ABSTRACT

Title of Document: THE POLITICS OF METABOLISM: THE METABOLIC SYNDROME AND THE REPRODUCTION OF RACE AND RACISM

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Biomedical researchers, government agencies, and the pharmaceutical industry increasingly use the term *metabolic syndrome* to define the observed co-occurrence of the major biological risk markers for heart disease, type II diabetes, and stroke. The metabolic syndrome is a new feature in what I call *the politics of metabolism,* or the discourses, social processes, and institutional relationships that governs the metabolism of individuals and groups. The emergence of the metabolic syndrome reflects a growing network of scientific, state, and corporate actors and institutions that are invested in studying, regulating, and profiting from control over metabolism. Drawing on insights from critical race theory, science and technology studies, and Foucauldian studies of biopower, I analyze the metabolic syndrome as a new discourse about metabolism that continually draws upon racial meanings to construct individual and group differences in different kinds of metabolic risk.

The metabolic syndrome not only constitutes a new way of constructing, studying, and treating metabolic health problems, it also constitutes an emerging site for the production of racial meanings. Researchers use race in metabolic syndrome research and to study, prescribe, and label prescription drugs that may be related to the metabolic syndrome. I investigate the use of race and the metabolic syndrome in biomedical research on prescription drugs and African Americans. I develop the metaphor of *killer applications* to examine how prescription drugs operate in the politics of metabolism. A killer application is a superior technology that combines human and non-human elements that structure bodily practices in a wide range of social, commercial, and scientific contexts—prescription drugs have become the new killer applications in biomedicine. I argue that the search for killer applications has transformed the ways that pharmaceutical corporations study prescription drugs, metabolism, and race. I compare how drug researchers use race and the metabolic syndrome to study antipsychotics and statins in African Americans, how physicians' race-based diagnoses of schizophrenia and high cholesterol structure the prescribing patterns of antipsychotics and statins, and how scientists' assumptions about the genetic basis of racial differences in drug metabolism structure the debate about race-based drug therapies.

THE POLITICS OF METABOLISM: THE METABOLIC SYNDROME AND THE REPRODUCTION OF RACE AND RACISM

By

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Dedication

I dedicate this study to Rebekah and Ruth whose sacrifices of time, patience, and love made it possible. I would also like to dedicate this study to my family and friends, peers and colleagues, and to all of my advisors, especially Dr. Patricia Hill Collins, in thanks for all of their labor that found its way into these pages. I also dedicate this study to all of us who work to resist the politics of metabolism.

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Chapter 1: Introduction

According to the National Library of Medicine, the central library of the National Institutes of Health (NIH), metabolism encompasses all the physical and chemical processes within the body that create and use energy.[1] While metabolism encompasses a litany of bodily processes that are linked to the development of poor health, a particular cadre of metabolic problems has increasingly assumed a new spotlight in the biomedical research community. Heart disease, type II diabetes, and obesity have all become predominant health problems and constitute the leading causes of death in the contemporary United States.

An expanding set of descriptions connects these problems of human metabolism to the social and economic conditions of modernity, like increasing leisure time and more widespread economic prosperity. Some have called these metabolic conditions diseases of comfort, referring to the observation that these conditions are increasingly prevalent among the populations of Western nations that enjoy an overabundance of food and leisure (Choi, Hunter, Tsou, and Sainsbury 2005). Others have called these conditions diseases of affluence, a label that emphasizes the positive statistical correlations between social class and metabolic illness (Ezzati, Vander Hoorn, Lawes, Leach, James, Lopez, Rodgers, and Murray 2005). This shift from infectious and communicable diseases to chronic metabolic conditions as leading causes of death has been called "the epidemiologic transition" (Olshansky and Ault 1986; Omran 1971; Omran 2005).

Since 1956 biomedical researchers, government agencies, and the pharmaceutical industry increasingly used a new term, *the metabolic syndrome,* to

describe the observed co-morbidity of metabolic conditions linked to heart disease, diabetes, and obesity.[2] The metabolic syndrome is *metabolic* because it concerns the biological processes by which bodies metabolize nutrients derived from food and describes these processes in terms of physiological or biochemical indicators of disease processes that are measured at the level of an individual's biology/biochemistry. Specifically, the metabolic syndrome is comprised of so-called abnormal levels of several clinical and laboratory measurements that represent the development of metabolic health problems: elevated blood pressure, elevated cholesterol, elevated blood sugar, and elevated weight. Elevated blood pressure, or hypertension, is defined as having systolic pressure of at least 140 mmHg and diastolic pressure of at least 90 mmHg. Elevated cholesterol, or dyslipidemia, is defined as having total serum cholesterol higher than 240. Elevated blood sugar, or hyperglycemia, is defined as having fasting blood glucose of at least 126 mg/dL. Elevated weight, or obesity, is defined as having a body mass index (BMI) greater than 30.

The metabolic syndrome is a *syndrome* precisely because it is an aggregation of clinical and laboratory measurements that has not yet reached designation as a disease (Hall et al 2003:414). The metabolic syndrome represents the collection of measurements of hypertension, dyslipidemia, hyperglycemia, and obesity, each of which biomedical researchers and epidemiologists have identified as major so-called risk factors for heart disease, diabetes, and stroke. According to an analysis of the 1988-1994 National Health and Nutrition Examination Survey (NHANES), a nationally representative study of the major adult populations in the US, nearly 1 out

of 4 (23.7%), over 60 million people, could potentially be classified with the metabolic syndrome (Ford, Giles, and Dietz 2002).[3]

The fact that high proportions of Americans can be classified with the metabolic syndrome has helped to establish a context where a range of biomedical, government, and corporate social actors has taken up the metabolic syndrome in their programs and protocols. For example, in 2000, an iteration of the metabolic syndrome (then named dysmetabolic syndrome X) was given a diagnostic code in the World Health Organization's International Classification of Disease (ICD-9). In 2002, a group of biomedical researchers started *The Metabolic Syndrome Institute*, an independent and not-for-profit organization that is the first organization dedicated to the dissemination of knowledge about the metabolic syndrome.[4] In 2003, a new academic journal was established to publish research articles specifically on the metabolic syndrome (Metabolic Syndrome and Related Disorders). The metabolic syndrome is also the subject of numerous medical books and monographs intended for the lay public (Reaven, Strom, and Fox 2000), physicians (Grundy 2005), mental health professionals (Mendelson 2008), and animal and biomedical researchers (Hansen and Bray 2008).

The metabolic syndrome is a discursive formation whose meanings and applications vary widely across biomedical, political, and commercial contexts. Foucault defines a discursive formation as a series of regularities or patterns in statements in terms of the objects to which they refer, the concepts used, and the thematic choices that circumscribe them over time (Foucault 1972, p. 38). While early iterations of the syndrome were formulated between the 1940s and 1980s,

research on the metabolic syndrome began to accelerate in the 1990s and into the millennium for a host of reasons I will explore in this study. The volume of published biomedical research literature on the metabolic syndrome is substantial and the rate of new publications has steadily increased in recent years.[5] In 1989, as Figure 1.1 shows, there was only one article published on the metabolic syndrome. However, by 2008, 2,613 articles were published on the metabolic syndrome, representing a two thousand percent increase in publications over a 20-year time period.

[Figure 1.1 The number of scientific studies published on the metabolic syndrome, 1989-2008].

Despite the increasing visibility of the metabolic syndrome, it would be a mistake to assume that the name, definition, and purpose of the syndrome have been consistent within this exponentially growing body of biomedical literature.

Scientists from across biomedicine, the government, and pharmaceutical corporations are using the discourse of metabolic syndrome as a new way to describe and respond to the increasing challenge that having multiple chronic metabolic conditions presents to Americans' health. Using widely accepted statistical techniques like factor analysis and linear regression, biomedical researchers have correlated the metabolic syndrome with an impressive and sobering array of health conditions including stroke, kidney failure[6], polycystic ovarian syndrome[7], cancer[8], HIV[9], and erectile dysfunction.[10] These statistical associations are possible because

the metabolic processes that encompass the metabolic syndrome unfold via every biological system of the body. Cardiologists and endocrinologists use the syndrome as a statistical predictor of who is most likely to develop heart disease and type II diabetes.[11] Psychiatrists and mental health researchers have noted the associations between the metabolic syndrome and mental disorders, like schizophrenia, bipolar disorder, and depression.[12]

In addition to biomedical researchers, government institutions that conduct and regulate biomedical research, such as the National Institutes of Health and the Food and Drug Administration, have also focused on the metabolic syndrome. In 2001, the National Cholesterol Education Program (NCEP) of the National Institute of Heart, Lung, and Blood Disorders (NHLBI), one of the National Institutes of Health, defined the metabolic syndrome as a potential target of biomedical intervention in its landmark guidelines on how to address the problem of high cholesterol among Americans.[13] The federal government also coordinates clinical trials for prescription drugs that might be associated with the metabolic syndrome. A search of the clinicaltrials.gov website that solicits research subjects for federally regulated clinical trials and found 180 studies that list the metabolic syndrome as a condition under study. Twenty percent of these trials (36 studies) are funded by pharmecutical industry; the federal government is the sponsor of the remaining 150 studies. Figure 1.2 shows a geopolitical map of these studies. The vast majority of these open studies are recruiting in the United States and Europe.

[Insert Figure 1.2 Global map of clinical trials recruiting for the metabolic syndrome, April 2009]

Ninety percent of the global market for prescription drugs is in the United States, Europe, and Japan. However, given the deepening global recession, patented prescription drug sales are at 30-year lows (adjusted for inflation) due to the increasing market share of generic medications, slower FDA approval processes, and fewer blockbuster drugs.[14]

In this context, pharmaceutical corporations are interested in developing prescription drugs that could be sold to people who might be classified with the metabolic syndrome. Indeed, the health problems encapsulated by the metabolic syndrome currently account for one fifth of health care spending in the United States and much of that money is spent on prescription drugs. In 2005, Americans spent $200.3 billion dollars on prescription drugs, five times more than they spent in 1990 (KFF 2007). In 2004, four of the ten most dispensed prescriptions manage hypertension or dyslipidemia, two core components of the metabolic syndrome (KFF 2007).[15] For example, Lipitor and Zocor, which manage hypertension and high blood pressure, respectively, were the two biggest prescription drugs sold in 2004, bringing in $7.7 and $4.6 *billion* in sales. Any prescription drugs that treat hypertension, dyslipidemia, hyperglycemia, and obesity are all potentially useful in treating patients who might be classified with the metabolic syndrome.

The Politics of Metabolism

Collectively, these increases in biomedical, government, and pharmaceutical attention on the metabolic syndrome reflect a growing *apparatus* of scientific, government, and corporate actors and institutions that are deeply invested in studying, regulating, and profiting from problems associated with metabolism. French social philosopher Michel Foucault defined the apparatus as the heterogeneous network of power and knowledge that can be established between discourses (including scientific, philosophic, moral, and philanthropic statements), institutions, structural arrangements, policy decisions, laws, and administrative measures (Rabinow and Rose 2003). An apparatus forms as a strategic response to a specific scientific discovery, political crisis, or economic opportunity (Rabinow and Rose in Foucault 2003: xvi).

In this context, the metabolic syndrome is a new discourse in the apparatus I call *the politics of metabolism,* which I define as the discourses, social processes, and institutional relationships that govern the metabolic health of individuals and groups. These relationships operate on several different levels of analysis. At the macro level, big social institutions like government health research institutions, pharmaceutical corporations, and professional medical doctors represent the three groups of social actors who are produce discourses of the metabolic syndrome and race. At the micro level, these discourses, technologies, and practices operate at the levels of biochemicals, DNA, and prescription pills.

The Metabolic Syndrome and Race in the Politics of Metabolism

The metabolic syndrome not only constitutes a new way of constructing, studying, and treating human metabolism, it may also constitute an emerging site for the construction of new racial meanings in the politics of metabolism. Specifically, the metabolic syndrome may draw upon and extend knowledge-making practices that have long constructed race as natural, biological, and genetic. In the contemporary politics of metabolism, the metabolic syndrome draws upon and extends scientific practices that have long used race to categorize individuals into groups. To document and understand the relationships between the metabolic syndrome and race, and to analyze the racial meanings produced through the science of the metabolic syndrome, this study focuses on the use of racial and ethnic categories in metabolic syndrome research and research on prescription drugs that may be related to the metabolic syndrome.

The first set of relationships that might link the metabolic syndrome to race concern the specific constructions of race, and relatedly, ethnicity, that are used in metabolic syndrome research. Race and ethnicity are both socially constructed systems of categorization that are used to identify, group, and rank human beings, albeit based on different criteria. Race is a socially constructed category that emerged in the 1700s to classify individuals into so-called races based on presumed biological differences between population groups. Ethnicity is a socially constructed category that emerged in the 1920 to classify individuals into so-called ethnic groups based on presumed differences in culture, geographic origin, and ancestry. Race and ethnicity are related in that ethnicity emerged in large part in response to critiques of biological

notions of race. Given this historical relationship, race and ethnicity are not interchangeable systems of categorization. However, there is meaningful overlap between what are considered racial and ethnic groups. For example, African Americans are considered to be both a racial and an ethnic group. Race and ethnicity are both controversial systems of categorization especially in the context of biomedical research because individual biological and genetic differences do not fall neatly along racial and ethnic lines. In other words, despite their shared origins in response to biological interpretations of individual and group differences, race and ethnicity are social constructions.

Because of historical and current federal research policies that regulate demographic data collection, statistical information about a research subject's race and/or ethnicity is routinely collected along with anthropomorphic, molecular, and genetic information about a subject's metabolism.[16] Therefore, the sampling frame, analytic strategy, and research findings of metabolic syndrome research studies are often framed using these racial and ethnic categories. In this regulated scientific environment, it is also common to see published review articles that are focused exclusively on particular racial and ethnic minority groups.[17] In this context, many researchers also frame their research on OMB racial groups as ethnic instead seemingly to avoid talking explicitly about race in ways that could be interpreted as racial bias, or worse, scientific racism.

A second set of relationships that might link the metabolic syndrome to race concern the use of race to study, prescribe, and label drugs that may be related to the metabolic syndrome.[18] Unsurprisingly, drug companies are actively recruiting

individuals who seemingly have the metabolic syndrome in their clinical research. For example, the African American Rosuvastatin Investigation and Efficacy Study (or, ARIES Study) investigated the ability of Crestor, a powerful new member of the statin class, to lower both blood pressure *and* cholesterol in a self-identified African American population (Flack, Victor, Watson, Ferdinand, Saunders, Tarasenko, Jamieson, Shi, and Bruschi 2008). A second recent study, the Clinical Utility of Caduet in Simultaneously Achieving Blood Pressure and Lipid Endpoints in a Specific Patient Population (or, CAPABLE Study) investigated whether Caduet, a combination of two drugs Lipitor and Norvasc, was effective at lowering African Americans' blood pressure and cholesterol (Flack et al. 2008). Both of these prescription drug studies were conducted in a manner similar to the way that African Americans were targeted in the research and marketing of BiDil, an anti-hypertensive medication that is the first drug approved by the FDA for specific use in a specific so-called ethnic group: African Americans (Duster 2005; Kahn 2006; Sankar and Kahn 2005). Yet, coupled with recent research findings that suggest that members of racially and ethnically categorized groups might require different medications, dosages, and routes of administration of prescription drugs trials and because of new federal guidelines about the inclusion of racial and ethnic minorities in clinical trials, this research has a new racial dimension.[19]

Through these practices, the metabolic syndrome may have become a new discursive tool used to produce new meanings of race in the politics of metabolism. The overarching theoretical challenge of understanding the possible linkages between the metabolic syndrome and race is grasping how they operate in what Foucault

called a polyvalent manner. Foucault used the term polyvalence to describe how discourses can be used as both a technique and an outcome of power (Foucault 1978: 101-102). For example, laws are both produced by and constitute state power. In other words, discourse is not only the documented effect of power relations, as in the case of the legal discourse that is produced by the state (discourse as an effect of power), discourse can also be used to mark its own material effects on bodies themselves by virtue of an existing set of power relations (discourse as a technique of power). Understood in this context of polyvalence, discourses establish the scientific knowledges that are used to justify unequal power arrangements. In turn, these arrangements structure the production and content of scientific knowledge.

Therefore, this study explores how the metabolic syndrome and race may operate together as polyvalent forms of power and knowledge in the politics of metabolism. Three questions guide the study. First, how did the metabolic syndrome emerge as a new discursive formation in the politics of metabolism? Second, how are current conceptions and meanings of race constructed through the discourses of the metabolic syndrome? Third, what are the implications of this emerging relationship between the metabolic syndrome and race for understanding the construction of racial meanings in the politics of metabolism?

Research Methods: Genealogy and Discourse Analysis

Foucault's genealogy serves as the central methodology for this study of the relationship between current constructions and practices of race and the emergence of the metabolic syndrome. Stated differently, this study is a genealogy of the metabolic

syndrome and race in the United States. I ground my articulation and execution of genealogy based upon what Foucault wrote in books, essays, and lectures, as well as secondary interpretations of key Foucauldian scholars.[20] One of the main practical challenges of executing this study was how to transform the seemingly obtuse method of genealogy into a set of procedures that I could follow consistently to analyze different kinds of documents and construct a critical narrative that challenge the relationships between the metabolic syndrome and race. Any reasonable interpretation of genealogy is complicated by the fact that Foucault never codified specifically how he believed genealogies ought to be carried out in various disciplines, and when he did state his method in recognizable terms, these definitions shifted over time in various contexts. Indeed, Foucault was not forthcoming with a codified checklist of procedures a researcher might follow to conduct a genealogy, and since his death, his interpreters have continued to struggled to do the same.[21] To this point, leading Foucault scholars Nikolas Rose and Paul Rabinow argue that the methodological construct of genealogy might need to be re-imagined through the lenses of comparative or ethnographic research methods, depending on how social arrangements unfold in the contemporary moment (Rabinow and Rose 2003). Despite the spirited debates about Foucault's codification of a genealogical method, and disagreement about how these methods ought to be deployed, this study attempts to utilize a grounded interpretation of what genealogy entails.

Genealogy is a historical methodology that traces the emergence and descent of technologies and practices used to produce discourses about the body and the political contexts through which these elements are constructed as self-evident,

natural and universal. Genealogy is also a form of political critique that diagnoses how such discourses, practices, and technologies are embedded in and rationalize unequal power arrangements. The intellectual and political intent of genealogy, therefore, is to contest discourses that are used to instantiate, enable, and support repressive and/or productive forms of modern social power by showing how those discourses have determined (in a limited way) what constitutes our present understanding of ourselves, our social world and the social relationships therein. It is in this sense that Foucault and others have referred to genealogy as a "history of the present." There are two intertwined analyses that comprise genealogy, namely, the analyses of descent and emergence. Because the analyses of descent and emergence helped me specify the data sources that I analyze with discourse analysis, I will briefly summarize these important components of the genealogical method.

The analysis of descent and emergence

The analysis of descent documents the heterogeneous sites of knowledge production by tracing the actual research techniques and procedures used in scientific practice (Foucault 2003 [1971]; Foucault 2003 [1976]; May 1993). The analysis of descent shows how these techniques and procedures structure what kinds of scientific practices are acceptable and how social arrangements shape the production of scientific knowledge (Foucault 2003 [1978]).[22] In this Foucauldian sense, practices can be defined as "places where what is said and what is done, rules imposed and reasons given, the planned and the taken-for-granted meet and interconnect" (Foucault 2003 [1971]). Thus, the analysis of decent narrates actual historical events

where the objects of genealogical analysis are inscribed by particular constellations of discourses, techniques, and practices.

However, in contrast to a more conventional form of historiography that might produce a linear and modern history of these technologies and practices, the analysis of descent highlights what I call the disjunctures that are central to the inscription of power/knowledge relationships. A disjuncture is an accident, error, shift, or deviation that challenges the assumption that historical events are homogeneous and represent self-evident truths. Disjunctures represent distinctive moments where the structure of discursive possibilities either opens up or contracts depending on the particular configuration of the field of power and knowledge in play in that moment. Foucault's approach to analyzing the disjunctures of modern history is directly linked to one of the central epistemological aims of genealogy; namely, to challenge self-evident discourses and practices that justify unequal social arrangements. Foucault worked to carry out this aim without resorting to presentism, which Foucault interpreter Mitchell Dean defines as "the unwitting projection of a structure of interpretation that arises from the historian's own experience or context onto aspect of the past under study" (Dean 1994: 28). Based on this understanding, I use the construct of disjunctures to work against the notion that this study is a modern history of ideas and to highlight the multiple open-ended processes that undergird the production of the metabolic syndrome and race as taken-for-granted truths.

In conjunction with the analysis of descent, the analysis of emergence situates the emergence of a practice or discourse within in a broader network of institutionally based power/knowledge relationships.[23] What is distinctive is that the analysis of

emergence should avoid describing the causes, motives, or perceived intent of a given social practice as self-evident, natural, and universal. To the contrary, practices can emerge in multiple sites of power, can take radically different forms in different historical moments, and do not result from one unitary cause. For example, drawing upon an analysis of Foucault's The Birth of the Clinic: An Archeology of Medical Perception (1975), Foucauldian scholar Todd May (2003) argues that it was not always self-evident that individual bodies required examination to determine the cause of illness. May argues that genealogy challenges three epistemological assumptions about the search for the emergence of a discourse or practice. The first assumption that genealogy challenges is that there is an essence behind a discourse or practice of interest, that there is "a being behind the becoming" (May 2003: 74). The second assumption is that the beginning of a discourse or practice is a highly visible social production when they are just as likely to be hidden from plain sight. Third, the concept of origins assumes a foundational notion of truth, that the origin is the "pristine instant" or "moment of pure communication [of a discourse or practice] with itself" (p. 74) (May 1993) Thus, this analysis recognizes that social practices are historically structured in a multiplicity of institutional and discursive contexts and that no single institution or individual is ever solely responsible for the emergence of a practice or discourse.

Foucault's treatment of the body illustrates these dual concepts of descent and emergence. For instance, again in The Birth of the Clinic: An Archeology of Medical Perception (1975), Foucault argued that the surveillance of the body was historically organized via a clinical gaze, a way of seeing and knowing the body and nature, that

sought to rationalize the space-time between life and death by classifying and organizing the body scientifically (Foucault 1975b). Foucault argued that genealogies should make an ascending analysis of power that traces the descent and emergence of the technologies used in the scientific study of the body and the social regulations of institutional power (Foucault 2003[1976]: 30). In this way, the analysis of descent should reveal how the body itself becomes inscribed through the production of discourses that are produced in service of power arrangements (Foucault 2003 [1971]). In other words, Foucault believed that the body was the canvas upon which power paints history. The analysis of emergence investigates what Foucault called the hazardous play of dominations, which represents the practices, theories, and regulations of social institutions that impose various rights, obligations, and practices on the body (Foucault 2003 [1971]). Taken together, these components of genealogy examine the polyvalent ways in which the body, the spaces around it, and the materials inside of it became both the object of knowledge and subject to new forms of social power.

Understanding the politics of metabolism requires a methodological framework that can analyze the relationships between power and knowledge, which I believe lies at the heart of my core questions about the metabolic syndrome and race. I have selected this method because I see genealogy as a necessary methodological alternative to conducting a standard quantitative analysis of racial health inequality and the metabolic syndrome that often aims to produce truth claims about the metabolic syndrome and race. In contrast, a genealogical account of the relationships between the metabolic syndrome and race would not assume nor does it seek to posit

any so-called scientific hypotheses about the metabolic syndrome and race. Rather, a genealogical account would examine the social structures of power and knowledge that made it epistemologically possible for biomedical scientists to produce scientific claims about the metabolic syndrome and race in the first instance.

Discourse analysis

Drawing on this notion of genealogy as a form of social historiography and political critique, I also rely heavily on the tools of discourse analysis as a method for analyzing my documents.[24] Rather than only analyzing the meaning of a discourse, discourse analysis also analyzes the structure of the discursive themes by which a particular discourse is produced. Specifically, discourse analysis asks three core questions about the production of discourses: (1) who produced the discourses and with what resources? (2) Under what political, economic, and historical conditions were the discourses produced? (3) How are the meanings of the discourse shaped by these economic, political, and historical conditions? Thus, my discourse analysis aims to interpret how the discourse of metabolic syndrome emerged in ways that draw upon constructions of race in service of producing new meanings of race. I analyze the explicit and implicit assumptions about race that structured the discourses, practices, and technologies of the metabolic syndrome.

Data sources and procedures

The methods of genealogy and discourse analysis bring into focus the types of documentary evidence required to analyze the relationships between race and the metabolic syndrome. Each of these documents contains specific information about the discourses, techniques, and practices used in the scientific study of the body. This study analyzes three types of documents: (a) published research, commentaries, and editorials on the metabolic syndrome and race in professional and academic biomedical journals; (b) corporate documents from pharmaceutical companies including yearly reports, regulatory submissions to the FDA, and clinical trial documentation; and (c) government documents including NIH and FDA regulatory guidelines on the collection of data on race and ethnicity in U.S. biomedical research and clinical trials, published reports and scientific documents from the NIH and its institutes, and other relevant government agencies. More information on my data sources and procedures can be located in the Appendix.

My data collection proceeded as follows. I employed three basic strategies to traverse and circumscribe the universe of documents about the metabolic syndrome and race. As I demonstrated earlier, thousands of research articles have been published on the metabolic syndrome, and while it was not possible to analyze all of the documents about the metabolic syndrome in this study, it was important to establish a subset of this universe of documents to analyze. The overall purpose of this three-step process was to identify the primary documents that formed the evidentiary bedrock of my study.

First, I conducted extensive searches of multiple biomedical research databases in order to compile a comprehensive bibliography of documents pertaining to the metabolic syndrome and race. Specifically, I repeatedly searched three prominent databases in this first strategy: (1) www.science.gov, the federal government's central search engine for published scientific research both within and outside the purview of the government and its scientific agencies; (2) Medline and PubMed™ Central, the premier bibliographic databases for the National Library of Medicine of the National Institutes of Health; and (3) ISI Web of Science, Science Citation Index.

A second strategy was to use the ISI Web of Science cited citations index to conduct citation counts on the published documents I found on the metabolic syndrome and race to determine the extent to which a particular document has traveled and gained scientific currency throughout biomedicine. I employed this strategy in the full recognition that some sites of biomedical knowledge production have more political and scientific influence than others. For instance, a document published by one of the National Institutes of Health wields more influence than a document published in a relatively obscure biomedical journal that deals with a narrow subject matter. When appropriate, I make reference to this information throughout the study.

A third strategy was to place special emphasis on the relatively smaller number of government and corporate documents pertaining to the metabolic syndrome and race. The fact that governments and corporations publish documents about the metabolic syndrome and race is significant for how I conceptualize the

institutional relationships between social power and biomedical knowledge that have converged around the metabolic syndrome and race.

Organization of the Study

Recall the three questions guide the study. First, how did the metabolic syndrome emerge as a new discursive formation in the politics of metabolism? Second, how are current conceptions and meanings of race constructed through the discourses of the metabolic syndrome? Third, what are the implications of this emerging relationship between the metabolic syndrome and race for understanding the construction of racial meanings in the politics of metabolism?

To investigate these questions, this study proceeds as follows. In chapter two (Theorizing Race, Biomedicine, and Power), I outline the theoretical frameworks that I use to explain the relationships between the metabolic syndrome and race by drawing on ideas from three bodies of scholarship: critical race theory, biomedicalization, and the social theory of Michel Foucault, especially his framework of biopower. Each of these frameworks provides a unique perspective on the relationships between the metabolic syndrome and race, but together they provide a power and nuanced interpretive framework through which to articulate a genealogy of the metabolic syndrome and race.

In chapter three (Disciplining Bodies & Regulating Populations: The Racial Formation of the Metabolic Syndrome), I analyze the emergence of the metabolic syndrome, highlighting some of what has made it controversial, and analyze some of the central ways it is connected to conceptions and meanings of race. I argue that

race was central to the emergence of the metabolic syndrome and that the metabolic syndrome, in turn, serves as dynamic new site for the production of race. This chapter is divided into two main sections. In Part I, I trace the emergence of the metabolic syndrome across three historical periods in American biomedicine: Technical and Conceptual Foundations (1947-1986); From Syndrome X to Dysmetabolic Syndrome X (1987-2000); and The Ascendance of the Metabolic Syndrome (2001-present). Each of these periods represents a distinct conceptual moment in the attempt to establish the metabolic syndrome as a biological clinical disease as a legitimate object of biomedical knowledge production. Across these periods, I demonstrate that the metabolic syndrome has had several different names and empirical definitions that each has different implications for how the syndrome constructs racial meanings and explains racial inequality. In the second part of the chapter, I trace the different racial meanings that are produced during the three conceptual periods. In some contexts, this racial production is explicit and in others it is implicitly woven into the everyday practice of doing biomedical science. In Part II of the chapter, I trace the production of racial meaning during the three conceptual periods of the emergence of the metabolic syndrome: Sampling Normal Subjects, 1956-1987; Is Race Really To Blame? 1988-2000; and The New Special Populations, 2001-present.

