both material and semiotic. Reading BiDil in these terms provides occasion to consider the simultaneous appeal and unpalatability of a “black drug.”

Drugs are objects that are readily accepted as material-semiotic, but many have tried to use BiDil to settle race on one side or the other of the material-semiotic divide. Arguments about BiDil are happening on different registers that cannot quite subsume or refute each other. By engaging with the polyvalence of BiDil at the dense transfer point of race, this chapter will show the limits of arguments that seek to purge race and medicine of either its material or semiotic aspects.

Heart failure as a disease category

In a thesis that has combined consideration of heart disease with that of pharmaceuticals and race, it is helpful to consider the particular characteristics of the disease for which BiDil is indicated: heart failure. What does it mean for hearts to fail? Heart failure as a disease category is both a social construction and an embodied reality.

The work of medical historians, sociologists, and anthropologists offers a number of ways to conceive of disease as socially constructed. “Social construction of disease” can mean many things, including inducement of disease by social causes, pathologization of ways of life, or organization of diffuse bodily experiences into discreet pathologies. It is with respect to the contingency of the ways that we organize diffuse bodily experiences that Robert Aronowitz described the challenge raised to him by a colleague: “now you’re going to tell me heart attacks are socially constructed.”³⁹⁶ And indeed he does, laying out the historical development of the concept of myocardial infarction, and suggesting that even those disease categories that seem so clear to us in our current context and moment have definitions contingent on medical technologies and ontologies from which they emerge and are negotiated. He is particularly interested in the ways that tensions between ontological notions rooted in the doctor’s expertise and holistic notions rooted in the patient’s experience have played roles in those negotiations.

Like myocardial infarction, heart failure has a contingent, technologically-dependent history. Its clinical definition can be confirmed on the basis of either a scan or an arbitrary dividing line. Where normally a well-functioning heart relaxes to allow in fresh blood and has an ejection fraction of over 50%, a heart that either does not relax enough to fill or has an ejection fraction of less than 40% is considered failing. These processes and fractions can only be determined through visualization technologies in a doctor’s office. In this sense, heart failure is also like hypertension, defined by an arbitrary dividing line assessed by a diagnostic test.

³⁹⁶ Robert A. Aronowitz, *Making Sense of Illness: Science, Society and Disease* (Cambridge: Cambridge University Press, 1998), 84.

Importantly, however, unlike the stages of hypertension, the stages of heart failure are not determined numerically. Rather, symptoms define the stages: Classes I and II are defined by whether symptoms like shortness of breath, palpitation, and fatigue cause no limitation on physical activity or slight limitation on physical activity respectively. Classes III and IV are based on whether those symptoms occur only during normal physical activity, or whether they are present even at rest.³⁹⁷ Heart failure, then, is not (in Aronowitz's terms) as dominantly a "physician's disease" as hypertension is; it is also a "patient's illness." In this sense, then, BiDil is very different from thiazide. Like thiazide, it is imagined to be taken for the rest of a person's life, yet the prognosis for the length of that life is quite short. And unlike thiazide, BiDil does not expand the category of the illness it seeks to treat. It is a drug for people who are sick, not for an infinitely expandable category of those at risk for becoming so. It also often does make them feel better in the short term, and has impacts on prevention of hospitalization and death that are on a scale that a patient can experience as a real improvement.

STS work on pharmaceuticals has focused on drugs such as Viagra and Prozac, which have been described as "lifestyle drugs," "health enhancing" rather than "life-saving."³⁹⁸ More recent scholarship has also attended to drugs for the pre-disease categories of cardiovascular disease: hypertension, hypercholesterolemia, and diabetes.³⁹⁹ Dumit has connected these two trends to understand the lifestyle aspects of cholesterol-

³⁹⁷ For an excellent overview of heart failure, see George S.M. Dyer and Leonard S. Lilly, "Heart Failure," in Leonard S. Lilly, ed., *Pathophysiology of Heart Disease: A Collaborative Project of Medical Students and Faculty (3rd Edition)* (Philadelphia: Lippincott Williams & Wilkins, 2003), 211-236.

³⁹⁸ Laura Mamo and Jennifer R. Fishman's "Potency in All the Right Places: Viagra as a Technology of the Gendered Body," *Body and Society* 7 (2001): 13-55.

³⁹⁹ Jeremy A. Greene, "Releasing the Flood Waters: Diuril and the Reshaping of Hypertension," *Bulletin of the History of Medicine* 79 (Winter 2005): 749-794; Jeremy A. Greene, *Prescribing By Numbers: Drugs and the Definition of Disease* (Baltimore: Johns Hopkins Press, 2006).

lowering drugs, at a historical moment in which people manage diet and drugs together, consuming both steaks and statins (or neither).⁴⁰⁰ Moreover, Dumit has shown the ways that both psychotropic and cholesterol-lowering drugs operate on a logic of surplus health. That is, they do not alleviate experienced suffering, but they teach patients-in-waiting both about risks of which they were unaware, and about the potential for limitless health that exceeds their consciousness. In effect, this renders normality dependent on pharmaceuticals.⁴⁰¹ Other modes of pharmaceuticals as prevention – for example, for the prevention of breast cancer – have also garnered critique for the administration of toxic drugs to healthy women without evidence of their need or effectiveness.⁴⁰² Amid successful appeals by other drugs for life enhancement and risk management, there is something peculiar in BiDil’s poor success in getting into bodies to date. This is a drug that is actually effective for patients who are easily defined as needing it, but it might be a poor fit for dominant contemporary pharmaceutical discourses focused on the slipperiness of disease categories and potential risks.⁴⁰³

⁴⁰⁰ Joseph Dumit, *A Pharmaceutical Grammar: Drugs for Life and Direct-to-Consumer Advertising in an Era of Surplus Health*, Forthcoming, Duke University Press.

⁴⁰¹ Joseph Dumit, “The Depsychiatrisation of Mental Illness,” *Journal of Public Mental Health*, September 2005.

⁴⁰² Bonnie Spanier, “‘Your Silence Will Not Protect You:’ Feminist Science Studies, Breast Cancer, and Activism,” in *Feminist Science Studies: A New Generation*, Maralee Mayberry, Banu Subramaniam, and Lisa H Weasel, eds. (Routledge: New York, 2001), 258-274. She advocates for ‘strong objectivity’ in analyzing breast cancer research, skepticism of genetic and pharmaceutical approaches that receive more attention than environmental toxins. She questions whether feminist breast cancer organizations are risking their strong objectivity—ability to carefully read both the scientific data and the role of power—by accepting money from pharmaceutical companies. Yet what she holds out hope for the promise of evidence-based medicine, extricated from aggressive overtreatment and undue influence of pharmaceutical and profit-driven interests.

⁴⁰³ In this sense, BiDil may be distributed by a biotech company but it does not really fit with biocapitalism, in for example Kaushik Sunder Rajan’s terms. “One can see, however, that there is a pressure on pharmaceutical companies that stems from a downstream logic to move *therapeutic* intervention to earlier and earlier stages of disease manifestation, indeed toward a regime of therapeutic intervention against *suggestion* rather than explicit manifestation of disease, which has been seen particularly in the increased prescription of psychotropic drugs... This move... stems not just from a desire on the part of pharmaceutical companies to enlarge markets but also from a manifestation of the pharmaceutical industry *acting as its*

The limits of the commercial success of BiDil may be connected with the limits of heart failure as a disease category as currently defined. Heart failure as a disease category is in some ways the opposite of hypertension. As the NHLBI fact sheet on the topic puts it: “It is often the end stage of cardiac disease.” Where hypertension is a way of understanding pre-disease, heart failure is a way of understanding end stage disease. The trial that led to BiDil’s approval, A-HeFT, included only those who were quite sick in terms of symptoms (Classes III and IV), and there has not yet been much discussion of using it in anyone who is not very sick. On the contrary, the overwhelming majority of African Americans with the advanced stages of heart failure are not receiving the drug, making diagnosis creep a concern that does not seem salient in this case at this time.

BiDil’s call has often been described as edgy because of its explicit racialization. However, its call is also old-fashioned, not only because it is based on old drugs, but also because it treats people for a highly morbid condition that has a relatively short duration, often making them feel better and live longer. Because drugs are productive (they ‘do work’) even when they don’t necessarily deliver on their promised effects (they don’t ‘work’), we don’t have a well-developed language for critiquing them when it turns out that drugs *both* are culturally productive *and* have beneficial physiological effects – that is, when they *both* ‘do work’ and ‘work.’ When this type of drug is racialized, critique

own insurance industry. The only way for pharmaceutical companies to insure against diagnostic encroachment on the domain of therapeutics is to shift the domain of what counts as disease to earlier and earlier stages of its manifestation – to the point, of course, that new diseases often get created, and old ones get significantly redefined on the basis of shifting the moment that demands therapeutic intervention.” Kaushik Sunder Rajan, *Biocapital: The Constitution of Postgenomic Life* (Durham: Duke University Press, 2006), 158.

that recycles old arguments about risk-averting or lifestyle-enhancing drugs is not sufficient.⁴⁰⁴

This is not to say that BiDil does not have the potential to transform into a larger preventative drug. Indeed, there is another aspect of heart failure staging that might help its market expand. According to cardiologists, heart failure is understood as a progression from a compensated to a decompensated state. This means that, in the face of some problem that is making the heart have to work harder (coronary disease, for example, or hypertension), the heart initially expands to compensate. Because heart cells do not divide, the cells themselves must expand to increase heart volume. Patients do not generally experience symptoms at this stage. But this is not a sustainable solution, and the heart eventually becomes *decompensated*, unable to meet the demands of circulation in spite of its increased size. This is when patients start to suffer symptoms. This progression could therefore be reframed as risk. It may be that eventually we will all be defined as having pre-heart-failure, which is in any case analogous to (and could even be synonymous with) our status as having “high” cholesterol and hypertension as well as “abnormalities” that appear on visual diagnostic technologies. We might be imagined to be all “compensating” for the pathologies unknown to us, ready to decompensate unless we are protected by pharmaceuticals. If this happens, BiDil will fall into the “dependent normality” that Dumit has described. But the drug is not yet being tested in or given to this potentially expansive population.

⁴⁰⁴ Medicine is thus an urgent site for the important call Evelyn Fox Keller has made for biology. Where Fox Keller has argued that those who analyze discourses of science must develop a critique that can account for the *effectiveness* of science without using that effectiveness to justify an embrace of traditional philosophy of science, a similar project is essential in understanding these multiple levels of “fixing race” and “doing work” in processes of medicating race. See Evelyn Fox Keller, *Reflections on Gender and Science*, 10th Anniversary Edition, (New Haven: Yale University Press, 1996), 6.

BiDil as small Pharma

If we want to understand both the promise and limits of BiDil, we need to understand not only its racialization and the promise and limits of the disease it treats, but also its participation in a market economy. Although the discussion of BiDil has for the most part invoked an undifferentiated notion of “pharmaceutical companies,” BiDil’s story is not only one of Pharma gone racial, but also of the challenges and promises of small Pharma. Its business model was conceived of in a brief historical moment in which many were captivated by the idea that the little guy focused on one good idea was the way of the future. In the 1990s, there was excitement about this model for pharmaceutical companies, even optimism that it would redefine the terms for Big Pharma.

BiDil’s market is not on the scale of a blockbuster drug. Even if every single African American with heart failure in the US took the drug, the estimated 750,000 prescriptions would be a tenth of a percent of Lipitor prescriptions (or for that matter thiazide prescriptions).⁴⁰⁵ Even if the racial indication were ignored, and every person with heart failure in the US took BiDil, that would still be a tiny market of 5 million,⁴⁰⁶ in contrast to that for statins or antihypertensives. Moreover, more than half of those diagnosed with heart failure will die within five years, and 80% will die within 8-12 years.⁴⁰⁷ Although the prevalence of heart failure as a diagnosis is growing, the potential for growth of the heart failure treatment market is hindered by the poor longevity of its

⁴⁰⁵ An estimated 63,219,000 Lipitor prescriptions were dispensed in the US in 2005, and 42,757,000 for the top thiazide (hydrochlorothiazide) as well as many more for other thiazides.

<http://www.rxlist.com/top200.htm>

⁴⁰⁶ The American Heart Association states that five million Americans have heart failure, and that 550,000 are diagnosed every year.

⁴⁰⁷ NHLBI Congestive Heart Failure Fact Sheet. <http://library.thinkquest.org/27533/facts.html>.

sufferers (even with BiDil). The heart failure market is quite a bit more limited in terms of total patient-days of consumption than the market for “lifelong” drugs for young healthy people. Market expansion happens with antihypertensives and statins because more people become diagnosed with the risk factors than die with the diagnosis, but does not happen with heart failure because time between diagnosis and death is limited. In order to increase the total number of patients living with the diagnosis, the increase in newly diagnosed patients would have to be of a greater scale than those who die of the diagnosis. As it is, heart failure remains far from the scale of the pre-heart-diseases that have become our preoccupations. This is not to say that money cannot be made on a market that small, but rather that we cannot assume that the business logics of blockbuster drugs operate in quite the same way as niche ones.

BiDil is the brainchild of University of Minnesota cardiology professor Jay Cohn.⁴⁰⁸ Since the 1970s, he has believed for elegant physiological reasons that the combination of isosorbide dinitrate and hydralazine (I/H) – generic drugs already in wide use for other indications – would be highly beneficial for heart failure. Cohn applied for a patent on the method in 1987, and received it in 1989. He put his combination to the test in collaboration with Veterans Affairs in two cooperative studies between 1980 and 1991, in studies called the Veterans Heart Failure Trials (V-HeFT) I and II.⁴⁰⁹ Both studies had a base treatment of digoxin and diuretics for both the control and experimental groups. On top of that base treatment, V-HeFT I compared the addition of

⁴⁰⁸ A more comprehensive timeline can be found in Jonathan Kahn, “How a Drug Becomes “Ethnic”: Law, Commerce, and the Production of Racial Categories,” *Yale Journal of Health Policy, Law, and Ethics* 4 (2004): 1–46.

⁴⁰⁹ J.N. Cohn, et al. “Effect of vasodilator therapy on mortality in chronic congestive heart failure. Results of a Veterans Administration Cooperative Study,” *New England Journal of Medicine* 314 (1986): 1547–1552; J.N. Cohn, et al. “A comparison of enalapril with hydralazine-isosorbide dinitrate in the treatment of chronic congestive heart failure,” *New England Journal of Medicine* 325 (1991): 303–310.

I/H head to head with that of another agent, prazosin (an alpha-blocker), and placebo. I/H did better than placebo and other agent, but the result did not reach statistical significance. V-HeFT II did not have a placebo arm and compared I/H again head to head with another agent, enalapril (an ACE-Inhibitor). This time, both agents had similar results but I/H did not do quite as well as the other agent.

Like many scientists confronted with evidence that fails to prove their theory, Cohn still believed that his idea worked.⁴¹⁰ He started to think that perhaps the combination of ACE-Inhibitors with I/H would provide the long-sought benefit, but could not find the commercial or governmental support to test his idea. A pharmaceutical benefit management company called Medco (then owned by Merck, as it experimented with vertical integration⁴¹¹) secured property rights from Cohn and got a trademark for the drug combination as BiDil in 1995, and Cohn went with company representatives to the FDA to seek a new drug application based on the data from the V-HeFT trials. But the FDA was not supportive of a new drug application based on the data amassed, especially since the components of the new drug were available generically, and it rejected the application.⁴¹² Medco left the picture.⁴¹³

⁴¹⁰ After BiDil's approval, Cohn said: "This has been 30 years. I have to feel that I am finally vindicated." As quoted in Andrew Pollack, "Drug Approved for Heart Failure in Black Patients," *New York Times*, July 20, 2004. See also: Stephanie Saul, "U.S. to Review Heart Drug Intended for One Race," *New York Times*, June 13, 2005. Cohn's belief remains that the combination works (period) as he always predicted, rather than just for a smaller group: "Dr. Cohn said that despite any flaws in the first studies, the similar findings in the new trial suggest the original data was accurate. 'The replication gives me confidence that this combination is more likely to be effective in people who call themselves black than in people who call themselves white,' he said. 'Do I believe this drug should work in whites? Biology would tell me it should.'"