In chapter three (Killer Applications: The Racial Pharmacology of Metabolic Syndrome), I use the ideas developed in the preceding chapters to analyze one site of genealogical descent of the metabolic syndrome into biomedical research on differential access and response to prescription drugs among African Americans. I

interpret these two cases as part of a new racial pharmacology, or the scientific study of prescription drugs in racially categorized bodies and populations, that is a central feature of the politics of metabolism. I divide this chapter into four sections. In the first section, I develop the metaphor of killer applications to examine how prescription drugs operate in the new racial pharmacology. This metaphor is especially well suited for examining prescription drugs and for exploring the new ways that biotechnologies are involved in the production of racial meaning in the politics of metabolism. In the third and fourth sections, I compare the different ways that conceptions of race (and ethnicity) and the metabolic syndrome are used in the racial pharmacology[25] of two potential killer applications: antipsychotics and statins. I argue that race and the metabolic syndrome intersect in unique ways in the racial pharmacology of these two potential killer applications. The case of antipsychotics involves the pharmacokinetic effects that "atypical" antipsychotics have on the development of the metabolic syndrome, explicitly in populations with schizophrenia. The case of statins involves the development and marketing of statins, explicitly in populations with high cholesterol.

In the concluding chapter of the study (The Politics of Metabolism), I summarize the study's main interpretations, outline the implications of the study for critical social theory, and elaborate on the broader sociological significance of the study.

Figure 1.1. The number of articles published on the metabolic syndrome, 1989-2008

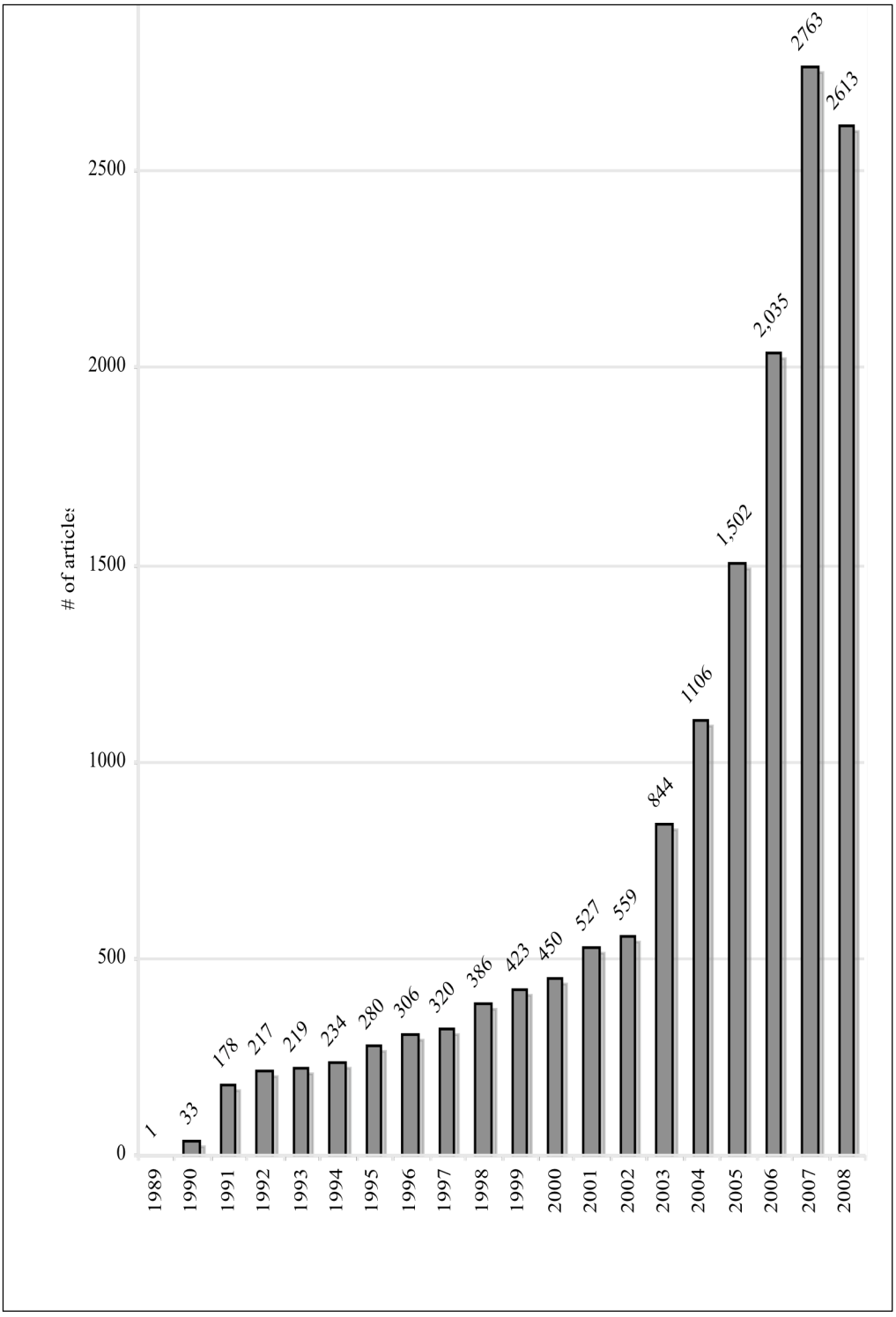

Figure 1.2. Global map of clinical trials recruiting for the metabolic syndrome, 2009.

Chapter 2: Theorizing Race, Biomedicine, and Power

This study investigates the relationships between the emergence of the discourses and practices of the metabolic syndrome and current conceptions of race in the U.S. Three questions guide this study. First, how did the metabolic syndrome become a discourse and a technique for producing biomedical knowledge? Second, how are current conceptions and meanings of race and racial difference forged through the discourses and practices of the metabolic syndrome? Third, what are the implications of this emerging relationship between the metabolic syndrome and race for understanding the construction of racial meaning in the politics of metabolism?

To situate these questions in a broader theoretical context, I draw upon core themes across three related bodies of scholarship: critical race theory, biomedicalization, and the social theory of Michel Foucault, especially his framework of biopower. In this chapter, I first develop these frameworks to provide the theoretical vocabulary for the second and third chapters of this study. Then, I consider how each framework sheds light on different aspects of the relationships between race, biomedicine, and power, which together form an important context through which the politics of metabolism play out in the United States. I conclude the chapter by outlining some of the areas of convergence and divergence across these theoretical frameworks.

The Framework of Critical Race Theory

Because critical race theory provides a framework for analyzing the metabolic syndrome as an emerging site of racial formation and the production of

racial meaning in the United States, it is important to outline some of its distinguishing features. Critical race theory refers to a historical and contemporary body of scholarship that aims to interrogate the discourses, ideologies, and social structures that produce and maintain conditions of racial injustice. At the most general level, critical race theories analyze how race and racism are structural elements of global, national, and local social organizations and in the life experiences of people living in racialized social orders. In particular, critical race theories have analyzed how race and racism were produced as elements of social structure in earlier periods and continue to be reproduced through contemporary social arrangements.

Critical race theories understand race as a constitutive feature of global social, political, economic, and cultural organization since the 1600s and not as a naturalized system of biological essences. Critical race theorists Michael Omi and Howard Winant defined race as "a concept that signifies and symbolizes sociopolitical conflicts and interests in reference to different types of human bodies" (Omi and Winant 1994: 55). This definition reflects the centrality of the body to critical racial theories, because black and brown bodies have born the brunt of racism. Race concepts and their accompanying racisms were used to establish colonial social systems (McClintock 1995; Stoler 1995), modern nation states and global political economies (Goldberg 2002; Omi and Winant 1994; Winant 2001), and the human biological sciences and medicine of the eighteenth, nineteenth, and twentieth centuries (Barkan 1992; Duster 2003a; Graves 2001; Reardon 2005; Stephan 1982).

Critical race theories understand racism as a vast and complicated system of institutionalized practices that structure the allocation of social, economic, and

political power in unjust and racially coded ways. While some race theorists have examined racism as a form of maligned individual prejudice, critical race theorists tend to embrace a more institutional understanding of racism that aims to identify how racism is embedded in the racially patterned practices of social institutions (Bonilla-Silva 1997; Bonilla-Silva 2003; Carmichael and Hamilton 1967). In examining the institutionalized aspects of racism, critical race theorists challenge the idea that people of color are responsible for their own oppression (Brown, Carnoy, Currie, Duster, Oppennheimer, Shultz, and Wellman 2003). These theories continue to challenge entrenched racial inequalities in health, education, criminal injustice, political representation, and social class (Brown et al. 2003; Guiner and Torres 2002; Shapiro 2004). The body of knowledge is too broad to review in great detail here, so in the section that follows, I develop four central themes from critical race theory that inform this study. These core themes are: 1) racial formation and racial projects; 2) science and medicine as sites of racial formation and racial projects; 3) scientific racisms and essentialisms; 4) the nation state as a sites of racial formation in the context of biomedical research.

The first core idea from critical race theory that is germane to this study is racial formation, which refers to the social and historical process by which racial categories are created, transformed, and destroyed (Omi and Winant 1994). Racial formation theory emerged in the 1990s in response to contemporaneous theories of race that viewed race as an epiphenomenon of ethnicity, social class, and/or nationality. From this perspective, interpreting the meaning of race analytically in the context of racial formation involves framing race social structurally and recognizing

racial dimensions in social structures (Omi and Winant 1994: 57). For example, analyzing race in the context of criminal justice would involve examining how the laws and practices of social institutions structure the unequal treatment of racially categorized individuals, not just comparing statistical rates of incarceration across groups or conducing psychological experiments to determine the inherent criminality of raced inmates. Race and racism, then, must always be understood in the context of the institutional relationships that are brought to bear in shaping racial conflicts and interests.

In this way, the notion of racial projects is also central to racial formation theory because it articulates how discursive and institutional elements of race work together in the process of racial formation. According to Omi and Winant (1994), a racial project is "simultaneously an interpretation, representation, or explanation of racial dynamics, and an effort to organize and distribute resources along particular racial lines" (Omi and Winant 1994: 56). Racial projects combine what race means in a particular discursive practice and the ways in which both social structures and everyday experiences are racially organized based upon that meaning (Winant 2001; Winant 2004).

A second core theme of critical race theory that is germane to this study is that critical race theory has long recognized the centrality of science and medicine to the construction of racial concepts and meanings that in turn influence the practices of social institutions, multiple levels of society, as well as the changing meaning of race over time. In other words, science and medicine are primary sites of racial formation. As many critical race scholars have observed, the meaning of race in the

context of science and medicine has shifted dramatically over the past 100 years.[26] In the wake of World War I, scientific conceptions of race began to shift from the so-called "typological view" to a population-based conception of race that has dominated biological theory in the post-war period (Barkan 1992; Gannett 2001).[27] In the typological approach to race, every conceivable physical and mental characteristic of the human body was measured and compared across racial types, in an effort to validate pre-existing racial taxonomies. In a population approach to race, anthropomorphic, mental, and social characteristics were compared across groups classified into races to perform the same ideological work, to confirm the hardness and impermeability of racial categories.

The population approach to race did not replace the typological approach, nor did the concept of population first emerge during this period following WWII, rather race was increasingly conceptualized as a population phenomenon, a new way to talk about presumably different populations. However, typological and population-based explanations of racial inequality can both draw upon essentialist understandings of race. By the 1950s, the UNESCO statements on race signaled the emerging scientific consensus that race concepts were socially constructed and were without foundation in human biology or nature (Reardon 2005). However, in contemporary biomedical theory and practice, racial categories are still assumed to be proxies for genetic or biological variation (IOM 2002).

A third core theme that is germane to this study is that critical race theories have challenged scientific racial projects that were used to create racial hierarchies and that were used to justify white supremacy—these racial projects are called

scientific racism. Scientific racism consists of meanings of race based on presumed physiological, biological, and/or genetic differences and the practices of deploying such ideas as explanations for racial stratification and oppression. Scientific racism emerged as an ad hoc justification of colonial subjugation and slavery in the eighteenth century and is most easily associated with the social practices of eugenics and Nazi racial hygiene in the nineteenth and early-to-mid twentieth centuries. Racial formation theorist Howard Winant argues that scientific racism functions by severing the effects of racism from the causes of white capitalist supremacy by attributing systematic racial inequalities to the nature of things, namely, to science (Winant 2001: 296).

Critical race theories have refuted scientific racism as a racial project by dismantling one of its core philosophical ideas: racial essentialism. Racial projects can be defined as racist if they create or reproduce structures of racial oppression based on essentialist understandings of race. Racial essentialism is the assumption that race categories reflect some inherent hierarchical organization of human bodies based on essences. Racial essentialism is the philosophical underpinning of scientific racism because the presumptive essential differences between bodies are 1) permanent and 2) cannot be caused by social forces. Racial essentialisms disallow institutional explanations of racism because. In his analysis of how European nation states used race to justify colonial domination and European expansion during the 19th and 20th centuries, philosopher of race David Theo Goldberg articulates a useful philosophical distinction between racial naturalism, racial primitivism, and racial historicism that

illustrates how different racial essentialisms operated in the context of modern government formation.

Racial naturalism is the view that racial inequalities are the outcome of natural law and racially subjected peoples are considered to be objective features of the natural environment meant for political appropriation and economic exploitation. Through the racial naturalism, race is the conduit for collapsing what is social and historical into and upon what is considered natural (Goldberg 2002). For Goldberg, primitivism emerges during the mid-twentieth century as a discursive bridge for racial essentialisms, as the forms of racial rule began to shift from racial naturalism towards historicism. As Goldberg writes, the logic of primitivism was to transform the subjects of colonial subjugation into idealized versions of themselves, frozen-in-time and taken-for-granted (Goldberg 2002: 93).[28] In contrast to naturalism and primitivism where non-Europeans are naturally inferior, through the racial historicism, racially subjugated peoples are historically immature and are thus subject to the civilizing process that constituted Manifest Destiny. Thus, racial historicism views racial inequality as the outcome of history, a history guided by the hidden hand of Enlightenment progress and modernity, the production of the "facts" of European racial superiority.

A fourth core theme of critical race theory that is germane to this study is the recognition that the US government is a central site of racial formation, especially in terms of the knowledge production apparatuses of the government that produce and enforce racial classification.[29] Since the taking of the first federal census in 1790, race has been a central feature of the United States' political data collection system.

The 1790 census measured the numbers of "free white males" as well as the "the number of slaves."[30] For centuries, the standard practice was for the census taker to make a determination as to the racial classification of individual members of the population. The federal government has employed numerous taxonomies of race in the Census. In response to the changing terms and meanings of race and ethnicity used in federal data collection, in 1997, the Office of Management and Budget issued new regulations on maintaining, collecting, and presenting federal data on race and ethnicity in the United States. The minimum categories for data on race and ethnicity for Federal statistics, program administrative reporting, and civil rights compliance reporting are defined as follows in Table 2.1:

[Insert Table 2.1. Office of Management and Budget racial and ethnic categories, 1997]

These federal regulations were intended to provide a standardized and universal language for defining the major population groups of the country and applies to all federal data collection efforts, including all clinical and biomedical research funded by the government (Office of Management and Budget 1997). According to these regulations, the U.S. government and its agencies consider self-identification as the preferred means of obtaining information about an individual's race and/or ethnicity.

Nonetheless, the implementation of standardized federal racial and ethnic categories, and the technique of self-identification, permitted the continued expansion of recruitment of racial and ethnic groups into biomedical research (Epstein 2007;

Evelyn, Toigo, Banks, Pohl, Gray, Robins, and Ernat 2001; Stevens 2003). This represents a substantive shift from earlier forms of racial knowledge that were grounded in the institutions of science and medicine. Yet, the OMB states that its racial and ethnic categories were developed to represent social-political constructs, and are not anthropologically or scientifically based (OMB 1997).[31] In other words, the federally mandated racial and ethnic categories are intended to be interpreted and applied in administrative and legal contexts, not scientific and biomedical contexts.

Sociologist Steve Epstein (2007) examined these and other recent changes in US biomedical research policies and practices in the mid-1990s regarding the inclusion of racial and ethnic groups and women in clinical research and trials. Drawing on racial formation theory, Epstein analyzes the US government's regulation of racial categories in biomedical and clinical trials research. As of 1994, the National Institutes of Health policy was that women and members of minority groups and their subpopulation must be included in all NIH-supported biomedical and behavioral research projects involving human subjects (NIH 1994).[32] Epstein's analysis raises important questions about what types of bodily difference the government should measure, how this measurement should be carried out, and how such differences should be interpreted.

Critical race theorists have focused explicitly on illuminating and contesting the social, economic, and political arrangements that undergird racism as a system of oppression. The core themes I have outlined provide a sense of critical race theory's multiple interventions into these arrangements. Critical race theory provides an organizing theoretical framework for my study because of this central preoccupation

with the social reproduction of race and racism, particularly in American society. As I have argued, critical race theory has long recognized the centrality of science and medicine to the construction of racial concepts and meanings that in turn influence the practices of social institutions. Drawing on this critical race framework, I argue that the metabolic syndrome is an emerging site for the reproduction of race and racism in American society.

In the next section, I turn to the framework of biomedicalization to understand how recent shifts within science, medicine, and technology can help to account for the relationships between the metabolic syndrome and race.

The Framework of Biomedicalization

The framework of biomedicalization constitutes a second important theoretical framework shaping this project. Biomedicalization is a historical and analytic framework for understanding the series of institutional, scientific, and technological processes that have transformed American biomedicine on multiple levels of social organization, especially since the mid-1980s (Clarke, Mamo, Fishman, Shim, and Fosket 2003). Whereas medicalization refers to a process whereby social practices, bodily processes, and bodily materials were subsumed under the jurisdiction of clinical medicine (Starr 1982), biomedicalization refers to the ways that medicalization is shaped by the powerful intersection of medicine, biology, *and* technology. Biomedicalization has been theorized from within a broader interdisciplinary body of knowledge called science and technology studies (STS). In constructing the framework of biomedicalization, I highlight six themes

that are especially relevant for this study: 1) technoscience; 2) molecularization; 4) the increased importance of risk; 5) the development of biomedical capitalism; and 6) stratified biomedicalization.

The first core theme of biomedicalization is technoscience, which is a way of understanding these increasingly technological and biological aspects of the practice of medicine in the contemporary world (Haraway 1997; Latour 1987). This framing of the relationship between biology and technology requires analyzing the practices by which scientific discourses about the body are culturally and collectively produced by scientists and their technologies (Haraway 1997; Jasanoff 2004; Oudshoorn 2002). Stated differently, bodies have to be manipulated to make them produce biomedical knowledge. This bodily manipulation occurs through the use of biotechnologies such as diagnostic tools, screening tests, drugs, and other regulatory devices. Thus, an important understanding of technoscience, and one shared by many scholars in the field known as science and technology studies (STS), is that biomedical scientists gain cultural authority and produce scientific objectivity by concealing the institutional practices that construct and constitute such knowledges and the unequal power relationships in which those practices are embedded.

For example, in her Foucauldian-inspired archaeology of sex hormones, STS scholar Nelly Oudshoorn shows how cultural norms and ideas about sex difference shape the institutional practices that produce knowledge about masculine and feminine sex hormones. Whereas once the essential nature of femininity had been located in specific organs especially the uterus and the ovaries, Oudshoorn argues that with the technoscientific interventions in the early 20th century in organic chemistry,

femininity increasingly became associated not with specific organs, but their chemical substances. Oudshoorn argues that prescientific ideas about sex difference influenced the interpretation of which hormones were labeled as male and female. However, the social and cultural contexts in which these ideas about sex difference influenced knowledge production do not become part of the record of so-called scientific truth. The epistemology of technoscience recognizes the seamless relationships between biomedical technologies, their bodily applications, and the scientific knowledge they are used to manufacture (Oudshoorn 2002). "Science," feminist technoscience scholar Nelly Oudshoorn states plainly, "is not just words" (Oudshoorn 2002: 13).

As Oudshoorn's work demonstrates, a second core theme of biomedicalization is molecularization or the emergence and dominance of scientific practices, technologies, and theories that conceptualize and conduct surveillance of human life at the molecular level (Kay 1993; Rose 2001; Shostak 2004). Molecularization encompasses processes of institutional and structural reorganization, the creation and application of new technologies, and the production of new theoretical ideas about molecules and their relationship to human disease. Beginning in the late 1800s and continuing to the present, molecularization was a central feature of the ways that the biological sciences conceptualized the body and its processes.

More recently, STS scholars have examined how scientists construct meanings of race at the molecular level. For instance, Sara Shostak (2004) analyzes two trends in genomic research on racial differences in environmental health that illustrate this theme. First, scientists in the fields of molecular epidemiology and

toxicogenomics are measuring the effects of environmental exposures at the molecular level, i.e. on DNA, genes, and gene expression. Second, scientists are using race to search for genetic differences that may shape individual and population responses to environmental exposures. On one hand, these new molecular tools are viewed as enabling new strategies of disease prevention that might help to interrupt the process from environmental exposure to illness. On the other hand, these molecular techniques might be used to create new scientific conceptions of race that sustain "a new era of molecularized scientific racism" (Shostak 2004: 547).

A third core theme of biomedicalization that emerges out of a technoscientific approach to studying life at the molecular level is an increasing emphasis on risk in biomedicine. The so called risk factor paradigm has been the dominant theoretical framework for chronic disease epidemiology in the second half of the twentieth century (Susser 1998; Susser and Susser 1996a; Susser and Susser 1996b). This methodological focus on risk in epidemiology reflects a influence of the dominant biomedical theory that human illness is caused by an interaction of environmental, physiological, and behavioral factors: so-called risk factors. In the risk factor paradigm, researchers produce risk statistics from population-level surveillance data that show that particular variables, often conceptualized at the molecular level are statistically associated with an undesirable health outcome. Analysts then interpret these population-level risk statistics as individual-level risk factors that, by virtue of their expression of molecular processes, become transformed into biologically meaningful causes of poor health at the individual level.

Sociologist Janet Shim's analysis of the implications of this risk factor paradigm for constructions and meanings of race illustrates this third core theme of biomedicalization. Shim's research illustrates how practices of surveillance and discourses of risk are coproduced, which means that scientists use practices of surveillance to produce knowledge about risks, and then risks are used to justify further practices of surveillance (Clarke et al. 2003: 172). For example, as biomedical researchers analyze population surveillance data collected using race categories, these practices contribute to the construction of race as an individual-level cause of disease. In practice, race variables are often statistically associated with undesirable health outcomes, and in this context race is often interpreted as an individual-level risk factor. In this context, bodies marked with risk *as* race suggests that race itself becomes an indicator of risk.

A fourth theme of biomedicalization that is relevant to this study is that biomedicine is a profitable global capitalist system that exploits human health as a commodity, especially the pharmaceutical industry(Hegecoe 2004; Kremer and Glennerster 2004; Moynihan, Heath, and Henry 2002). Drawing on Marxist social theory, scholars Catherine Waldby and Nikolas Rose argue that contemporary biomedicine is increasingly driven and organized by the search for biovalue, or the production of a surplus out of life itself (Rose 2006; Waldby 2000). Feminist science studies scholar Charis Thompson takes this argument one step further to argue that biomedicine has helped to establish a new mode of capitalism in the United States—the biomedical mode of capitalist reproduction (Thompson 2006a).

Thompson identifies five distinguishing features of the biomedical model of reproduction that comprise biomedicalization as a capitalist system. First, whereas traditional forms of capitalism have focused on modes of production, biomedical capitalism has shifted to a focus on reproduction through the deployment of biotechnology. Second, as I suggested earlier, whereas traditional forms of capitalism produced profit through the extraction of surplus labor, biomedical capitalism has shifted to the extraction and maximization of profit out of bodies and their bodily products. Third, whereas traditional forms of capitalism alienated workers from their labor and the products of their labor, biomedical capitalism has shifted to a situation where bodies are alienated from the profits of their own reproduction. Fourth, whereas traditional capitalism is premised on the accumulation of capital in the present moment, biomedical capitalism is characterized by the success of procedures and processes that lead to promised future returns (i.e. developing future cures). Fifth, and finally, whereas traditional capitalism produced by-products or externalities that require disposal, the by-products of biomedical capitalism are often ethically sensitive materials (such as embros) or are desirable in themselves (such as donated organs).

One central feature of the rise of biomedical capitalism is new biomedical-government-industry collaborations that involve the production, legitimation, and commercialization of biomedical knowledge (Etzkowitz, Healey, and Webster 1998; Swann 1988; Teeling-Smith 1965). These new relationships form the institutional bases out of which growing volumes of research are produced. For example, pharmaceutical companies pay academic biomedical researchers to conduct clinical

trials for their new investigational drugs, and then pay federal drug regulators at the Food and Drug Administration to review their drug for regulatory approval.

In the context of a biomedical mode of capitalism and new ways of producing profitable biomedical knowledge, Sandra Soo-Jin Lee investigates the corporate research, development, and marketing of pharmaceutical drugs targeted toward specific racial groups.[33] In the context of pharmaceuticals, Lee outlines what she calls an infrastructure for racialization, a set of scientific and institutional practices that inscribe bodies and their bodily products with racial meaning. These practices consist of (1) new research on human genetic variation that overlays genetic data onto social categories of race; (2) the continued and widespread use of race as a proxy for risk in clinical medicine; and (3) the commercial development of racially inscribed niche markets by the pharmaceutical industry (Lee 2005).

Collectively, the scholarship of Sara Shostak, Janet Shim, Charis Thompson, and Sandra Soo-Jin Lee suggests that the scientific, technological and economic processes that encompass biomedicalization do not operate uniformly on all social groups. Thus, a fifth core theme is that biomedicalization is a *stratified* and *stratifying* social process. Clarke et al (2003) define cooptative biomedicalization and exclusionary disciplining as two oppositional processes within biomedicalization that target and exclude particular bodies and populations, respectively. Drawing on the framework of medicalization, cooptative biomedicalization entails the expansion of medical jurisdiction over areas previously not deemed medical in terms of interventions targeted towards particular social groups. For example, Sandra Soo-Jin Lee's work shows how racial groups are targeted in pharmaceutical development

through the cooptation of race in biomedical research. Exclusionary disciplining refers to the institutionalized practices that erect barriers to the social process of biomedicalization for selected social groups. For instance, drawing on my earlier example, members of racial and ethnic population groups who lack prescription drug coverage are excluded from the cooptative practices of pharmaceutical companies.

The framework of biomedicalization provides a set of powerful analytic tools through which to analyze the relationships of the metabolic syndrome and race. In the next section, I turn to Foucault's framework of biopower. I argue that biopower provides a synthetic conceptual framework for this study that views racial formation and biomedicalization as part of the same apparatus of power/knowledge.

The Framework of Biopower

The social theory of Michel Foucault, especially his analytic framework of biopower, provides a third and synthetic theoretical lens shaping this study. In this section, I will define Foucault's framework of biopower as a theory of power and knowledge, and then outline the core themes from which I draw upon to analyze the relationships between the metabolic syndrome and race. Foucault uses the concept of biopower as a way of understanding the transitional period beginning in the seventeenth century when modern institutions of power began to take human life as their objective and target. The framework of biopower focuses on the relationships through which the life and health of bodies and populations become the objects of scientific discourse and institutional regulation by governments and corporations.

Foucault conceived of biopower as the convergence of disciplinary power and a new kind of regulatory power over the life processes of entire populations, two "poles" of power that converge at the level of concrete arrangements (Foucault 1978: 140). These concrete arrangements were bodies and populations themselves.