⁴¹¹ Bloomberg Business News, "Merck Profits Were Higher in Second Quarter," *New York Times*, July 19, 1996. At the time, this sort of vertical integration was viewed as a model. See the case study, "Merck-Medco: Vertical Integration in the Pharmaceutical Industry," HBS 9-598-091.

⁴¹² "Panel Recommends FDA Reject BiDil Application," *Wall Street Journal*, February 28, 1997, p. 3.

⁴¹³ Andrew Pollack, "Drug Approved for Heart Failure in Black Patients," *New York Times*, July 20, 2004.

But by the late 1990s, a time when focus on health disparities was growing at the FDA and beyond, two things had changed: ACE-Inhibitors were widely argued to be less effective in blacks, and Cohn and a colleague conducted a retrospective analysis of the few blacks in the V-HeFT trials. That analysis that seemed to suggest that I/H showed a stronger benefit for them—much better than the placebo in V-HeFT I and slightly better than the ACE-I in V-HeFT II. In 1999, Cohn and others published the retrospective analysis by race,⁴¹⁴ and shortly thereafter a biotech company that focused on nitrous oxide, NitroMed, bought intellectual property rights to BiDil. Cohn got a new methods patent, this time for the race-specific benefit of the combination, and licensed it to the company. NitroMed had never developed or marketed drugs, but teamed up with the Association of Black Cardiologists to design a trial. When Cohn and NitroMed, backed by ABC and the Congressional Black Caucus, went back to the FDA in 2001, it was supportive of a trial that would focus on this population that seemed poorly treated both because of high morbidity and mortality and because current guidelines seemed less effective in that group. Thus emerged the trial that would lead to BiDil's approval: the African American Heart Failure Trial (A-HeFT).

A-Heft began in 2001 with a cohort of 1050 self-identified blacks with Class III or IV heart failure.⁴¹⁵ It added BiDil or placebo to the current standard-of-care, which now generally included not only digoxin and diuretics but also ACE-Inhibitors and a similar-acting drug class, ARBs. This combination proved to be very potent. By the time

⁴¹⁴ P. Carson, S. Ziesche, G. Johnson, and J.N. Cohn, "Racial differences in response to therapy for heart failure: analysis of the vasodilator-heart failure trials. Vasodilator-Heart Failure Trial Study Group," *Journal of Cardiac Failure* 5 (1999): 178-187.

⁴¹⁵ Taylor AL, Ziesche S, Yancy C, et al for the African-American Heart Failure Trial Investigators. Combination of isosorbide dinitrate and hydralazine in blacks with heart failure. *N Engl J Med*. 2004;351:2049-2057. Remarkably, none were lost to follow-up.

the trial was ended two years later, in 2004, (early, because of the strength of the results), 43% fewer in the standard+BiDil group had died compared with the standard+placebo group, while 39% fewer had been hospitalized, and those in the treatment arm also reported better quality of life.

The FDA approved BiDil “for heart failure in blacks” in June of 2005. Its package insert describes its indications:

BiDil is indicated for the treatment of heart failure as an adjunct to standard therapy in self-identified black patients to improve survival, to prolong time to hospitalization for heart failure, and to improve patient-reported functional status. There is little experience in patients with NYHA class IV heart failure. Most patients in the clinical trial supporting effectiveness (A-HeFT) received a loop diuretic, an angiotensin converting enzyme inhibitor or an angiotensin II receptor blocker, and a beta blocker, and many also received a cardiac glycoside or an aldosterone antagonist.⁴¹⁶

No one knows why BiDil was so much more effective in A-HeFT than it had been in the V-HeFT trials. Perhaps it was because of the synergistic effects with ACE inhibitors, with which it had not been combined previously.⁴¹⁷ Perhaps the synergy of drugs was even more comprehensive, by attacking multiple mechanisms at the same time.⁴¹⁸ Perhaps it had to do with the differences in etiology of the heart failure, with a higher share being due to hypertension than coronary disease in the latter trial. Perhaps it had to do with the inclusion of women. Perhaps it had to do with the increased prevalence of nitrous oxide deficiency in the A-HeFT population compared with that of the V-HeFT

⁴¹⁶ <http://www.bidil.com/pdf/PI.pdf>

⁴¹⁷ These suggestions are offered, among other places, by M. Gregg Bloche, “Race-Based Therapeutics,” *New England Journal of Medicine*, 351 (November 11, 2004), 2035-2037.

⁴¹⁸ Investigator Anne Taylor has described this possibility in speaking engagements, and the slide in the slide deck from A-HeFT suggests that BiDil adds a fourth mechanism upon which it is possible to intervene. reno-angiotensin system (RAS) blockade with ACE-Inhibitors; beta blockade; and aldosterone blockade. Combining all four was what BiDil did in most patients.

population. But the marketing decision was to be agnostic on these and let the explanation rest at efficacy in “self-identified blacks.”

This decision to target “self-identified blacks” rather than those with the presumably underlying markers for responsiveness to BiDil can be read as connected to my argument in Chapter 4, about the appeal of African American Hypertension as a disease category. Physicians do not necessarily prefer to make decisions based on characteristics they can *measure*, but rather on characteristics they can *record*. It is easier to communicate a pragmatic application for “self-identified blacks” than it is to make complicated guidelines based on test values. (Cohn has responded to criticisms that race is an arbitrary category by saying that age and presence/absence of ventricular abnormalities are arbitrary, too, but that doesn’t mean that a study cannot be constructed around them.⁴¹⁹) But just because it is easier to target a drug this way does not mean that the enterprise is easy. Real challenges confront a small Pharma player in a big Pharma field, and the polyvalence of the intersection of race and drugs exceeds NitroMed’s control.

In the constellation of brands

In discussions of BiDil, there have been considerable attempts to situate it in the constellation of other branded pharmaceuticals. As peculiar as BiDil’s trajectory has

⁴¹⁹ Jay Cohn, “The Use of Race and Ethnicity in Medicine: Lessons from the African American Heart Failure Trial,” *Journal of Law, Medicine and Ethics*, 34 (Fall 2006): 552-554.

been, it has been necessarily invoked with references to much more widespread drugs that have come before it and also been used in identity and justice claims.

BiDil as high risk like Vioxx

If BiDil is characterized as a “new” drug with a high profit potential, that carries valences of being little-studied and risky. Is BiDil “like Vioxx?” This is the principal specter raised by Margaret Kimberly on blackcommentator.com. Pointing out the financial conflicts of interest on the part of investigators like ABC and supporters like NAACP, she talks about BiDil in terms of the risks of taking a new drug:

No one knows if BiDil is very effective or safe. If it isn't it will not be different from other drugs given FDA approval that were later discovered to be dangerous. Fenfluramine was marketed as Redux, a drug used to treat obesity. It was taken off the market after causing cases of heart valve damage and pulmonary hypertension. How many commercials exhorted consumers to ask their doctors about Vioxx and Celebrex? We now see commercials from law firms exhorting us to pursue malpractice suits against the makers of those drugs.

Past experience indicates that BiDil shouldn't be greeted as a health care panacea for anyone. It should be treated like all newly approved pharmaceuticals, with great caution if not suspicion. As Raymond Woosley, vice president for Health Sciences at the University of Arizona, advised Public Television, “Americans need to recognize that every time they put a pill in their mouth, especially a new pill that they've never taken before, it's an experiment. How big an experiment depends on the pill and how well it's been studied.”⁴²⁰

I will return to this unspecified danger, which seems out of place given that the component drugs have actually been widely used and are being given only to those with a prognosis worse than many cancers. I flag it now to consider that this concern coexists with the claim that the problem with BiDil is that it is *not* new. Even Kimberly in the same article points out that it is a rebranding of two generic drugs. In this sense, BiDil is

⁴²⁰ Margaret Kimberly, *The Black Commentator*, “Rx for Black Hearts,” http://www.blackcommentator.com/143/143_freedom_rider_rx.html, also available at Alternet as “A Bitter Pill for Black Hearts,” <http://www.alternet.org/story/23185/>.

undermined *both* by the danger that attends newness and by the shoddiness that attends oldness.⁴²¹ Is the problem with BiDil that it could save lives cheaply but the drug companies are profiteering off of it?

BiDil as a human right like HAART

In the sense that it is profiteering off those that are in desperate need, some of the rhetorical strategies around critique of BiDil have been similar to those used around HIV drugs. Drug regimens like highly-active anti-retrovirals (HAART) are of course not precisely brands, though they are made up of branded drugs. But they are like BiDil in the sense that they are drugs for diseases understood as ‘death sentences’ that can be the subject of varied kinds of politics and identification.

One difference between HAART and BiDil in the US is that Medicare/Medicaid and insurers pick up the tab on the former but, in large part, not the latter. So even if we establish that the drug is too expensive for those in need, it is not altogether clear whose fault that is. Part of the NAACP/NitroMed partnership has been around advocating for inclusion in health insurance formularies at a lower tier and in Medicare Part D. (More consideration will be given to price below.)

BiDil as like Viagra

One brand name that has been rhetorically attached to BiDil from disparate quarters is Viagra. Prominent BiDil critic Jonathan Kahn has invoked it that way in describing

⁴²¹ I wonder whether at the nexus of race and drugs we engage in Freud’s overjustification as in dreams: the kettle I returned to you is brand new; the holes in it were there when you loaned it to me; I never borrowed the kettle...

NitroMed's success in raising money: "Where drugs such as Viagra may target one sex or another, BiDil promises to lead the way in ethnic niche marketing of pharmaceuticals."⁴²²

Viagra was also used by Juan Cofield to situate BiDil at the New England Conference of the NAACP. He is quoted as introducing the presentation by a NitroMed spokesperson with a half-joke that induced some giggles: "I would like to see the name BiDil as common in our community as Viagra is in the general public."⁴²³ The NitroMed speaker, Gerald Bruce, came back around to this referent after a speech in which he emphasized the importance of grassroots education because insurers' refusal to cover it would change in the face of demand. "What you can do to help is just building awareness in the community," Bruce said. "Talk to your family members, your constituents, your friends, your neighbors. Tell them you heard about a lifesaving medication that makes patients feel better and be able to function better. Let's, as Juan said, make BiDil as commonly known in the African American community as Viagra is in the broader community."⁴²⁴

There is a slippage in this conversation, at least as it has been reported, between whether the drug should be "as common" or "as commonly known."⁴²⁵ In drugs' mobilization in cultural debate, they can become something of the same thing. The audience is described as laughing. In a sense, Cofield seems to be making fun of their

⁴²² Jonathan Kahn, "How a Drug Becomes "Ethnic": Law, Commerce, and the Production of Racial Categories," *Yale Journal of Health Policy, Law, and Ethics* 4 (2004): 24.

⁴²³ Dan Devine, "NAACP goes to the grassroots for BiDil," *Bay State Banner*, Oct 5, 2006, p. 7.

⁴²⁴ Dan Devine, "NAACP goes to the grassroots for BiDil," *Bay State Banner*, Oct 5, 2006, p. 7.

⁴²⁵ In a sense, Cofield is arguing that BiDil should become a preoccupation among African Americans, despite the relative rarity of heart failure in comparison with erectile dysfunction. As mentioned in the introduction, diseases need not be common in order to become preoccupations – and diseases that remain more or less hypothetical for Americans such as Bird Flu, SARS, and West Nile, do gain traction in worries on the scale of diseases that are actually prevalent whether depression or cancer. But heart failure does not at this moment seem to be rich for preoccupations, for a visceral engagement with heart disease in order to talk about self-value and social experience in disease.

knowledge of erectile dysfunction drugs. Taunting them a little: why do you know more about restoring sexual function than heart function? BiDil and erectile dysfunction drugs are actually contraindicated – the combination of them can cause dangerous hypotension. So in some literal sense you have to choose whether to have to salvage your erection or your heart. (Good thing the heart failure population has so many women.)

BiDil as like Serafem

Considering the connections between raced and gendered drugs, it becomes salient to ask: is BiDil “like Serafem?” The blogger Drugnazi (now called DrugMonkey) considers them together, with both BiDil and Serafem making his list of “the most pointless, inane, rip-off piece of crap peddled by Big Pharma.”⁴²⁶ Here are his descriptions of the two:

Bidil- A combination of isosorbide dinitrate and hydralazine, two meds that have been on the market for years and are also cheap as dirt. Bidil combines the two old meds into one tablet, then markets itself to African Americans as a race specific remedy for heart failure. Of course there's no reason you can't prescribe the two drugs separately and save money, but hey, maybe it is easier for people to just swallow one tablet, and at least there is research to support the claim this combination of meds is more effective for black folks.

Moving up a level on the rip off scale we have:

Sarafem- The exact same fucking thing as Prozac. But hey, at least it comes in a different box so someone snooping in your medicine cabinet will just see that you're PMSing, not crazy.

Extending patent protection through rebranding a drug for a new indication is something BiDil and Serafem have in common, but the volatility of gender versus race turns out to

⁴²⁶ Drugnazi [aka DrugMonkey], “If You Are A Doctor That’s Ever Written A Prescription For Pexeva, You Are By Definition A Numnut,” October 22, 2006, <http://drugnazi.blogspot.com/2006/10/if-you-are-doctor-thats-ever-written.html>

be very different.⁴²⁷ For all the emphasis in the second wave of feminism on distinguishing between sex that is biological (and presumably fixed in the sense of unchangeable) and gender that is social (and presumably amenable to a social-change-oriented fix), special medicine for women as a biological group does not have the same volatile power as special medicine for blacks.

BiDil as one more drug class in a boring-to-treat condition

In the comments by DrugMonkey above, there is another point often mentioned by NitroMed and others: that its status as a combination pill might be what makes BiDil worthwhile. There is something fast-and-loose in this enumeration of pills taken, since BiDil is only given on top of a daily regimen of multiple pills from multiple classes of drugs. In A-HeFT, most participants received six pills of BiDil itself daily in addition to a standard of care that included dozens of pills per day. Pill consolidation may well be a worthy goal, but BiDil is a paltry attempt in that direction.

Given the wide range of physicians who deal with heart failure with already intensive drug regimens, it has not been easy for BiDil to find its way in this terrain. Most general practice physicians keep the patients on whichever drugs they were prescribed in the hospital where their heart failure diagnosis was most likely received, meaning that advertising to hospitals is particularly important. But for a tiny company

⁴²⁷ Although I have not heard any comparisons between BiDil and the other most gendered pill – *The Pill* – it might be an evocative line of thought to consider. If participation in American citizenship is indeed related to the consumption of pills, the figuration of “birth control” in the United States as oral hormonal contraception this could be another piece of this. Andrea Tone, *Devices and Desires: A History of Oral Contraceptives*, Hill and Wang; Nelly Oudshoorn, *Beyond the Natural Body: An Archeology of Sex Hormones*. London: Routledge, 1994.

like NitroMed, getting hospitals and formularies to change their practices has been a huge challenge.

One critic of BiDil, Harriet A. Washington, has suggested that the fact that it does not replace any of the other drugs for heart failure but rather adds to them means that BiDil will not face competition:

NitroMed stock rode the good news from the A-HeFT trials to a 73 percent leap in share price. Because it was tested only with other drugs, BiDil typically will be prescribed for use in concert with other drugs, not instead of them, so that BiDil will not compete in the marketplace with established heart medications. This will help BiDil's sales and this could even explain why BiDil was only tested against a placebo: Had BiDil been tested alone, researchers would have run the risk that the study results could have been different, finding that BiDil provided less protection to black patients than standard medicines.⁴²⁸

It's not clear precisely how Washington would design a proper trial: with one wing getting less than standard of care plus BiDil versus one getting full standard of care? But what is clear is that she assumes that, absent direct competition, a drug naturally reaches its markets.

Although Washington's assessment of the stakes of the terrain seems off base, she puts BiDil into an apt context: the heavily-medicated landscape of only partially-effective drugs. BiDil, like ACE-Inhibitors or ARBs when used for heart failure, is one more drug in a boring-to-treat condition – and yet because it lacks ACE-Inhibitors' broader appeal as an antihypertensive, BiDil has nowhere near their reach.⁴²⁹ Considering BiDil in the context of these other drugs also helps to put its racial indication into starker relief: ACEs

⁴²⁸ Harriet A. Washington, *Medical Apartheid: The Dark History of Experimentation on Black Americans from Colonial Times to the Present* (New York: Double Day, 2006), 323.