The first core theme of biopower that is relevant to this study is that the two technologies of biopower, disciplinary power and regulatory power, represent distinct institutional locations for the operation of power and the production of knowledge. Disciplinary power is the means to extract political and economic productivity from individual bodies and the use of tactical procedures used to observe, judge, and examine bodies. Achieving the disciplining of the body requires hierarchical observation, normalizing judgment, and the physical examination (Foucault 1975a). Through these techniques and practices, disciplinary power establishes the relations of docility and utility of the body—this is how discipline makes docile bodies (Foucault 1975b). Hierarchical observation involves the continuous and uniform monitoring of the processes of the body, in order to achieve its maximal productive efficiency. Normalizing judgment involves the introduction of a system of rewards and punishments whose goal was to induce the body to conform to the laws of efficient movement corresponding to the activity it was being asked to perform under disciplinary conditions. The examination is the recurrent and culminating event in the disciplinary process through which the body is gazed upon as "both a ritual of power and a procedure for the establishment of truth" (May 1992: 43). Disciplinary knowledges are the scientific truths about the body produced through the observation, judgment, and examination of bodies.

Whereas disciplinary powers operates through strategies that target the individual body, regulatory power operates through "massifying" strategies that deal strictly with *populations* as "a political problem, as a biological problem, and as power's problem" (Foucault 2003[1976]: 245). Regulatory power gained increasing prominence in the nineteenth century with the rise of demography, epidemiology, and sociology. The primary techniques of regulatory power involve the use of demographic averages, comprehensive and comparative measures, and statistical assessments that are derived from the surveillance of populations. Foucault provides the familiar example of the birth rate as such a measure. The birth rate is a statistical measurement of the population that used to evaluate the relative health of the population. In a recursive fashion, these measures are then used to establish further regulations that are intended to act on the population as a whole. If the birth rate is low, interventions are required to improve the population's health. Whereas disciplinary power makes docile bodies so as to increase utility, regulatory power constructs populations more regulated so as to maximize health and life.

A second important theme of the framework of biopower is that governments and corporations create and use the disciplines and regulations to conform bodies and populations to unequal political and economic arrangements. With its explicit focus on the life processes of human populations, Foucault articulates biopower as a critique and synthesis of the liberal-juridical and Marxist conceptions of power. The juridical or liberal conception of power maintains that the governments exercise the Law and threat of death to rule over its subjects. Indeed, the institution of the nation state itself required the production of a discourse about a bounded population of

individuals, citizens. Foucault argues that governments had historically exercised their right to kill their enemies, both foreign and domestic, and that this management of death was central to the extension of government power, especially military power. While governments continued to kill and still do, during the transition to biopower, governments began to add a biopolitical management approach to their repertoires. As I have mentioned, this new approach was primarily concerned with investing in, interrogating, and controlling the biologies of *all* populations. To explain the significance of this new political relationship relationship, Foucault writes "One might say that the ancient right to *take* life or *let* live was replaced by a [bio]power to *foster* life or *disallow* it to the point of death" (Foucault 1978: 138).

A Marxist conception of power maintains that corporations exercise power to extract surplus value from the labor of workers. In contrast, Foucault's conception of biopower maintains that scientific institutions, governments, and corporations construct and deploy biological relationships for the regulation of populations. In Foucault's words, biopower was central to the development and success of capitalism because it enabled "the adjustment of the phenomena of population to economic processes" (Foucault 1978: 141). Modern capitalism requires the control of large numbers of individual bodies, not only in terms of a need for a population of laborers, but with the health of bodies as objects of investment and sources of revenue *in themselves*. The increasing commodification of health that comprises biomedicalization is good evidence of this treatment of bodies and their products as sources of profit.[34]

A third and crucial theme of the framework of biopower is that it outlines a Foucauldian theory of race and racism. Foucault's recently published lectures at the College of France in 1975-1976, provide a unique elaboration of the concept of biopower and its connections to the emergence of the concept of race and the early formations of state racisms (Foucault 2003 [1976]). In these lectures, Foucault argues that race emerged historically as a way to create a caesura, a break, within the biological and population phenomena addressed by biopower, a break used to separate out perceived biological risks to the health and vitality of the population (Foucault 2003 [1976]). For Foucault, race serves as a transfer point between the production of biological knowledge about population health and the exercise of political power; race becomes a means of "transcribing a political discourse into biological terms" (Foucault 2003 [1976]: 266). Thus, there existed a quick linkage between the exercise of biopower and nineteenth century biological theories of race.[35]

Relations of biopower both enable and justify the practices of racism that have taken place in the name of strengthening or improving population health (Foucault 2003 [1976]: 258). Modern racisms function in the context of biopower by establishing a perpetual relationship of war between the so called races in which racial categorization emerges as a way to identify biological enemies, again internal and external to a particular state government, and mark them for improvement, purification, or extermination. Given this framework, it is clear how and why racial discourses were deployed to institutionalize ideas and practices of population eugenics, which were presumably aimed at improving the health of populations through the purification of the racial stock.[36]

In this section, I argued that Foucault's framework of biopower contributes three important themes to this study. The first core theme of the framework of biopower is a new emphasis on the relationships through which the life and health of populations become the objects of power and knowledge. The second core theme of the framework of biopower is that institutions of power use these techniques in a polyvalent fashion to guarantee exploitive economic and political relationships by conforming bodies and populations to unequal political and economic arrangements. The third and crucial theme of the framework of biopower is that it specifies a Foucauldian theory of race and racism that emphasizes how biological and political relationships are deployed through racial categorization.

Convergence, Divergence, and Synthesis

Thus far, I have presented critical race theory, biomedicalization (hereafter in this section called STS), and biopower as three distinct and independent bodies of scholarship that each offers unique contributions to this study. However, sharp lines demarcating these areas are not so easily drawn as these areas have been shaped by and continue to influence each other in the broad context of sociological theory. While there are multiple points of convergence across these areas, they also diverge in meaningful ways that are germane to my synthetic interpretation of the relationships between the metabolic syndrome and race.

A primary point of convergence across critical race theory, STS, and biopower is a focus on the multiple linkages between bodies and populations and in particular how scientific ideas about the body emerge out of practices that are

targeted towards populations. Yet, within this convergence, these three frameworks each treat this body-population link in different ways. Historically in the United States, critical race theorists have been first and foremost concerned with the lived experiences of people of African descent living under unjust conditions of colonialism, slavery, and capitalism. The idea of race itself provided a pseudo-scientific pretext for enacting various forms of racial subjugation under European rule that operated by linking ideas about the inferiority of particular racial types to population-based exploitations. In the same way, Foucault's thinking about race as a system of biopower draws upon this understanding of race, but emphasizes how the operation of this new kind of power was enabled by a new focus on maximizing the life and health of dominant groups that emerged in the 1800s, nearly a century after the first racial taxonomies were codified in the European academy. Scholars in the field of STS begin their analysis of the body-population link with Foucault, accompanied by a heavy reliance on feminist ideas about the gendered body, and examine the multiple ways that bodies and populations are constituted via new technologies in science and medicine.

This first point of convergence on the body-population link suggests a second key point of convergence across critical race theory, STS, and biopower—a shared focus on institutionally based forms of social stratification and inequality that predominate Western society. Each of these bodies of knowledge draws upon a political orientation to the production of scholarship that recognizes that objective intellectual production does not, and cannot, occur in a just and fair society. To the contrary, scholars in these areas have traditionally focused on how particular social

institutions like governments, corporations, and science shape the life chances of people and have worked to illuminate the social and political conditions necessary for the fostering of a more just society. This shared focus on justice stems from a shared understanding about the nature of modern forms of power that have operated largely since the Enlightenment. Major works in each of these areas have analyzed the role of scientists and scientific practices in the formation of unjust social arrangements and have deconstructed those forms of knowledge that undergird those arrangements.

A second point of convergence across these frameworks concerns the research methods utilized to answer research questions in each area. Specifically, contemporary practitioners in critical race theory, biomedicalization, and biopower have all drawn upon discursive and historical methodologies to study different aspects of the relationships between power and knowledge. For example, in her recent book (Panic Diaries: A Genealogy of Panic Disorder, 2006), sociologist Jackie Orr uses Foucault's genealogical method to analyze the relationships of power and knowledge developed by a normalizing society to regulate the psychological life, health, and disorders of individuals and entire populations—a concept she calls pychopower (Orr 2006:11). Drawing on Foucault's formulation of biopower and the frameworks of technoscience and biomedicalization, Orr argues that psychopower has emerged since the late 19th century, but has gained new operational capacities with the rise of twentieth-century information and communication technologies. Orr uses genealogy to identify three distinctive ways that the panic disorder serves as a site for the operation of psychopower. First, psychopower disciplines individuals and entire populations through the surveillance, scientific classification and management, and

public administration of the psychic realms of perception, emotion, and memory (Orr 2006: 11). Second, the techniques of public opinion polling, attitude measurement, and psychological testing both govern populations and have the additional effect of intensifying and multiplying the communicative feedback loops between governing bodies and the bodies they would govern (Orr 2006: 12). Third, by utilizing these new techniques and knowledges focused on perception, emotion, and memory psychopower can blur the boundaries between the real and the unreal (Orr 2006: 13).

Through her articulation of psychopower, Orr contributes to a political understanding of how scientific practices and institutional relationships reproduce particular kinds of subjectivities and materialities. Orr shows how U.S. government propaganda about nuclear annihilation during the Cold War was informed by and proactively informed the social psychology of group trauma, fear, and panic, which were themselves financed by the state. Similarly, she makes a similar genealogical argument about clinical trials for the killer application Xanax. The pharmacological effects of Xanax were tightly linked with the classificatory schema for panic disorder because of the new institutional relationships between biomedical psychiatry, the federal government (Department of Defense and FDA) and pharmecutical corporations (Upjohn), which were the institutional locations for the classification, administration, and treatment of panic disorder (Orr 2006: 255).

While serving as an exemplar of a way to synthesize a framework of biopower in the context of science and technology studies, Orr's research also demonstrates a first point of divergence with critical race theory. While some critical race scholars draw upon Foucault's ideas (e.g. David Theo Goldberg) and the organizing principles

of science and technology studies (e.g. Troy Duster), precious few Foucauldian and/or STS scholars draw upon the insights of critical race theory to address questions of race and racism in direct terms. Stated differently, these frameworks do not share equally across each other's domains of inquiry. A counterexample to this point of divergence is the work of Melbourne Tapper, whose 1999 book In the Blood: Sickle Cell Anemia and the Politics of Race draws upon ideas about African American citizenship, medicalizing discourses about disease, and the operation of biopower to explore how sickle cell anemia became an object of scientific intervention targeted on people of African descent.

A second point of divergence across these areas concerns the use of different theoretical vocabularies to describe what I increasingly see as analogous social practices and arrangements regarding the reproduction of race and racism. For example, critical race theorists Michael Omi and Howard Winant advance the construct of racial formation to describe the processes by which racial categories are created, transformed, and destroyed. Racial formation, in their way of speaking, consists of the integration of the discursive meanings of race and the institutionalized practices of racism that function based on that meaning. In comparison, Michel Foucault advances a similar idea that race is reproduced through the convergence of the (racial) disciplining of bodies and the regulation of (raced) populations and that this reproduction takes place in order to propagate the unequal power/knowledge arrangements that comprise modern racisms. While it is likely that Omi and Winant were influenced by the so called discursive turn in critical social though that was

popularized by Foucault, nonetheless, these two frameworks ostensibly describe the same social process using different linguistic formulations.

Taken together, critical race theory, biomedicalization, and biopower provide a powerful synthetic framework that I use to interpret the politics of metabolism. These frameworks are synthetic because they speak to the similar social process and institutional relationships of these politics of metabolism. Table 2.2 presents a summary of the core themes I developed in this chapter and positions them relative to the core components of the politics of metabolism.

[Insert Table 2.2. Summary of Core Themes & the Politics of Metabolism]

Column one summarizes the elements the politics of metabolism that are germane to the guiding questions of this study: social processes, institutional relationships, and the constructions of racial meaning and the metabolic syndrome. Recall the three questions that shape this study: First, how did the metabolic syndrome emerge as a new discursive formation in the politics of metabolism? Second, how are current conceptions and meanings of race constructed through the discourses of the metabolic syndrome? Third, what are the implications of this emerging relationship between the metabolic syndrome and race for understanding the construction of racial meanings in the politics of metabolism?

Columns two, three, and four summarize the frameworks I developed in this chapter that shape my analysis of the emergence of the metabolic syndrome and the construction of racial meaning. These frameworks highlight the relationships between

biomedical scientists, the government, and corporations through which the metabolic syndrome has emerged as a racialized phenomenon. This table arrays these core elements of these frameworks against the principle components of the politics of metabolism. Namely, the social processes and institutional relationships of the politics of metabolism structure the emergence of the metabolic syndrome and the construction of racial meaning. The *social processes* of racial formation and biomedicalization illustrate the combined use of racial categorization and biotechnologies to enact relations of biopower. The framework of biopower helps to reframe the *institutional relationships* between biomedical scientists, the government, and corporations that are involved in racial formation and biomedicalization.

In the next two chapters, I use the main ideas summarized here to explore how the metabolic syndrome emerged as a new discourse of biopower by tracing the social processes and institutional relationships that are involved in the production of new racial meanings. In the concluding chapter of the study, I will address how my interpretation of the relationships between the metabolic syndrome and race speak back to these theoretical frameworks that informed my analysis.

Table 2.1. Office of Management and Budget (OMB) racial and ethnic categories, 1997.

(1) American Indian or Alaska Native: A person having origins in any of the original peoples of North and South America (including Central America), and who maintains tribal affiliation or community attachment.
(2) Asian: A person having origins in any of the original peoples of the Far East, Southeast Asia, or the Indian subcontinent including, for example, Cambodia, China, India, Japan, Korea, Malaysia, Pakistan, the Philippine Islands, Thailand, and Vietnam.
(3) Black or African American: A person having origins in any of the black racial groups of Africa. Terms such as "Haitian" or "Negro" can be used in addition to "Black or African American".
(4) Hispanic or Latino: A person of Cuban, Mexican, Puerto Rican, Cuban, South or Central American, or other Spanish culture or origin, regardless of race. The term, "Spanish origin," can be used in addition to "Hispanic or Latino".
(5) Native Hawaiian or Other Pacific Islander: A person having origins in any of the original peoples of Hawaii, Guam, Samoa, or other Pacific Islands.
(6) White: A person having origins in any of the original peoples of Europe, the Middle East, or North Africa.

Table 2.2. Summary of Core Themes & The Politics of Metabolism

The Politics of Metabolism	***Critical Race Theory***	***Biomedicalization***	***Biopower***
Social processes	Unfolds via discursive and institutional processes	Unfolds through synergies between technologies and science	Reflects convergence of disciplines of the body and regulations of populations
Institutional relationships	Biomedicine and nation state as racial projects	New forms of biomedical-government-industry collaboration	Frames relations between biomedical, state, and corporate institutions as political
Construction of racial meaning	Race is constructed via process of racial formation	Race is constructed in new structures of racial stratification	Uses racial categorization to construct unequal scientific, political, and economic relationships
Construction of metabolic syndrome	New forms of scientific racism and racial essentialism	Entails emphasis on molecularization and risk in the study and treatment of metabolic health	Metabolic health of bodies and populations as new objects of power/knowledge

Chapter 3: The Racial Formation of the Metabolic Syndrome

In chapter one, I introduced the metabolic syndrome as a concept that has been used to describe the co-morbidity of several prominent chronic metabolic conditions. I noted that I would use the term "metabolic syndrome" to refer to a larger group of terms that encapsulate these new relations. In chapter two, I outlined the theoretical vocabulary from critical race theory, biomedicalization, and biopower that I would use to analyze the relationships between the metabolic syndrome and race. In this chapter, I analyze the emergence of the metabolic syndrome by examining the central ways it is involved in the production of new racial meanings. In the genealogical analysis of emergence, the metabolic syndrome appeared in multiple sites of knowledge production in biomedicine and has taken different forms in these various sites over time.[37] I demonstrate that the phenomena that came to be called the metabolic syndrome had several different names and empirical definitions that each represents disjunctures that have differential implications for the production of racial meanings and the interpretation of racial inequality.

One way to frame the puzzle that race has presented for metabolic syndrome researchers is how to measure and interpret *metabolic* differences, both in terms of bodies and populations, in ways that were consistent with prevailing ideas about *racial* differences between bodies and populations. In this chapter, I show how the emergence of the metabolic syndrome forged a technoscientific process for the production of biomedical and genetic conceptions of race. Stated differently, I argue that race was central to the emergence of the metabolic syndrome and that the

metabolic syndrome, in turn, serves as dynamic new site for the production of race and racial meaning. Yet, by using the construct of disjunctures I described in chapter one, which represent the structure of discursive possibilities, I showed how these processes of emergence and racialization did not result from conscious and obvious choices on the part of biomedical scientists working in their respective fields. To the contrary, the social processes and institutional relationships that comprise the politics of metabolism seem to be more open ended and heterogeneous, and have created a set of possible conditions through which the metabolic syndrome could emerge as a racial phenomena in some historical moments, and apparently non-racial in other moments.

I have divided this chapter into two main parts, each of which is further divided into three subsections. While these subsections appear to be organized according to chronological time, each of these moments represents arbitrary disjunctures in the attempt to establish the metabolic syndrome as a biological clinical disease as a legitimate object of biomedical knowledge production and intervention. In Part I, I trace the emergence of the metabolic syndrome across three thematic moments in American biomedicine: Technical and Conceptual Foundations (1947-1986); From Syndrome X to Dysmetabolic Syndrome X (1987-2000); and The Ascendance of the Metabolic Syndrome (2001-present). In the first section, Technical and Conceptual Foundations, I highlight the early biotechnical and theoretical work on bodies and populations, molecular processes, and clustering that shaped early discourses and practices of the metabolic syndrome. The second section, From Syndrome X to Dysmetabolic Syndrome X, 1988-2000, follows the emergence of the

syndrome through a second moment, where researchers continued to advance new ideas about the definition and causes of the syndrome focusing on the bodily measurement of insulin resistance. The third section, The Ascendance of the Metabolic Syndrome, chronicles a third turning point in the emergence of the syndrome, namely, when in 2001 the syndrome comes under federal biomedical jurisdiction through the National Cholesterol Education Program and then accelerates out into the vast network of biomedical disciplines and research specialties.

In Part II of the chapter, I retrace the production of racial meaning during the three thematic periods that I described in Part I. For the first moment, Sampling Normal Subjects, I show how researchers' approaches to the study of human metabolism both explicitly and/or implicitly targeted bodies and populations based upon prevailing racial categorizations that marked the metabolic processes of white male bodies as normal. For the second moment, Is Race Really To Blame?, I examine how race was constructed as reflecting a genetic causation of the metabolic syndrome. For the third moment, I use the notion of the "special populations" to show how race becomes fully incorporated into the technological measurement, scientific definition, and causal theories of the metabolic syndrome. In the contemporary moment, all people who could be classified with the metabolic syndrome and all racial and ethnic minorities now seem to comprise high-risk populations that require permanent forms of examination, surveillance, and regulation.

Cutting into the emergence of the metabolic syndrome in these ways allows me to highlight the institutional and discursive forces that have shaped and continue

to shape the unfolding of the relationships between the syndrome and race. The two main parts reflect my effort to sift the ostensibly non-racial features of the metabolic syndrome from the racial ones. In fact, the production of racial knowledge related to the metabolic syndrome in and of itself suggests an important and organizing disjuncture in its emergence. It was not always obvious that the metabolic syndrome could emerge as a biomedical construction that relied so heavily on racial distinction and administrative classification. In addition to each of the main parts of chapter three representing a major disjuncture, I have divided each of the subsections in parts I and II according to a particular historical periodization that maps onto important disjunctures within the non-racial and racial narratives of the emergence of the syndrome. In other words, while these subsections are organized according to a Western notion of continuous and linear time, I intend for them to represent arbitrary disjunctures where the emergence of the syndrome shifted in important ways that mattered for the production of racial meaning.

Part I: The Emergence of the Metabolic Syndrome

Technical and Conceptual Foundations, 1956-1987

During 1956-1987[38], the technical and conceptual foundations of the metabolic syndrome were tightly linked to three main sets of ideas and practices that operated in polyvalent fashion. Taken together, these polyvalent discourses and practices comprise the technical and conceptual frameworks through which the metabolic syndrome would emerge as a new technique of biopower. They were (1) the increasing technical focus on measuring the body's metabolic processes at the

molecular level of a body; (2) the institutionalization of measuring the metabolic processes of populations; and (3) the increasing focus on the clustering of metabolic problems and risk that resulted from these new practices. By interpreting these technical and conceptual foundations of the metabolic syndrome as technologies of biopower, I explore how the syndrome emerged as a result of the synergy of molecularization and the risk factor paradigm in biomedicalization. In the sections that follow, I explain each of these technical and conceptual foundations in greater detail, highlighting how they each contributed to the emergence of the metabolic syndrome.

The development of a range of techniques to measure molecular processes constitutes a first development during these moments that shaped the emergence of the metabolic syndrome. In the late 1800s and early 1900s, new scientific theories and concepts were developed and new technologies deployed to understand the body's metabolic processes at the molecular level. The creation of new technologies that would be used to study metabolism in terms of molecular processes, such as the discovery of insulin in 1920, helped to reinforce the early scientific imperative to know more about how bodies metabolized molecular structures like lipids, glucose, and insulin. The technical development of the physical examinations and laboratory tests that comprise the metabolic syndrome occurred mostly prior to this first period, thus making it technically and discursively possible to construct a metabolic syndrome of any kind in this period. Table 3.1 lists selected technical developments that contributed to the metabolic syndrome between 1896 and 1985.

[Insert Table 3.1 Selected technical developments contributing to the metabolic syndrome, 1896-1985]

Not only did these technologies structure the content of biomedical knowledge about metabolic processes of bodies at the molecular level, they also opened up and closed off particular possibilities in terms of the emergence of the syndrome. Once each of these technologies was in use, the conceptual foundations of the metabolic syndrome could be readily forged in laboratories and doctor's offices. Yet, the creation of technologies in a given historical moment delimits the technical aggregation of the various elements of the syndrome at later moments. In other words, the technical and conceptual foundations in this early period form a set of disjunctures that continue to structure the range of discursive possibilities for the metabolic syndrome in the contemporary moment.

A growing focus on measuring various aspects of the body and the construction of certain ideas about particular populations based on these bodily measurements constituted a second important development during this period that enabled the subsequent development of the metabolic syndrome. Take, for example, the work of University of Marseilles physician Jean Vague, who proposed an alternative anthropometric method that "traces the thickness of the fatty tissue on the surface of the body" (Vague 1956: 20). Vague becomes a central figure in this period and is routinely cited as one of the primary so-called fathers of the metabolic syndrome concept[39] because of his investigation of the causal relationships between obesity, heart disease, and diabetes (Vague 1947; Vague 1956).

Vague articulates two hypotheses that illustrate the way he defined, measured, and conceptualized the IMD in relationship to sex difference. His anthropometric method required the measurement, enumeration, and tabulation of the fatty tissue found at ten points on the trunk and limbs of the body. The first hypothesis is that "the relationship of the thickness of the fold of the nape of the neck to that of the sacral fold is much greater than unity in the normal male, but much less in the female" (Vague 1956: 21). The second hypothesis is that the brachio-femoral adipo-muscular ratio, a comparison of the adipo-muscular ratio [the relationship between fat and muscle tissue] on the arm compared to the thigh, is above unity [greater than zero] in the normal adult female, while the inverse is true in the male" (Vague 1956: 21). He uses these measurements to construct a statistical representation on sex-differentiated obesities, an *index of masculine differentiation* (IMD). The IMD is "the average of the nape:sacrum ratio and the brachio-femoral adipo-muscular ratio" (corrected in terms of the total thickness of the fat in the two regions) (Vague 1956: 21). This information is significant because Vague developed a body of conceptual and technical language that emphasized the measurement of the body, and the statistical comparison of different parts of the body, that were essential to his ideas about bodily difference.

Vague's hypothesis proposes that men and women are essentially different if measured and compared at these locations. Based on his analysis of 600 subjects, Vague constructs five mutually exclusive groups, standardized around a value of "0" for the standard male body, comprising the following categories: "hyperandroid" (+15 IMD), "android" (+15 to -15 IMD), "intermediate" (-15 to -45 IMD), "gynoid"

(-45 to -75 IMD), and "hypergynoid" (+75 IMD). These categories are meant as descriptions of the different types of distributions of obesity typically found in women and men, with android referring to men and gynoid to women. The binary logic of sex drives Vague's interpretations of his statistics. In this regard, he notes "fat distribution is very definitely a sexual characteristic, but there is a high percentage of overlapping between one sex and the other, especially at the two extremes of life" (Vague 1956: 24). In other words, while the categories gynoid are meant to refer to a particular statistical construction of populations, men can exhibit with gynoid forms of obesity and women with android forms of obesity.

Vague concedes the non-exclusive nature of his gendered categories as demonstrated by Figures 3.2 and 3.3. The text of Vague's paper was published along with several tables and figures, as well as eight photographs of four of his research subjects, whose bodies are fully exposed to show the distributions of their obesities. Figure 3.2 shows one woman and one man, both classified with gynoid obesity, whereas Figure 3.3 shows one woman and one man both classified with android obesity.

[Insert Figures 3.2 and 3.3]

The images of Vague's subjects and the captions that accompany them represent the visual forms of bodily evidence that Vague used to present ideas. The captions note specific information about the subject's metabolism including age, height, weight, blood pressure, the presence/absence of diabetes, and other bodily indicators

including hair, the status of genitals and noted sexual practices, and the subjects' value for the index of masculine differentiation. Undoubtedly, disciplinary conventions about publishing photographs of nude research subjects in professional biomedical journals have changed somewhat since the 1950s, but the visual representation of these subjects shows the ease with which physicians might have differentiated between gynoid and android forms of obesity.

Vague's work illustrates the growing emphasis of measuring the body, but it also points to how specific ways of interpreting the bodily measurements begin to make the metabolic syndrome possible. In particular, one important ways Vague's ideas preview the emergence of the metabolic syndrome lies in his claims about android obesity as a common cause of heart disease and diabetes. In this brief passage, he describes how android obesity is the common cause of atherosclerosis and diabetes:

> The inconstancy of diabetes in the course of atherosclerosis when the islets of Langerhans[40] offer a sufficient genetic resistance to the overwork imposed by the pituitary-adrenal overactivity, in contrast to the constancy of atherosclerosis in adult diabetes and its relative independence to the degree of hyperglycemia, cease to surprise us if we regard arterial legions and diabetes as the consequences of an identical cause [android obesity] acting against a backdrop which may suffer from a genetic fragility of the islets or be free from it (Vague 1956: 31).

In other words, Vague posited that heart disease and diabetes share android obesity as a cause. According to Vague, the development of diabetes, gout, uric calculous disease, and atherosclerosis is "very strongly favored by android obesity, especially when weight and the index of masculine differentiation are very high" (Vague 1956: 29). In contrast, gynoid obesity "does not exercise any direct influence on the metabolic disorders" (Vague 1956: 29). In Vague's thinking, genetic differences between bodies caused the differential development of android obesity, and consequently, diabetes and heart disease.

A second important way Vague's ideas preview the syndrome lies in the predictive power Vague attaches to the IMD for classifying bodies that will develop metabolic disease. By the 1956, Vague proposed that a particular combination of bodily measurements results in the best statistical predictor of android obesity. Stated differently, Vague's method was significant because it represented an important shift from diagnosing the body through physical examination to compiling the results of physical examinations to construct statistical ideas about population-based risk. In other words, the IMD is a statistical construction that successfully identifies obese bodies that are predisposed to diabetes and to heart disease because of a common genetic mechanism. He states that the index of masculine differentiation has "always indicated to us the exact position of these forms [of obesity] in our classification and, in addition, has provided prognostic data" (Vague 1956: 24). His hope was that physicians would calculate the IMD and use it to predict, with great accuracy in his view, which bodies and populations are likely to develop metabolic disease.

Increasing reliance on the notion of clustering to guide biomedical research on metabolism constitutes a third important development during this period that enabled the subsequent emergence of the metabolic syndrome. Clustering refers to the observation that several different metabolic conditions are more likely *to occur together* in one individual than would be expected by chance alone. The notion of clustering is significant because the production of knowledge about the metabolic syndrome is made possible by the physical examination and biochemical surveillance of bodies and the aggregation of that individual level biological data to the level of populations. These conceptual developments in epidemiology are directly linked to the technical and conceptual foundations of the metabolic syndrome because of the widespread use of the metabolic syndrome as statistical predictor of heart disease and stroke in federally funded biomedical research.