⁴²⁹ Though even the profit potential of antihypertensives is less dramatic than other types of drugs. The highest revenue antihypertensive of the moment, NORVASC, which is a CCB, is the only antihypertensive in the top ten drugs by US sales volume amid lots of cholesterol, stomach acid, pain, asthma, and antidepressant meds. ACE-I's, ARBs, and CCBs are more exciting than diuretics, but not as exciting as statins or Vioxx much less Viagra.

and ARBs often include in their package insert that their benefits have not been proven in black patients⁴³⁰ or are less effective in black patients.⁴³¹ This grammar is slightly but crucially different from that of BiDil. BiDil could have, like ACEs and ARBs, been approved for everyone while being accompanied by a package insert indicating that “this drug’s benefit has not been proven in non-Black patients.” Instead, absence of evidence in nonblack patients was treated as tantamount to evidence of absence. The blackness of the subjects on whom the drug was tested could not be detached from the drug – neither for the FDA, nor, as we will see below, for many of BiDil’s critics.

BiDil as Tuskegee

The very marked status of an African American drug has led BiDil to be invoked together not just with pharmaceuticals, but also with other racialized practices in medicine. Thus,

⁴³⁰ For example, the patient product information for the ARB COZAAR suggests that one reason to prescribe it may not apply to blacks: “to lower the chance of stroke in patients with high blood pressure and a heart problem called left ventricular hypertrophy. COZAAR may not help Black patients with this problem.” http://www.merck.com/product/usa/pi_circulars/c/cozaar/cozaar_ppi.pdf. It also notes that “Analysis of age, gender, and race subgroups of patients showed that men and women, and patients over and under 65, had generally similar responses. COZAAR was effective in reducing blood pressure regardless of race, although the effect was somewhat less in Black patients (usually a low-renin population).” “Race: In the LIFE study, Black patients treated with atenolol were at lower risk of experiencing the primary composite endpoint compared with Black patients treated with COZAAR. In the subgroup of Black patients (n=533; 6% of the LIFE study patients), there were 29 primary endpoints among 263 patients on atenolol (11%, 26 per 1000 patient-years) and 46 primary endpoints among 270 patients (17%, 42 per 1000 patient-years) on COZAAR. This finding could not be explained on the basis of differences in the populations other than race or on any imbalances between treatment groups. In addition, blood pressure reductions in both treatment groups were consistent between Black and non-Black patients. Given the difficulty in interpreting subset differences in large trials, it cannot be known whether the observed difference is the result of chance. However, the LIFE study provides no evidence that the benefits of COZAAR on reducing the risk of cardiovascular events in hypertensive patients with left ventricular hypertrophy apply to Black patients.”

⁴³¹ For example, the package insert of the ACE-Inhibitor ALTACE notes that “Although ALTACE was antihypertensive in all races studied, black hypertensive patients (usually a low-renin hypertensive population) had a smaller average response to monotherapy than non-black patients;” and “In considering use of ALTACE, it should be noted that in controlled trials ACE inhibitors have an effect on blood pressure that is less in black patients than in non-blacks. In addition, ACE inhibitors (for which adequate data are available) cause a higher rate of angioedema in black than in non-black patients.” <http://www.altace.com/altace/pdf/PI.pdf>

BiDil has been described as “like Tuskegee” and “like eugenics.” As Susan Reverby has pointed out, a similarity between Tuskegee and A-Heft is that both were framed and justified in part on the basis of the idea that there are physiological differences between the races, that black syphilis and black heart failure are each a ‘different disease.’⁴³² Moreover, Tuskegee is often mentioned in articles about BiDil to explain suspicion around it, with open-endedness about what the connection might be. Similarity with Tuskegee is a suggestion that African American cardiologists, in particular, bristle at. For them, Tuskegee is principally defined as first deceit (lack of informed consent) and second denying available treatment to African Americans, while A-Heft is defined as well-consented and providing treatment to patients who are suffering and at high risk of death.⁴³³

Since BiDil emerged into a rhetorical field of diverse pharmaceuticals, its meaning has been negotiated in relationship with brands ranging from Viagra to Cozaar. The discussion of the intersection of financial and medical interests has focused on this, such that when commentators are asked about the role of marketing in BiDil they speak also in relationship with these drugs. BiDil is so invoked with these other drugs, despite a generally poor discursive fit, because the vocabularies we have on hand to critique drugs are developed toward lifestyle-enhancing and risk-averting drugs. The analogous presence of Tuskegee in the grammar of comparative brands should alert us that there are

⁴³² Susan Reverby, “BiDil as Tuskegee’s Child: What Does It Mean?” presented at *Race, Pharmaceuticals and Medical Technology*, MIT, April 7, 2006. A version of this talk will be published in the *Journal of Law, Medicine and Ethics* in Fall 2008. Reverby is responding in particular to the point of view of Clyde Yancy, who suggests that heart failure in blacks is a different disease, Clyde Yancy, “The role of race in heart failure therapy,” *Current Cardiology Reports* 2 (May 2002): 218-25.

⁴³³ This point was made by Keith Ferdinand at the conference, in his talk “BiDil and Race,” that followed Reverby’s. *Race, Pharmaceuticals and Medical Technology*, MIT, April 7, 2006. A version of his paper will also be published in the *Journal of Law, Medicine and Ethics* in Fall 2008.

gaps between BiDil and the landscape of cutting-edge-drugs. There is a deep ambiguity about the status of race that puts BiDil into categories that exceed the bounds around these other brand-name drugs. Consider: it is possible to talk about Vioxx without having an opinion about either arthritis or about aging. Yet although it is possible to talk about BiDil without having an opinion about heart failure, it is not possible to talk about BiDil without having an opinion about race and medicine. Even if that opinion is relegated to disavowal, race remains present, and with it shadows of history – including Tuskegee.

Advertising BiDil

Many BiDil critics have suggested that it would be easy to market BiDil. For example, Helen Wallace from GeneWatch answers a question about whether financial rather than medical reasons drive race-based medicines like BiDil:

Well if you look at how drug development works, then it's very clearly driven by the markets where the biggest profits are. We already know that many diseases are neglected altogether because they occur mainly only in developing countries, and we also know that lifestyle drugs - drugs for baldness or drugs to prevent conditions in the worried well - sell and get a lot of money in their research. So I think it's very clear that those financial factors will play an important role.⁴³⁴

The idea that in the new economy start-ups stood to make billions and challenge the big companies' dominant claims on pieces of the pie was a fleeting one, but one that had a lot to do with the path that BiDil took. BiDil commercials are not on television, its

⁴³⁴ http://www.kenanmalik.com/tv/analysis_race+medicine.html

marketing force is tiny, and it has taken a quirky grassroots approach to reaching potential patients.⁴³⁵

Although BiDil has generated a good deal of publicity, it has generated very little advertising. Direct-To-Consumer advertising has been only a minuscule part of the discourse generated around BiDil, and so we can see that in a sense it is less different than we might assume from the generic drug considered in the previous chapter. Even within the category of “patient education” marketing by NitroMed, most of the resources have targeted black churches and communities on a low-budget grassroots level. However, there was a print advertisement that appeared in black newspapers in Houston, Detroit, and the District of Columbia in October and November 2006 that is worth review.⁴³⁶

The half-page advertisement is dominated by a photograph of an old man and a young girl, smiling together.⁴³⁷ They appear to be grandfather and granddaughter, which resonates with the campaign by the Association of Black Cardiologists, framing the importance of heart health around “Children Should Know Their Grandparents.” The large print ad copy under the picture reads “Live longer...Live Better.” Also in

⁴³⁵ This is not to say that Big Pharma doesn't like grassroots. Pfizer is a “partner” with the NAACP, too. <http://www.naacp.org/advocacy/health/>

⁴³⁶ There was also a radio ad that played in black stations in Houston, DC, and Detroit, which I have not been able to track down. A description of it can be found in: Mark Jewell, “Ad campaign for blacks-only heart drug touches lightly on race,” *Associated Press*, September 1, 2006. He describes it this way: “Although BiDil is approved only for use by the estimated 750,000 U.S. blacks with heart failure, a 60-second radio spot that will kick off the media campaign makes just one direct reference to race, noting that BiDil is ‘FDA-approved to treat heart failure in African-American patients.’ The spot opens with a grandfatherly voice asking, ‘Pass the string beans please, and those sweet potatoes!’ Then a girl's voice pleads with her grandfather to play ‘hide and go seek’ after dinner. The response from the girl's mother: ‘Honey, granddad's too tired. You go play with your sister.’ A voice then lists common heart-failure symptoms such as fatigue, shortness of breath and swollen ankles or legs, and suggests, ‘If this sounds like you or someone you love, ask your doctor about BiDil.’

⁴³⁷ The example of the advertisement I am using appeared in the *Houston Defender*, October 29-Nov 4, 2006, p. 4.

prominent typeface is “BiDil®” and underneath is its chemical name “isosorbide dinitrate/hydralazine HCl” and the slogan “More Life to Live.” The smaller text tells a story:

Life is made for living. And you deserve to enjoy every moment of it. BiDil is FDA approved to treat heart failure in African-American patients. When taken with routine heart failure medicines, BiDil can help you feel better, stay out of the hospital and live longer (see indication below). BiDil is available only by prescription. Only a healthcare professional can decide whether BiDil is right for you. For more information and approved package label, visit: www.bidil.com or call 1-888-MYBIDIL. Ask about NitroMed Cares™. You may be eligible to save on BiDil prescriptions.

The contraindications are given in typeface equal to this, and full small print appears on the facing page.⁴³⁸ This advertisement reads like a copy-cat ad of high-profile drugs, done at lowest possible cost. The advertisement engages in some of the dominant grammars of DTC advertising, emphasizing in the bold type general things that everyone could be presumed to want – longer and better life – rather than the specific condition that the drug is designed to treat. However, the small type does not engage in the typical interpellative strategy of pharmaceuticals in DTCs that address the patients along the lines of “do you feel tired?” The big type could portend “diagnostic creep,” because it is so general, yet the small type does not enable people to diagnose heart failure in themselves. Moreover, race is not particularly emphasized—there is no mention of disparities in heart failure, for example, or the importance of heart health for African Americans. The fact that the direct-to-consumer advertising ran so briefly suggests that

⁴³⁸ The indication reads: “BiDil is proven in African-American heart failure patients, in addition to routine medicines, to help you feel better, stay out of the hospital and live longer. There is little experience in patients with heart failure who experience significant symptoms while at rest. Most patients in the clinical study of BiDil received routine heart failure medicines. Tell your doctor about any allergies you have, especially if you are sensitive to nitrates, such as nitroglycerin. If you are taking any erectile dysfunction or pulmonary hypertension drugs, mixing them with BiDil can cause a sudden drop in blood pressure, fainting, chest pain, or heart attack. The most common side effects of BiDil are headaches and dizziness. Please see important product information on back.”

it was not effective enough in increasing prescription rates in those areas to merit its continuation.

NitroMed has also launched a patient-education website, hearthealthheritage.com, and it mobilizes some of the most typical grammars of pharmaceutical websites.⁴³⁹ It is addressed to a reader who is framed as black. The references are understated, with the “Heart.Health” part standing in for the less positive heart failure and “Heritage” standing in for black. On its homepage, the site proclaims itself to be “An online resource for African Americans concerned about heart failure and their family's heart health.” Tabs guide a browser through answers to questions like “What is heart failure?”; “Risks to African Americans”; “Treating Heart Failure”; “What you can do”; and “Other Resources.”

Nitromed has fired most of its sales staff and shifted its focus to “elite” cardiovascular experts, health plans, and medical centers.⁴⁴⁰ This is normally the first line of marketing for any drug, since sales generated by direct-to-consumer advertising are small as a percentage of investment (even if still profitable for blockbusters). This traditional approach might be more in tune with who actually has more determining power in the prescribing patterns of African Americans. A limitation in relying on this website, DTC ads, and the community education in black churches is that they rest on the assumption that doctors listen to their black patients in the same way that they do their white patients, that ‘patient empowerment’ to demand drugs works whether the patient in

⁴³⁹ <http://www.hearthealthheritage.com/index.asp>.

⁴⁴⁰ Dan Devine, “Heart pill maker slashes sales staff,” *Bay State Banner*, October 19, 2006.

question is imagined to be a ‘compliant’ one or a ‘drug-seeking’ one.⁴⁴¹ The difference in valence between a ‘typical patient’ and a ‘typical black patient’ is one that has threaded through this dissertation.

Drugs do not always reach their markets

Although it is too early to say that BiDil has been a commercial failure, its success has been slow if it is to come. BiDil’s new CEO has tried to look at the positive, saying: “One way is to say, ‘Gee, I am very disappointed we only have about 3 percent of eligible patients taking BiDil. The other way is, very optimistically, to say: ‘I have 97 percent of patients to penetrate.’”⁴⁴² NitroMed’s stock had fallen by an order of magnitude from its high point just after BiDil was approved to the following August (\$27/share to \$2.50), and the business pages that had described it as a model of the future of medicine have radically changed their tune.

Many of the reasons that have been given for why BiDil has not been a commercial success so far are connected with its list price, which is widely considered to be high at over \$10 a day. But given the complexity of drug pricing on the one hand and the role of price in the fetishism of commodities on the other, explanations cannot simply rest on price.

There is no necessary relationship between low price and high demand. For some drugs, like the breast cancer drug trastuzumab (Herceptin), extraordinarily high prices of

⁴⁴¹ “Typical black patient” connects to the description that Richard Allen Williams gave of the “typical” black hypertension patient who is both sick and heretofore poorly cared for, discussed in Chapter 4.

⁴⁴² Steven Syre, Knight Ridder Tribune Business News, *Boston Globe*, Aug 10, 2006, pg. 1.

tens of thousands of dollars for a course of treatment do not serve to squelch demand. Demand for Herceptin is not limited to the advanced metastatic disease for which it was approved, but there is also demand for access to the drug for treatment of early stages.⁴⁴³ Its contribution to survival is less impressive than that of BiDil, but its price has had no apparent downward effect on demand. This is because when it comes to breast cancer, societal willingness to pay is essentially infinite. Price is irrelevant for those drugs understood to be absolutely vital. No matter what price the drug companies assign, people (and their insurance companies) will still buy Herceptin. Indeed, high price can add to the allure of a drug, as discussed in the previous chapter—it must be the best if it is the most expensive.

Some of the complexities of price and drug success are captured by the *Wall Street Journal*, which reports:

Yet after more than a year on the market, BiDil is reaching only about 1% of the 750,000 African-Americans who suffer from heart failure.

Sluggish acceptance for a new drug is not unusual. But BiDil's rollout sharply illustrates how the interests of patients, insurers and drug companies can easily collide. In this case a company's decision to price a drug steeply, resistance by insurers, and a change in federal policy all proved to be impediments. The drug's unavailability, say some medical experts, may also be symptomatic of a deeper problem in the health-care system, where issues affecting minorities and the poor sometimes fall through the cracks.⁴⁴⁴

Moreover, the *Wall Street Journal* reported, although noting high price concerns for a drug that combines two generics, they report that most black patients aren't getting the treatment at all – branded or generic. The figure from the *Bay State Banner*, and

⁴⁴³ Indeed, access to it even for early disease has been framed as a human rights issue in lawsuits in the UK. See for example, Sarah Lyall, "British Clinic Is Allowed to Deny Medicine," *New York Times*, February 16, 2006, p. A6.

⁴⁴⁴ Sylvia Pagan Westphal. "Tough Prescription: Heart Medication Approved for Blacks Faces Uphill Battle; As Insurers Debate Costs And Generics Loom, BiDil Fails to Reach Needy; The Role of Medicare Part D," *Wall Street Journal*, Oct 16, 2006, p. A.1.

presumably also from NitroMed, is 6% of the 750,000 receiving either branded or generic.⁴⁴⁵ If price were the barrier, rather than the story told by BiDil, then why are physicians not adopting the generics in droves?

Considering the very low rate of adoption of the generics here, we can see that the NPR story on the topic was a bit misleading. When reporting on the “dismal” rates of BiDil prescriptions so far, NPR reported that doctors are prescribing the generic components instead, without mentioning that the percentages are very small.⁴⁴⁶ The host said that “that hasn’t sat well with the NAACP.”⁴⁴⁷ Thus, the response from the NAACP is framed as if what is at stake is whether generics are as good or less than, which we have understood in the previous chapter as caught up in the aspect of the commodity fetish that imputes more value to what is more expensive. NAACP’s Juan Cofield is quoted on that program, saying “This is a serious enough issue that we are not going to sit back and just let it happen, or fail to happen.” The host went on to mention the “1.5 million dollar partnership” between the NAACP and NitroMed, before moving on to discuss whether race is “in some way biological.” But this left hanging the question of whether blacks are being denied access to lifesaving drugs in a way that whites would not be.

Indeed, lack of access to BiDil has been used as evidence of poor treatment of African Americans:

⁴⁴⁵ Dan Devine, “NAACP goes to the grassroots for BiDil,” *Bay State Banner*, Oct 5, 2006, p. 7.

⁴⁴⁶ Sarah Varney, “A Drug Just for Blacks?” *National Public Radio: News and Notes*, December 13, 2006, available at: <http://www.npr.org/templates/story/story.php?storyId=6618872>.

⁴⁴⁷ The NAACP partnership has occasioned considerable press, and the story told is a more interesting one than a simple use of the recognized civil rights name providing cover for NitroMed. NAACP President and Chief Executive Officer Bruce Gordon is quoted in the *Baltimore Afro-American*: “We have been on a health care march for some time. ... African Americans as a community do not have all the equal-access opportunities to health care capabilities in this country.” Zenitha Price, “NAACP to partner in bridging health care gap,” *Baltimore Afro-American*, Dec 17-Dec 23, 2005.Vol.114, Iss. 18; pg. A1.

If that were white patients, nobody in America would tell them, ‘Excuse me, I want you to go buy a drug for angina and another for hypertension, and I want you to go home and cut them, and I want you to take multiple pills a day on top of all the [other drugs] you got,’ says Gary Puckrein, a NitroMed shareholder and executive director of the National Minority Health Month Foundation, a nonprofit organization that has received educational grants from the company. If the medical establishment ‘were doing it to whites there would be an uproar,’ he says.⁴⁴⁸

There is something disingenuous about these comments given that these patients are on many, many pills a day, not just two or one. The added benefit from the pill’s combination aspect may not be worth a great deal, but the added benefit of the pill over previous standard of care is significant. In the prevention of hospitalization and death, BiDil remains clearly “cost-effective” even if “overpriced.”⁴⁴⁹ For Puckrein, claiming value for convenience for black patients becomes a consumerist citizenship demand.⁴⁵⁰

Not everyone agrees that high value necessarily justifies high price. Charles Curry, a prominent Howard University cardiologist who had minor participation in A-Heft and has been a NitroMed speaker, said on NPR both that BiDil was one of the “most

⁴⁴⁸ Sylvia Pagan Westphal. “Tough Prescription: Heart Medication Approved for Blacks Faces Uphill Battle; As Insurers Debate Costs And Generics Loom, BiDil Fails to Reach Needy; The Role of Medicare Part D,” *Wall Street Journal*, Oct 16, 2006. pg. A.1. “With insurance plans balking, BiDil’s sales of \$5.2 million in the first half of this year were significantly below analysts’ prelaunch estimates, which had the drug achieving around \$130 million in sales for 2006. NitroMed’s stock has plummeted to under \$3 from about \$23 a share at the time of the launch of BiDil, its only product on the market.”

⁴⁴⁹ Derek Angus, “Heart Failure; Study finds that African-American heart failure drug is cost effective,” *Heart Disease Weekly*, Jan 8, 2006, 84.

⁴⁵⁰ This could be productively brought into dialogue with Rose and Novas: “Another page suggests that there may be differences between brand name Prozac and its generic equivalent, fluoxetine hydrochloride, explaining to potential customers that there is no such thing as ‘generic Prozac’ – for example they come in different packaging - and that if they feel uncomfortable about changing to a generic, they should ask their doctor to prescribe brand name Prozac (www.prozac.com/generic_info.jsp). What kind of scientific literacy is being promoted here? What kinds of active biological citizens are being shaped, and to what ends? This is the citizenship of brand culture, where trust in brands appears capable of supplanting trust in neutral scientific expertise. The weaving together of Eli Lilly’s commitment to education and brand marketing gives us the title of this section of our paper – from public value to biovalue – for this is just one example of the way in which biovalue is supplanting public value in the biological education of citizens-consumers.” Nikolas Rose and Carlos Novas, “Biological Citizenship,” for Aihwa Ong and Stephen Collier, eds., *Global Anthropology*, Blackwell, 2003. Accessed at: <http://www.lse.ac.uk/collections/sociology/pdf/RoseandNovasBiologicalCitizenship2002.pdf>, p. 17.]

valuable” drugs to come on the market in his long career since its improvement of survival rates is so much higher than most in cardiology, and criticized the drug maker for the high price.⁴⁵¹ He spoke of “practical” doctors using generics instead.⁴⁵² Yet the head of the NMA was described as saying that even with its high cost, BiDil could be cost-saving by keeping patients out of the hospital and allowing some to go back to work.

And so the market’s willingness to pay is up for debate, and could change in response to political pressure. But the pharmaceutical company’s sponsorship of physicians and civil rights organizations turns out not to be able to buy sufficient political pressure to force insurers and the government to pay.

From the beginning of BiDil’s racialization, ideas about the nature of the market have led the way.⁴⁵³ As Kahn among the other many critics have pointed out in the lead-in to BiDil, A-HeFT was not designed to actually show black/white differences or target any genetic markers presumed to be associated with race. Rather, it was designed around a market. As sociologist Troy Duster put it in the popular press:

While the new mantra of biotechnology is to claim that pharmaceuticals will someday soon be marketed to individuals based upon their DNA, the fundamental truth is that selling drugs is about markets. These markets are not about individual

⁴⁵¹ Joanne Silberner, “Race-Specific Drug Comes In at High Cost,” *NPR: All Things Considered*, July 12, 2005.

⁴⁵² Curry also said, elsewhere: Charles Curry, the recently retired head of cardiology at Howard University Hospital, was concerned about racial profiling, but he supplied patients for the BiDil trial and was listed as a coinvestigator. “I objected to the hypothesis that blacks have a ‘sick’ blood vessel—that their veins and vessels are different,” Curry said. “I still don’t believe it. Then I decided that it [the drug trial] can’t hurt. These were very sick patients, and they improved their life expectancy. Sometimes I sound like I’m flip-flopping. But I bet the data will show that BiDil helped in lowering hypertension.” The connection between hypertension and heart disease, he noted, is somewhat different among blacks and whites. ““Human, Study Thyself: Learning Series: Genes, Race, and Medicine,” *Discover*, Vol. 26 No. 03, March 2005.

⁴⁵³ Jonathan Kahn and Pamela Sankar, “BiDil: Race Medicine or Race Marketing?” *Health Affairs* 24 (Jul-Dec 2005): 455-463.

designer drugs, but about groups and population aggregates that become the target market.⁴⁵⁴

Duster is right that treating blacks as a target market is a practice we are accustomed to. Why has it not successfully been reached in this case? The assumption that Duster and other commentators are making is that drugs naturally reach their markets.

Heart failure as a disease category may be ripe for pharmaceutical development, although unlikely to produce blockbusters, but it is not necessarily ripe for identity practices around race.⁴⁵⁵ In the lead-up to BiDil, there was alignment of interests by NitroMed and ABC, but they were not necessarily seeing BiDil as a solution to the same problem. For NitroMed, the principal problem was how to get approval for the drug combination in a way that would be profitable. For ABC, the problem was and is more diffuse: how to get the funding to run trials and thus participate in the construction of evidence based medicine, and how to find solutions for black morbidity and mortality from heart failure.

In the A-HeFT trial, stakeholders who were not necessarily previously aligned came together around a practical project. The “pragmatism” here is like that of those working on the basic science of cancer in Joan Fujimura’s work. Heart failure in African Americans was a problem that was “ripe,” or “both intellectually interesting and doable,” in Fujimura’s terms. As she points out, the doability does not rest only on the technical (whether in her case, of oncogenes, or this one, a racially specific trial).

⁴⁵⁴ Troy Duster, “Medicine and People of Color: Unlikely Mix – Race, Biology, and Drugs,” *San Francisco Chronicle*, Monday, March 17, 2003, p. B7.

⁴⁵⁵ Perhaps part of the failure of BiDil is about the limits of Illness as Metaphor in Sontag’s terms, a desire to see the disease as the abjection it is beyond language and not immediately turn it into stories for society beyond the suffering individual. Just because cancer takes an interest in you, do you have to take an interest in cancer? Just because heart failure takes an interest in you, do you have to take an interest in heart failure?

Whether the A-HeFT trial was an optimal way to get at efficacy or not, it had been a way to make a “doable problem” out of a conundrum.⁴⁵⁶ Or in this case, two conundrums: salvaging a drug, and serving the professional needs and patient populations of African American cardiologists. The black trial is not only “production,” but also “articulation,” of both problems and potential solutions. The FDA, clinical researchers, and drug companies make problems doable by reorganizing their work. What made African-American heart failure ripe for NitroMed, ABC, and the FDA was the deluge of data around African-American responses to ACE-inhibitors. The A-HeFT investigators framed their project quite unambitiously, adding a quality of life score in addition to the harder endpoints of death and hospitalization because they did not expect so robust a response on the latter. But the articulation of BiDil exceeds that in the study around which ABC, NitroMed, and the FDA intersected, and its poor success to date may be connected to difficulties in that articulation.

Both NitroMed and its critics dramatically overestimated the amount of money that would be made after BiDil’s approval. Where Kahn has been critical of dramatic overreporting of the death rate disparity as sensationalism that plays into ideas of biological race that suit pharmaceutical approaches, he and coauthor Sankar have indulged in overreporting the money that stands to be made. This feeds a parallel aspect of the hype. Kahn and Sankar report uncritically the hype of the NitroMed CEO:

Shortly before the FDA approval, NitroMed had predicted that BiDil revenues could reach \$120 million in its first year of sales, increase to \$350 million within a few years, and conceivably top \$ 1 billion annually. This projection, by

⁴⁵⁶ Joan Fujimura, “Constructing ‘Do-Able’ Problems in Cancer Research: Articulating Alignment,” *Social Studies of Science*, 17 (May, 1987): 257-293. Joan Fujimura, *Crafting Science: A Sociohistory of the Quest for the Genetics of Cancer*, Cambridge, MA: Harvard U Press, 1996.

NitroMed chief executive officer Michael Loberg, was based in part on comparison to an existing heart failure drug, Coreg, which cost \$3.56 per day.⁴⁵⁷

Kahn and Sankar then suggest that the CEO had actually underreported the amount of money that stands to be made:

A week after the FDA approval, BiDil's projected market opportunity nearly tripled to \$3 billion as NitroMed announced BiDil pricing at \$1.80 per pill, or \$10.80 per day, based on the target dose of six pills per day. This dwarfs the estimated cost of generic equivalents at \$0.25 per pill.⁴⁵⁸,⁴⁵⁹

Although Kahn and Sankar begin by reporting of hype, they go further and engage in hype as they use the highest possible figures to make their estimates. Further, they suggest that part of the problem with BiDil's "success" is that it will "set in motion a trend in the pharmaceutical industry for turning other widely used and cost-effective generics into patented, expensive drugs in the name of alleviating disparities."⁴⁶⁰ But is this such an easy task to accomplish?

Kahn is also concerned about the money that investigators stand to make:

Make no mistake, BiDil will benefit heart patients. Some of them will be black. Cohn, the drug's champion for nearly thirty years, has achieved a valuable objective. He also is likely to make a lot of money. Indeed, in addition to royalties and licensing fees, Cohn received \$1 million in milestone payments for approval and first marketing of BiDil. Most of the other coauthors of the article announcing AHeFT's findings also could benefit from a BiDil launch. Nine of eleven have direct financial ties to NitroMed (K. Ferdinand and M. Taylor do not). Some report receiving research funding, and several disclose that they are stockholders. Careful management of these conflicts likely protected A-HeFT data from bias. However, conflicts of interest do not influence research only by biasing results. They also can assert themselves into research by allowing potential financial gain to distort the design and trajectory of research in ways that might ultimately

⁴⁵⁷ Jonathan Kahn and Pamela Sankar, "BiDil: Race Medicine or Race Marketing?" *Health Affairs* 24 (Jul-Dec 2005): 458.

⁴⁵⁸ Jonathan Kahn and Pamela Sankar, "BiDil: Race Medicine or Race Marketing?" *Health Affairs* 24 (Jul-Dec 2005): 458.

⁴⁵⁹ Note that even this extraordinarily optimistic maximum number for amount of money standing to be made is more than an order of magnitude less than Lipitor's revenue of \$12.2 billion last year.

⁴⁶⁰ Jonathan Kahn and Pamela Sankar, "BiDil: Race Medicine or Race Marketing?" *Health Affairs* 24 (Jul-Dec 2005): 455.

undermine broad and affordable access to important therapies that help eliminate health disparities.⁴⁶¹

Here, Kahn and Sankar's cagey implication is that pharmaceutical companies are buying off otherwise good investigators and organizations. This kind of implication is one that has fostered ill will in the debate, and is connected to what we saw in the previous chapter about doctors' defensiveness about whether the money they receive colors their expert judgment.

Physicians often respond to critiques of their participation in gift-exchange with pharmaceutical companies with righteous indignation, that *they* would never be swayed by such things.⁴⁶² But what makes the gifts powerful is precisely this space: they might well believe, or they might not. Critics of BiDil address ABC and NAACP comments as if they are simply paid ventriloquists for NitroMed, but there are multiple sets of stakes here.

There should be room for some conceptual space here between being bought off and, in a limited way and with an independent voice, buying in. This is not to diminish the power of the gifts circulating here, but rather to capture the way that gifts' power is actually more interesting than a simple purchase of support. Mauss taught us that there are no free gifts,⁴⁶³ and funding for the physicians' organization that supports a drug trial

⁴⁶¹ "BiDil: Race Medicine or Race Marketing?" *Health Affairs* 24 (Jul-Dec 2005).

⁴⁶² It is useful to divide the grounds of critique of the physicians' practice into two parts. Karl Manheim, in *Ideology and Utopia*, divides ideology into two meanings: particular (directed at an opponent) and total (referring to an era or group). Where particular ideology assumes common criteria with the opponent, the total assumes fundamentally different grounds and analytic systems. These two kinds of allegations of ideology often become the sites of debate over pharmaceuticals: on the one hand critiques of fraud or corruption in some particular case and on the other hand critique of the general ideology of when-in-doubt-drug. It is important not limit our understanding of the ideology of pharmaceuticals in only Manheim's particular sense.

⁴⁶³ Marcel Mauss, *The Gift: Forms and Functions of Exchange in Primitive Societies* (Glencoe Ill: Free Press, 1954).

comes at a cost. And yet this critique applies not just to BiDil and ABC, but to the more general alliance of pharmaceutical-funding (or government-funding), clinician-scientists, and social justice organizations that campaign around disease. For both physicians and their patients, the power of gifts is salient: the gift is something that at once should be received, and yet which is dangerous to take. When NitroMed, ABC, and NAACP align, more than mere utility circulates, obligation and liberty intermingle, the force of the objects of gift-giving are both practical and mystical.

Polyvalence of drugs as remedy and poison

As discussed in the previous chapter, the meaning of a given drug exceeds that given by its marketers or medical experts. In the case of BiDil, the meanings that have circulated around the drug have been polarized and volatile. This situation is not, of course, unique to BiDil. As Asha Persson has pointed out, “For all their innocuous appearance, every pill is a potent fusion of ingredients, including scientific practices, political agendas and commercial interests, along with social activism and media spin.”⁴⁶⁴ She turns to “the original Greek word for drug, *pharmakon*, meaning both ‘remedy’ (medicine, cure) and ‘poison’ (toxic), but also ‘a means of producing something’” to understand the interpretive flexibility around the often excruciatingly visible side effects of anti-

⁴⁶⁴ Asha Persson, “Incorporating *Pharmakon*: HIV, Medicine, and Body Shape Change,” *Body and Society* 10 (2004): 46.

retrovirals against the invisible but medically salient decrease in the viral load of HIV.⁴⁶⁵

The *pharmakon* is also a useful theoretical tool for considering BiDil.