By the early 1920s, several European physicians were the first to document and publish research about the clustering of metabolic problems they observed in their patients, and the potential risks such clustering could pose to metabolic health (Hitzenberger 1922; Kylin 1923; Maranon 1922). While none of these physicians explicitly codified a syndrome, they had similar theoretical ideas about how different metabolic processes worked together in the body. For example, in his 1936 study of insulin action, endocrinologist H.P. Himsworth created the distinction between insulin sensitivity and insensitivity, the latter being most likely to precede and then accompany the development of type II diabetes (Himsworth 1936).

While the focus on the measuring the body's metabolic processes in terms of clustering represented a shift in the biomedical approach to studying chronic

conditions, it also formed the basis of later struggles to subsume the metabolic syndrome under different disciplinary specialties. For example, Himsworth's research on insulin metabolism in the 1930s anchored the structure of contemporary endocrinology, and the efforts of contemporary endocrinologists to study the metabolic syndrome make sense given this technical and conceptual anchor. Early scholars, like Vague, drew explicitly upon notions of risk-based clustering in their theories about the nature of metabolic problems, but it was not always with respect to the same outcome. Whatever the outcome, these notions of clustering formed the logic upon which risk-based syndromes, like the metabolic syndrome, would be constructed in later decades. Different clusters of conditions drew the attention of newly developing medical specialties like endocrinology and cardiology. In this early period, endocrinologists were concerned mostly with glucose metabolism, insulin, and diabetes; cardiologists were concerned with heart disease and the processes underpinning vascular function; rheumatologists were concerned with gout and so on. Increasingly, over this thematic period, physicians would continue to conduct clinical research on the interrelationships between basic metabolic processes with a growing list of new molecular compounds and physical examinations.

While many of these early metabolic researchers developed and used statistical methods of analysis in their clinical research, in the late 1940s, the federal government assumed a new role in producing information about the metabolic health of populations. Indeed, the incorporation of a population-approach to metabolism represents a major disjuncture in the emergence of the metabolic syndrome. In epidemiology, the statistical computation of incidence and prevalence data are made

possible only through the numerical comparison of individuals within a defined population. In 1948, the National Heart Institute[41] provided funding for the Framingham Heart Study, the first population heart study to include all of the physical exams and laboratory tests required to make a classification of the metabolic syndrome in the United States (Kannel, McGee, and Gordon 1976).[42] The Framingham Study is also noteworthy for its role in identifying cholesterol as a so-called risk factor in the development of coronary heart disease. Following the successes of the Framingham Study at identifying risk factors for heart disease, the U.S. Congress passed the National Health Survey Act of 1956, which authorized "a continuing survey and special studies to secure accurate and current statistical information on the amount, distribution, and effects of illness and disability in the U.S. and the services rendered for such conditions."[43] According to the National Health Survey Act of 1956, the empirical data for these new government studies would be drawn from at least three sources: (1) the people themselves by direct interview, (2) clinical tests, measurements, and physical examinations on sample persons, and (3) places where persons received medical care such as hospitals, clinics, and doctor's offices.

This law was significant because it mandated that the US government now conduct routine surveillance of its populations by use of physical examinations and laboratory tests that had hitherto been focused on individual bodies. This act led to the creation of the National Health Interview Survey (NHIS), first conducted in 1957, the National Health Examination Survey (NHES) beginning in 1960, and the National Health and Nutrition Examination Survey (NHANES), which began in 1967. The

NHANES study is the study upon which the National Cholesterol Education Program would later base its construction of the metabolic syndrome in 2001. These government epidemiological studies have been and still are the largest population health surveys conducted in the United States each year. Thus, population health studies over the next five decades were designed using the Framingham study as gold-standard model.[44]

These conceptual and technical developments at the level of bodies and populations converge in risk-based-syndromes. Risk-based syndromes are sites where ideas and practices about molecular processes, bodies and populations, and clustering come together in a polyvalent fashion. For example, the International Diabetes Federation (IDF) published their own version of the metabolic syndrome and draw upon a definition of syndrome from a 1995 dictionary of epidemiology (Last 1995), which states that what distinguishes syndromes from diseases is their lack of a clearly defined cause. They note:

> "A syndrome is defined as a recognizable complex of symptoms and physical or biochemical findings for which a direct cause is not understood. With a syndrome, the components coexist more frequently that would be expected by chance alone. When causal mechanisms are identified, the syndrome becomes a disease" (Alberti, Zimmet, and Shaw 2006: 473).

Currently, the National Library of Medicine's online medical dictionary defines a syndrome as "a group of signs and symptoms that occur together and characterize a particular abnormality."[45]

Throughout the 1960s and into the 1970s, given the increasing proliferation of laboratories across the US, and the increasing availability of epidemiological data, more researchers would have access to the technologies and interpretive frameworks required to produce knowledge about risk-based syndromes. In the 1960s, there are several noteworthy contributions to the emergence of the metabolic syndrome, but still unresolved was the little issue of what to call the syndrome. First, in 1966, French researcher Camus theorized that gout, diabetes, and hyperlipidemia comprised "a metabolic trisyndrome" (Camus 1966). The following year in 1967, two Italian researchers advanced the notion of a "plurimetabolic syndrome" that included diabetes, obesity, and hyperlipidemia (Avogaro, Crepaldi, Enzi, and al 1967).[46] And finally, in 1968, Dutch researchers Mehnert and Kulmann published an article in a prominent Dutch medical journal about the relationships between hypertension and diabetes (Mehnert and Kuhlmann 1968).

It was during the 1970s that the term "metabolic syndrome" would first appear in the biomedical research literature. In 1976, Gerald Phillips, drawing heavily on Vague's earlier work, theorized that the "constellation of abnormalities" that comprised increased heart disease risk could be explained by sex hormones (Phillips 1978; Phillips, Jing, and Heymsfield 2003). In 1977, three studies were published that each codified specific formations of "the metabolic syndrome" into the biomedical literature for the first time (Haller 1977; Singer 1977; Ziegler and Briggs 1977). A few years later, in 1981, two German researchers were also among the first to publish research on the "the metabolic syndrome" (Hanefeld and Leonhardt 1981). While the notion of the metabolic syndrome would continue to change in the coming

years, the increasing scientific focus on the clustering of condition in bodies and populations that was newly possible with new biomedical technologies, made that change possible.

The different names, definitions, and disciplinary homes for the metabolic syndrome represent central disjunctures in the emergence of the syndrome. The incommensurability of the syndrome across cardiology, endocrinology, and epidemiology meant that there would continue to be struggles over its meaning in biomedicine. At the same time, the technologies and conceptual developments that undergird the syndrome in polyvalent fashion made it possible for the syndrome to travel across these disciplinary boundaries with remarkable ease. For example, medical practitioners and biomedical researchers regardless of specialty area utilize measurements of blood pressure and obesity. Yet, when these same researchers study the metabolic syndrome within the confines of their own respective areas, it becomes possible for them to include and/or omit particular features of human metabolism that are deemed relevant or irrelevant to their biomedical perspective. In the next section, I explore how the search for a cause of the metabolic syndrome inside of endocrinology reflects this kind of struggle.

From Syndrome X to Dysmetabolic Syndrome X, 1988-2000

In a binomial equation, the letter "X" stands for an unknown variable that bears a measurable relationship to another variable "Y". In order to solve for Y in such an equation, the value of X must be known, and vice versa. This simple logic framed the second period of emergence of the metabolic syndrome with physicians'

efforts to take existing observations about the clustering of multiple risk factors and use them to construct new forms of knowledge about the interrelationships between these risk factors. Specifically, this period consisted of professional physicians, mostly endocrinologists, trying to advance new theories of what *caused* the metabolic syndrome. Such theories were intended to help galvanize the syndrome as a biological disease and formal clinical diagnosis. Perhaps by discovering the cause of the metabolic syndrome ("X"), their logic suggested, researchers might then be able to discern the real value and meaning of the metabolic syndrome ("Y"). During this period, different research groups hoped to explain the statistical associations between heart disease risk factors with causal theories focused on the metabolic syndrome. In other words, researchers made continued efforts to establish the causes of the metabolic syndrome.

One major event in this process occurred in 1988, when Dr. Gerald Reaven accepted the Banting Award, named in honor of Sir Fredrick Banting who synthesized human insulin in 1920, and gave the Banting Lecture to the American Diabetes Association based on his research on the role of insulin resistance in the development of heart disease (Reaven 1988).[47] In this lecture, Reaven defined "syndrome X" as a series of six related variables that tend to occur in the same individual—resistance to insulin-stimulated glucose uptake, hyperglycemia, hyperinsulinemia, an increased plasma concentration of VLDL triglyceride, a decreased plasma concentration of HDL-cholesterol, and high blood pressure (Reaven 1988). In his 2000 "Syndrome X: Overcoming the silent killer that can give you a heart attack" Reaven metaphorically calls syndrome X "the silent killer", a not-so-

subtle reference to the paradox that while the syndrome has no visible symptoms, he argues that it may be responsible for up to 50 percent of heart disease in the United States (Reaven, Strom, and Fox 2000).

Reaven's hypothesis is that insulin resistance is the common cause of the five other components of syndrome X, and therefore is a primary cause of heart disease. This framing of insulin resistance as the cause of syndrome X stands in stark contrast to Vague's earlier theory that android obesity was the cause of heart disease and diabetes. While the notion of syndrome X would not acquire the cache of similar terms, due to his omission of obesity in its definition, Reaven's influence on the science of the metabolic syndrome is noteworthy. Reaven's hypothesis was that insulin resistance was responsible for up to 50% of heart disease. Despite the existence of multiple methods for measuring insulin resistance, none of them have been institutionalized in population survey research to the extent that other biological measurements of diabetes have, like fasting blood glucose, in large part due to their expense.[48]

Dr. Reaven's book or his 1988 lecture surprisingly do not include technical definitions of syndrome X. Whereas Vague went to great lengths to include highly specific physiological measurements and statistical procedures in his codification of the metabolic syndrome, Reaven's omission of these details represents a disjuncture in the emergence of the syndrome. Specifically, whereas clustering was the central conceptual anchor in the earlier thematic moment, in this moment, the cultural power of biological causality serves to anchor and promote the truth properties of the metabolic syndrome. In his published lecture, Reaven did not intend to establish a

new statistical concept in the biomedical landscape. Instead, his introduction and reference to syndrome X is more of a passing reference to the unknown nature of these metabolic processes. He writes:

> Based on available data, it is possible to suggest that there is a series of related variables—syndrome X—that tends to occur in the same individual and may be of enormous importance in the genesis of coronary artery disease. These changes include resistance to insulin-stimulated glucose uptake (insulin resistance), hyperglycemia (glucose tolerance), hyperinsulinemia, increased of very low-density lipoprotein (VLDL) triglyceride, a decreased plasma concentration of HDL-cholesterol, and high blood pressure (Reaven 1987: 1605).

While in 1988 Reaven's hypotheses about syndrome X were more tentative, by 2000, he was calling it "the silent killer." Since his book seems to have been written for a general audience, it includes a "Self-Assessment for Risk of Syndrome X" rather than a formal scientific definition.[49]

After Reaven's original hypothesis, what remained unknown, or at least unsettled, about the pathophysiology of the metabolic syndrome, was more than made up for with the growing list of heart disease risk factors that were correlated with the syndrome. By the end of the 1990s, other groups of researchers advanced several similar constructions that aimed to encapsulate these hidden physiological relationships and to challenge Reaven's syndrome X. These constructions all draw upon the early conceptual and technical foundations and propose different iterations of risk-based syndromes: the deadly quartet (Kaplan 1989), the insulin resistance

syndrome (DeFronzo and Ferrannini 1991), the multiple metabolic cardiovascular syndrome (Hjermann 1992), and the chronic cardiovascular risk factor clustering syndrome (Zimmet, Collins, Dowse, Alberti, Tuomilehto, Knight, Gareeboo, Chitson, and Fareed 1994), and multiple metabolic syndrome (Liese, Mayer-Davis, and Haffner 1998).

Perhaps the hope for each of these constructions was that they could derail and shift the subsequent development of a science of the metabolic syndrome. In 2000, the American Association of Clinical Endocrinologists secured a petition to have a diagnosis code assigned to "dysmetabolic syndrome X" in the World Health Organization's International Classification of Disease (ICD-9-CM) (Dickey 2000). This meant that physicians could now use a specific code, 277.7, to specify a diagnosis of the dysmetabolic syndrome X in their patients. According to the new diagnostic criteria, dysmetabolic syndrome X is "a multifaceted syndrome characterized by hyperinsulinemia; dyslipidemia (hyperlipidemia); essential hypertension; abdominal obesity; and glucose intolerance in individuals with insulin resistance.[50] With the codification of the dysmetabolic syndrome X in the ICD, what had started out for Reaven as an unknown with syndrome X, could now be known through classification with a simple diagnostic code.

The Ascendance of the Metabolic Syndrome, 2001-Present

The third moment, which began in 2001 and continues into the present, is characterized by continued institutional and scientific battles over what the metabolic syndrome is, what it means, and who gets to define it as a legitimate disease. The

culmination of past disjunctures directly impacts the structure of contemporary institutional power struggles over the authority to produce a science of the metabolic syndrome. In the broadest terms, whereas Vague had been fundamentally concerned with obesity and its multiple effects on metabolic health, and Reaven's work puts insulin resistance at the center of the analysis of syndrome X, the effort to establish the metabolic syndrome as a derivative of cholesterol metabolism represents a defining disjuncture of the emergence of the metabolic syndrome. In this section, I explore this institutionalized effort and analyze how earlier disjunctures shaped the subsequent ascendance of the metabolic syndrome as a formalized object of biomedical knowledge.

In 2001, what began as a multi-year, multi-agency government effort to study cholesterol, turned into a pivotal shift in the emergence of the metabolic syndrome. The National Cholesterol Education Program (NCEP) began in 1985 as part of a National Heart, Lung, and Blood Institute effort to examine the dynamics of high cholesterol among American adults. The NCEP brought together experts from across the government, academy, and professional medicine. Stated differently, the National Cholesterol Education Program was an example of an industry-academy-government collaboration that, given its centrality in defining the metabolic syndrome during this period, wielded significant influence in biomedical research on cholesterol and its relationship to heart disease.

In addition to publishing aggressive new standards for the clinical management of cholesterol, the NCEP defined the metabolic syndrome by calling it a secondary target of intervention in its final report (NCEP 2001). By framing the

metabolic syndrome as a secondary target of intervention, the NCEP argued that clinicians could not adequately address the heart disease risk from cholesterol without acknowledging the role these additional risk factors played in the causes of heart disease. Under this logic, codifying the construct of the metabolic syndrome was intended to make practicing physicians aware of the need to address the clustering of risk factors for heart disease.

According to the NCEP, in order to be classified with the metabolic syndrome, a research subject would have to submit a blood sample (for analysis of cholesterols, fasting blood sugar, and other molecules) and submit to a physical examination including measurement of blood pressure, height, weight, and abdominal circumference. If the subject's levels met or exceeded three of five predetermined empirical cutpoints, the individual was said to "have" the metabolic syndrome. The five components and their values for the NCEP definition are: (1) blood pressure (higher than 130/85); (2) fasting blood sugar (higher than 110 mg/dl); (3) LDL or "bad" cholesterol (higher than 150 mg/dl); (4) HDL or "good" cholesterol (lower than 40 mg/dl for men and 50 mg/dl for women); (5) abdominal circumference (greater than 40 inches for men and 35 for women).

However, the move of framing the syndrome as a *secondary* target of intervention seems to have had the unintended effect of signaling to the biomedical research community, pharmaceutical corporations, and to the government itself that the metabolic syndrome required special attention as a *primary* object of knowledge. In a 2003 meeting at the National Institutes of Health on the metabolic syndrome, Dr. Scott Grundy, the chairman of the NCEP, reflected that the group of scientists was

"concerned that the NCEP guidelines would be seen as only drug treatment guidelines for LDL [cholesterol], they decided to define a set of medical conditions related to obesity, physical inactivity, and nutrition and define these conditions as a metabolic syndrome" (NIH 2003: 9). In other words, the NCEP's action to define the metabolic syndrome seems to have been a way to cloak the practice of setting cholesterol control standards to drug regimes in a scientific garb.

The additional significance of this disjuncture is that, through the NCEP's codification of the metabolic syndrome, the syndrome came under the province of government scientific authority. Whereas in earlier periods, the syndrome has been debated among practicing physicians who specialized in endocrinology and cardiology, the syndrome, however conceptualized, would now be understood as falling under government biomedical jurisdiction. This moment is also significant because, despite the earlier ICD-9-CM classification for dysmetabolic syndrome in 2000, as a result of the NCEP action, the metabolic syndrome increasingly came to acquire significant currency across biomedicine. Table 3.4 shows the citation counts for major definitions of the metabolic syndrome.[51]

[Insert table 3.4 citation counts of major definitions]

The NCEP's definition of the metabolic syndrome also created both controversy and opportunities for more research in the biomedical community. Partly in response to the NCEP definition of the metabolic syndrome, two years later the American Association for Clinical Endocrinologists and the American College of

Endocrinology, including Dr. Reaven, began a renewed campaign to use the construct "insulin resistance syndrome" over the NCEP's metabolic syndrome (Einhorn, Reaven, Cobin, Ford, Ganda, Handelsman, Hellman, Jellinger, Kendall, Krauss, Neufeld, Petak, Rodbard, Seibel, Smith, and Wilson 2003). Dr. Reaven has continued to be a critical voice in the debates about the metabolic syndrome and similar concepts, despite being a key member of the National Cholesterol Education Program, and has advocated for the use of different terms at different times (Reaven 1999; Reaven 2004a; Reaven 2005a; Reaven 2004b; Reaven 2005b).

To add to the ongoing struggles to name and define the syndrome, funded by an educational grant from AstraZeneca pharmaceuticals[52], the International Diabetes Foundation (IDF) convened a 2004 workshop in London consisting of 21 experts in the fields of diabetes, public health, epidemiology, lipidology, genetics, metabolism, nutrition, and cardiology. The workshop aimed to establish a new unified definition of the metabolic syndrome that could be used specifically to compare different populations around the world (Alberti, Zimmet, and Shaw 2006). They present several different hypothetical causes of the metabolic syndrome: insulin resistance, obesity, genetic profile, physical inactivity, aging, and a proinflammatory state.[53]

In 2005, the NCEP and the American Heart Association (AHA) published an official statement affirming the metabolic syndrome as a useful and valid construct (Grundy, Cleeman, Daniels, Donato, Eckel, Franklin, Gordon, Krauss, Savage, Smith, Spertus, and Costa 2005). The NCEP/AHA ground this affirmation in their view that the metabolic syndrome clinically identifies a person at increased risk for cardiovascular disease and/or type 2 diabetes mellitus (Grundy et al 2005: 2736).

However, what is significant about their defense of the syndrome is that they argue that the *clinical* significance of the metabolic syndrome comes through its power as an indicator of statistical risk of disease, not through its existence as a disease with a unique pathogenesis. Yet, the authors argue that getting "a better understanding of the cause(s) of the syndrome may provide an improved estimate for developing ASCVD or type 2 diabetes for individuals" (Grundy et al 2005: 2737).

These discourses about the metabolic syndrome signal that the long-standing cultural power attached to diseases with known biological causes has accompanied, and perhaps in some was supplanted by, the increasing influence of risk-based syndromes with predictive power. The disjuncture in this discursive moment is that after risk-based syndromes are identified via statistical manipulation, then scientists work to uncover the assumed-to-exist biological causes of those manufactured associations. In this context, the authors of the NCEP update assume that the syndrome has a pathogenesis that can be discovered by studying genetics, molecular biological, and cellular signaling:

> Moreover, a lack of understanding of the genetic and metabolic contributions to the causation of the syndrome stands in the way of developing new therapeutic approaches. The need exists, therefore, for additional basic and clinical research designed to better understand [the] pathophysiology [of the metabolic syndrome] from the standpoint of genetics, molecular biology, and cellular signaling (Grundy et al 2005: 2745).

The implication of their argument is that proof of a cause for the metabolic syndrome will help improve its prediction of which groups will develop diseases, not to help establish it as a disease in and of itself. Here, the effort to establish the metabolic syndrome as a disease with a cause in a body seems to be combined with, or perhaps supplanted by, the need to use the metabolic syndrome as an indicator of risk across populations.

Nonetheless, it was precisely these types of arguments about the metabolic syndrome that encouraged the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD) to publish an eight point critique of the metabolic syndrome that called into question its legitimacy within biomedical science and use within clinical practice (Kahn, Buse, Ferrannini, and Stern 2005). While the ADA/EADS authors believe that the metabolic syndrome may have been useful for educational purposes—to educate doctors about the clustering of risk factors for chronic disease—in the final analysis the metabolic syndrome has "taken on meaning and import greater than is justified by our current knowledge" (Kahn et al 2005: 2299). A citation count of this study as of April 30, 2009, shows that 672 authors have cited this ADA critique of the metabolic syndrome (see Table 3.4 for more information)

In the paper, the ADA/EASD advance a critique that lists the top eight reasons to be concerned about the metabolic syndrome

[Insert Figure 3.5]

The authors also raise a new issue about the metabolic syndrome namely, that because of the ways that some definitions of the syndrome use race to determine statistical cutpoints of obesity, these definitions classify different proportions of racial and ethnic minority groups. For example, the prevalence of metabolic syndrome in Mexican Americans varied up to 24% between the WHO and NCEP definitions of the syndrome (Kahn et al 205: 2291).[54] Despite raising this issue, which I take up in greater detail later in this chapter, the ADA/EASD missed an opportunity to frame any broader implications for race and ethnicity as one of the top reasons to challenge the legitimacy or meaning of the metabolic syndrome.

Since the NCEP codified the metabolic syndrome in 2001, different agencies within the government have published information about the metabolic syndrome on their websites. Medline Plus, the web-based medical library provided by the National Library of Medicine[55] and the National Institutes of Health, both define the metabolic syndrome and its causes. Currently posted on the National Heart, Lung, and Blood Institute's website, is the following claim: "Genetics (*ethnicity* and family history) and older age are other important underlying causes of metabolic syndrome."[56] The next section addresses how, in 2009, it is possible for the federal government to construct race and ethnicity as a genetic cause of the metabolic syndrome.

Part II: The Production of Race

In this section, I interpret the metabolic syndrome as a racial project, an unfolding representation of bodily and population difference that continually draws upon racial meanings to make sense of human metabolic difference, and also uses

race to classify bodies and populations. In other words, I reconstruct the emergence of the metabolic syndrome as a polyvalent process of racial formation and biomedicalization. I also use the construct of disjunctures to show how in some moments, this process of racial formation was explicit, and in other moments, it seems to be implicitly woven into the everyday practice of doing biomedical research in the United States. In the broadest and most generous terms, the specific approaches to race within metabolic syndrome research were consistent with the broader treatment of race in biomedical research and clinical medicine. Yet, the metabolic syndrome also becomes a new way that researchers can construct molecular and genetic discourses about racial difference. In the three sections that follow, I show how biomedical researchers used and produced conceptions of race in metabolic syndrome discourse that affirmed essentialist, biological, and genetic conceptions of race. By organizing part II using the same thematic schema I developed in part I, I aim to demonstrate how the racialization of the syndrome itself represents a major disjuncture in the emergence of the metabolic syndrome.

Sampling Normal Subjects, 1956-1987

As World War II ended and the horrors of the Holocaust were revealed in public sight, the range of scientific ideas and practices that had supported white supremacy, racial superiority, and eugenics lost their normalcy. The pronouncements of the United Nations' Education, Scientific, and Cultural Organization (UNESCO) that scientific conceptions of race used to justify Nazi extermination policies had no basis in evolutionary biology (a typological view of race) began to shift biomedical

approaches to race towards a population approach to race. As I described earlier in my critical race theory framework, in the post-War period, population-based approaches to race began to accompany and supplant a view of race as reflecting natural human types. As I describe in this section, this disjuncture was apparent in research on the metabolic syndrome. However, the early period of emergence of the metabolic syndrome (1956-1987) still contained the seeds of typological race thinking.

The physical examinations and laboratory tests that comprised the early technological foundations of the metabolic syndrome were developed using white European research subjects. Thus, the European body comprised the empirical data for the construction of early ideas about the metabolic syndrome. In other words, the metabolism of the European allegedly normal body became the norm, against which other bodies would be compared, in the total absence of any explicit discourse on race. None of the samples of these early studies contained any visible racial minorities, and there was no explicit or implicit reference to whether the observed clustering varied across population groups classified according to race (Hitzenberger 1922; Kylin 1922; Maranon 1922, Vague 1956). Rather, the assumption seemed to be that knowledge produced with white research subjects was valid and would apply universally to all bodies.

Jean Vague's (1956) article on the index of masculine differentiation provides a good example of how race was present by virtue of its absence. The ways in which Vague's core concept, the index of masculine differentiation, overlays the socially constructed category of sex over standardized anthropometric data provided a

conceptual blueprint for how the metabolic syndrome would overlay sex and race categories over standardized biochemical and anthropometric data. However, from a contemporary perspective, the unmarked and yet unremarkable white skin of his subjects is also noteworthy.[57]

In contrast to this implicit racial discourse in Vague's published research, race and ethnicity were explicit organizing principles of population research beginning in the late 1970s and through the 1980s. As I described earlier, beginning in the 1940s the federal government took on a new role in monitoring the metabolic health of the population of the United States. While epidemiological studies like the Framingham Study provided the empirical basis for biomedical information about risk factors for heart disease in white populations, it was not until the 1980s that the US government began to fund population studies on non-European population groups specifically in terms of metabolic health problems (Pollock 2008). This more explicit focus on race, and use of race to sample populations, was in part a response to community-studies of diabetes, heart disease, and stroke that showed rates of disease on the rise in communities of color beginning in the 1960s and 1970s (Williams and Collins 1995). This new focus on race, and new use of race, was also a consequence of broader efforts to include racial and ethnic minorities in clinical and biomedical research (Epstein 2004).

Population studies were instrumental for the emergence of the metabolic syndrome because they provided institutional mechanisms *by* which and a discursive framework *through* which conceptions about race and ethnicity could become attached to the metabolic syndrome. Here, I highlight technical and racial

frameworks of four of the earliest of these federally funded studies, which were all modeled after the 1948 Framingham Study.[58] These four studies were significant for at least three main reasons: 1) they each included examinations and laboratory tests; 2) they sampled and collected data from populations they conceptualized as racial; and 3) their data has been used to analyze the metabolic syndrome. The timing of these studies from the mid-1980s through the present is significant because they would provide the racial data to study the metabolic syndrome in later years.

The first study, the San Antonio Heart Study, 1979-1988, was a longitudinal cohort study that sampled 5,000 residents of three areas of San Antonio, TX—from low SES 'Mexican American', middle SES 'Mexican and White', high SES 'White' (Gardner, Stern, Haffner, Relethford, and Hazuda 1982; Hazuda, Stern, Gaskill, Hoppe, Markides, and Martin 1981). The study was designed to determine factors beyond obesity that contribute to diabetes and cardiovascular risk in Mexican immigrants and Mexican Americans as compared to whites. The physical examination of this study included "blood pressure, obesity, body fat distribution, [and] skin color, the latter to estimate percent Native American genetic admixture."[59] Measurements of insulin resistance were compared to skin color to test the hypothesis that at any given level of adiposity Mexican Americans will be more insulin resistant than Anglos and that the insulin resistance in Mexican Americans is proportional to the degree of Native American ancestry."[60] The San Antonio Heart Study is important because it was the first major study after Framingham to measure all of the components of the metabolic syndrome *and* to focus on a particular ethno-national group: Mexicans.

A second study, the Coronary Artery Risk Development in Young Adults Study (CARDIA), 1985-2006, was a prospective longitudinal, multi-site, cohort study that sampled 5,115 black and white men and women aged 18-30 in Birmingham, Chicago, and Minneapolis (Hughes, Cutter, Donahue, Friedman, Hulley, Hunkeler, Jacobs, Liu, Orden, Pirie, Tucker, and Wagenknecht 1987). The CARDIA Study has been used to evaluate the relationship between racial discrimination and blood pressure (Krieger and Sidney 1998), as well as the relationships between dairy consumption and the insulin resistance syndrome (Pereira, Jacobs, Van Horn, Slattery, Kartashov, and Ludwig 2002). This study is significant because an explicit effort was made in the sampling strategy for CARDIA to achieve approximately balanced subgroups of race, gender, and education across age and geographic groups.[61]

The Atherosclerosis Risk in Communities Study (ARIC), 1987-1998, constituted a third study that was significant because it also was designed to "investigate the etiology and natural history of atherosclerosis, the etiology of clinical atherosclerotic diseases, and variation in cardiovascular risk factors, medical care and disease by race, gender, and location."[62] ARIC was a prospective longitudinal study that sampled 15,792 individuals (aged 45-62) across Minneapolis, MN; Washington County, MD; Forsyth County, NC; and Jackson, MS (Williams 1989) (Schmidt, Duncan, Watson, Sharrett, Brancati, and Heiss 1996).

The fourth study is the Jackson Heart Study (JHS) (1987-2003), the largest prospective study ever of the "inherited (genetic) factors that affect high blood pressure, heart disease, strokes, diabetes and other important diseases in African

Americans."[63] JHS initially began as one site of the aforementioned ARIC study. It sampled 6,500 African Americans, aged 35-84, living in Jackson, MS (Taylor, Liu, Wilson, Golden, Crook, Brunson, Steffes, Johnson, and Sung 2008). According to the study description at the NHLBI website, the Jackson Heart Study included an extensive examination including a questionnaire, physical assessments, and laboratory measurements of conventional and emerging risk factors that may be related to CVD. The physical assessment of subjects in JHS includes height, weight, body size, blood pressure, electrocardiogram, ultrasound measurements of the heart and arteries in the neck, and lung function. The laboratory measurements collected from subjects in JHS includes cholesterol and other lipids, glucose, indicators related to clotting of the blood, among others. With these techniques, the Jackson investigators have been able to examine the "physiological relations between common disorders such as high blood pressure, obesity, and diabetes, and their influence on CVD."[64]

[Insert Table 3.6]

These four studies are significant because they produced forms of racial data that emerged in polyvalent locations and were then incorporated into subsequent research on the metabolic syndrome.[65] Table 3.6 presents the citations for selected research articles based on data from these four studies. For example, according to the study's website, the ARIC study data have been used to publish at least eighteen studies on the metabolic syndrome, metabolic syndrome X, and multiple metabolic syndrome since the publication of its first wave of data in 1989. An analysis of the

list of publications on the study website shows that as of February 2009 at least 16 studies have used CARDIA data to analyze the metabolic syndrome and race.

Is Race Really to Blame? 1988-2000

While population studies would come to produce the majority of data used in published studies of the metabolic syndrome in the 1990s and into the millennium, clinical researchers continued to use racial categorization in their research on metabolic syndrome. In fact, the data that emerged out of race-based population studies provided a basis upon which practicing physicians might treat patients differently based upon their racial classification. From the epidemiological perspective that shaped government funded race-based population studies, there was a need to understand whether and to what extent risks for metabolic health problems might differ across the major population groups of the nation. As will become apparent in this next section, these questions about the *distribution* of metabolic health problems across racially categorized groups began to intersect with new questions about the *causes* of metabolic health problems.

Gerald Reaven's early and later publications constitute a useful documentary case to examine how scholars conceptualized race and ethnicity in relationship to the population dynamics and individual-level causes of the syndrome X. Along side of Jean Vague, who I discussed early in this chapter, Reaven is revered as a second so-called father of the contemporary metabolic syndrome, and for this reason his ideas about race warrant detailed scrutiny. Perhaps because it was delivered as a public lecture and not a scientific study, in his Banting Lecture, Reaven did not note any

special racial/ethnic distinctions in the syndrome X construct nor in the etiological theories that he proposed connected insulin resistance, cholesterol, blood pressure, and heart disease risk. For that matter, he did not mention his sample population at all in the lecture.

In his early research on insulin resistance during the 1970s, Reaven seems to have drawn upon mostly European research subjects when he was part of a group of medical researchers in the Department of Medicine in the Stanford University School of Medicine. Different members of the group (both including Reaven) published two studies in the *Journal of Clinical Investigation*, one in 1970 that tested a new technique for measuring insulin-mediated uptake (Shen, Reaven, and Farquhar 1970), and another in 1975 that demonstrated that this new method of insulin resistance tends to identify subjects with diabetes (Ginsberg, Kimmerling, Olefsky, and Reaven 1975). Both studies seemingly use of one of the samples upon which Reaven built his later research on Syndrome X. The first and last initials of one of the research subjects ("L.K.") are printed in both articles, thus strongly suggesting they are using the same sample. The descriptions of the sample, which contains people with diagnosed diabetes and those without diabetes, are different in each paper in one exceptional case. In the 1970 paper, the authors describe how the diabetics in the sample were selected from their patient referral group, matched by weight, age, and percent adiposity with the normal control group. In this brief passage, they describe the sampling procedure for the normal population, which is notably absent from the 1975 paper:

> Normal individuals were selected after interviews with a group of volunteers who had recently been discharged from a local minimum-security prison. Volunteers responded to a notice asking for assistance in a research project which would furnish their living expenses during a 2 week hospital stay (Shen, Reaven, and Farquhar 1970: 2151).

In the 1975 paper, the recently released inmates who likely participated in the study in order to get shelter are described simply and neatly as "healthy adult male volunteers." While neither study reveals or refers to the race or ethnicity of its subjects, both the age and sex of each subject is noted in printed tables. Without any evidence one way or the other, the only safe assumption is that Reaven's subjects were predominantly white.

In a book on syndrome X in 2000 (*Syndrome X: overcoming the silent killer that can give you a heart attack*), Reaven argues that "ethnicity" plays a role in causing syndrome X, with people of non-European origin being at a much greater risk for the syndrome (Reaven, Strom, and Fox 2000: 20). In the introductory chapter of the book, the reader is confronted with a section labeled "Who is likely to develop syndrome X?" Here, Reaven lists the people who are likely to develop syndrome X: 1) people with genetic abnormalities; 2) people of non-European origin; 3) people with a family history of diabetes, heart attack, and hypertension; 4) and people who eat poorly and exercise little. Why does Reaven believe that people of non-European origin are more likely to develop syndrome X?

The answer to this question about how Reaven conceptualizes the relationship between ethnicity and syndrome X lies in chapter four in a section labeled "Are

Genes Really to Blame?" This was my inspiration for the title of this section because Reaven's central argument in this passage is that ethnicity identifies genetic differences between individuals and populations. In adjudicating the respective role of genes in the development of syndrome X, Reaven cites three lines of genetic evidence, two of which are drawn from research in which he participated, that taken together treat race and ethnicity as genetic categories.

For the first line of evidence, Reaven cites a 1985 study that he co-authored that compared fifty-five Pima Indian men living near Phoenix to thirty-five Caucasian men living in California (Bogardus, Lillioja, Mott, Hollenbeck, and Reaven 1985).[66] The investigators measured the levels of obesity, physical fitness, and insulin resistance in the two groups (who are not explicitly labeled as racial groups in any way) and used statistical techniques to determine the degree to which differences in their levels of obesity and physical fitness contributed to the variability of their insulin action (Reaven, Strom, and Fox 2000: 56). Reaven, writing now in 2000, claims that this 1985 study showed that "half of the variability of insulin action was due to lifestyle, *the other half presumably to our genes*. Of the 50 percent attributed to lifestyle, half was due to fitness, half to obesity" (Reaven, Strom, and Fox 2000: 57). Here, the authors claim that the other half was due to racial differences in genetics because their operating assumption about race is that by comparing Pima Indians and Europeans, they were uncovering underlying genetic differences between them.

The second line of genetic evidence upon which Reaven draws to claim that syndrome X is heritable also comes from research conducted on the same sample of

Pima Indians (Lillioja, Mott, Zawadzki, Young, Abbott, Knowler, Bennett, Moll, and Bogardus 1987). This study compared levels of insulin resistance within Pima families to levels of insulin resistance across families and demonstrated, again according to Reaven in 2000, that the clustering of insulin action is greater within families than it is across families.[67] In effect, this claim constructs familial heritability and genetic susceptibility as the same biomedical phenomenon when it plays out within a tribal group known to have high rates of intermarriage.

The third line of evidence that Reaven cites to substantiate the role he sees for genetics in causing syndrome X is not as well cited, making it more difficult to analyze his claims about race in great detail, but the implications of his argument are clear. According to Reaven's theory, genetics play a role in the development of syndrome X, and whatever the guilty genes might be, people of non-European ancestry are more likely to have them (Reaven, Strom, and Fox 2000: 58). How can Reaven make such a claim? He refers to a body of population studies that purportedly shows that American Indians, South Asian Indians, Japanese-Americans, African-Americans, Mexican-Americans, Australian Aboriginals, and various Pacific Islander populations are more insulin resistant compared to those of European ancestry (Reaven, Strom, and Fox 2000: 57). Reaven does not cite any studies after making these sweeping claims, but instead inserts a parenthetical statement after listing these group differences that crystallizes his ideas about the causes of racial difference: the observed differences in insulin resistance reflect genetic differences between racial groups. The authors argue that while its possible that some racial groups might be more insulin resistant because of lifestyle habits and other factors,

several studies [again, not cited in the book] *did take* group differences in all known factors into account, the differences in insulin resistance found as a result of these comparisons result from heritable genetic differences between groups (Reaven, Strom, and Fox 2000: 58).

At the conclusion of the section, Reaven writes that the comparison of a racial group with insulin resistant genes to one that does not have these genes is a way to test the hypothesis about non-European ethnicity as a genetic cause of syndrome X (Reaven, Strom, and Fox 2000: 58). With reference to a comparative study of South Asian Indians and Europeans living in the United Kingdom, Reaven argues that despite the fact that the South Asian Indian individuals ate little fat and had lower cholesterol levels than the Europeans, they had fifty percent higher incidence of diabetes and heart attacks. "Clearly," Reaven concludes, "genes played a major role in the development of insulin resistance and Syndrome X in these South Asian Indians" (Reaven, Strom, and Fox 2000: 58).

The New Special Populations, 2001-Present

In the contemporary period, the uses and conceptions of race in biomedical research on the metabolic syndrome seemingly have expanded in ways reminiscent of earlier periods and extended in new and unanticipated directions. These expansions and extensions have taken place through the increasing interaction between new forms of clinical biomedicine and government public health research, both of which are focused on racial health disparities. Due to these converging forces, there is no lack of data about race and health in American biomedicine. The term *special*

populations is used specifically within *government biomedicine* to refer to pregnant women, children, racial/ethnic minorities, elders, and any other population group that is not white/European and male. In the contemporary moment, people who are classified with the metabolic syndrome or who think they have it comprise a new special population that is constructed out of and produces race. In this final section, I describe some of the central ways that race is taken up in contemporary research on the metabolic syndrome and discuss a special focus on African Americans as a special population that has been organized around the metabolic syndrome.

The 2001 National Cholesterol Education Program (NCEP) approach to race was to acknowledge existing racial inequalities in heart disease risk, and to advocate an approach to managing cholesterol that treated all groups as if they were the same, but otherwise to leave questions about race unasked. In a section titled "Special Considerations for Different Population Groups", the authors of the 2001 NCEP report imply that the high presence of the metabolic syndrome among African Americans is a partial explanation of racial inequalities in heart disease risk:

> African Americans have the highest overall CHD [coronary heart disease] mortality rate and the highest out-of-hospital coronary death rates of any ethnic group in the United States, particularly at younger ages. *Although the reasons for the excess CHD mortality among African Americans have not been fully elucidated, it can be accounted for, at least in part, by the high prevalence of coronary risk factors.* Hypertension, left ventricular hypertrophy, diabetes mellitus, cigarette smoking, obesity, physical inactivity, and multiple CHD risk factors

> all occur more frequently in African Americans than in whites [emphasis added] (NCEP 2001: 2495).

What this is saying is that African Americans have higher risk for heart disease because, as a group, they experience multiple heart disease risk factors more often than do whites. Despite making this claim, the NCEP concludes that there was *insufficient evidence* to make racial and ethnic-specific recommendations for studying or treating cholesterol for African Americans or other ethnic population groups. They continue,

> Other ethnic groups and minority populations [other than African Americans] include Hispanics, Native Americans, Asian and Pacific Islanders, and South Asians. Although limited data suggest that racial and ethnic groups vary somewhat in baseline risk for CHD, this evidence did not appear sufficient to lead the ATP III panel to modify general recommendations for cholesterol management in these populations (NCEP 2001: 2495).

And while it is technically true that there are groups other than European and African Americans living in the United States, this has nothing to do with cholesterol management *per se*. By not addressing the potential implications of their new definition of the metabolic syndrome for racial and ethnic groups in the context of the ongoing dialogue about racial health disparities, the NCEP helped to establish the epistemic conditions for race and ethnicity in contemporary metabolic syndrome research.

Since 2001, despite the claims about race (and not made) in the NCEP definition of metabolic syndrome, scientists have increasingly raised questions about the use, measurement, and interpretation of the metabolic syndrome construct across different racial and ethnic populations. These new questions about the relationship between race and the metabolic syndrome have several disjunctive features. For one, since the World Health Organization recommended standardizing obesity measurements in different racial and ethnic groups in first 1997 (WHO 1997) and again in 2004 (WHO 2004), race and ethnicity are explicitly used in the practice of validating group-specific empirical cutoff points (endpoints) for the physical examinations and laboratory tests (biomarkers, for short) that comprise the syndrome. The argument for using race-based endpoints is that they improve the generalizability and validity of comparisons of disease risk across individuals and populations. Statistical validity is determined with respect to the outcome, the metabolic syndrome, by evaluating whether the syndrome successfully identifies all of the individuals at increased risk within specific populations groups. For example, the body mass index for an individual who is classified "African American" would be statistically adjusted for two reasons: first, to account for the differential relationship between obesity and CVD risk in African Americans as compared to other groups. These standardizations construct statistical norms against which racial and ethnic populations can be validly compared to one another.

A second feature of the relationships between race and the metabolic syndrome concerns how research institutions use new conceptions of the metabolic syndrome to compare racial and ethnic groups. In 2003, in their joint definition of the

insulin resistance syndrome, the American Association of Clinical Endocrinologists and the American College of Endocrinology provided optional standardizations of obesity for different ethnic groups (Einhorn 2003). They also repeat the thesis that "Non-Caucasian ethnicity (e.g. Latino/Hispanic American, African American, Native American, Asian American, Pacific Islander)" is a "risk factor" for the syndrome, reaffirming Reaven's earlier racial hypotheses from the 1980s.

Three years later in 2006, the International Diabetes Federation incorporated racial and ethnic measurements of waist circumference because "…there are clear differences across ethnic populations in the relationship between overall adiposity, abdominal obesity, and visceral fat accumulation" (Alberti, Zimmet, and Shaw 2006: 473). The authors elaborate a list of country/ethnic-specific values for waist circumference for "Europids," "South Asians," "Chinese," and "Japanese" populations. Several other groups do not yet have their own standardized values: "Ethnic South and Central Americans," "Sub-Saharan Africans," and "Eastern Mediterranean and Middle East." In the meantime, the authors advocate that the South and Central American ethnic groups should use "South Asian" values, the Africans and the "Arab populations" should use "European" values until "more specific data are available." The authors provide special instructions for applying these "country/ethnic specific values" in clinical and epidemiological research. They write,

> It should be noted that the ethnic group-specific cut-points should be used for people of the same ethnic group, wherever they are found. Thus, the criteria recommended for Japan would also be used in

> expatriate Japanese communities, as would those for South Asian males and females regardless of place and country of residence (Albert, Zimmet, and Shaw 2006: 476).

These recommendations imply that these standardizations are not country-specific values, but racial ones that transcend "place and country of residence."

Since 2005, these institutional practices have resulted in a new line of biomedical research that investigates the implications of using the metabolic syndrome to compare heart disease risk across different racially categorized groups (Banerjee and Misra 2007; Unwin, Bhopal, Hayes, White, Patel, Ragoobirsingh, and Alberti 2007). Scholars in this emerging field of research have investigated racial and ethnic differences in the relationships between obesity and heart disease risk (Zhu, Heymsfield, Toyoshima, Wang, Pietrobelli, and Heshka 2005), body composition and metabolic risk factors (Desilets, Garrel, Couillard, Tremblay, Despres, Bouchard, and Delisle 2006), the power of trigycerides to predict insulin resistance (Bovet, Faeh, Gabriel, and Tappy 2006; Sumner, Finley, Genovese, Criqui, and Boston 2005; Sumner and Cowie 2008), and the relationship between HDL cholesterol levels and CVD risk (Amarenco, Labreuche, and Touboul 2008).

African Americans, and theories of African American health, occupy a prominent place in special populations research that links race and the metabolic syndrome. A review article on the metabolic syndrome in African Americans was published in the journal *Ethnicity & Disease* in 2003.[68] All of the authors of this review article are members of the African-American Lipid and Cardiovascular Council (AALCC), a non-profit health professional advisory group that is sponsored

from an unrestricted educational grant from Bristol-Myers Squibb Company, and many of them have published widely on the metabolic syndrome and African Americans.[69] Like the NCEP, Hall and colleagues (2003) situate their review of metabolic syndrome and African Americans in the context of the epidemiological fact that African Americans have the highest overall CHD mortality and out-of-hospital coronary death rates of any racial group in the United States. Yet to explain the racial disparities in the metabolic health between "Native Americans", "Mexican Americans", and "African Americans" compared to "European Americans", the group advances a "genetic admixture theory" (Hall et al 2003: 415).[70]

Theories of genetic admixture assume that individual level susceptibility to disease is related to their shared genetic admixture with populations known to be susceptible to the disease. According to this theory, before the 1960s, European Americans had historically had higher rates of diabetes than African Americans, Hispanics, and Native Americans but increasing racial miscegenation that has occurred since the colonialism explains the increasing rates of diabetes in these racial and ethnic minority groups (Tull and Roseman 1995: 614). The central assumption of this theory is that racial groups at an earlier moment were pure and segregated and it is their intermingling since the "discovery" of race that explains racial disparities in modern times. They argue that the degree of genetic admixture is related to the "susceptibility" of different racial groups to the risk factors that constitute metabolic syndrome. They write

> Whites of European origin appear to have greater predisposition to atherogenic dyslipidemia [high levels of LDL or bad cholesterol],

> whereas Blacks of African origin are more prone to HBP [high blood pressure], type 2 diabetes and obesity. Native Americans and Hispanics are less likely to develop HBP than Blacks, but appear particular susceptible to type 2 diabetes. Of particular note is the considerable genetic admixture among Native Americans and Mexican Americans (Hall et al 2003: 415).

Also like the authors of the NCEP report, the authors homogenize all non-white population groups in terms of recommendations for treating the metabolic syndrome. They write that most of the discussion and recommendations for African Americans probably also apply to Native Americans, Mexican Americans, and South Asians (Hall et al 2003: 415).

Conclusion

In part I of this chapter, I demonstrated that the metabolic syndrome emerged through the technoscientific integration of molecularization and the risk factor paradigm, two social processes that were increasingly focused on understanding metabolism from a biomedical perspective. Based on this analysis, I argue that the extension of legitimate government authority over the metabolic syndrome marks the emergence of a new discourse of biopower. This emergence created a context in which the molecular processes of the body were used to constructed risk-based syndromes of populations that social institutions like professional biomedicine and the federal government could deploy to understand and improve metabolic health, especially among racially categorized groups. Table 3.7 provides a summary of the

social processes and institutional relationships involved in the racial formation of the metabolic syndrome.

[Insert Table 3.7. Summary of social processes and institutional relationships in the racial formation of the metabolic syndrome]

In part II, I argued that the emergence of the metabolic syndrome created a discursive and institutional context for the production of race. In other words, the constructions of the metabolic syndrome have changed over time, but they emerge out of processes that consistently draw upon and produce racial meaning. The construction of racial meaning that accompanied the emergence of the metabolic syndrome was consistent with broader biomedical ideas about race. In the first moment between 1956 and 1988, the science of metabolism became a site for the integration of typological and population-based approaches to the study of race and racial difference. This process would continue to inform the emergence of the metabolic syndrome. For example, in the second moment between 1988 and 2000, discourses that constructed race as genetic shaped the biomedical debate about the genetic causes of the metabolic syndrome. In the third moment since 2001, the metabolic syndrome has become a new site of special populations research in which racially categorized groups are compared using standardized biological, genetic, and metabolic measurements.

In the next chapter, I explore how constructions of the metabolic syndrome and meanings of race are taken up in new special populations research on prescription drugs in the politics of metabolism.

Table 3.1 Selected technical developments contributing to the metabolic syndrome, 1896-1985.

Metabolic process	Measurement	Technical Development[71]
Blood Pressure/ Hypertension	Systolic/diastolic blood pressure measurement	(1896) Riva-Rocci develops the mercury manometer. (1897) Hill and Bernard develop the aneroid manometer. (1906) Janeway publishes "The Clinical Study of Blood Pressure" which influences the medical director Northwestern Mutual Life Insurance Company, Dr. J.W. Fisher, to include blood pressure in its physical examinations. By 1918, most insurance companies measured blood pressure in their examinations. (1917, 1921, and 1927) the American Bureau of Standards published major reports on the improvement and standardization of blood pressure measurement and equipment.
Blood Sugar/ Insulin Resistance Glucose Tolerance	Fasting Plasma Glucose	(1929) Horgaard and Thayssen develop what they call the insulin-tolerance test. (1983) DeFronzo and colleagues develop the "Eeuglycaemic hyperinsulinaemic clamp technique"-- the proverbial 'gold standard' for measuring insulin resistance in vivo (Defronzo, Ferrannini, and Koivisto 1983). (1985) Mathews and colleagues construct the homeostasis model assessment-insulin resistance index. (1998) Belfiore and colleagues develop the oral glucose tolerance test (Belfiore, Iannello, and Volpicelli 1998).
Cholesterol	LDL and VLDL triglycerides, HDL lipoprotein analysis	The history of the science lipid metabolism originates in the late 1800s. (1948-present) The Framingham Heart Study was central to the establishment of the risk factor paradigm, especially the role of total cholesterol in the development of cardiovascular disease (Kannel, McGee, and Gordon 1976). (1964) Konrad Bloch and Feodor Lynen were awarded the Nobel Prize in Physiology or Medicine for their discoveries concerning the mechanism and regulation of the cholesterol and fatty acid metabolism. (1985) Goldstein and Brown also win a Nobel for their research on the cellular synthesis of cholesterol.
Obesity	Body Mass Index (weight in kg/ height in meters^2)	(1942) Metropolitan Life Insurance Company issues weight-for-height tables that measure the "ideal weight" for men (Metropolitan Life Insurance Company 1942). (1959) MetLife includes women in its weight-for-height schema (Metropolitan Life Insurance Company 1959) (1980) The USDA Dietary Guidelines for Americans attempts to standardize the measurement of body mass index, although the measurement of obesity would continue to undergo significant revision in the intervening years.

Figure 3.2. Photograph scanned from Vague (1954) showing gynoid obesity in male and female subject.

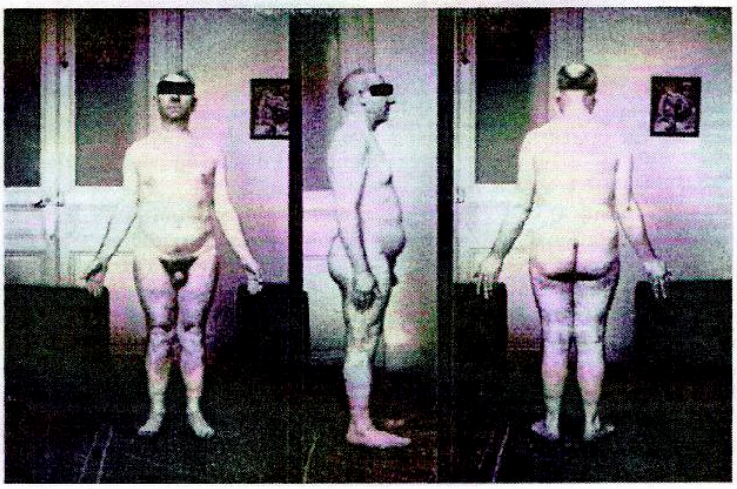

Fig. 3. Gynoid obesity in the male. 47 years. Height, 159 cm. Weight, 70 kg. IMD −63. Normal distribution of hair. Blood pressure 110/70 mm Hg. Hyposthenia, anxiety neurosis, mediocre sexual activity, rectal obsession. Testicles 3/4 right-left. Spermogram deficient (3000 spermatozoa per cu mm). FSH 10 mouse units. Ketosteroids normal, but elevated pregnandiol (25 mg).

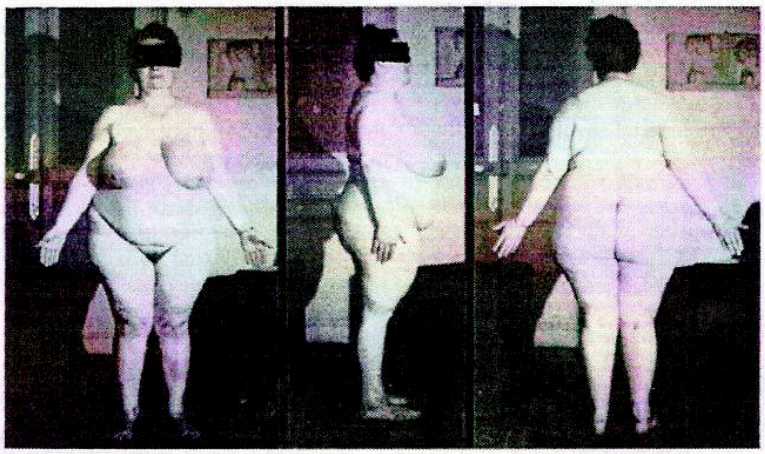

Fig. 4. Gynoid obesity in the female. 47 years. Height, 158 cm. Weight, 102 kg. IMD −60. Blood pressure 130/70 mm Hg. Blood sugar normal. Normal genital system. Menopause at age 42. Normal distribution of hair.

Figure 3.3. Photograph scanned from Vague (1954) showing android obesity in male and female subject.

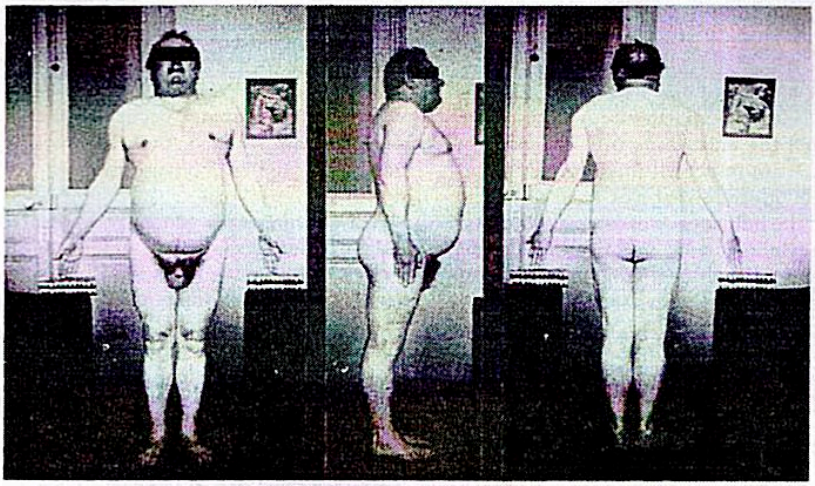

Fig. 5. Android obesity in the male. 60 years. Height, 168 cm. Weight, 98 kg. IMD +83. Masculine distribution of hair. Blood pressure 170/90 mm Hg. Marked muscular activity. Good appetite and digestive capacity—2 liters of wine daily. Rages with flushing. Strong sexual activity. Known diabetic for 2 years. Intermittent claudication. Impotence. Adrenals normal on pneumoretroperitoneal examination. Urinary steroids normal. Diet and dienestrol brought the weight to 90 kg and the blood sugar to 90 mg/100 ml. with disappearance of intermittent claudication and return of erections.