Derrida, writing about the polyvalent meanings of the Greek term *pharmakon*, critiques the translation of it as “remedy,” with its sense of inherent beneficence.⁴⁶⁶ He argues, rather, that it cannot be so constrained: “As opposed to ‘drug’ or even ‘medicine,’ *remedy* says the transparent rationality of science, technique, and therapeutic causality, thus excluding from the text any leaning toward the magic virtues of a force whose effects are hard to master, a dynamics that constantly surprises the one who tries to manipulate it as a master and a subject.”⁴⁶⁷ Poison and remedy are inseparable parts of the *pharmakon*.

Mike Fortun has described the *pharmakon* in relation to DeCode genomics as a promise, as a gift/poison, and the difficulty of separating out the *pharmakon* from the Pharma Con.⁴⁶⁸ Similarly, Tavis Smiley has described the problem of BiDil as one of separating hope from hype.⁴⁶⁹ But such a separation is impossible.

⁴⁶⁵ Asha Persson, “Incorporating *Pharmakon*: HIV, Medicine, and Body Shape Change” *Body and Society* 10 (2004): “Drugs, as is the case with antiretroviral therapy, have the capacity to be beneficial and detrimental to the same person at the same time.”

⁴⁶⁶ Jacques Derrida, “Plato’s Pharmacy,” in *Dissemination*, Translated, with an Introduction and Additional Notes, by Barbara Johnson (Chicago: University of Chicago Press, 1981). There are even more definitions than Derrida fully explores. The *pharmakon* is also, according to the Tufts *Greek-English Lexicon*: (1) drug, whether healing or noxious; (2) healing remedy, medicine; (3) enchanted potion, philter, hence charm, spell; (4) poison; (5) lye for laundering; II generally remedy, cure; (2) means of producing something; (3) remedy or consolation in; III. Dye, paint, color; IV chemical reagent used by tanners.

⁴⁶⁷ Jacques Derrida, “Plato’s Pharmacy,” in *Dissemination*, Translated, with an Introduction and Additional Notes, by Barbara Johnson (Chicago: University of Chicago Press, 1981), 97.

⁴⁶⁸ Mike Fortun, *Promising Genomics: Iceland, DeCode Genomics, and a World of Speculation*, Forthcoming. As Fortun demonstrates, promises are complex. We might consider critics of BiDil’s use “promise” in a way that seems to be a poetic way to say portend or predict, in for example Dorothy Roberts’ words: “Because federal patent law permits the PTO to issue patents on ‘anything under the sun made by man’ race-based pharmaceuticals promise to be a lucrative field of invention.” Dorothy Roberts, “Legal Constraints on the Use of Race in Biomedical Research: Toward a Social Justice Framework,” *Journal of Law, Medicine and Ethics* 34 (Fall 2006): 529. But biotechnology companies cannot always deliver on their promises.

⁴⁶⁹ Tavis Smiley, “Dr. Ian Smith: Race-Specific Drugs,” *NPR: The Tavis Smiley Show*, November 17, 2004.

BiDil has promised remedy on (at least) three registers: survival and quality of life benefit of the pill for individual patients, a potential mechanism in progress toward the alleviation of health disparities, and as a path toward pharmacogenomics. Similarly, the danger associated with BiDil has been on (at least) three registers: that of potential literal toxicity of the pill, that of harm to the cause of anti-racism in medicine and society, and that of profits for amoral drug companies at the expense of suffering patients.

A good deal of discursive boundary work has gone into trying to address the different registers of remedy and harm separately. For example, commentators such as Kahn want to accept the physical benefit of the pill for individual patients while rejecting the claim that it leads to progress in race relations or medicine. Perhaps part of the reason that Kahn so often gets criticized for wanting to deny the pill to patients in need is that these registers cannot quite be separated. The *pharmakon* is messy.

A *Black Enterprise* article on the BiDil connects the debate over race in research with the safety of the drug itself:

Since being approved by the U.S. Food and Drug Administration in June, BiDil, the first drug marketed specifically for treatment of heart failure among African Americans, has aroused suspicion about its safety and set off a debate about attitudes surrounding race and medical research.⁴⁷⁰

The ready association between drugs for blacks and lack of safety suggests that there may be a symbolic mistake⁴⁷¹ in targeting drugs at African Americans. This is in part because the associations between race and drugs (unlike gender and drugs) are overwhelmingly

⁴⁷⁰ Ron Chepesiuk and Joyce Jones, "Are Race-Specific Drugs Unethical?" *Black Enterprise*, Nov. 2005, pg. 37.

⁴⁷¹ For consideration of the concept of "symbolic mistake," see: Nathan Greenslit, "Pharmaceutical Branding: Identity, Individuality, and Illness," *Molecular Interventions*, 2:342-345 (2002); Nathan Greenslit, "Depression and Consumption: Psychopharmaceuticals, Branding, and New Identity Practices," *Culture, Medicine and Psychiatry* 29 (December 2005), 477-502.

illicit.⁴⁷² Can a pharmaceutical emerge well out of this context? Moreover, there is another, related resonance of drugs and race, that leads to another kind of symbolic mistake. In the context of Tuskegee and rumors of crack as a government invention to suppress Black Power, there is a sinister connotation to “a drug for black people.” A cartoon from the K Chronicles captures this tangled genealogy:



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⁴⁷² A quick google search for “African Americans” and “drugs” does successfully pull up both BiDiL and an NPR story about it, as well as a reference to the Medicare drug benefit on the first page of results. But the other seven entries direct the searcher to the racism of the drug war, drugs and crime, drugs to be overcome through spirituality, and drugs and AIDS.

⁴⁷³ Keith Knight, “BiDiL Me This, Man,” available at <http://www.buzzle.com/showImage.asp?image=3273>.

The potential unpalatability of a black drug was not predicted by many critics or proponents of BiDil. On the contrary, social science critic George Ellison said:

There is a political demand for treatments for African Americans in America that makes the production of an ethnic drug for African Americans a highly palatable political event. At the same time, as we're well aware within our racialised societies, African Americans have a social and political identity that makes them a powerful lobbying group and a powerful market in their own right. So it's possible to develop a drug for African Americans because we can identify them, we can market to them, we can sell it to them, we can justify it to them, and that makes it a commercially sensitive, commercially successful enterprise.⁴⁷⁴

Yet, as its poor market performance so far should alert us, BiDil's "palatability" is not so clear. Indeed, I argue, some unpalatability of a "black drug" is overdetermined. Black drugs connote black markets. Even licit drug markets are narrated as shady in BiDil's case, as debate around BiDil's approval renders visible both what would have otherwise been a black box around its own contested approval and the unsavory interests underlying all drug approval processes.⁴⁷⁵ And "black drugs" are for black people, who are not generally characterized as ideal patients. First, neither drug companies nor the government are necessarily trusted authorities for this population.⁴⁷⁶ Moreover, in the

⁴⁷⁴ George Ellison, as heard on Kenan Malik, "A Colour Coded Prescription," BBC Radio 4, November 17, 2005. http://www.kenanmalik.com/tv/analysis_race+medicine.html

⁴⁷⁵ As Arjun Appadurai points out, shifts in the form of commoditization toward a more profitable form are common, but that perceived diversion of commodities from their normal channels bears a taint: "The diversion of commodities from their customary paths always carries a risky and morally ambiguous aura...the spirit of entrepreneurship and that moral taint enter the picture simultaneously." Arjun Appadurai, "Introduction: Commodities and the Politics of Value," in *The Social Life of Things*, edited by Arjun Appadurai (Cambridge, UK: Cambridge University Press, 1988): p. 27.

⁴⁷⁶ See, among other sources, W. Michael Byrd and Linda A. Clayton, *An American Health Dilemma: Vol. 1: A Medical History of African Americans and the Problem of Race: Beginnings to 1900* (New York: Routledge, 2000).

medical literature, black patients are framed overwhelmingly as refractory, noncompliant, and untrusting.⁴⁷⁷

FDA approval does not by itself determine drug palatability in a differentiated society. We might read FDA approval as certification of edibility, albeit one not always trusted as completely reliable. Yet what is “good to eat” is not a category as simple as edibility, but rather is connected (as we learn from Levi-Strauss) with what is “good to think.”⁴⁷⁸ As we have seen above, BiDil was “good to think” from the perspective of turn-of-the-millennium biotech investors and the FDA. But do many want to swallow the black pill?

It is not altogether clear, a priori, whether more access to drugs is evidence of discrimination – as it is in the distribution of crack sales, or prescriptions for haldol⁴⁷⁹ – or whether less access to drugs is, as it is in medical disparities literature more generally. Indeed, both kinds of indictments can operate together. Less access to antihypertensives and statins leads to more heart failure – and hence, potentially a role for BiDil.

Moreover, the dangers pointed to in raising the specter of race and drugs together are not necessarily understood as a contraindication for a racial drug. When Kahn describes the ABC and Congressional Black Caucus support for the “purportedly benign” use of race as biology in BiDil, he operates on the assumption that if its malignance were

⁴⁷⁷ See, among other sources, David Barton Smith, *Health Care Divided: Race and Healing a Nation* (Ann Arbor: University of Michigan Press, 1999).

⁴⁷⁸ As categories of food fit with the categories of society, categories of drugs must as well. Categories of different drugs for different groups do not only divide, they also unite all groups under an overarching pharmaceutical grammar. Claude Levi-Strauss, *Totemism*, trans. Rodney Needham (Boston: Beacon Press, 1963), 89.

⁴⁷⁹ Jonathan Metzl, “Race, Stigma, and Schizophrenia,” presented at *Race, Pharmaceuticals and Medical Technology*, MIT, Cambridge, MA, April 8, 2006.

known it would be rejected.⁴⁸⁰ But the dangers raised by the specter of BiDil can be read as analogs to a drug and its side effects. The poison that is part of the *pharmakon* is not separate from its efficacy, but immanent with it. This helps understand why a low side effect profile does not guarantee that a drug will be taken. Indeed, as we saw with thiazide, a low side-effect profile for a drug for an asymptomatic condition can inhibit compliance. If a drug causes no harm, it may not have any effect at all—*no pain, no gain*. Side effects, then, can either inhibit or inspire compliance. We can see a lateral but evocative parallel with HAART, as described in Persson: for some people the evidence of the drugs' toxicity on their bodies was evidence of its effectiveness against the otherwise invisible virus.⁴⁸¹

Sense of toxicity can add to the appeal of a *pharmakon*. The toxicity of the connection between race and blackness suggests, for some, that the unease must be worth it. The problem of black disease is widely recognized as serious, and yet hard to get a handle on. The cure might well need to be bad, too.

Many supporters of BiDil have expressed anxiety that social science and other critiques of the drug will make people afraid of it, and thus prevent access to the remedy it offers. They want to suppress expression of the poison aspects of this *pharmakon*. As in the opening vignette, there is a rhetorical strategy that suggests that the danger comes not from the drug itself but from the lack of consensus about its unqualified beneficence. Conservative commentator Steve Sailer concludes his defense of the use of race and medicine in research (contra the PBS series "Race: the Power of an Illusion") with an

⁴⁸⁰ Jonathan Kahn, "How a Drug Becomes "Ethnic": Law, Commerce, and the Production of Racial Categories," *Yale Journal of Health Policy, Law, and Ethics* 4 (2004): 33.

⁴⁸¹ Asha Persson, "Incorporating Pharmakon: HIV, Medicine, and Body Shape Change," *Body and Society* 10 (2004): 45-67.

accusation of violence perpetrated by those he calls “race flat-eathers:” “So for the foreseeable future, knowledge of race will play a highly useful role in saving lives - unless PBS and the Ford Foundation have their way.”⁴⁸²

Not all pro-BiDil voices employing this type of narrative approach are necessarily neoconservative. For example, an article from the black newspaper the *Jacksonville Free Press* argues against rejecting racial profiling in medicine:

If African-Americans suffering from heart problems benefit from BiDil, it should be celebrated instead of feared. The Human Genome Project, which mapped human DNA, found that humans share more than 99 percent of the exact same genes, but that leaves room for differences. Should a drug be withheld simply because it may play into the fear of a racist agenda?

Deliberately targeting someone for mistreatment or unmerited benefit due to the color of their skin is abhorrent, but the concept of racial profiling has acquired an unwarranted bad reputation over the past few years. It may now be a tool for improving the health of African-Americans. In the case of BiDil, thinking along racial lines is saving lives.⁴⁸³

Although the toxic effects of BiDil as racialized drug are operating at a different register from its effects as medical remedy, there is often conflation in the debate between those who critique the way that BiDil is framed with regard to race and those who would deny access to the actual pills. (This puts the BiDil critics in an odd position, as Kahn for example becomes an advocate for the chemical compounds and starts to say that the problem with BiDil might be that too few white people will be prescribed a drug that might benefit them. If the drug is effective, it should be approved for everyone.)

There has been a frustrating tendency for the debate around BiDil to be framed as “between” “science” and “social science” that is related to this suggestion that social

⁴⁸² Steve Sailer, “Race Flat-Earthers Dangerous To Everyone’s Health,” http://www.vdare.com/sailer/medicine_and_race.htm.

⁴⁸³ Sammons, Leah, “Racial Profiling: Not Always a Bad Thing,” *The Jacksonville Free Press*. Feb 16-Feb 23, 2006. Vol.20, Iss. 4; pg. 4.

scientists want to deny access to medicine for suffering patients. Epidemiologist Nancy Krieger comments on this inaccurate representation by calling us to look at the “stormy” situation at the intersection of race, genetics, and health disparities. She chooses this metaphor because “Storms, after all, are violent disturbances of the atmosphere resulting from the movement and collision of masses of warmer and cooler air. Arguments in turn are often polemically portrayed as ‘hot air’ and impassioned beliefs versus ‘cool reason’ and scientific logic.”⁴⁸⁴

Bioethicist Rick Carlson has epitomized this representation, which also employs the be-not-afraid-of-science rhetorical trope, and suggests that if social scientists didn’t get in the way, science and the market would be able to proceed more quickly in helping patients.⁴⁸⁵ He supports even the most crass and unscientific of clinical methods, arguing that if something simple like race explains something, and helps doctors to help patients, then “don’t keep looking.” However, the debate has been more accurately characterized when it has been seen not as between “scientists” and “social scientists,” but as among and between “scientists” and “clinicians.”⁴⁸⁶ The debate has generally been between scientists – whether of a social or genetic ilk – on the one hand, and clinicians on the other, with considerable heterogeneity in each group.

We need to be skeptical of claims that what is holding us back from solving racial disparities is squeamishness or fear of learning all we can about race. If Foucault is right that we need to question the tendency in the West to see power only in its negative

⁴⁸⁴ Nancy Krieger, “Stormy Weather: *Race*, Gene Expression, and the Science of Health Disparities,” *American Journal of Public Health* 95 (December 2005): 2155.

⁴⁸⁵ Rick J. Carlson, *Health Affairs*, “The Case of BiDiI: A Policy Commentary on Race and Genetics: The Debate Between Sociologists and Biologists Is Just the Beginning, When It Comes to Genomics And Race.” 24 (Jul-Dec 2005): pp. 464-468.

⁴⁸⁶ Joe Palca, “FDA Panel Weighs Heart Drug for Blacks,” *NPR: All Things Considered*, June 16, 2005.

manifestations, in what it prohibits, we need to consider too what it incites.⁴⁸⁷ This knowledge-against-repression framework is a familiar narrative trope of the history of medicine. In *The Birth of the Clinic*, Foucault argues compellingly that the rise of clinical medicine was not simply the rise of empiricism, but rather a reorganization of what was knowable. He argues that the move was not, as it claimed, from imagination to reason, but rather a change in the structure of the knowable that turned on the relationship between the visible and the invisible. This change made it possible to turn the patient's bed into a field of scientific investigation. Though the gaze claimed to let things come up to the surface to be observed without disturbing them, it actually entailed a reorganization of the depth of the body not only for medical discourses but also for the possibility of discourse about the body and about disease.

The attention to taxonomies of truth here should alert us to the peril of dwelling too long on any given scientific 'fact' about race, refuting each one in the hope of boxing the specter down. The problem in race in medicine is not locatable in any particular statistic or physiological claim, but in organizing the stakes in race and medicine around science or its repression. Foucault's interest is not so much in the actual truth claims, but in the "regimes of truth," or the grounds on which statements could be seen as true or not true. Foucault argued that this consensus about the beneficence of the medical gaze was also happening in the polity: liberation from tyranny and illness could be achieved through the shining light of the unyielding gaze. In debates about race and medicine, faith in or skepticism of the libratory capacity of the gaze is part of differentiating the terrain.