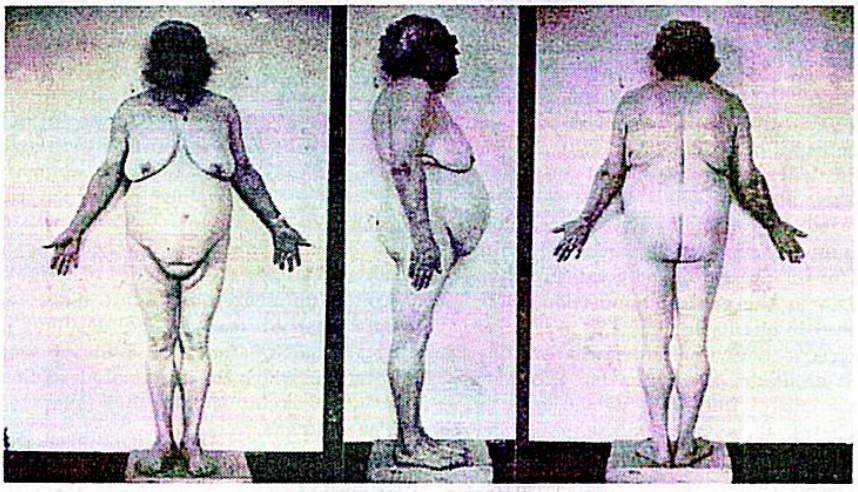

Fig. 6. Android obesity in the female. 66 years. Height, 161 cm. Weight, 85 kg. IMD +43. Normal genital system. Pubic and axillary hair rather scant. Blood pressure 190/110 mm Hg. Diabetes since age 56. Angina of effort. Death two years later from myocardial infarction.

Table 3.4. Selected major definitions of the metabolic syndrome and citation counts (as of April 30, 2009), 1999-2005.

	Reaven's Syndrome X (1988)	**World Health Organization: metabolic syndrome (1999)**	**National Cholesterol Education Program Adult Treatment Panel III: metabolic syndrome (2001)**	**American Association of Clinical Endocrinologists and American College of Endocrinology: Insulin Resistance Syndrome (2002)**	**American Heart Association and the Heart, Lung, and Blood Institute: Metabolic syndrome (2005)**
Criteria	*All of the following*	*Elevated glucose and dyslipidemia plus two or more of the following biomarkers*	*3 of 5 of any of the following biomarkers*	*All of the following*	*3 or more out of 5 of the following*
Elevated blood sugar	Insulin resistance	Elevated glucose >= 110 mg/dl	Elevated glucose > 110 mg/dl	*excluding type 2 diabetes fasting glucose of 110-125 mg/dl *or* 2hr post-glucose (75g) > 140 mg/dl)	Elevated glucose >=100 mg/dl *or* Drug treatment for glucose
"Bad" LDL cholesterol dyslipidemia	Increased VLDL	LDL triglycerides >=150 mg/dl *or* HDL < 35 mg/dl	LDL triglycerides > 150 mg/dl	LDL triglycerides > 150 mg/dl	LDL triglycerides >= 150 mg/dl *or* drug treatment for cholesterol
"Good" HDL cholesterol	Decreased HDL	See above panel	HDL cholesterol men < 40 mg/dl women < 50 mg/dl	HDL cholesterol men < 40 mg/dl women < 50 mg/dl	HDL cholesterol men < 40 mg/dl women < 50 mg/dl
Blood pressure	High blood psressure	Blood pressure > 160/90 *or* drug treatment for hypertension	Blood pressure > 130/85	Blood pressure > 130/85	Blood pressure >= 130 systolic *or* 85 diastolic *or* drug treatment for hypertension
Obesity	Not included	Abdominal Circumference weight/height ratio > .90 *or* BMI > 30kg/m^2 *or* waist circumference >= 94 cm	Abdominal circumference men > 40 inches women > 35 inches	See below panel	Abdominal circumference men >= 102 cm women >= 88 cm
Other Criteria		Also includes microalbuminuria, or the urinary albumin excretion rate		Body mass index (BMI) adjusted by ethnicity, waist circumference, and family history of type 2 diabetes	
Citation Count	**6,261**	**2,843**	**7,897**	**294**	**1,005**

Table 3.5. American Diabetes Association & European Association for the Study of Diabetes Critique of the metabolic syndrome, 2005 (cited 672 times, April 2009).

Eight Challenges to the Metabolic Syndrome
1) The criteria for metabolic syndrome are ambiguous or incomplete and the rationale for threshold values of specific biomarkers are ill defined
2) The value of including diabetes in the definition is questionable
3) Insulin resistance as the unifying etiology of metabolic syndrome is unclear
4) There is no clear basis for including/excluding other CVD risk factors
5) CVD risk value is variable and dependent on the specific risk factors present
6) The CVD risk associated with the syndrome appears to be no greater than the sum of its parts;
7) Treatment of the syndrome is no different than the treatment for each of its components; and
8) The medical value of diagnosing the syndrome is unclear

Table 3.6. Selected articles on metabolic syndrome based on population studies.

San Antonio Heart Study (SAHS)

- Ferrannini E, Haffner SM, Mitchell BD, Stern MP. 1991. Hyperinsulinaemia: the key feature of a cardiovascular and metabolic syndrome. *Diabetologia* 34(6):416-22.
- Han TS, Sattar N, Williams K, Gonzalez-Villalpando C, Lean ME, Haffner SM. 2002. Prospective study of C-reactive protein in relation to the development of diabetes and metabolic syndrome in the Mexico City Diabetes Study. Diabetes Care 25(11):2016-21.
- Meigs JB, Williams K, Sullivan LM, Hunt KJ, Haffner SM, Stern MP, Gonzalez Villalpando C, Perhanidis JS, Nathan DM, D'Agostino RB Jr, D'Agostino RB Sr, Wilson PW. 2004. Using metabolic syndrome traits for efficient detection of impaired glucose tolerance. *Diabetes Care*. June; 27(6):1417-26.

Atherosclerosis in Communities Study (ARIC)

- Liese A, Mayer-Davis EJ, Tyroler HA, Davis CE, Keil U, Schmidt MI, Brancati FL, Heiss G. 1997. Familial components of the multiple metabolic syndrome: the ARIC study. Diabetologia 40:963-70.
- Schmidt MI, Duncan BB, Watson RL, Sharrett AR, Brancati FL, Heiss G. 1996. A metabolic syndrome in whites and African-Americans; The Atherosclerosis Risk in Communities baseline study. Diabetes Care. May;19(5):414-8.
- Liese AD, Mayer-Davis EJ, Tyroler HA, Davis CE, Keil U, Duncan BB, Heiss G. 1997. Development of the multiple metabolic syndrome in the ARIC cohort: joint contribution of insulin, BMI, and WHR. Annals of Epidemiology 7:407-16.

Coronary Artery Risk Development in Young Adults Study (CARDIA)

- Rathmann W, Funkhouser E, Dyer AR, Roseman JM. 1998. Relations of hyperuricemia with the various components of the insulin resistance syndrome in young Black and White adults: The CARDIA Study. *Annals of Epidemiology* 8(4):250-261
- Shen W, Punyanitya M, Chen J, Gallagher D, Albu J, Pi-Sunyer X, Lewis C, Grunfeld C, Heshka S, Heymsfield S. 2004. Waist circumference correlates with metabolic syndrome indicators better than percentage fat. *Obesity* 14(4):727-736.
- Carnethon M, Hill J, Loria C, Sidney S, Savage P, Liu K. 2004. Risk factors for developing the metabolic syndrome in young adults. *Diabetes Care* 27:2707-2715.

Jackson Heart Study (JHS)

- Burchfiel CM, Skelton TN, Andrew ME, Garrison RJ, Arnett DK, Jones DW et al. 2005 Metabolic syndrome and echocardiographic left ventricular mass in blacks: the Atherosclerosis Risk in Communities (ARIC) Study. *Circulation* 112(6): 819-827.
- Taylor H, Liu J, Wilson G, Golden SH, Crook E, Brunson CD, Steffes M, Johnson WD, Sung JH. 2008. Distinct component profiles and high risk among African Americans with metabolic syndrome: the Jackson Heart Study. Diabetes Care 31(6):1248-53.

Table 3.7. Summary of social processes and institutional relationships in the racial formation of the metabolic syndrome.

The Politics of Metabolism	***1956-1987***	***1988-2000***	***2001-Present***
Construction of racial meaning	Integration of typological and population-based study of race and metabolism	Race is constructed as a genetic and biological cause of metabolic syndrome	New special populations research that measures and compares racially categorized groups
Construction of metabolic syndrome	Technoscientific development and institutionalization of molecularization and risk factors focused on metabolism	Proliferation of risk-based syndromes and discourses about the biological and genetic causes of the metabolic syndrome	Extension of state legitimacy and authority over the metabolic syndrome

Chapter 4: The Racial Pharmacology of Killer Applications

In chapter one, I stated that pharmaceutical corporations are interested in developing prescription drugs that could be sold to people who might be classified with the metabolic syndrome and that this research has a new racial dimension. In chapter three, I argued that the emergence of a discourse about the metabolic syndrome involved social processes and institutional relationships that accompanied and produced new racial meanings. Specifically, I suggested that the emergence of the metabolic syndrome is a racialized process of biomedicalization that has drawn upon the techniques of biopower that are focused on disciplining and regulating the metabolic health of individuals and groups. In chapter three, I also used the term *special populations* as a way of referring to the biomedical study of population groups who are not white men. This term has added significance in the context of drug research and development because just as biomedical research needed to include these non-white and male groups, drug manufacturers are now required to study the safety and efficacy of prescription drugs in these groups.

The metabolic syndrome and race are deployed at the intersection of an increasingly technological, biomedical, and racially organized approach to the study of prescription drugs and drug metabolism. In this chapter, I analyze the genealogical descent of race and the metabolic syndrome in biomedical research on the metabolism and use of prescription drugs among African Americans.[72] This new biomedical research encompasses three distinctive elements that connect race, the metabolic syndrome, and prescription drugs. First, researchers use conceptions of race and the metabolic syndrome to study prescription drugs and drug metabolism in African

American populations. Second, physicians and clinical decisions play a role in terms of the differential diagnoses in health conditions that lead to differential forms of pharmacological treatment of health problems in African Americans. Third, this new special populations research is a potential site for the deployment of biological and genetic explanations of racial differences in the metabolism of prescription drugs among African Americans.

To investigate these issues, I divide this chapter into three sections. In the first section, I develop the metaphor of *killer applications* to examine how prescription drugs operate in the politics of metabolism. Recall that the politics of metabolism encompasses the discourses, social processes, and institutional relationships that structure the metabolic health of individuals and groups. Killer applications is a metaphor for novel combinations of human and non-human technologies that structure bodily practices in a wide range of social, commercial, and scientific contexts. The metaphor of killer applications is especially well suited for examining how prescription drugs operate in the politics of metabolism by transforming the ways that pharmaceutical corporations design and market drugs for racially categorized groups. Stated differently, the search for and research on killer applications have seemingly incorporated the metabolic syndrome and race.

In the second and third sections, I compare the different racial meanings in the pharmacological study of two potential killer applications: atypical antipsychotics and statins. Statins and atypical antipsychotics are prescribed for the treatment of schizophrenia and high cholesterol, respectively, and each has a unique relationship to the metabolic syndrome. First, statins are a class of drugs that physicians prescribe

to treat dyslipidemias, which are fundamental to the construction of the metabolic syndrome.[73] Second, atypical antipsychotics are a class of drugs that mental health practitioners prescribe to treat the collection of symptoms known as schizophrenia, but that produce the negative side effects of weight gain, hyperglycemia, and dyslipidemia—side effects that together comprise the metabolic syndrome.[74] After describing the metaphor of killer applications and the field of racial pharmacology, I elaborate on the specific comparisons made in this chapter.

The Metaphor of Killer Applications

A biomedical-government-industry collaboration formed in 2002 focused on the relationships between diabetes and heart disease.[75] At this meeting, a pharmaceutical company representative encouraged a new line of pharmaceutical research on what he called "killer applications." He argued that so called killer applications research on the metabolic syndrome was needed because patients, like those with metabolic syndrome, are taking multiple drugs for multiple health problems and a new killer application in this area might obviate the need for multiple drug regimens, or replace existing therapies by increasing efficacy or decreasing side effects. What is a killer application and what does it have to do with prescription drugs and the metabolic syndrome?

Donna Haraway suggests that *killer applications* constitutes a useful metaphor for novel combinations of human and non-human technologies that structure bodily practices in a wide range of social, commercial, and scientific contexts (Haraway 1997).[76] Companies strive to develop killer applications to gain technological

superiority and maintain market supremacy over their competitors (Downes and Mui 1998). As Larry Downes and Chunka Mui point out, killer applications are a new good or service that "establishes an entirely new category and, by being first, dominates it, returning several hundred percent on the initial investment" (Downes and Mui 1998: 4). For example, killer applications structure the social practices of technology users, as was the case of the iPod. Within a few short years, the iPod revolutionized how people listen to music, interact with each other, and as a result of being the first of its kind, it still enjoys widespread popularity and brisk sales. In other words, killer applications enact the power to change modes of cultural and economic organization. Moreover, because of the myriad ways that killer applications impact our social lives, they have the potential to change our bodies and identities in profound ways.

This metaphor of killer applications suits prescription drugs in four central ways. First, prescription drugs fit the classic definition of killer applications, namely, they are technoscientific commodities that combine non-human and human elements. The non-human element of prescription drugs consists of the drugs themselves. Prescription drugs are mostly synthetic chemical compounds and fillers that have been mass-produced in laboratories and factories since the 1950s. The human elements of prescription drugs as killer applications can be seen in the field of clinical pharmacology, the branch of biomedical science that studies the intended and unintended effects of drugs on the body. These human elements of prescription drugs consist of the relationships between medical professionals, typically doctors, who prescribe the drugs and the individual patients who purchase and consume the drugs.

The interaction of these non-human and human elements of prescription drugs qua killer applications represents a network of technoscientific and commercial relationships that fundamentally change human bodies. In this sense, prescription drugs are "applied" to bodies through a formalized process that involves drug companies, federal regulatory agencies, medical professionals, and consumers.

Second, because prescription drugs have revolutionized how American medicine treats illness and disease, the search for killer applications has taken on new cultural meaning within the pharmaceutical industry. As the pharmaceutical industry has grown in scope and reach over the past fifty years, taking prescription drugs has become Americans' preferred practice for treating illness. When Americans get sick, they turn to their doctors and pharmacists for help, assuming that they have access to doctors and pharmacists and the financial means to pay them. If one is not feeling well, often the first question people ask is "Are you taking anything?" When individuals develop illnesses, all they need to do is ask their doctors for a prescription. Every day, Americans are bombarded with television and print advertising from the pharmaceutical industry that encourages them to ask their doctors about taking new drugs to treat what ails them.

Third, risk management is the central tool for creating successful and profitable killer applications. The production of knowledge about risk was central to the emergence of the metabolic syndrome and no less has risk influenced a biomedical and statistical approach to population-based drug development and research. Some prescription drugs like Lipitor acquire market supremacy by doing the best job of helping patients lower their risk of developing a particular condition,

whereas other prescription drugs become successful because they do the best job of minimizing the risk of experiencing side effects from other drugs. The better a drug is at managing different kinds of risk in bodies and populations, the more likely it will become a killer application. Recently, the term *comparative efficacy* has emerged in the current debate over the future of the US health care system as a way to make America's health care system more cost efficient (Malozowski 2008). Comparative efficacy refers to a process through which researchers *compare* possible treatments for a health problem in order to determine which treatment, or combinations of treatments, is most likely to be effective at treating the problem.

Fourth, because of the rise of metabolic health problems in the American population, the pharmaceutical industry has a special interest in killer applications. The health problems encapsulated by the metabolic syndrome currently account for one fifth of all health care spending in the United States and much of that money is spent on prescription drugs. In 2005, Americans spent $200.3 billion dollars on prescription drugs, five times more than they spent in 1990 (KFF 2007). The pharmaceutical industry has been *the* most profitable industry in the United States for years, in large part due to prescription drugs that are sold to the millions of individuals who suffer from metabolic conditions like heart disease and high cholesterol. In 2004, four of the top ten most dispensed drugs treat hypertension or high cholesterol, two central pillars of the metabolic syndrome (KFF 2007).[77] Globally, the biggest selling drug is Lipitor, a cholesterol drug, which brought in sales *of $7.7 billion* in 2004. With the appearance of the metabolic syndrome, the

pharmaceutical industry has sixty million new potential customers and, potentially, some new challenges, some of which are centered on race.

Defining Racial Pharmacology

In recent years, numerous scholars have raised a series of questions about how drug researchers use race to develop, study, and market prescription drugs (Jones and Perlis 2006; Kahn 2006; Lynch, Lynch, and Dubriwny 2006; Sankar and Kahn 2005). Many of these new questions about race in pharmaceutical research have emerged in response to the controversy over BiDil, the first drug approved by the FDA in June 2005 for specific use among African Americans. BiDil (isorbide dinitrate/hydralazine hydrochloride) is a not a new chemical compound—rather it is a new patented combination of two existing generic drugs. As these scholars have identified, one of the central questions in the BiDil case was how industry researchers used racial categories to frame their investigation of whether subpopulations varied with respect to drug response and metabolism. Thus, the central challenge race poses for killer applications is this: If race is a socially constructed category, then how can biomedical researchers use race to identify which bodies and populations need particular killer applications, or particular doses of killer applications?

These developments are part of what I refer to here as racial pharmacology, or the biomedical study of prescription drugs, their effects, and their metabolism in racially categorized bodies and populations. Clinical pharmacology is the branch of biomedicine that studies the intended and unintended effects of drugs on the body. Clinical pharmacology can be understood as comprising three interconnected fields of

study: pharmacokinetics, pharmacodynamics, and pharmacogenomics. Each of these fields of clinical pharmacology has a racial structure that shapes the research and development of killer applications. Pharmacokinetics studies the biological processes by which bodies absorb, distribute, metabolize, and excrete drugs. Pharmacodynamics studies the effects of drugs on bodies, the mechanisms of drug action, and the relationships between drug concentration and effect. Pharmacogenomics investigates the relationships between drug pharmacokinetics, pharmacodynamics, and genetics.

I argue that the racial pharmacology of killer applications is a central feature of the politics of metabolism. Three interrelated developments comprise the central questions in this emerging field of racial pharmacology. First, as illustrated in the BiDil case, pharmaceutical companies are interested in creating racially circumscribed markets for their killer applications. Because the pharmaceutical industry is part of an economic system that exploits human health as a commodity, constructions of racially categorized risk groups are easily adopted into drug research and marketing strategies that seek to profit from presumed forms of racial difference that are thought to have a meaningful relationship to individual-level differences in drug metabolism. Second, in 2005 the Food and Drug Administration published new guidelines the use of racial classifications in clinical trials advocate that pharmacologists use the Office of Management and Budget (OMB) racial categories in clinical trials in order to study group differences in these metabolic processes that may be related to variability in drug responses (Food and Drug Administration 2005).[78] Third, as I have suggested, drug researchers are concerned that

pharmacokinetic, pharmacodynamic, and pharmacogenomic differences between individuals map onto racial categorizations that organize groups of individuals.

In the following sections, I compare and contrast the racial meanings that emerge from the racial pharmacology of two potential killer applications, antipsychotics and statins, that are both associated with the metabolic syndrome, yet in different ways. Specifically, I compare these two killer applications across four central dimensions of racial pharmacology. First, I ask how scientists use race to study the underlying health conditions that are related to each potential killer application. In the case of antipsychotics, the underlying health condition is schizophrenia and in the case of statins, the underlying condition is high cholesterol. The racial dynamics of each of these conditions is linked to how race is taken up in killer applications research. Second, given the treatment of race in the study of the underlying condition, I ask how race is used to organize clinical trials for these killer applications. How well are African Americans represented in clinical research on these drugs and what are the implications of this participation? Third, I ask how race is used to organize the routes of administration and consumption of these killer applications. Are African Americans underprescribed or overprescribed particular killer applications? How might ideas about race shape these practices? Fourth, I ask how race is deployed to frame questions about group differences in African Americans' drug metabolism. How do assumptions about genetic meanings of racial difference shape the science of drug metabolism? By comparing antipsychotics and statins along these four dimensions, I hope to gain a richer understanding of how race and the metabolic syndrome intersect in the study of killer applications.

Prescribing Antipsychotics: Schizophrenia and the Metabolic Syndrome

In this section, I analyze antipsychotics as a potential killer application that is a site for the descent of the metabolic syndrome and race in clinical pharmacology. Analyzing the side effects of atypicals using the discourse of the metabolic syndrome has become a new focus of schizophrenia drug research. Specifically, in the context of atypicals, the metabolic syndrome has become a way of measuring whether racially categorized bodies require different modes of antipsychotic therapy because of the risks of weight gain and type II diabetes associated with their consumption.

A diagnosis of schizophrenia is traditionally a prerequisite for the prescription of any antipsychotic medicine. And, like the category metabolic syndrome, the diagnostic category "schizophrenia" must be understood in relationship to the knowledge-making practices that have produced psychiatric illness taxonomies since the 1800s. According to the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV), an individual can be classified with schizophrenia if he/she reports the following:

> …a disturbance lasting at least 6 months and […] including two or more of the five symptom groups: (1) delusions; (2) hallucinations; (3) severely disorganized speech; (4) grossly disorganized or catatonic behavior, or (5) negative symptoms (e.g. affective flattening, alogia/poverty of speech, and avolition/inability to initiate and interfere with social and occupational functioning (Bruce 1999).

The contemporary professional psychiatric model understands mental illnesses like schizophrenia as, "a spectrum of syndromes that are classified by clusters of symptoms and behaviors considered clinically meaningful in terms of course, outcome, and response to treatment" (Bruce 1999). Based on this technical and conceptual understanding of mental illness, schizophrenia is a mental illness that defines one percent of the American population—roughly four million people (Keith, Regier, and Rae 1991).

In the 1950s, a biological view of schizophrenia gained prominence before the publication of the Diagnostic and Statistical Manual. The biological model of mental illness maintains that psychiatric symptoms, and the taxonomies they are used to construct, reflect undetected biochemical and genetic processes in the body. In contrast contemporary social constructionist models of mental illness argue that illness categories, like schizophrenia, represent cultural definitions applied to different types of bodies and behaviors, and whose contours and meanings change over time (Foucault 1965; Horowitz 1999).

Beginning in the 1950s, based on a biological view of psychiatric illness, psychiatrists began to treat schizophrenia using powerful new medications called antipsychotics. The first generation, or so-called typical antipsychotics, instantly became the killer applications for schizophrenia. However, typical antipsychotics had a series of undesirable side effects: they caused significant weight gain, elevated risk for developing type II diabetes, and increased cholesterol levels (Remington 2006). The typical antipsychotics also cause a neuromuscular disorder called tardive

dyskinesia, a disorder that causes involuntary movements including tongue thrusting, repetitive chewing, jaw swinging, and facial grimacing.[79]

In the 1990s, pharmaceutical companies began to develop a second-generation of antipsychotics called "atypical" that were supposed to avoid these metabolic and neuromuscular side effects. The six atypicals and their year of FDA approval are: Clozaril® (clozapine) in 1990; Risperdal® (risperdone) in 1994; Zyprexa® (olanzapine) in 1996, Seroquel® (quetiapine) in 1997, Geodon® (ziprasidone) in 2001, and Abilify® (ariprazole) in 2003. Since their introduction in the 1990s, atypical antipsychotics have become the new killer applications for the treatment of schizophrenia and have become a major source of profit for pharmaceutical companies by costing as much as 10 times more than typical antipsychotics (Daumit, Crum, Guallar, Powe, Primm, Steinwachs, and Ford 2003: 121). In 1999, the status of atypicals as killer applications was affirmed when professional psychiatric treatment guidelines were modified to name atypical antipsychotics as "first-line drug therapy" in the treatment of schizophrenia (McEvoy, Scheifler, and Francos 1999).

Atypicals continue to dominate the antipsychotics market, yet they, too, create serious side effects. In November 2003, a biomedical-government-industry collaboration, led by the American Diabetes Association and the American Psychiatric Association, met to discuss the causes and consequences of the observed correlations between atypical therapy and diabetes (Barrett, Blonde, Clement, David, Devlin, Kane, Klein, and Torrey 2004). The conference, titled "Consensus Development Conference on Antipsychotic Drugs and Obesity and Diabetes," brought together representatives from the American Diabetes Association, the

American Psychiatric Association, the American Association of Clinical Endocrinologists, the North American Association for the Study of Obesity, the FDA, and AstraZeneca, Bristol-Myers Squibb, Janssen, Lilly, and Pfizer pharmaceutical companies. A consensus view emerged that psychiatrists should closely monitor their patient's metabolic biomarkers because of the known metabolic side effects of atypicals. The 2003 ADA/APA group also urged researchers to determine whether the risks of therapy are increased in certain ethnic groups (e.g., African Americans) (Barrett et al. 2004: 600). It is in this context that two noted schizophrenia researchers, Wayne Fenton and Mark Chavez, claim the metabolic syndrome is emerging as the tardive dyskinesia of the second-generation antipsychotics (Fenton and Chavez 2006).

In 2004, the FDA issued a warning that atypical antipsychotics increased the risk of developing diabetes on its "news show"—FDA Patient Safety News.[80] The FDA asked drug manufactures of atypicals to add new warnings to their labels informing patients of these risks; they also recommended that patients taking atypicals have their blood sugar levels checked periodically. At the same time in 2004, a group of psychiatrists issued specific recommendations for monitoring the metabolic health of people diagnosed with schizophrenia (Marder, Essock, Miller, Buchanan, Casey, Davis, Kane, Lieberman, Schooler, Covell, Stroup, Weissman, Wirshing, Hall, Pogach, Pi-Sunyer, Bigger, Friedman, Kleinberg, Yevich, Davis, and Shon 2004). For instance, one study found that African Americans may be more likely to gain weight while taking atypicals (Basson, Kinon, Taylor, Szymanski, Gilmore, and Tollefson 2001).

Slow Metabolizers: Treating African Americans With Schizophrenia

Since the rise of professional psychiatry in the nineteenth century, psychiatrists have assumed, asserted, and eventually accepted that African Americans were more likely to suffer from schizophrenia (Adebimpe 2003; Adebimpe 1994; Adebimpe 1981; Keck, Arnold, Collins, Wilson, Fleck, Corey, Amicone, and Adebimpe 2003; Strakowski, Flaum, and Amador 1996). One major reason for this prevailing view about African Americans and schizophrenia had to do with the statistical methods that were widely used to produce knowledge about population rates of mental illness. Prior to the 1980, the treated-case-method was the preferred method for psychiatric epidemiology, which only counted subjects who received inpatient treatment in mental health institutions (Grob 1985). During this period, African Americans comprised a disproportionate portion of those individuals who were institutionalized for schizophrenia, especially during the era of mass institutionalization of the mentally ill, between 1900 and 1940 (Dowdall 1999). Because psychiatrists assumed that African Americans were more likely to have schizophrenia, they institutionalized them at higher rates. Because African Americans were overrepresented among the institutionalized, the treated-case method produced inflated estimates of group illness, which reaffirmed the prevailing view of African Americans' mental inferiority.

By the 1990s, community based studies, like the Epidemiologic Catchment Area (ECA) Study and the National Comorbidity Survey (NCS) were established as the accepted method for determining population rates of mental illness (Agbayani-

Siewart, Takeuchi, and Pangan 2003). These population studies showed that African Americans did not have higher rates of schizophrenia or other affective disorders when compared to Whites, nor were there other significant racial or ethnic group differences in other mental illness (Kessler and Zhao 1999). Nonetheless, these epidemiological studies used the diagnostic categories for schizophrenia provided by the first edition of the DSM, which was published in 1980.

Despite the fact that racist ideas about the prevalence of schizophrenia among African Americans had been successfully challenged, the emerging field of racial pharmacology created new problems and new questions. Beginning in the 1980s, scholars began to study group differences in access to and use of antipsychotics as a drug class. In the 1980s and 1990s, researchers found that psychiatrists prescribed typicals at higher rates and in doses to African Americans in both inpatient and outpatient psychiatric settings (Chung, Mahler, and Kakuma 1995; Rudorfer and Robins 1982). More recently, scholars have documented group differences in the route of administration of antipsychotics: African Americans categorized with schizophrenia are *more likely* to receive atypicals via injection as opposed to pill therapy (Kuno and Rothbard 2002; Segel, Bola, and Watson 1996; Walkup, McAlpine, Olfson, Labay, Boyer, and Hansell 2000; Woods, Sullivan, Neuse, Diaz, Baker, Madonick, Griffith, and Steiner 2003). Several groups of scholars have documented that African Americans with schizophrenia are *less likely* to receive atypicals (Daumit et al. 2003; Herbeck, West, Ruditis, Duffy, Fitek, Bell, and Snowden 2004; Mark, Dirani, Slade, and Russo 2002; Wang, West, Tanielian, and Pincus 2000).

Researchers also documented patterns between African Americans' access to and use of atypicals compared to other racially categorized groups. A 2001 Surgeon General Report titled "Mental Health: Culture, Race, and Ethnicity" included a listing of the representation of racially categorized groups in twenty-five randomized controlled trials for the treatment of schizophrenia that took place between 1986 and 1996. While sixteen of the twenty-five studies collected and reported data on the race and/or ethnicity of their research subjects, none of these conducted (or at least reported) analyses by race or ethnicity. The remaining nine studies did not collect information on the race or ethnicity of research subjects, and did not conduct analyses by race or ethnicity (DHHS 2001).[81] In other words, there is a lack of statistical information about the safety and efficacy of antipsychotics in African Americans.

The 2001 Surgeon General's report on Mental Health and Race also advanced a biological explanation of the different pharmacokinetics, or the processes by which bodies absorb, distribute, metabolize, and excrete drugs, of antipsychotics in African Americans: more of them are "slow metabolizers." Citing a 1977 study (Ziegler and Briggs 1977), a 1982 study (Rudorfer and Robins 1982), and a 1998 study (Bradford, Gaedigk, and Leeder 1998), the report claims "a greater percentage of African Americans than whites metabolize some antidepressants and antipsychotic medications slowly and might be more sensitive than whites" (DHHS 2001: Chp. 3). They offer two contrasting arguments about the clinical significance of race in the pharmacological treatment of schizophrenia. On one hand, they argue "*biological similarities* between African Americans and whites are such that effective medications are suitable for treating mental illness in both groups." On the other

hand, they cite recent that suggests that "African Americans and white Americans *sometimes* have different *dosage* needs" [emphasis added] (DHHS 2001)

The Surgeon General's report mentions the P450 system as a possible genetic source of observed racial pharmacokinetic differences among schizophrenic patients taking atypicals. Relling and colleagues found racial differences between "American black and white subjects" in debrisoquin hydroxylase (P450IID6) activity, a biochemical and genetic process that is implicated in drug metabolism (Relling, Cherrie, Schell, Petros, Meyer, and Evans 1991). Walkup and colleagues (2000) explain the slow metabolizer theory of racial group difference in the P450 system in the following manner:

> Drug metabolism is mediated through the cytochrome P450 micosomal enzyme system. Small numbers of individuals lack the P450 microsomal enzyme and, consequently, are "poor metabolizers." Their plasma levels tend to be high. Recent studies have identified a larger group who are genotypically heterogeneous "slow metabolizers." Recent estimates suggest that the prevalence of slow metabolizers of antipsychotic medications is higher among African Americans and Asian groups than whites (Walkup et al. 2000: 346).

In their conclusion, the authors insinuate, it is logically possible that *unmeasured physical differences* in pharmacokinetics might be responsible for differences in the metabolism of antipsychotics between white and African American or Hispanic individuals (Walkup et al. 2000: 346).

Selling Statins: Cholesterol and the Metabolic Syndrome

In this section, I analyze statins as a second potential killer application that is a site for the descent of the metabolic syndrome and race. Statins have become the killer application for the treatment of cholesterol problems (dyslipidemias), a central component of the metabolic syndrome. Statins are a class of cholesterol drugs that were first approved for sale in the United States in 1986 and went on the market in 1987 (Junod 2007).[82] As of 2004, there were seven statins available in the United States for the treatment of dyslipidemia (Gotto 2004).[83]

The first statin approved by the FDA in 1987, lovastatin, introduced the practice of treating "surrogates" into the FDA drug approval process and private drug research and development (Greene 2007). A surrogate is a biological marker that is transformed into a statistical stand-in for a hypothesized disease process or outcome. So, LDL cholesterol is a surrogate for the development and growth of plaque in the arteries and in the heart.[84] Rather than needing to demonstrate that lovastatin reduced the incidence of heart attacks or strokes in a long-term prospective clinical trials, the investigators only needed to show that the drug agent effected the surrogate in expected (and desirable) ways in order to gain FDA approval (Junod 2007). The pharmacological treatment of cholesterol as a means of reducing heart disease risk is an outgrowth of the so-called "lipid hypothesis," namely, that lowering LDL cholesterol alone will stop or slow the development of heart disease.

Formal clinical guidelines for the pharmacological management of cholesterol identify statins are used to determine the adequacy and equitability of patient care. As I discussed in chapter three, the 2001 guidelines issued by the National Cholesterol

Education Program stand as the expert recommendations for the management of cholesterol (Ito, Cheung, Gupta, Birtcher, Chong, Bianco, and Bleske 2006; NCEP 2001). Given the stringent nature of these recommendations, many individuals with dyslipidemia will not be able to achieve optimal LDL cholesterol levels without pharmacological therapies, even with adequate exercise and changes in dietary practices (Grundy et al. 2004)

Race-Based Therapies: Treating African Americans with High Cholesterol

Crestor™ is one of the newest statins made by the AstraZeneca that lowers LDL cholesterol and raises HDL cholesterol. Just one year after its FDA approval in 2003, 15 million individuals filled prescriptions for Crestor™, spending $908 million dollars. Crestor™ (rouvastastin) is a member of a class of drugs called statins that are prescribed primarily to treat forms of hyperlipidemia (they are prescribed to people who have high LDL cholesterol and low HDL cholesterol, two criteria of the syndrome). In August 2008, AstraZeneca began marketing Crestor™ a new clinical finding that, along with diet and exercise, it can slow the progression of atherosclerosis.

Cholesterol researchers use the metabolic syndrome and race as ways to identify which bodies and populations are most likely to benefit from statin therapy. Are the makers of Crestor, a new statin, framing the racial pharmacology of statins in order to be able to market them to racially categorized groups? In 2003, the pharmaceutical giant AstraZeneca started the Galaxy Programme™ Studies, which according to their website, is a "large, comprehensive, long-term and evolving

research initiative designed to address unanswered questions in statin research and to investigate the impact of Crestor™ on cardiovascular risk reduction and patient outcomes."[85] The Galaxy Programme™ funded three six-week randomized, controlled, open label, multi-center clinical trials were designed to evaluate the safety and efficacy of Crestor™ in populations with hyperlipidemia—populations that were sampled using racial and ethnic categories.[86]

To organize these trials, AstraZeneca followed the FDA's guidelines for including racially categorized groups in its clinical trials; however, they divided the groups into single race trials. For example, the African American Rosuvastatin Investigation of Efficacy and Safety (AIRES) trial is a randomized, controlled, open-label, multi-center trial designed to evaluate the efficacy of Crestor® in 774 African American subjects by comparing it to Lipitor® (Ferdinand et al. 2006).[87] In this study, Dr. Luther T. Clark and his colleagues evaluated so-called ethnic differences in the achievement of cholesterol treatment goals in a sample of African Americans and non-Hispanic Whites. The rationale for the study is that racial and ethnic differences in cholesterol management may partially account for the excess risk and mortality experienced by racial and ethnic minority groups. The study's cholesterol treatment goals were based on the 2001 National Cholesterol Education Program (NCEP) Adult Treatment Panel III definitions for dyslipidemia, the same risk category criteria that are included in the metabolic syndrome. Subjects were classified into risk categories based on the NCEP criteria.

The ARIES patient sample consisted of non-Hispanic Whites and African Americans, although the methods of determining the subjects' ethnicities are omitted

from the description of the study's methodology.[88] The investigators found that body mass index (BMI), total cholesterol, and LDL cholesterol were higher in African American subjects (which confers higher disease risk), but HDL and triglycerides biomarkers were better (which confers lower disease risk). African Americans were significantly less likely to meet ATP III LDL-C treatment goals within each risk category and overall. African Americans were less likely to be taking statins than whites (75.7% versus 70.6%) and less likely to be taking high-efficacy statins (54.8% versus 45.6%).[89] Even among subjects taking statins and high efficacy statins, fewer African Americans reached ATP-III targets for LDL-C.

According to the researchers, the explanations for the alleged ethnic disparity in cholesterol management were "not immediately apparent" (Clark et al. 2006: 324). The disparity cannot be explained by differences in dyslipidemia diagnoses or access to health care because both the African Americans and non-Hispanic Whites in the sample were recruited because they were receiving treatment for dyslipidemia. Fewer African American subjects were taking statins and received treatment from lipid specialists less frequently. Therefore, less aggressive treatment on the part of treating physicians may partially explain the disparity. However, the statistical association between ethnicity and cholesterol goal achievement remained after controlling for these differences.

Group differences in rates of drug compliance may also account for these differences in cholesterol management. The authors cite three studies published since 2000 that suggest that African Americans are less compliant with statin drug therapy than are non-Hispanic Whites (Charles, Good, Hanusa, Chang, and Whittle 2003;

Chong, Tzallas-Pontikes, Seeger, and Stamos 2000; Williams, Morris, Ahmad, Yousseff, Li, and Ertel 2002). The authors conclude, "It is likely that the explanation for lower frequencies of treatment goal achievement among African American patients for lipids and other therapies is multifactorial" (Clark et al. 2006: 324). The authors cite socioeconomic status, educational level, and type of medical and prescription drug coverage as the multiple other factors affecting goal achievement, but their data did not permit the analysis of these other factors.

Clark and colleagues (2006) cited evidence that serum lipid (cholesterol) responses to lifestyle modifications and drug therapies are "generally similar" in African American and non-Hispanic White subjects (Clark, Maki, Galant, Maron, Pearson, and Davidson 2006). However, three of the four studies that they cite analyzed samples that only consisted of African Americans, including the ARIES trial, and thus could not have compared racial and ethnic groups (Ferdinand et al. 2006; Jacobson, Chin, Curry, Miller, Papademetriou, Schlant, and Larosa 1995; LaRosa, Applegate, Crouse, Hunninghake, Grimm, Knopp, Eckfeldt, Davis, and Gordon 1994; Prisant, Downton, Watkins, Schnaper, Bradford, Chremos, and Langendorfer 1996). In the conclusion of the paper, the authors cite these same four studies again and state that data from clinical trials of lipid-lowering drug therapies suggest that African American and non-Hispanic White subjects exhibit similar physiological responses (Clark et al 2006: 324). Because the effects of lifestyle intervention and drug therapy do not vary across African American and non-White Hispanic populations, the authors hypothesize that the lower rates of cholesterol management among African Americans must result from a) less aggressive

management by treating physicians, b) suboptimal compliance by African American patients, or c) some combination of these factors.

In a 2007 article titled "Metabolic Syndrome in African Americans: Implications for Preventing Coronary Heart Disease," Drs. Luther T. Clark and Fadi El-Atat review several therapeutic approaches to metabolic syndrome in African Americans. While the magnitude of LDL-C reduction with statins appears to be similar in blacks and whites, the authors cite data from the Antihypertensive and Lipid Lowering Treatment to Prevent Heart Attack Trial (ALLHAT-LLT) study showed that statin therapy lowered the risk of CHD death and non-fatal heart attacks more in black than in non-black subjects, but did not decrease the mortality gap overall between the two groups (Clark and El-Atat 2007).

In 2008, Dr. Karol E. Watson, an Associate Professor at the Geffen School of Medicine at UCLA and Director of the UCLA Center for Cholesterol and Lipid Management, published a review article in the Journal of the National Medical Association entitled "Cardiovascular Risk Reduction among African Americans: A Call to Action" (Watson 2008). As a member of the speaker's bureau for AstraZeneca, Merck, Schering-Plough, and Sanofi Aventis, Dr. Watson reviews evidence that the major risk factors for cardiovascular disease require special study and intervention in the African American population. She argues that while data on racial and ethnic differences in response to lipid-lowering drugs are limited, two studies have shown that statins are not as effective at lowering African Americans' LDL levels compared to European Americans (ALLHAT 2002; Simon, Lin, Hulley, Blanche, Waters, Shiboski, Rotter, Nickerson, Yang, Saad, and Krauss 2006)

Towards the end of her review article, Dr. Watson has a subsection titled "Race-Based Therapeutics," which is in quotation marks. The section begins "The development and use of so-called "Race-based therapeutics" remains controversial. Results of some clinical trials indicate that racial/ethnic differences in vascular function may have implications for the treatment of CVD risk factors" (Watson 2008: 22). Here, she makes the argument that African Americans may have a different endothelial response to ACE inhibition that European Americans (see also (Ferdinand 2007); and

> Individual response to the pleiotropic effects of statins, such as their beneficial effects on renal function independent of lipid lowering, may also be affected by race. In one study of short-term rosuvastatin treatment, estimated glomerular filtration rate increased by >3-fold in African American patients compared with the overall study population (Watson 2008: 24).

To substantiate this second claim, she cites the study (Vidt, Harris, McTaggart, Ditmarsch, Sager, and Sorof 2006). She then argues that "the fact that African Americans and European Americans appear to exhibit differences in endothelial and vessel wall response suggests that alternative strategies may be needed to customize therapy appropriately for patients of different races/ethnicities" (Watson 2008: 24). For Watson, the case of BiDil serves as an exemplar of these alternative strategies for race-based therapeutics. She concludes this section by rejoining that BiDil may work equally well in other racial/ethnic groups and that more research is needed in this area (Watson 2008: 24).

Conclusion

The convergence of racial pharmacology, killer applications, and the metabolic syndrome created an important synergy that is the focus of this chapter. The racial pharmacology of killer applications has several distinguishing features in the politics of metabolism. Table 4.1 presents a summary of the social processes and institutional relationships that comprise the racial pharmacology of killer applications.

[Insert Table 4.1. Summary of social processes and intuitional relationships in the racial pharmacology of killer applications]

The analysis presented in this chapter suggests that racial pharmacology, the biomedical study of prescription drugs, their effects, and their metabolism in racially categorized bodies and populations, is an emerging site for the production of racial meaning. First, pharmaceutical companies and drug researchers use constructions of race are used to organize clinical trials, the study of killer applications, and the genetics of drug metabolism. In the case of antipsychotics, researchers use the metabolic syndrome as a discourse about the undesirable side effects of killer applications like antipsychotics. African Americans have historically received differential diagnoses of schizophrenia, have been under represented in clinical trials, are over prescribed antipsychotic injection therapies, and are said to differ genetically from other groups in terms of antipsychotic metabolism. Because the treatments for

schizophrenia have become racialized, the diagnostic category of schizophrenia becomes racialized through the deployment of killer applications.

In contrast, in the case of statins, researchers use the metabolic syndrome as a discourse about potentially broader uses of existing killer applications like statins. African Americans are constructed as having differential rates of high cholesterol, are the primary subjects in new clinical trials, are under prescribed the most effective statin therapies, and are said to differ genetically from other groups in terms of statin metabolism. In order to justify race-based treatments, high cholesterol is being framed as a new racial disparity that requires new studies in drug efficacy and safety.

Second, race and the metabolic syndrome intersect *in different ways* in the racial pharmacology of antipsychotics and statins that matter differently for African Americans. African American bodies are deployed in unique ways in the racial pharmacology of antipsychotics and statins. Yet, in both cases, assumptions about unobserved genetic differences across racially categorized groups shape the racial pharmacology of killer applications. Drug scientists are using the metabolic syndrome to study the interactions Black bodies and drugs, and are seeking to produce pharmacological knowledge that can be translated into profits for drug companies.

Table 4.1. Summary of social processes and institutional relationships in the racial pharmacology of killer applications.

The Politics of Metabolism	***Killer applications***	***Antipsychotics***	***Statins***
Killer applications	-----	Killer applications for schizophrenia	Killer applications for high cholesterol
Construction of racial meaning—racial pharmacology	Use of race to study underlying health conditions and construct drug markets Use of race to organize clinical trials Use of race to study consumption of drugs Use of race to study genetics of drug metabolism	Schizophrenia as racialized mental illness African Americans underrepresented in clinical trials African Americans overprescribed injection vs. pill therapy Genetics as source of racial differences in antipsychotic metabolism	High cholesterol as new kind of racial health disparity New clinical trials focused on African Americans African Americans underprescribed statins Genetics as source of racial differences in statin metabolism
Construction of metabolic syndrome	Use of metabolic syndrome to develop new drugs	Use of metabolic syndrome to study side effects of antipsychotics	Use of metabolic syndrome to broaden the applications of statins

Chapter 5: The Politics of Metabolism

In the previous two chapters, I explored how the metabolic syndrome and race emerged as features of biopower by tracing the social processes and institutional relationships that are involved in the production of new racial meanings. In this concluding chapter, I provide the central interpretations, implications, and significance of this study. First, I summarize the main interpretations of this study in terms of how they might contribute to a more robust understanding of how race shapes the politics of metabolism. Second, I discuss the implications of this study for the critical social theory and for the theoretical frameworks that shaped this study. Third, I discuss the sociological significance of the metabolic syndrome in the broader struggle for social justice.

Understanding the Politics of Metabolism

In chapter one, I asked what are the implications of this emerging relationship between the metabolic syndrome and race for developing a more robust understanding of race in the politics of metabolism? The sociological relationships between the metabolic syndrome and race in the United States seem to have emerged at the intersection of scientific racism—a set of scientific discourses and practices that served to ignore, explain away, and/or justify racial inequalities—and the practices of an increasingly biological and technological approach to the study of human metabolism. Because the metabolic syndrome emerged in 20th century American biomedicine, it was inexorably shaped by the social structures of race and racism.

Here, I develop two central interpretations that emerge from this study that can inform a richer understanding of the politics of metabolism. First, understanding the politics of metabolism requires that we shift our thinking from an epidemic perspective to one that embraces an endemic perspective of metabolic health problems. Second, developing a more robust understanding of the politics of metabolism also involves further analysis of the biomedical-government-industry collaborations that lie at the center of biomedical knowledge production in the United States. Taken together, these interpretations underscore both the importance of ideas and institutional practices in the politics of metabolism.

Shifting from an epidemic to an endemic perspective

Developing a more robust understanding the metabolic syndrome and race in the politics of metabolism requires that we shift our thinking from an *epidemic* perspective to one that embraces an *endemic* view of metabolic health problems. Recent public scientific discourse about the metabolic problems that comprise the metabolic syndrome refers to each of them as epidemics in their own right (Grundy 2008; Kereiakes and Willerson 2003; Zimmet, Alberti, and Shaw 2001). While it is true that most Americans will most likely experience, and/or die from one or more metabolic problems over the course of their lives, these conditions are not epidemics in the historical meanings of the term. In this historical context, epidemics killed nearly every individual living within a circumscribed geographic region both quickly and indiscriminately. Therefore, the historical response to controlling and eradicating epidemics has been to rapidly target individuals who are most likely to fall within the epidemic's reach.

In stark contrast, the politics of metabolism are characterized by endemic problems. According to Foucault, endemics are discriminating, widespread, and long-term population-level phenomena that weaken societies' energy because treating them is expensive and they lead to the decreased economic productivity of working populations (Foucault 2003 [1976]: 244). Because endemics represent both a political problem for those who govern, endemic problems quickly become objects of scientific knowledge and commodification. In the context of biopower designed to make the population more productive and healthier, this problematization of endemic phenomenon is required to excise as much scientific discipline, economic profit, and political utility from populations as possible. According to data published by leading authorities, the direct and indirect healthcare costs from heart disease, diabetes, and stroke exceed one trillion dollars per year (ADA 2003; Finkelstein, Ruhm, and Kosa 2005; Thom 2006).[90]

Shifting our perspective from an epidemic to endemic view is critical for understanding the how the biological realities, political rationalities, and economic opportunities of the politics of metabolism shaped the emergence and descent of the metabolic syndrome. While the metabolic syndrome may not exist as a biological reality in precisely the same ways that cancers exist, the metabolic syndrome emerged in the context of a massive biomedical, government, and corporate response to the endemic problems of metabolism.

Analyzing biomedical-government-industry collaborations

Developing a more robust understanding of the politics of metabolism also involves further analysis of the biomedical-government-industry collaborations that lie at the center of biomedical knowledge production in the United States. Throughout this study, I showed how biomedical-government-industry collaborations on the metabolic syndrome shape the production of racial meanings. The discourses, technologies, and practices of these social institutions are the tools with which researchers construct the metabolic syndrome and race in the contemporary United States. Here, I briefly highlight how these discourses, technologies, and practices are linked across biomedical, government, and corporate contexts.

First, biomedicine is a term that denotes the centrality of a biological approach to the ideas and practices that comprise contemporary medicine. Biomedical researchers and institutions combine new forms of molecularization and risk assessment surveillance to produce the construction of the metabolic syndrome. Whether the metabolic syndrome construct will be widely adopted by practicing physicians to diagnose patients is not clear. However, the inclusion of a diagnostic code for the metabolic syndrome in the International Classification of Disease certainly signals that physicians and health care institutions would be operating within accepted guidelines if they started classifying patients using this new category. Also not clear is what a diagnosis of the metabolic syndrome would actually mean for patients, physicians, and the practice of medicine. What is clear is that the metabolic syndrome has the potential to revolutionize the way that biomedicine conceptualizes

and investigates metabolic health problems. As the biomedical discourses and practices of the metabolic syndrome continue to unfold, they intersect with the ways in which race shapes the theories and practices of medicine in terms of disease surveillance, diagnosis, and treatment.

Second, the federal government plays several important roles in the production of the metabolic syndrome and race. The federal government enforces the racial categorizations used in biomedical research on the metabolic syndrome, funds and produces research on the metabolic syndrome, and regulates the labeling and safety of prescription drugs related to the metabolic syndrome. In chapters three and four, I analyzed the institutional practices of government that are central to the production of discourses about the metabolic syndrome and race. For example, in chapter three, I documented the moments when the government institutionalized the measurement of the metabolic processes of Americans at the molecular level. As these new technologies were incorporated into the routine practices of epidemiology, they became integral to the establishment of the risk factor paradigm that operates today.

Third, the relationships between the metabolic syndrome and race cannot be understood without analyzing the pharmecutical industry as a central actor in the politics of metabolism. The metabolic syndrome and race are used to study and target killer applications produce toward racially categorized individuals and groups. In chapter four, I examined how government racial categories are used to structure killer applications research on racial and ethnic groups. I showed how research on

antipsychotics and statins has taken place in a commercial context where administrative racial categories are invested with pharmacological uses and meanings.

Implications for Critical Social Theory and Social Justice

In chapter two, I outlined the core concepts from three critical social theories that shaped the questions and analysis in this study. Critical social theories can provide several unique insights as to how the metabolic syndrome and race might operate together in the politics of metabolism. Patricia Hill Collins defines critical social theory as bodies of knowledge and sets of institutional practices that actively grapple with the central questions facing groups of people differently placed in specific political, social, and historical contexts characterized by injustice (Collins 2005). Critical social theories are committed to producing ideas and engaging in practices that serve the interests of social justice. In the context of this study, this commitment to a social justice context involves linking an analysis of institutional racism and racial health disparities to the analysis of racial meaning in biomedical constructions of health. As a way of framing the contribution of this study for critical social theory, in this section I explore the implications of this study for these ideas and practices.

What are the implications for critical race theory?

The metabolic syndrome appears to be a new site of racial formation, the social and historical process by which racial categories are created and invested with

meaning. As I argued in chapter two, critical race theory has long recognized the centrality of science and medicine to the construction of racial concepts and meanings that in turn influence the practices of social institutions. In other words, the metabolic syndrome is an emerging site for the reproduction of race and racism in American society.

Drawing on this critical race framework, I have argued that the metabolic syndrome represented a new way that biomedical researchers could construct scientific knowledge about racial difference. In chapter three, I documented how the metabolic syndrome became a racial project, an unfolding process that drew upon different racial meanings to make sense of human metabolic difference, and simultaneously used race to classify bodies and populations. Based upon this analysis, I believe that the metabolic syndrome seems to draw upon earlier formations of race that link racial inequality to the essential properties of purportedly biologically and genetically meaningful groups.[91] As I argued in chapters three and four many of the biomedical theories of metabolic differences across racially categorized populations can rely on assumptions about color-coded predispositions, susceptibilities, and genetic admixture.[92]

These practices and theories are reminiscent of historical formations of scientific racism that explained racial inequalities as biological, natural, and immutable. Racial health disparities have long constituted a major site of struggle over the meaning of race and explanations of racial inequality in the United States. When analyzed in the specific context of racial health disparities in the United States, using essentialist notions of race to explain away racial inequality takes on special

significance in the history of comparative racial biology and eugenics. Racial essentialism is central to the operation of scientific racism, a set of discourses and practices that served to explain and justify racial inequalities using the tools and authority of science. For example, critical race theorist Tukufu Zuberi analyzed how the field of biometrics, an 19th century science that applies the methods of social statistics to biological problems, was used to justify the practices of the American and European eugenics movements (Duster 2003 [1990]; Zuberi 2001).

More recently, other critical race theorists have described emerging developments in biomedicine and genomics as "the biological reification" or "biological rewriting" of race (Duster 2005; Fausto-Sterling 2004; Gannett 2004; Thompson 2006b).[93] For example, Troy Duster argues racial categories are increasingly interpreted through the new genetic prism, a phrase he uses to describe the increasing centrality of genetics and genomics as the primary lens for understanding racial differences in so-called multifactorial diseases. Duster argues that social, economic, and political interests profoundly influence the production of scientific knowledge about race. While biomedical researchers theorize that multifactorial diseases, like heart disease, are caused both environmental and genetic factors, through the new genetic prism, individual differences in disease are often explained as the consequence of inherent population-based genetic susceptibilities (Duster 2005: 1050). Thus, understanding the relationships between sociopolitical processes, knowledge-making practices, and racial categorization is fundamental to understanding what remains contested and problematic about the concept of race in science (Duster 2003b).

Duster also reminds us that the first principle of knowledge construction is which question gets asked first in the research enterprise. In the context of race and pharmaceuticals, a priori assumptions about biological differences between racially classified populations shape the kinds of questions that biomedical researchers ask about race. Study samples are treated as populations in a narrow sense of the term, even when there is little evidence that they represent a geographically localized, reproductively isolated group. These narrowly and scientifically defined populations are then analyzed in terms of existing racial taxonomies. Alternatively, Duster argues that biomedical researchers should treat race as "a stratifying practice in societies that can lead to different frequencies of alleles in different modern populations but also to different access to health-related resources" (Duster 2005:1050). So, not only do questions about racial difference create a context where disparities themselves are used as marketing tools, but they also may unwittingly reinforce biological and genetic explanations of racial inequality.

However, perhaps in part due to prevailing assumptions within critical race theories that familiar formations of scientific racism had been discredited, critical race theorists have been slow to recognize the productive power of biotechnologies to transform race in the contemporary moment. While scholars like Troy Duster have been talking about the importance of biotechnologies for racial formation since 1990, this study makes a meaningful contribution to critical race theory by demonstrating two important ways that race and biotechnology intersect through the lens of the metabolic syndrome. Specifically, I investigated how biotechnological developments in measuring metabolic processes and manufacturing prescription drugs became

racialized in the discourses on the metabolic syndrome. Thus, new intersections of race and technology will require sustained attention from racial theorists going forward.

What are the implications for biomedicalization?

The framework of biomedicalization provides a set of powerful analytic tools through which to analyze the relationships of the metabolic syndrome and race. The metabolic syndrome was also forged in the context of biomedicalization, which encompassed an increasingly biological and technological approach to the study of human metabolism. Technoscience provides a way of understanding how the increasingly technological and scientific aspects of the metabolic syndrome come together in politics of metabolism. Thus, in chapter three, I showed how the emergence of the metabolic syndrome was made possible through the technoscientific integration of molecular approach in clinical medicine and a risk factor approach in epidemiology. This integration led to a new emphasis on the production of knowledge about disease risk and a new practice of targeting allegedly at risk populations for behavioral, pharmacological and social interventions. Taken together, these practices resulted in the construction of raced bodies as inherently and always at risk and the very constitution of so-called at risk populations as always explicitly or implicitly racial.

In a 2005 article published in the journal *Science*, sociologist Troy Duster cautions biomedical researchers of race against committing the fallacy of reification—the tendency to assume that our categories of thought coincide with the

obdurate character of the empirical world (Duster 2005). Duster questions how NitroMed, the producer of BiDil, presented statistical information about racial disparities in hypertension in ways that misrepresented the extent and etiology of the racial gap in the prevalence of hypertension between African Americans and Whites. This misrepresentation in the case of BiDil (and in the case of new forms of genetic criminology) comprises the reification of race by arguing that different racial groups have "genetically sufficiently distinctive features…which are used to explain health disparities between racially categorized populations" (Duster 2005: 1050). As I discussed in chapter two, these types of essentialist discourses about race historically established an important justification for scientific racisms.