⁴⁸⁷ Michel Foucault, "Truth and Power," in *Power/Knowledge : Selected Interviews and Other Writings, 1972-1977*, edited by Colin Gordon, New York, N.Y.: Pantheon Books, 1980.

Skepticism or embrace of BiDiI can be part of a skepticism or embrace of the beneficence of medicine toward marginalized groups in liberal society. Foucault argued that two great myths emerged about the role of medicine in liberal society: a nationalized medicine would be organized like clergy to care for the population, and the contradictory myth that it would be possible to reach a state of total disappearance of disease.⁴⁸⁸ Liberalism in politics and in medicine had a convergence that liberation could be achieved through shining the light of the unyielding gaze, hiding their actual discursive productions. Critics of the critics of BiDiI often point to the need not to be afraid to open racial difference up for investigation. Leaders of black liberalism have been outspoken in BiDiI's favor, arguing that previous regimes of medical knowledge had rendered the poor health of blacks invisible. These leaders are correct that the status quo is intolerable, but is the repression of knowledge really what is at stake? In a society and medical establishment so visibly founded in slavery and participant in discrimination, faith in its epistemological purity is particularly hard to maintain.

Yet claims about the nature of citizenship are important in the stakes of the argument on all sides. Sharona Hoffman has argued that "race-based" medicine may violate anti-discrimination mandates because they use unreliable race as a basis rather than sound medical indications.⁴⁸⁹ Indeed, at medical conferences the enthusiasm of ABC support has been audibly mitigated by disappointment at narrowness of the applicability of their study compared with all-white studies. If black cardiologists prescribe drugs tested only on white patients for their overwhelmingly black patients,

⁴⁸⁸ Michel Foucault, *The Birth of the Clinic: An Archeology of Medical Perception*, trans. A.M. Sheridan Smith (London: Tavistock Publications, 1973).

⁴⁸⁹ Sharona Hoffman, "Racially-Tailored Medicine Unravelling," *American University Law Review* 55 (2005): 425.

why isn't their trial enough to suggest the drug's benefit for all patients? And so even though the drug gets full official support from ABC, enthusiasm is crucially limited by this problem of the sense that black doctors and black patients cannot represent the norms in clinical trials. In general (though not universally), ABC members have shown what we could read in Goffman's terms⁴⁹⁰ as "front stage decorum" with regard to the press, but the "back stage intimacy" shows considerable ambivalence. The citizenship demands around BiDil can be made in plural directions and on different registers.

BiDil and the move to put race on one side of the material-semiotic

It is helpful to think about the relationship between BiDil and race-as-biology on the same levels that were considered in the previous chapter. If black heart failure is to be "fixed" through this pharmacological intervention, it is in at least three ways: by rendering race identifiably stable, by focusing attention upon it, and by promising easy repair. As we saw in the chapter on African American Hypertension as a disease category, pharmaceuticals are mobilized not only on the basis of existing biological categories, but also on their capacity for changing biology, on the very malleability of race. One aspect of this process is that race is a difference that is imagined to be fixed enough for action, but at the same time potentially able to be medicated away. Thus pharmaceuticals are part of the argument for mutability in the face of longstanding

⁴⁹⁰ Irving Goffman, *The Presentation of Self in Everyday Life* (Garden City, NY: Doubleday, 1959).

practice, not just reification. As a NitroMed CEO has said: “These disparities have been around so long, they risk being accepted as the norm.”⁴⁹¹

Drugs are an easy case for objects that are “material-semiotic” in Haraway’s terms.⁴⁹² That is, people generally accept that drugs have both physical and social aspects. Their ability to move between the material and the semiotic make drugs appealing for invocations in boundary work with regard to other categories on that divide. Drugs are an appealing tool for those who want to get other categories settled onto one side or the other of the material/semiotic. For example, arguments over the ‘reality’ of mental illness are sometimes staked out over the terms of antidepressants. Similarly, the ‘reality’ of race is staked out in terms of BiDil. The move to use BiDil to try to put race onto one side or the other of a material/semiotic divide ultimately fails because BiDil keeps its dual aspect. I argue that STS scholarship must move beyond critique of BiDil’s role in the ‘biological basis’ of race, and attend to the productive work it is doing *at* the social/biological boundary. Because the principal scholar of BiDil within STS has been Jonathan Kahn, the example of his attempts to purge race of material-semiotic ambiguities through critique of this drug will be a central part of this section.

When NitroMed’s critics argue that BiDil naturalizes disparities, there is a disconnect that stems from different assumptions about what race-as-biological must mean. For Kahn among others, race-as-biological means race-as-genetic.⁴⁹³ But it is

⁴⁹¹ Zenitha Price, “NAACP to partner in bridging health care gap,” *Baltimore Afro-American*, Dec 17-Dec 23, 2005. Vol. 114, Iss. 18; pg. A1.

⁴⁹² Donna Haraway, “Situated Knowledge: The Science Question in Feminism as a Site of Discourse on the Privilege of Partial Perspective,” *Feminist Studies* 14 (1988): 575-99.

⁴⁹³ Dorothy Roberts, too, suggests a purification process to put race wholly on the social side, purged of a biological side framed as synonymous with the genetic: “Thus, the legal regulation of biomedical research should discourage or prohibit the use of “race” as a genetic or biological category, but encourage or require the use of “race” as a socio-political category to understand and investigate ways to eliminate disparities in

Kahn, not NitroMed or the ABC, who reduce biology in this way. In this sense, Kahn is an example of the kind of scholarship that Bruno Latour criticizes in *We Have Never Been Modern*. In Latour's terms, Kahn takes modernism at its word that it can reduce the world into clean categories without producing hybrids.⁴⁹⁴ The emergence of BiDil can provoke the uncanny, in Freud's sense, for these scholars because it seems that the repressed genetic inscription of race recurs.⁴⁹⁵ But scientific medicine is not even capable of fully purifying and rationalizing its objects of study into piles of genes – the human organisms that are the necessary intersection between genes and society continually get in the way.

In other sites, such as the regulation of boundaries of membership in indigenous communities and the patenting of their genes,⁴⁹⁶ as well as eugenic projects,⁴⁹⁷ the inscription of race with genetics has mattered very differently. Thus, what might be required is what Alondra Nelson has called for with regard to distinguishing commercial genetic ancestry tests from spheres such as state-sponsored eugenics: “site specificity in race and genetics.” Analyzing different sites of race and genetics should yield both familiar tropes of race and genetics and new ones, and we miss important opportunities for furthering critique of race and medicine if we attend only to those ways that BiDil

health status, access to health care, and medical treatment.” Dorothy Roberts, “Legal Constraints on the Use of Race in Biomedical Research: Toward a Social Justice Framework,” *Journal of Law, Medicine and Ethics* 34 (Fall 2006): 532-533.

⁴⁹⁴ Bruno Latour, *We Have Never Been Modern*, trans. Catherine Porter (Cambridge, MA: Harvard University Press, 1993). Like Boyle's laws, laws of medicine and genetics have not really become universal, merely stabilized in larger social networks.

⁴⁹⁵ Sigmund Freud, *The Uncanny*, trans. David McLintock (London: Penguin, 2003). If race merely happened its presence would not provoke the uncanny, it can only do so after being repressed.

⁴⁹⁶ See Jenny Reardon, *Race to the Finish: Identity and Governance in an Age of Genomics* (Princeton, NJ: Princeton University Press, 2004).

⁴⁹⁷ See Daniel J. Kevles, *In the Name of Eugenics: Genetics and the Uses of Human Heredity* (New York: Knopf, 1985).

allows the repetition of old grammars without attending to how its specific contingency matters. We should attend more closely to the political economy and identity of biological race claims in this context of BiDil. Neither NitroMed nor ABC is actually picky about whether patients or others accept one or another etiology of black heart failure. One thing that both NitroMed and ABC share a concern about is that patients and know that BiDil is part of changing things for the better—and take the drug. For both NitroMed and ABC in this debate, plural arguments about the etiology of black heart failure – not just genetic ones – can provide a basis for arguments to treat it with BiDil.

Interestingly, considering the heavy criticism that NitroMed relies on a genetic notion of racial disparity, their patient education website answers the question: “Why are African Americans at greater risk?” with an agnostic answer: “African Americans are at a much higher risk for heart failure in part because more of them develop high blood pressure and diabetes than other ethnic groups. Some scientists suspect that low levels of nitric oxide, which relaxes blood vessels, may also play a role.”⁴⁹⁸ The disparity statistic

⁴⁹⁸ <http://www.hearthealthheritage.com/risk.asp>. The further discussion, on the next page, also does not frame hypertension as a black genetic disease: “High blood pressure is the most common cause of heart failure in blacks. It affects a greater percentage of African Americans than other Americans. Researchers have also learned that in African Americans high blood pressure tends to cause more damage to their organs, including the kidneys and heart. High blood pressure is called a “silent killer” for good reason. Without regular blood pressure checkups, it can go unnoticed, gradually wearing down your heart and blood vessels. High blood pressure can also run in families, so if your parents or grandparents had this condition, you’re at greater risk for developing it. Taking medication that lowers your blood pressure as needed and adopting some heart-healthy habits can go a long way toward preventing heart failure.” In addition to a section on diabetes, the one on nitrous oxide is also agnostic: “The nitric oxide connection Researchers have also begun to suspect that some aspects of basic body chemistry may work differently depending on ethnicity. This theory, as yet unproven, is based on studies showing that African Americans sometimes respond differently to medications than Americans from other ethnic backgrounds. For example, studies show that African Americans may be more likely to have an impairment in the ability of their blood vessels to relax. Some researchers suggest this may have something to do with the amount of nitric oxide available in their bodies. When blood vessels stay narrow, it can interfere with the heart’s ability to pump blood as well as it should. Nitric oxide is a compound that works to relax blood vessels — including those in your heart — so more blood can flow through as needed.”

NitroMed uses here is not the two-to-one that Kahn has criticized,⁴⁹⁹ but rather that blacks are 50% more likely than the general population to develop heart failure, and to do so younger, more severely, quicker, and with more hospitalization. The causes for the differential are described on the next page: high blood pressure, diabetes, nitric oxide, and “other possible causes” that include less access to health care, more exposure to environmental risks like pollution, and a greater tendency toward being overweight and sedentary.

The eclecticism in this etiology confounds easy divisions between the genetic material of the lived body and the history of the lived body. As Judith Butler reminds us, nature itself is not a given: matter has a history.⁵⁰⁰ In her *Bodies That Matter*, Butler explores the dual meaning of the word “matter”: matter as the stuff of the world and matter as a verb of meaning. Critiquing feminism’s reliance on the division of sex (stuff) from gender (meaning), her goal is to show the immanence and inseparability of meaning and stuff. For Butler, the interaction of meaning and matter is not simply one-way, with discourse shaping all, but rather a dynamic process between regulatory norms of sex—sometimes reached, sometimes contested and resisted—and the performance of identity. In this way, Butler is arguing that the body should be understood not as just meaning or just stuff, but, in Haraway’s terms, as a material-semiotic object.

There is a disconnect in the debate over BiDil in the sense that its critics’ most central worry is about attributing a genetic basis to race while for its proponents that is

⁴⁹⁹ Kahn emphasizes that the statistics do not show that the incidence of heart failure in African Americans is much higher, but rather merely earlier onset and earlier mortality. But isn’t that at least as important difference? If whites die in old age at as high a rate as blacks do, does that mean that we should care less about the real and striking differences among those ‘at the prime of their lives?’ Jonathan Kahn, “How a Drug Becomes “Ethnic”: Law, Commerce, and the Production of Racial Categories,” *Yale Journal of Health Policy, Law, and Ethics* 4 (2004): 21.

⁵⁰⁰ Judith Butler, *Bodies That Matter: On the Discursive Limits of “Sex,”* (New York: Routledge, 1993).

simply not the case. For example, in her book cataloguing abuses of African Americans in medical research, Harriet Washington puts her discussion of BiDil in a chapter called “Genetic Perdition: The Rise of Molecular Bias.”⁵⁰¹ It seems that the purging of genetics from race is the central project of scholarship of race that it becomes a unity ritual in Douglas’ sense, the repetition of the “race is not genetic” mantra becoming a ritual of purity and impurity that creates a unity of experience among scholarly critics, allowing symbolic patterns to be worked out in both instrumental ways (in which laws of nature are dragged in to enforce a moral code) and expressive ways (in which a general view of the social order is culled from untidy experience).⁵⁰² Yet for BiDil’s proponents—with the exception of right-wing pundits and the like—race’s connection with genetics is usually framed as a side issue they are comfortable either dismissing or proclaiming agnosticism on.

Jonathan Kahn has emphasized his concern that BiDil makes race seem genetic, even though every time he’s debated someone from ABC they have accepted the premise that race is social. For example, ABC’s Paul Underwood responded to Kahn on an episode of “Democracy Now!” in this way: “I think there’s no implication at all that genetics has anything to do with BiDil’s mechanism of action. In fact, there’s no claim that there’s genetics at all. I believe that race in the United States is a social construct, not really a genetic construct.”⁵⁰³ For ABC, the drug is precisely a way to renegotiate the

⁵⁰¹ Harriet A. Washington, “Chapter 12: Genetic Perdition and the Rise of Molecular Bias,” in *Medical Apartheid: The Dark History of Experimentation on Black Americans from Colonial Times to the Present* (New York: Double Day, 2006): pp. 299-324. BiDil specifically is discussed on pp. 318-324, under a subheading “Heart of Darkness.”

⁵⁰² Mary Douglas, *Purity and Danger* (London: Routledge, 1966).

⁵⁰³ Paul Underwood, on Amy Goodman, “The FDA Approves a Race-Specific Drug for the First Time in History. Will it Address the Real Health Issues Facing African-Americans?” *Democracy Now!*, Monday, August 1st, 2005.

social imbalance of biologies. Arguing against BiDil on the basis that racial differences are “not biological” sets up the debate in a way that cannot be held on a useful level, because it reinforces an artificial binary between originary states and living bodies.

NitroMed’s CEO Michael Sablonski is quick to say “We know that skin pigmentation is at best a poor surrogate marker for looking at responses to medication.”⁵⁰⁴ This poor correlative potential is simply not threatening to BiDil as a solution, because the response can simply be to continue studying it in more groups while providing it to one group in the meantime. Kahn thinks that the “caveats” about race being a rough proxy are somehow buried and not enough. Kahn characterizes BiDil researchers’ caution about using race as a surrogate marker for genetics as “ironic,”⁵⁰⁵ but it is actually part of a racial narrative that exceeds any narrow reliance on genetics as determinative.

This disconnect over the stakes of the debate should alert us that we have more work to do than simply repeatedly and in each case reiterate “race is not genetic.” As discussed in the chapter on hypertension, for physicians in the field there is no necessary conflict between social causes and medical solutions. The constant criticism that BiDil focuses attention away from social aspects and a more holistic critique of racial injustice underestimates the NAACP and the ABC. Those groups may be prominent in the promotion of BiDil, and their financial links with NitroMed do make obvious their noninnocence as participants in the debate. But promotion of BiDil is a miniscule portion

⁵⁰⁴ Fard Johnmar, “Conversations About Race-Based Medicine: NitroMed’s Michael L. Sabolinski,” posted July 27, 2006, <http://fardj.prblogs.org/2006/06/27/conversations-about-race-based-medicine-nitromeds-michael-l-sabolinski-md/>

⁵⁰⁵ Jonathan Kahn, “How a Drug Becomes “Ethnic”: Law, Commerce, and the Production of Racial Categories,” *Yale Journal of Health Policy, Law, and Ethics* 4 (2004): 25.

of what these organizations do, and there is scant evidence of the specter of the abandonment of social justice that Kahn and others point to as a danger of BiDil.⁵⁰⁶ It is historians and social scientists, not the NAACP or ABC, that are preoccupied with genetics above all other arguments about race. Historians and social scientists of race have made this preoccupation with the relationship between race and genetics a principal part of our occupation, such that insofar as STS as a field has focused on race it has focused overwhelmingly on genetics and critique thereof. But ABC and NAACP have much broader takes on race than either NitroMed on the one hand or STS as a field on the other.

Kahn and Sankar among others have suggested that a peril of BiDil is “not only that it biologizes race but also uses race as biology to create the impression that the best way to address health disparities is through drug development.”⁵⁰⁷ But there is conceptual space between drug development as “a way” to address disparities and drug development as “the best way” to do so. *Any* way to address disparities might be good given the intolerable present. Moreover, drug development is a node of progress and power in America, and suggestions that the NAACP and ABC have no business being there is to suggest a less democratic debate.

For Kahn, the private is isomorphic with the genetic, but that is actually a conceptual leap. Kahn argues:

⁵⁰⁶ A chart of NAACP health actions is here: <http://www.naacp.org/advocacy/health/goals/>, with BiDil clearly marginal. A search on the NAACP website for BiDil yields no hits at all, and for NitroMed a few along these holistic lines – access to high quality affordable health care, and although in the Medicaid Part D there is a reference to collaboration with NitroMed in ensuring access to effective drugs no reference to specific drugs.

⁵⁰⁷ Jonathan Kahn and Pamela Sankar, “BiDil: Race Medicine or Race Marketing?” *Health Affairs* 24 (Jul-Dec 2005): 462.

Attempts to address social disparity generally implicate the power of the state or other nonmarket institutions consciously to intervene both in the allocation of resources and the sanctioning of racist practices. In contrast, attempts to locate genetic differences may be located at the level of the molecule and targeted by pharmaceuticals developed and dispensed through the purportedly impersonal forces of the market.⁵⁰⁸

But the *individual* is a necessary conceptual step between the institutions and the molecules, and it is the focus on the individual that more accurately assesses most conservative positions on BiDil. It seems to be Kahn, more than the investigators, who conceptualizes racial molecules as easy to fix: “The appeal of taking a predominantly biomedical approach to addressing health disparities is undeniable—instead of fixing social inequality you simply fix molecules.”⁵⁰⁹

Even Clyde Yancy, the ABC member whose comments about genetics have earned him the special ire of Kahn, does not limit his proposed fixes to molecular ones in these terms. Rather, Yancy, like many conservatives, emphasizes individualism in ways that are both social and biological. He has emphasized the same “lifestyle” arguments that are central to notions not only of “black heart health” but “heart health” as a whole.

In *Ebony*, a profile about him emphasizes not on the deep genetic nature of black heart

⁵⁰⁸ Jonathan Kahn, “Misreading Race and Genomics After BiDil,” *Nature Genetics*, Correspondence, 37 (July 2005), 655. The passage continues: “Implicit in the logic of conservatives such as Satel and Entine, who use BiDil to characterize disparate health outcomes in terms of genetics, is an argument for privatizing efforts to address what are currently characterized as health disparities.”

⁵⁰⁹ Jonathan Kahn, “How a Drug Becomes “Ethnic”: Law, Commerce, and the Production of Racial Categories,” *Yale Journal of Health Policy, Law, and Ethics* 4 (2004): 7. He says later in the same article: “Hydralazine and isosorbide dinitrate do not address the social causes of heart failure, only the individualized biological ones.” (p. 36). But socially-caused or genetically-caused, at this stage, the level at which it makes sense to intervene is biology. He outlines BiDil’s logic his way: “The case for BiDil can thus be roughly summarized as follows: 1) blacks die from heart failure at a rate twice that of whites; 2) given this great disparity it seems that there must be some underlying biological or genetic (as opposed to “merely” social or environmental) factor accounting for the difference; 3) supporting this hypothesis are studies that control for socioeconomic factors and still show racial differentials in outcome; 4) moreover, additional studies indicate that blacks do not respond as well as whites to certain front line heart failure therapies; 5) therefore, a response is called for that addresses this different biology; 6) enter BiDil, a pharmaceutical response to the statistical disparity that appears to have a differentially beneficial effect on blacks at the molecular level.” Jonathan Kahn, “How a Drug Becomes “Ethnic”: Law, Commerce, and the Production of Racial Categories,” *Yale Journal of Health Policy, Law, and Ethics*, 4 (2004): 11.

disease but on the very mutability of it through diet and preventative health: “I would love for every African American woman to get the message that you can become heart healthy and the steps you need to take are simple: Get your blood pressure checked; if you have diabetes, get it treated; if you have a weight problem, reduce the weight. You can get heart-healthy.”⁵¹⁰ BiDil gets a brief mention, taking up a very small place in the larger context an article that makes the heart health disparities seem almost *too* easily solved. What unites Yancy’s comments here on lifestyle modification and elsewhere on genetics and BiDil is the sense that *individualism* can solve the problem. This makes him a bit of an outlier within the ABC, whose members tend to have more social understandings of the nature of problems and solutions. But if we critique his ideology based only on the genetics side of individualism, we miss an important part of his larger framing of the problem and its solutions. Indeed, the ideal promise of pharmacogenomics is that once it gets past race to the individual it will be able to solve problems after all. This is a fantasy that individuals are the only meaningful entity for medical research and practice.

BiDil’s critics like Kahn operate as if an all black trial in itself limits applicability of the findings. He has criticized the lack of a “control group” in A-HeFT and written that “A study could have been conducted instead in a racially diverse population using the generic drugs (H/I), which might have resulted in the broader availability of a good, low-cost medication.”⁵¹¹ But “racially diverse” trials tend to under-represent blacks, (and, as importantly from the perspective of groups like ABC, multiracial trials also tend

⁵¹⁰ Kendra Y Mims, “Learning to Love Your Heart: A Few Simple Steps Can Lead To a Longer, Healthier Life,” *Ebony*, October 2006, 61: 12, p. 162.

⁵¹¹ Jonathan Kahn and Pamela Sankar, “BiDil: Race Medicine or Race Marketing?” *Health Affairs* 24 (Jul-Dec 2005):. 460.

to underrepresent black investigators). Even when black patients are represented, as we saw in the previous chapter on ALLHAT, the results can get so focused on differences that the actual implications for best serving the population can get obscured.

Fundamentally, it is a fallacy to assume that multi-racial trials are more “diverse” than all-black trials, since after all more diversity occurs within groups than between them. Kahn suggests that a racially diverse trial would have shown that BiDil works for “everyone,” taking for granted both that white “controls” make some sort of ontological sense and that a black trial cannot be argued to show that. NitroMed has exploited ambiguity about the question of whether A-HeFT is meant to be a trial of efficacy in blacks *in contrast to* the overwhelmingly white populations previously studied, or whether A-HeFT stands on its own to prove efficacy. For both the NAACP and the ABC, the A-Heft trial and BiDil as a product are explicitly *not* comparative projects. If the goal had been comparison, there would have been a more typical trial that actually included white people as “controls” as well as blacks. But as we saw in the chapter on Framingham and Jackson, the field is going in a different direction. In the Jackson Heart Study, understanding disparities is understood as demanding not ever more comparative data (which is essentially *contrastive* data), but rather understanding diverse African Americans on their own terms.

Among black cardiologists, there is both frustration that BiDil was not approved for everyone based on a trial about blacks and frustration that even after an all-black trial the argument focuses on nonblacks. Must all things about blacks be comparative? Must the question to answer always be how to apply the knowledge blacks gain about blacks “beyond” them? The point of BiDil for A-Heft investigators is not necessarily to say that

blacks are different, not necessarily that blacks unlike whites benefit, but rather to say blacks do benefit. As for the question of whether whites do, they generally say they would give it to their white patients as well, though this is rather abstract—black doctors overwhelmingly see black patients. In terms of approval, BiDil could have been handled in an analogous way to Cozaar: “these results have not been shown in white people.” But constantly being asked about white people is a frustrating demand to turn, again, away from the needs of suffering black patients.⁵¹²

As discussed in the previous chapter, there is dishonesty in the common rhetorical move in social science critiques of positing naive belief in categories social science has abandoned. The belief in biological race by black cardiologists individually or as a group can only be understood as complex. Critiques of the ABC for supporting race-reifying drug research are missing something important if they rely on a projection that black Cardiologists “really believe” in genetic race despite their statements to the contrary.⁵¹³ Congratulating ourselves on not being so mistaken about race misses something central about ABC’s practice and shuts down exploration of how we social thinkers might have our own unexamined real beliefs. In this case, that value of medical knowledge depends on whether it can be extrapolated to whites.⁵¹⁴

⁵¹² Harriet A. Washington also makes the same move: “Because heart disease is the number-two killer of blacks—and whites—BiDil should be embraced if it indeed conveys a racial benefit to blacks with CHF. So should any other therapy that accurately targets clinically meaningful disease vulnerabilities in African Americans.” Harriet A. Washington, *Medical Apartheid: The Dark History of Experimentation on Black Americans from Colonial Times to the Present* (New York: Double Day, 2006): 323. But why should BiDil have to show “racial benefit to blacks,” rather than merely “benefit to blacks?”

⁵¹³ This insistence that accepting pharmaceuticals necessarily means denying environmental causes is analogous to critiques of MIT students on SSRIs—we miss something if they rely on a projection that they (only) “really believe” in a chemical-imbalance cause of their distress, and are somehow blind to the stressors of the circumstance.

⁵¹⁴ Harriet A. Washington, like Kahn, has suggested that one harm of BiDil is that whites are excluded: “The study should have included whites in order to provide evidence that the drug works differently in blacks, but because the patents for use in all races will expire in 2007, there is no economic incentive to test

In these debates, Kahn, like Kaufman in Chapter 4, assumes that the connection between race and medicine can only lead one way: toward medicalizing injustice and against social change, citing discrimination against carriers of sickle cell trait as a precedent. But as Keith Wailoo points out, not all ethnically marked diseases have the same connotations.⁵¹⁵ Moreover, as Alondra Nelson has tracked in her dissertation and forthcoming book, diverse tactics of recontextualization are possible.⁵¹⁶ Nelson tracks the Black Panther Party advocacy for medical response to sickle cell and against medicalization of crime to suggest that social movements can be tactical rather than necessarily consistent. Participating in BiDiI does not actually prevent the NAACP from participating in non-medicalized social justice.

Trying to separate out that nexus of race as social/biological and material/semiotic is not the solution. A purifying process is what Kahn calls for: “to articulate an institutional mechanism of guidelines whereby relevant administrative actors would be required to distinguish between uses of race as a sociopolitical category from uses of race as a biological and/or explicitly genetic category.”⁵¹⁷ But purification is a dead end. When Kahn wants to make a choice for social justice against biology, he is solidifying

the drug in whites. (NitroMed will hold the patent for the use of BiDiI in blacks until 2020.) In an ironic twist, whites are being subjected to racial exclusion by being denied access to testing or use of a heart drug that could benefit them or even save their lives.” Harriet A. Washington, *Medical Apartheid: The Dark History of Experimentation on Black Americans from Colonial Times to the Present* (New York: Double Day, 2006): 323.

⁵¹⁵ Keith Wailoo, “Inventing the Heterozygote: Molecular Biology, Racial Identity, and the Narratives of Sickle Cell Disease, Tay-Sachs, and Cystic Fibrosis,” in *Race, Nature, and the Politics of Difference*, edited by Donald S. Moore, Jake Kosek and Anand Pandian, (Durham and London: Duke University Press, 2003), 235-53. See also: Keith, Wailoo and Stephen Pemberton, *The Troubled Dream of Genomic Medicine: Ethnicity and Innovation in Tay-Sachs, Cystic Fibrosis, and Sickle Cell Disease* (Baltimore: Johns Hopkins University Press, 2006).

⁵¹⁶ Alondra Nelson, *Black Power, Biomedicine, and the Politics of Knowledge*, (PhD diss, New York University, 2003); Alondra Nelson, *Body and Soul: The Black Panther Party and the Politics of Race and Health*, Forthcoming.

⁵¹⁷ Jonathan Kahn, “How a Drug Becomes “Ethnic”: Law, Commerce, and the Production of Racial Categories,” *Yale Journal of Health Policy, Law, and Ethics* 4 (2004): 44.

the validity of that boundary in the sense that, as Nancy Krieger points out, “The notion that scientific thinking and work must somehow ‘choose’ between social justice and biology is itself an ideological stance.”⁵¹⁸

Kahn wants to settle the intersection of BiDil and race exclusively on the biological side of the social/biological divide in order to stamp out traces of the biological in race, but neither BiDil nor race can be settled. Kahn’s efforts to close down the heterogeneous meanings of BiDil into separable components of molecules and meanings provides a parallel to the efforts to close down debate in the name of consensus that opened this chapter. Both are attempts to pin down the slipperiness of BiDil and race in order to make a clear embrace or denunciation on one register. If Cofield wants the only thing at stake to be African American access to drugs as remedies, Kahn wants the only thing to be at stake to be the purging of race from drug development and marketing. But BiDil retains what we can characterize in Derrida’s terms as undecidability. The goal of critical scholars should not be to claim the decision, but to engage with the tenacity of undecidabilities.

⁵¹⁸ Nancy Krieger, “Stormy Weather: *Race*, Gene Expression, and the Science of Health Disparities,” *American Journal of Public Health* 95 (December 2005): p. 2155.

Epilogue: Tracking Plural

Noninnocent Discourses

Vignette

It was the end of March, 2007, almost two years after the FDA approval of BiDil for “heart failure in blacks.” The scene was the second annual conference of the MIT Center for the Study of Diversity in Science Technology and Medicine, which I had been part of organizing. This conference followed up on the previous year’s theme of “Race, Pharmaceuticals and Medical Technology”, and marked something of a shift, now focusing on “The Business of Race and Science.”⁵¹⁹ The morning had been devoted to an invited lecture on the complicated uses of race in population genetics, and a panel on genetic ancestry testing. After lunch, I called the crowd of about a hundred back to order for the panel called “Debating Race in Biomedicine.” The three papers were rather disparate, the first arguing that social science critics should acknowledge that there are limited genetic correlations by race, the second analyzing physicians’ responses to race-based therapeutics, and the third considering racial valences in arguments about Alzheimer’s disease genetics. After the panel’s presentations, I called on questions from

⁵¹⁹ MIT Center for the Study of Diversity in Science, Technology and Medicine, Second Annual Conference, “The Business of Race and Science,” Cambridge, MA, March 30-31, 2007.

the audience. The first several came from academics I knew and could call by name, and dealt with methodology of one paper, a request to expand on a comment – the regular order of the day for an academic conference.

But then I called on someone standing in the back of the room by the door, a middle-aged African American man in a brown suit whom I did not know by sight. I had noticed his name among the nametags at the registration table because it was familiar to me from articles in support of BiDil, but I had not seen him come in. Having heard him on the radio, I was able recognize him as soon as he started to speak: Gary Puckrein, head of the National Minority Health Month Foundation. He had apparently entered the room after the panel's presentations. His comments were addressed initially to one of the panelists, George Ellison from the University of London, but unrelated to the specifics discussed, and slipped as he spoke from the individual to the general.

Puckrein's tone was belligerent from the outset, and he performed a crescendo with both his words and body language directed against a "you" that seemed to include everyone in the room. He said that he was having a hard time containing his outrage that we were up here at MIT debating these issues while people were dying. He said that we should be ashamed of ourselves for having this academic conversation at all, because insurance companies cite our academic debate in their refusal to cover this drug called BiDil that could save people's lives. Thus, we were responsible for killing people. He said that he was "down with the people," and that academic conversation was inappropriate when people are dying. Pointing out that his mother had died of heart failure, and that of the 750,000 African Americans with heart failure only a couple

percent are getting the potentially life-saving drug, he said it was unethical to deny access to this drug, and demanded to know: “Do you want people to live or die?”

Puckrein went on for minutes, and it was only with great difficulty that I cut him off to attend to other comments and questions. Even as other audience members and panelists joined in on the debate, Puckrein maintained a good share of the floor with a continuing denunciation that was expertly performative, speaking with a sharp but weighty tone and squaring his broad shoulders against others from the audience as well as those at the front of the room.

I closed the session with something of a plea for common ground. I said that everyone in the room was concerned about health disparities and dedicated to ameliorating them, and that it was important to realize that BiDil is not the only drug to which African Americans are being denied access. In fact, lack of access to antihypertensives is part of what leads to heart failure, and thus the need for BiDil. So I suggested that we could best proceed from that starting point: our shared commitment to reducing health disparities.

It was only upon further reflection that I realized just how much common ground Puckrein had with us “up at MIT.” In addition to sharing the concern about health care disparities, Puckrein had more in common with the majority of those in the room, including: indirect engagement with medical practice; noninnocence; and he wants to separate arguments about the approval of BiDil from those about its distribution.

First, although Puckrein gave the impression that he was a physician personally in the practice of saving people’s lives, his training and his advocacy are actually disconnected, and his engagement with medical practice is primarily indirect. His title of

“doctor,” like that of many in the room, comes not from an MD but from a PhD, in history (Brown, 1978; dissertation title: *The Acquisitive Impulse: Plantation Society, Factions, and the Origins of the Barbadian Civil War (1627-1652)*).⁵²⁰ Second, Puckrein’s position, like ours, is not disinterested. It is noninnocent. His alliance is not only “down with the people,” but also “up” with the pharmaceutical company behind the BiDil: according to the *Wall Street Journal*, he is a shareholder in NitroMed and his nonprofit organization has also received grants from it.⁵²¹ Third, like BiDil’s critics, Puckrein wants to separate arguments about BiDil’s approval from those about its distribution. Although he claimed that academics opposed the provision of BiDil to patients in need, to a person everyone had supported the distribution of the chemical compound to blacks with heart failure (among others), even if there was some disagreement about peripheral questions such as whether the branded or generic compound was superior.

Puckrein’s framing of the antagonism between those “down with the people” and those “up in the academy” was one that would resonate throughout the rest of the conference. Attending to Puckrein’s own plural interests behind his stance – as I have just done – does not discredit his intervention. Innocent engagement with race is not possible, and race is a topic about which neither academics nor activists should ever feel comfortable. Puckrein’s intervention was an important reminder to consider not only the residue of a horrific history and the specter of an unacceptable future, but also the

⁵²⁰ <http://www.brown.edu/Students/HGSA/Alumni/alumlist70s.htm>;
<http://www.brown.edu/Students/HGSA/Alumni/Puckrein/Puckrein.htm>

⁵²¹ Sylvia Pagan Westphal. “Tough Prescription: Heart Medication Approved for Blacks Faces Uphill Battle; As Insurers Debate Costs And Generics Loom, BiDil Fails to Reach Needy; The Role of Medicare Part D,” *Wall Street Journal*, Oct 16, 2006. p. A.1.

unbearable present. The kernel of truth in what he said was that social justice as such was not on the table in the academic discussion, and should be.

Moreover, Puckrein is by no means alone in refusing to acknowledge all of his own stakes or to engage with what is at stake for his interlocutors, or in his preference for denunciation over engagement. Most of BiDil's advocates including Puckrein are quick to disavow race as a genetic category. Yet academic critique of BiDil has focused heavily on genetic reification of race. That fits BiDil into the main framework of scholarship about race and medicine in the humanities and social sciences, but is not quite engagement.

One way to frame what is at stake in this impasse over race and biomedicine is a central question of this thesis: what aspect of medicating race is the most important subject of discussion. As this dissertation has tracked, medicine can best be understood as a heterogeneous field that is both a science and a practice – one that does not merely reify difference but seeks to act on it in plural ways. Medicine *both* represents and intervenes on racialized bodies. A central theme of this dissertation has been tracking the ways that medicine as a field is implicated in two aspects of mediating race: mediating in the sense of arbitrating, and mediating in the sense of intervening. Whether the arbitration or the intervention is primary is one source of the disconnect between Puckrein and many of BiDil's critics. If the preponderance of academic interest has been in how BiDil mediates race in the sense of arbitrating, Puckrein's call was for attention to the role BiDil could have in mediating race in the sense of intervening.

In Chapters 4 and 5, I connected this plural meaning of *medicating as mediating* with the plural senses in which medicine might *fix race*, attending to the capacity of

medicine as a social field that can be invoked both to fix (reify) racial differences and fix (repair) racial inequality. The identity practices that emerged around African American hypertension and thiazide also exceeded narrow medical data or financial interests. The medicating of race, as it arbitrates/intervenes and reifies/repairs, also opens up opportunities to fix attention on race as an object of analytical inquiry and to meditate upon race. One thing that doctors, advocates, and academics are doing when we argue about how to medicate race is *meditating upon* race, articulating emergent theories of a differentiated world through the languages of medical terminology and data. Putting the meditations about BiDil's implications for race in medicine in the context of the intersection of race, heart disease and pharmaceuticals over the past century allows us to see the ways that it is neither a mere repetition of older racial discourses nor a fresh one. This contextualization illuminates both BiDil's appeal to many parties as a site in which to articulate diverging opinions about race; and the continuing volatility of debates about the drug.

Connecting this impasse with those of the past century

In this vignette, we can see connections with and reverberations of the debates about race and medicine of the past century that have been the subject of this dissertation. The name of Puckrein's organization, "National Minority Health Month Foundation," is an evocative one that provides an opportunity to return to questions opened in the reinvestigation of Booker T. Washington's cause of death that opened this dissertation.

In its name, Puckrein's organization invokes a lineage to the "National Negro Health Week," mentioned in Chapter 2. That movement was founded by Booker T.

Washington in 1915, remained important as the Public Health Service took it on in the 1930s, and continued until 1951. It is with that historical precedent in mind that Puckrein's organization framed its goals and means: like the earlier movement it seeks uplift through black mass mobilization supported by black health professionals and business leaders, as well as the federal government and the philanthropy of major corporations. His approach also garners similar critiques. Like the NAACP physician Louis T. Wright's 1930s admonishment that black health is not a separate problem needing separate solutions but an American problem that should be solved through integration, critique remains strong about whether attention to African American health as a segregated enterprise is inevitably flawed. At the same time, absolutism with regard to holding out for integration provoked then, like now, justifiable critique as elitist, because this ideology disregards the immediate needs of suffering black patients. It also undercuts work done by black physicians striving to bring their practices and patient populations into modern medicine on their own terms.

This vignette also reopens questions attended to in Chapter 3, about the Framingham and Jackson Heart Studies. Attending to the postwar Framingham Heart Study and the post-2000 Jackson Heart Study showed that debates about whether research conducted in one racial group are applicable "beyond" them are not new. Tensions between representation and extrapolation remain open problems in the application of medical science. Though the earliest reports of the Framingham Study suggested that its scope of extrapolation was only as far as "the white race," over time the results of Framingham have been used to account for both white stories and universal

ones. It is still not clear whether research conducted on black patients can, as the Jackson Heart Study investigators hope, tell both black stories and universal ones.

A-HeFT, the trial that led to the approval of BiDil, included only self-identified blacks. The FDA's decision to make the indication for BiDil specific to that population suggests that, from its current leadership's perspective, studies in black patients cannot provide universally-applicable therapeutics. Many of BiDil's critics, too, have suggested that the all-black study-design meant that it could not be extrapolated to whites, and that that limitation presents a harm to white patients because it leaves unknown whether the drug would also benefit them. That line of argument is troubling for two reasons: first, because it assumes the inability of African Americans to stand in for the universal, and because preoccupation with what BiDil means for white people turns again away from the needs of suffering black patients.

At stake in this vignette, as in this thesis, is *both* whether studies by black doctors in black patients can provide knowledge that has the capacity to be as mobile and extrapolatable as studies done by white doctors in white patients, and whether the work of black doctors and the health of black patients matter for their own reasons independent of that extrapolation process. Puckrein's analysis of race and medicine seems less nuanced than those of the Jackson investigators, in that he assumes that the scope of representation in the study and the capacity for extrapolation are isomorphic. Yet that question of scientific arbitration is not where Puckrein's principal interest is, as is clear from his disavowals of race as a biological category in the discussion at the conference, and engagement with him on those terms does not seem to get at the disconnect. Pukrein's disavowal of biological (understood as genetic) race mirrors disavowals by

Kahn of the implications for treatment described in the previous chapter. Kahn's acknowledgement, for example in an article co-authored with Pamela Sankar, that "BiDil will help heart patients; some of them will be black,"⁵²² is not enough to satisfy Puckrein. Puckrein's acknowledgement that blackness is not genetic is not enough to satisfy Kahn. Both want to obligate the other to center the discussion on their own terms: problems of arbitration for Kahn and Sankar or promises of intervention for Puckrein. But BiDil speaks on all these aspects of medicating race simultaneously, and the argument reaches an impasse.

Similar to the black health advocates mobilized around African American hypertension as a disease category described in Chapter 4, Puckrein both supports scientific medicine's continuing investigation into racialized difference, and argues to close the debate so that treatment known to work can be given. He wants black heart failure, like black hypertension, to be durable enough to command intervention. He blames etiological ambiguity for its failure to do so. Yet we can tell by the etiological ambiguity that underlies African American hypertension that debate over the nature of race does not preclude but rather bolsters the durability of that disease category. Diverse actors accommodate considerable epistemological eclecticism underlying what is recorded as status as African American. That recorded status is then employed to tell stories about difference and make demands for medical intervention and social change. Thus, explanations for BiDil's failure so far cannot rest on the ambiguity provoked by our academic debates.

⁵²² Jonathan Kahn and Pamela Sankar, "BiDil: Race Medicine or Race Marketing?" *Health Affairs* 24 (Jul-Dec 2005): 461.

Puckrein's intervention in the conference is an example of performative identity practices and citizenship claims around a drug, practices and claims that exceed the literal in ways that resonate with the extra-medical discussion of thiazide discussed in Chapter 5. As illustrated in my analysis of the performative intervention by Henry Louis Gates, Jr., around thiazide, drugs' relationships with preoccupations with difference both include and exceed their role in narrow economic interests or medical data, and we should preserve some conceptual space between being bought off by pharmaceutical companies and buying in to pharmaceutical grammars. Looking at this debate over BiDil in light of those over thiazide shows that there is not just one way that pharmaceuticals can articulate participation in racialized American ways of life, but rather plural ways. While thiazide is proven, old, and cheap, in contrast, the scope of BiDil's scientific basis is shaky, and it is both old and new, and it is expensive. Thus debates over BiDil can articulate different narratives of the nature of inclusion and difference in American ways of life in comparison with thiazide, opening up ways to articulate demands for access to the cutting edge products of recent clinical trials that are both racially differentiated and branded and expensive.

The exchange also opens up another opportunity to read ineffective attempts to separate out the poison and remedy elements of BiDil as *pharmakon*, a subject addressed in Chapter 6. The blatancy of BiDil's racialization has attracted considerable scholarly critique, and yet much of the critique has leaned too heavily on old arguments about race and genetics on the one hand, or about blockbuster drugs on the other. Neither of these lines of critique are up to the task of deconstructing a drug that is simultaneously: effective in treating symptoms and delaying death, supported by diverse

epistemologically eclectic actors, and commercially unsuccessful. Puckrein is as unsuccessful as Kahn in the previous chapter in closing down the irredeemable polyvalence of a “black drug” in this current historical moment, but he is wrong that academic meddling alone sows doubt about this drug or impedes its rush to markets. Using BiDil to track plural noninnocent discourses about race and medicine is an endeavor that is more productive than attempts to decry racist villains. Discourses of BiDil are racial discourses not because they support particular political or commercial interests, but rather because it is impossible to talk about BiDil without talking about race. Thus, for example, while it is possible to talk about Vioxx without having an opinion about aging or arthritis, it is not possible to talk about BiDil without having an opinion about race – even if that opinion is a disavowal. We should not try to use this drug to put the material-semiotic category of race to one side (material) or the other (semiotic), but rather to engage with race as material-semiotic in both academic and social justice spheres.

Toward ethical noninnocence

In this dissertation and in the vignette that opened this epilogue, my goal has been not to settle race and heart disease generally or BiDil in particular, but rather to open up these emergent processes for analysis. A consequence of this process of opening and unsettling, I hope, is that the productivity of race in medicine emerges as more difficult to come to terms with after the thesis than before it. Attempts to settle both drugs and disease categories and their intersections with race are productive of new ambiguities.

My mode of critique is not to render a verdict, but to engage in a process that could be characterized as one of ethical noninnocence, that acknowledges the impossibility of innocent engagement without abdicating the imperative to strive to work toward justice.⁵²³ Although the processes of medicating race in America generally are designed by and for a racialized and gendered biocapitalism, that biocapitalism doesn't always get what it wants.⁵²⁴ Articulating the noninnocence of the plural discourses of race and pharmaceuticals is not the end of the inquiry, but a condition of continuing engagement. In the tracking of plural noninnocent discourses around race and pharmaceuticals, the critique should be framed in terms of how they can be used to indict injustices and articulate aspirations, rather than on the basis of assumptions of the superiority of the natural body over the pharmaceutically-enrolled one, or the privileging of the sameness of natural bodies over lived inequalities.

With Haraway, my goal has been not to reject one side of the boundary between the technical and the political, between the social and the biological, but to put those boundaries into permanent question.⁵²⁵ We might see this move as analogous to Harding's call to get "beyond spontaneous feminist empiricism" (which simply takes the standards of scientific rigor for granted and then measures a trial against those). Harding urges feminist science studies to move toward a successor science that considers

⁵²³ Noninnocence is a concept Donna Haraway has developed in much of her work, including "Manifesto for Cyborgs: Science, Technology, and Socialist Feminism in the 1980's," *Socialist Review* 80 (1985): 65-108.

⁵²⁴ In this sense, these pharmaceutical processes are related to commodification of bodies and kin more generally, which have both liberatory and oppressive aspects. As Cathy Griggers points out, the same reproductive technologies that generally serve to reinforce the repressive maternal economy of straight reproduction also allows a lesbian to bear her partner's genetic child, Cathy Griggers, "Lesbian Bodies in the Age of (Post)Mechanical Reproduction," in *The Lesbian Postmodern*, edited by Laura L. Doan (New York: Columbia University Press, 1994).

⁵²⁵ Donna Haraway, *Modest_Witness@Second_Millennium.FemaleMan©_Meets_OncoMouse*TM (New York: Routledge, 1997).

perspectives from those with diverse interested stakes, not only to criticize the science but also to make it better—more answerable to both truth and justice.⁵²⁶ In this dissertation, I hope to have situated my argument in a place “beyond anti-racist empiricism,” even if making science better is less central to my stakes. Interventionist medicine, too, must be held answerable to questions of truth and justice, both for its practitioners and for its publics. Yet we need to remember that answerability is not the same as an answer. If an analysis of race and medicine ever leaves a settled feeling, in which it is possible to feel comfortable that an unjust racial discourse has been taken down and a just racial discourse characterized, that is a symptom that the critique has strayed from engagement. Any racial discourse that seems to crumble in the hands of a sharp critique could not possibly be essential to race itself, because as we have seen in this thesis race is extraordinarily resilient. Contents shift, but durable preoccupation endures, and engagement with that preoccupation should remain uncomfortable for as long as injustice remains.

This project of ethical noninnocence might be allied with that of the prophetic pragmatism theorized by Cornel West, that “promotes the possibility of human progress” even as it acknowledges the “impossibility of human paradise.”⁵²⁷ Analysis of race and

⁵²⁶ Sandra G. Harding, *The Feminist Standpoint Theory Reader: Intellectual and Political Controversies* (New York: Routledge, 2004).

⁵²⁷ “Prophetic pragmatism is a form of tragic thought in that it confronts candidly individual and collective experiences of evil in individuals and institutions—with little expectation of ridding the world of *all* evil. Yet it is a kind of romanticism in that it holds many experiences of evil to be neither inevitable nor necessary, but rather the results of human agency, i.e. choices and actions. This interplay between tragic thought and romantic impulse, inescapable evils and transformable evils makes prophetic pragmatism seem schizophrenic. On the one hand, it appears to affirm a Sisyphean outlook in which human resistance to evil makes no progress. On the other hand, it looks as if it approves a utopian quest for paradise. In fact, prophetic pragmatism denies Sisyphean pessimism and utopian perfectionism. Rather, it promotes the possibility of human progress and the impossibility of human paradise.” Cornel West, “On Prophetic Pragmatism,” in *The Cornel West Reader* (New York: Basic Books, 1999), 166.

medicine is not a project that can perfect the world. There is no place of engagement that is above the fray, but abstention is not the solution. If there is hope for progress in both the social justice and academic debates about race and medicine, it comes from the recognition of the inevitability of injustice combined with the commitment to engage in the pursuit of justice.

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