In another recent article, philosopher of race Lisa Gannett asks whether federally created and self-reported race, ethnicity, and ancestry are good proxies for genetic similarities in drug metabolism (Gannett 2005). She argues that the debate about the use of racial categories in clinical trials and pharmacological research has often been framed in terms of realist versus social constructionist theories of race. The realist theory of race claims that our racial classifying practices identify things in nature, and that race is therefore a scientific and objective category. The social constructionist theory of race asserts that race is not a genuine natural category, but an invention of racialist/racists societies, hence subjective. Gannett steps through this debate by noting that this framing of the epistemological status of race assumes that boundaries can be inserted between "the social and scientific, the cultural and natural, and what is objective and subjective" (1235). According to Gannett, a priori racial

taxonomies of human groups, such as those used in pharmacological research, do not exist independently of social classifying practices in specific research contexts.

To analyze these emerging practices, Gannett advances a pragmatist epistemological framework that includes a normative ethical analysis of the use of group categories in pharmacogenomics research. What this means is that scholars need to ask questions about the potential harms enacted upon racially categorized groups if the very categories designed to track and rectify the effects of systemic racism become biologized through their incorporation in scientific research, clinical practice, and the marketing of pharmaceuticals. Rather, attention to health-related group differences need not perpetuate the racist history that has seen some communities shoulder a disproportionate share of the burdens associated with biomedical research while reaping fewer of the benefits.

In chapter four, I analyzed how biomedical researchers use the metabolic syndrome and race to target different population groups in killer applications research. I argued that the search for new killer applications has led pharmaceutical corporations to take greater advantage of the infrastructure for racialization in drug research and development.

What are the implications for biopower?

What can this study contribute to the ongoing evaluation of Foucault's ideas about biopower to analyze contemporary social arrangements of knowledge and power? In 2003, science and technology studies scholars Paul Rabinow and Nikolas Rose called for more studies that use biopower, which they believe is profoundly

relevant to contemporary social arrangements yet remains analytically underdeveloped (Rabinow and Rose 2003: 34). A growing handful of scholars are using Foucault's thinking on race in the context of biopower to understand racial formation and racism across different historical periods and social contexts.[94] Since 2000, scholars the science and technology studies have adopted and extended biopower to analyze the contemporary dynamics of biomedicalization in the life and human sciences.[95]

Biopower provides a way to think about how scientific disciplines like demography and epidemiology, and emerging biomedical specialties like cardiology and endocrinology, combined with government regulations on race and prescription drugs, to create a racial context through which the metabolic syndrome could emerge. The relations of biopower that encapsulate both the metabolic syndrome and race discipline bodies and regulate populations so that they can be more easily targeted for biomedical research, political utility, and economic exploitation.

In chapter three, I analyzed how ideas about race shaped the emergence of the metabolic syndrome in terms of the relationships between bodies and populations. In chapter four, I examined how race and the metabolic syndrome operate together to establish a political, economic, and scientific context in which racial groups are targeted in killer applications research. I argued that the metabolic syndrome is a new site where racial pharmacology shapes the deployment of killer applications in the politics of metabolism.

The Sociological Significance of the Metabolic Syndrome

Are we forever doomed to the politics of metabolism? How might this study highlight ways to navigate and shift the politics of metabolism in ways that foster human health and undermine entrenched inequalities and the institutional practices that create them? To conclude, I outline three contexts that I could not explore in depth in this study, but that highlight the broader sociological significance of the metabolic syndrome.

Context of racial health disparities

The first context for the significance of the metabolic syndrome is the use of the metabolic syndrome to represent and explain racial health disparities. The scope and impact of chronic metabolic conditions has intensified in the United States, especially among America's racial and ethnic minority groups. Recent data from the Centers for Disease Control and Prevention (CDC), documents substantial and persistent racial disparities in the distribution of and complications from these major chronic metabolic conditions (CDC 2005). Based on government data[96], 27% of whites, 42% of African Americans, and 27% of Mexican Americans have hypertension or are currently taking anti-hypertensive medication

This study suggests that the metabolic syndrome will continue to serve as a new way of representing and explaining racial inequalities in the politics of metabolism. For decades social epidemiologists have documented substantial group disparities in the distribution of and complications from the major chronic metabolic health conditions that comprise the metabolic syndrome between America's racial and ethnic groups (House and Williams 1996; Krieger 2004; Williams and Collins

1995). This research on racial health disparities reveals that African Americans and other racially categorized minority groups experience higher rates of death due to chronic metabolic diseases (Benjamin, Arnett, and Loscalzo 2005; Mokdad, Marks, Stroup, and Gerberding 2004; Zhang and Wang 2004) and higher rates of complications due for those diseases (Kington and Smith 1997), in large part due to the interactive dynamics of racism and social class on health (Clark, Anderson, Clark, and Williams 1999; Dressler 1993; Hayward, Crimmins, Miles, and Yang 2000; House 2001; House and Williams 1996; Krieger 1987; Krieger and Sidney 1998; Link and Phelan 1995; Marmot 2003; Smith and Hart 2002; Smith 1998; Williams 1990; Williams and Collins 1995).

This body of literature on racial health disparities has received less attention in terms of making a major theoretical contribution to critical race theory, science and technology studies, or political sociology, and instead has been more embraced in the fields of social epidemiology and public health. At its core, this research challenges the notion that racial health disparities are caused by natural and/or cultural differences between racially categorized groups. These scholars have long argued that racial health disparities result from group-based inequalities in access to the economic and political resources necessary to maintain and improve health, like having access to affordable and adequate medical care.

However, simply paying more attention to racially encoded health disparities in the context of the metabolic syndrome will not be enough. Currently, scientific comparisons of racially categorized groups in the metabolic syndrome and its correlates have become a veritable cottage industry. Nobly, metabolic syndrome

analysts often carry out their work with the purpose of devising better biomedical explanations for health disparities in heart disease, diabetes, and stroke. Yet, the dubious theories of racial inequality and essentialist discourses of race that emerge from metabolic syndrome research on racial and ethnic groups have not been adequately addressed in the research literature on the metabolic syndrome.

Context of colorblind racism

A second important context for assessing the sociological significance of the metabolic syndrome is that of colorblind racism. Colorblind racism is a new racial ideology that aims to explain racial inequality with reference to non-racial dynamics (Bonilla-Silva 2003; Brown et al. 2003; Guiner and Torres 2002). Colorblindness is an ideology whose effect is to obscure the material practices of racism and racial structure. Colorblind racism comprises four distinctive non-racial frames that social actors use to account for the effects of racially coded inequalities: liberal individualism, cultural racism, and minimization of racism, and naturalization (Bonilla-Silva 2003). Because the metabolic syndrome is constructed out of physical and biochemical markers, it is routinely interpreted as non-racial. However, this study has shown how colorblind racism operates in the politics of metabolism by obscuring the multiple ways in which this ostensibly non-racial syndrome is, in fact, racialized.

The discourse of the metabolic syndrome seems to draw upon each of these colorblind frameworks in specific ways. First, liberal individualism is a political philosophy that emphasizes the rational actions of autonomous individuals acting in a

free-market economic system. In terms of health, liberal individualism the stock stories (Guiner and Torres 2002) through which the role of individual responsibility is overemphasized in explaining group rates of chronic disease. The millions of dollars spent on public health interventions in communities of color reflects the belief that causes of and solutions to racism in health is in people of color themselves (Guiner and Torres 2002). The proliferation of techniques of examination and surveillance and the construction of risk-based syndromes for marking bodies becomes comprehensible only within this dominant ideological framework that views health an individual moral responsibility (Novas and Rose 2000).

Second, cultural racism refers to the explanations of racial health disparities that reference to the "lifestyles" and corrupted cultures of racially categorized groups, without any reference to the broader systems of institutional power that structure access to health or opportunity (Satel 2000). "Lifestyle" here explicitly refers to questions around individuals or groups' patterns of daily life in terms of diet, nutrition, and exercise (Tesh 1988). Third, minimization of racism is a frame that suggests that racism is no longer a reality that can impact the life chances of racially categorized minority groups. While it is nearly impossible to maintain that racial health disparities do not exist, the metabolic syndrome can be constructed in ways statistically that seem to *design* racial health disparities in such a way as to minimize them wholesale. As I described in chapter three, different definitions of the metabolic syndrome can produce different information about the prevalence of the metabolic syndrome in racially categorized groups. It is possible, therefore, for

scientists to construct so called racial differences in the metabolic syndrome in different ways for different purposes.

Fourth, as I have described in detail in this study, naturalization is a framework of colorblind racism that explains racial inequality as an outcome of natural and inevitable processes. The emergence of the metabolic syndrome represents an effort to naturalize a social and economic order that disproportionately increases rates of metabolic health problems in racially categorized groups. Genetics research on racial disparities and the metabolic syndrome is one potent site where colorblind racism and naturalization seem to be operating in different forms. These interpretive practices reflect the long-standing assumption that race is essentially linked to natural, biological, and immutable differences through the attempt to map genetic differences onto racial difference, and vice versa. This research often proceeds in the absence of any meaningful analysis of the economic and political arrangements that create racial health disparities.

In the field of genetic epidemiology, which investigates the molecular underpinnings of common chronic diseases, conceptions of race are used as conceptual tools for categorizing and explaining different levels of population risk (Cooper, Kaufman, and Ward 2003). In a first example of naturalization, Loos and colleagues sampled genetic information from individual members of 105 self-identified black and 99 white nuclear families in order to identify genomic regions harboring genes that may influence metabolic syndrome (Loos, Katzmarzyk, Rao, Rice, Leon, Skinner, Wilmore, Rankinen, and Bouchard 2003 :5935) The authors

report their findings in a manner that strongly suggests that the black and white samples have mutually exclusive genomic profiles:

> Blacks and whites had no QTLs [quantitative trait loci] in common for PC1 or PC2 [the two principal components of a multivariate analysis of phenotypic characteristics]. Although this may be due to the lack of power in the black sample, it is also possible that the loci for blacks and whites are truly distinct. (Loos et al. 2003 :5941).

A second example of naturalization comprises discourses of family history that construct race as scientifically valuable tool in the determination of genetic risks for heart disease without explicitly using discourses of race. This family history approach argues that the systematic collection and interpretation of family history is the best technique for identifying individuals with genetic susceptibility to heart disease (Scheuner 2004). This second genetic approach to the syndrome and race shifts the unit of analysis for assessing heart disease risk from the individual or population levels to the level of the nuclear family. The discursive effect of this approach is that researchers use family and ethnicity as colorblind substitutions for race potentially in order to avoid charges of scientific racism.

This method involves asking study participants specific questions concerning their nuclear and extended family's burdens of chronic disease and classifying their responses into categories of risk. This measure of family risk is then compared to the presence of the metabolic syndrome in a sample individual. This technique has also been used to study the heritable influences on the development of mental

illnesses, like schizophrenia (Shih, Belmonte, and Zandi 2004). The central assumption of this approach is that a family history of heart disease reflects the interaction of genetic, environmental, cultural, and behavioral risk factors *that is shared* among family members (Scheuner 2004 :2). Despite Scheuner's obvious care in not using race in his article, he believes the family history should include ethnicity and country of origin because certain conditions may be more prevalent in certain ethnic groups (Scheuner 2004:11).

A third colorblind approach of naturalization that is grounded in genetics involves tests of the theory of genetic admixture as an explanation for color-coded disparities in diabetes. The theory of genetic admixture assumes that the genetic susceptibility of different populations to the risk factors that constitute the metabolic syndrome are determined by the extent of *racial* admixture in any given individual. The theory of genetic admixture maintains since Europeans have historically had higher rates of diabetes than racially categorized groups (e.g., African Americans, Latinos, and Native Americans), the increasing racial miscegenation that has occurred since the colonialism explains the increasing rates of diabetes in these minority groups (Tull and Roseman 1995). The central assumption of this theory is that racial populations at an earlier moment in (pre) history were pure, distinctive, and segregated and it is their intermingling since the discovery of race that explains racial disparities in the contemporary moment times. One way to think about this theory is of it as a reverse degeneracy theory, in which genetic intermingling with whites reduces the life chances of people of color, rather than having the normative effect of "civilizing" them.

Context of intersectionality

A third important context for the significance of the metabolic syndrome is intersectionality. Analyzing the politics of metabolism using an intersectional framework would investigate how systems of race, gender, age, social class, and nation operate together in shaping the biological realities, political rationalities, and economic opportunities presented by the emergence of the metabolic syndrome. Scholars like Janet Shim, Donna Haraway, Dorothy Roberts, and Laura Mamo have documented how science, medicine, and technology are involved in propagating systems of racial, gender, and sexual stratification. For example, how did assumptions about gender shape the metabolic syndrome research of Jean Vague? The metabolic syndrome is associated with metabolic health conditions that disproportionately impact women of color especially heart disease, stroke, polycystic ovary syndrome, and or gestational diabetes. The children of women with metabolic syndrome have become a new object of biomedical scrutiny because of research and theories linking gestational conditions prior to birth to the development of metabolic health problems later in life.

These interlocking systems of oppression are also important for another reason that directly relates to the interpretation of racial data on the metabolic syndrome. Analysts have documented gender and class differences in the metabolic syndrome within racial and ethnic groups. For example, African American women and poor African Americans are disproportionately classified with metabolic syndrome compared to African American men and wealthier African Americans.

Metabolic syndrome researchers who are fixed on documenting and explaining racial and ethnic forms of difference while ignoring these important within-group differences may unwittingly interpret what are really gender or class differences as racial differences.

Context of food and nutrition

A final important context that highlights the significance of the metabolic syndrome is that of food politics and nutrition. It is not possible to provide a critical interpretation of the politics of metabolism without recognizing and acknowledging the synergistic relationships between food politics and metabolic health problems. Not only are food politics critical to the politics of metabolism, Foucault argued that agricultural innovation is one of the central features of the emergence of biopower—the power over food represents an emergent form of social control over life itself. The processed foods that are associated with the development of metabolic health problems have long been the objects of scientific study, government regulation, and corporate commodification within global capitalism. The historical period during which time the metabolic syndrome emerged also brought radical global changes to the ways that food was produced, marketed, and distributed, which in turn, shape the distribution of metabolic health problems. Most prominently, in the 1930s, 40s, and 50s, the privatization of food under conditions of global capital expansion led to the introduction of low-cost synthetic substitutes for raw sugar and vegetable oils.

The significance of food politics is also apparent in stark contrast to the deployment of killer applications and the cultural power that prescription drugs have

over people's metabolic lives. Indeed, the economic interests of transgenic (which means both agricultural *and* pharmaceutical) corporations operate in our bodies and shape the politics of metabolism in ways that go beyond posing food and drugs as disconnected political issues. If, as a society, we decided to invest in the sustainable production of foods that promote good metabolic health, as opposed to more and more killer applications, perhaps the politics of metabolism could begin to establish a context for human flourishing.

The Metabolic Fetish

> Fetishism is about interesting "mistakes"—really denials—where a fixed thing substitutes for the doings of power-differentiated lively beings on which and on whom, in my view, everything actually depends. (Haraway 1997: 135).

The names, definitions, and theories of the metabolic syndrome have indeed changed over time, but the discursive formation of the metabolic syndrome has never been consistent with itself. The category of the metabolic syndrome cannot contain the complexity and heterogeneity of its history and those social actors who participated in this complexity cannot contain its productive effects. To conclude, let me reflect on the ontological and epistemological status of the metabolic syndrome. How can we know anything about the metabolic syndrome and in what ways does it exist? Acting with the authority of scientific, state, and corporate power, social actors simply called the metabolic syndrome into existence within the government database, the corporate market report, and in the physiological substrata of bodies and populations. Drawing on a technoscientific notion of a fetishized commodity, it is

hard for me to imagine at this point a more unabashedly constructed thing-in-itself. Any critical questions about the socially constructed nature of the syndrome and its implications for race and racism might be quickly subsumed in molecular, genetic, and biological discourses about its etiology and population distribution. If a person is classified and/or diagnosed with the syndrome, do they "have" it? Of course, based upon this study the answer to this question is "no".

My final interpretation centers on the fetishism of the syndrome--the denial, disavowal, and error that undergird its production in a racialized and biomedicalized politics of metabolism. Situating the syndrome as a fetishized commodity helps to explain how this construct has become a new bright object in a biomedical gaze trained on metabolism. The reified heat of the syndrome glows as brightly as an imploding star, and biomedical scientists, corporate benefactors, and government regulators shave been unable to avert their eyes from it. In other words, while the metabolic syndrome does not exist as a thing-in-itself, scientists have constructed an epistemological framework in which claims about the metabolic syndrome have meaning for social actors and institutions that have an interest in the truth and validity of any such claims. This study attempted to develop a critical relationship to the knowledge-making practices that have produced a viable epistemological framework for the metabolic syndrome that has afforded it a provisional legitimacy in the world of scientific objects.

Appendix A

Research methods, data sources, and procedures

One of the main practical challenges of executing this study was how to transform the seemingly obtuse method of genealogical historiography into a set of procedures that I could follow consistently to analyze different kinds of documents and construct a critical narrative that demonstrated some of the relationships between the metabolic syndrome and race. Indeed, Foucault was not forthcoming with a codified checklist of procedures a researcher might follow to conduct a genealogy, and since his death, his interpreters have continued to struggled to do the same.[97] With this challenge in mind, this appendix restates my genealogical approach to discourse analysis and elaborates on the data sources I drew upon to construct this genealogy. I also use this appendix as a space to reflect on methodological roads not taken in the study.

Discourse Analysis

As I stated earlier, rather than only analyzing the meaning of a discourse, discourse analysis also analyzes the structure of the discursive themes by which a particular discourse is produced. Specifically, discourse analysis asks three core questions about the production of discourses: (1) who produced the discourses and with what resources? (2) Under what political, economic, and historical conditions were the discourses produced? (3) How are the meanings of the discourse shaped by these economic, political, and historical conditions? Thus, I aimed to interpret how

the discourse of metabolic syndrome emerged in ways that draw upon constructions of race in service of producing new meanings of race. In each document I analyzed, my goal was to identify the explicit and implicit assumptions about race that structured the discourses, practices, and technologies of the metabolic syndrome.

It is worth nothing that the theoretical frameworks that informed the study consistently shaped my thinking about which documents were important and the analytic procedures I used to highlight what might be important about a document. In other words, the multiple connections between theory, method, and data were exceptionally important in this case. At different points throughout the study, particular ideas in critical race theory, biomedicalization, and biopower informed my interpretation of the discursive themes that were important to document in the genealogical narrative. For example, from a critical race perspective, I was consistently interested in documenting any naturalizing discourses that constructed race as biological and genetic. However, naturalization is also an important theme in the frameworks of biomedicalization and biopower. Because naturalization is theoretically important within each of these frameworks, my analysis of the theme of naturalization took on greater significance as the study unfolded. Consequently, I increasingly focused on discourses within documents that suggested this theme, and then tried to analyze those discourses in a consistent manner.

Data Sources

As I described in chapter one, I employed three basic strategies to traverse and circumscribe the universe of documents about the metabolic syndrome and race. The

overall purpose of this three-step process was to identify the primary documents that formed the evidentiary bedrock of my study. As Table 1.1 suggests, thousands of research articles have been published on the metabolic syndrome, and while it was not possible or perhaps desirable to analyze all of the documents about the metabolic syndrome, it was important to establish a subset of this universe of documents to analyze in this study.

Biomedical Research Documents

First, I conducted extensive searches of multiple biomedical research databases in order to compile a comprehensive bibliography of documents pertaining to the metabolic syndrome and race. This group of documents consists of peer-reviewed research, reviews and commentaries on the metabolic syndrome and its connections to race published in academic and professional biomedical journals. Specifically, I repeatedly searched three prominent databases in this first strategy: (1) www.science.gov, the federal government's central search engine for published scientific research both within and outside the purview of the government and its scientific agencies; (2) Medline and PubMed™ Central, the premier bibliographic databases for the National Library of Medicine of the National Institutes of Health; and (3) ISI Web of Science, Science Citation Index. As my research continued, I also created publication alerts for the metabolic syndrome, which are search tools that emails the user when a document is published that makes reference to a particular set of search terms. This tool was extremely useful, as I was able to reference many *new* articles that were published on the metabolic syndrome over the past two years.

A second strategy was to use the ISI Web of Science cited citations index to conduct citation counts on the published documents I found on the metabolic syndrome and race to determine the extent to which a particular document has traveled and gained scientific currency throughout biomedicine. I employed this strategy in the full recognition that some sites of biomedical knowledge production have more political and scientific influence than others, and that not all journals are included in the ISI science citations index. For instance, a document published by one of the National Institutes of Health wields more influence than a document published in a relatively obscure biomedical journal that deals with a narrow subject matter. When appropriate, I made reference to this information throughout the study.

However, I do not feel that this technique was as useful as I had thought it might be because the production and consumption of a discourse are not one and the same process. For example, a document that is published in a specialized biomedical journal may not have been widely cited, but specialists in that subfield may consume and use the ideas contained in the document in ways that do not involve academic citation (e.g. practicing physicians may use that information in their practice, but never publish any research that cites that usage). One way to frame the limitations of this particular method is that citation counts are not meant to account for the polyvalence of discourses—they only assume one valid and legitimate usage of scientific knowledge.

Corporate Documents

A third strategy was to place special emphasis on the relatively smaller number of government and corporate documents. The corporate documents consist of published documents pertaining to AstraZeneca's Galaxy Program, Crestor™, including published clinical trial research, study documentation, regulatory submissions and letters to the FDA on behalf of AstraZeneca. I also collected documents that pertained to the research and development of antipsychotics. While I monitored and collected corporate documents relating to corporate practices relating to the metabolic syndrome and race that emerged over time, my analysis of racial pharmacology and killer applications focused more on published research articles in pharmacology and psychopharmacology journals. Because my study increasingly focused on the construction of racial meaning, published biomedical documents were a more useful kind of data for this kind of analysis.

Government Documents

The third group of primary documents consists of U.S. federal science policies, regulatory documents, administrative guidelines, and scientific documents that pertain to the conduct of biomedical research and clinical trials in the U.S. I analyzed these government documents in so far as they provide the overall regulatory context for the collection, standardization, and reporting of racial and biomedical data in all federal research on the metabolic syndrome. I considered the regulatory impact of four specific federal policies. The first policy is the National Health Survey Act of 1956, which authorized longitudinal surveys and special studies to secure accurate

and current statistical information on the amount, distribution, and effects of illness and disability in the U.S. and the services rendered for such conditions.[98] This act led to the creation of the National Health Interview Survey (NHIS), first conducted in 1957, the National Health Examination Survey (NHES) beginning in 1960, and the National Health and Nutrition Examination Survey (NHANES), which began in 1967. These government studies have been and still are the largest population health surveys conducted in the U.S., while the government has also funded many smaller yet still influential population health surveys over the past 50 years.[99]

The second policy is the 1997 Office of Management and Budget (OMB) regulations for the collection and presentation of federal data on race and ethnicity (Office of Management and Budget 1997). This regulation applies to all federal data collection efforts, including clinical and biomedical research sponsored by the NIH and FDA. According to the regulations, the U.S. government and its agencies consider self-identification as the preferred means of obtaining information about an individual's race and ethnicity. The regulations provide the minimum number of racial categories that must be used to ensure compliance with various civil rights statutes.[100] The OMB stresses that the racial and ethnic categories it recommends represent social-political constructs, and are not anthropologically or scientifically based (OMB 1997). The federal regulation of race provides a uniform, standardized, and common language for defining the major population groups of the country (OMB 1997).

The context and substance of the first two shapes the context and substance of the third and fourth policies. The third policy is the 2001 National Institutes of

Health guidelines on the inclusion of women and racial groups as subjects in clinical research (NIH 2001). These guidelines mandate that women and members of minority groups and their subpopulations be included in all NIH-supported biomedical research projects involving human subjects, unless a clear and compelling rationale and justification establishes to the satisfaction of the relevant Institute/Center Director that inclusion is inappropriate with respect to the health of the subjects or the purpose of the research. The fourth policy is the 2005 FDA guidance statement for the pharmecutical industry on the collection of race and ethnicity data in clinical trials (Food and Drug Administration 2005). This regulatory guidance statement says that drug companies must follow the NIH guidelines on the inclusion of racial and ethnic groups and use the OMB definitions of race in their regulatory submissions, drug research and development.

Notes

[1] (http://www.nlm.nih.gov/medlineplus/ency/article/002257.htm accessed August 22, 2006)

[2] For introductory purposes, I use the term the metabolic syndrome as an umbrella term to encompass many different concepts advanced by biomedical researchers to describe these relations including the metabolic syndrome, dysmetabolic syndrome X, insulin resistance syndrome, and syndrome X.

[3] The definition of metabolic syndrome used in this analysis was from the National Cholesterol Education Program (NCEP), which I will discuss along with the NHANES Study in chapter three. I conducted a works-cited search for the Ford et al study on December 11, 2006 in the Expanded Science Citations Index (ISI Web of Science) and found 1,114 articles published between 2002 and 2006 that cited this (Ford et al. 2002) study. By August 20, 2008, 1,676 articles cited this article, increasing by 562 citations in less than two years.

[4] (http://www.metabolicsyndromeinstitute.com/about/mission) accessed on March 5, 2009 at 4:15pm.

[5] This table reflects a search I conducted of the ISI Web of Science bibliographic database on February 5, 2007 and then again on October 15, 2008 for the terms "metabolic syndrome," "insulin resistance syndrome," "syndrome X", and "dysmetabolic syndrome X". This search showed that 16,040 original research articles were published on the metabolic syndrome and related terms between 1962 and 2007.

[6] For more on the proposed relationships between components of the metabolic syndrome and kidney and liver disease, see Bugianesi, E., A. J. McCullough, and G. Marchesini. 2005. "Insulin resistance: A metabolic pathway to chronic liver disease." *Hepatology* 42:987-1000; Chen, J., P. Muntner, L. L. Hamm, D. W. Jones, V. Batuman, V. Fonseca, P. K. Whelton, and J. He. 2004. "The metabolic syndrome and chronic kidney disease in US adults." *Annals of Internal Medicine* 140:167-174; Kurella, M., J. C. Lo, and G. M. Chertow. 2005. "Metabolic syndrome and the risk for chronic kidney disease among nondiabetic adults." *Journal of the American Society of Nephrology* 16:2134-2140; and Muntner, P., J. He, J. Chen, V. Fonseca, and P. K. Whelton. 2004. "Prevalence of non-traditional cardiovascular disease risks factors among persons with impaired fasting glucose, impaired glucose tolerance, diabetes, and the metabolic syndrome: Analysis of the Third Health and Nutrition Examination Survey (NHANES III)." *Annals of Epidemiology* 14:686-695.

[7] For more on the proposed relationship between components of the metabolic syndrome and polycystic ovarian syndrome, see Apter, D., T. Butzow, G. A. Laughlin, and S. S. C. Yen. 1995. "Metabolic Features of Polycystic-Ovary-Syndrome Are Found in Adolescent Girls with Hyperandrogenism." *Journal of Clinical Endocrinology and Metabolism* 80:2966-2973; Glueck, C. J., R. Papanna, P. Wang, N. Goldenberg, and L. Sieve-Smith. 2003. "Incidence and treatment of metabolic syndrome in newly referred women with confirmed polycystic ovarian syndrome." *Metabolism-Clinical and Experimental* 52:908-915; Morales, A. J., G. A. Laughlin, T. Butzow, H. Maheshwari, G. Baumann, and S. S. C. Yen. 1996. "Insulin, somatotropic, and luteinizing hormone axes in lean and obese women with polycystic

ovary syndrome: Common and distinct features." *Journal of Clinical Endocrinology and Metabolism* 81:2854-2864; and Solomon, C. G. 1999. "The epidemiology of polycystic ovary syndrome - Prevalence and associated disease risk." *Endocrinology and Metabolism Clinics of North America* 28: 247.

[8] For more on the proposed relationships between components of the metabolic syndrome and breast and colorectal cancer, see Argiles, JM and FJ Lopez-Soriano. 2001. "Insulin and cancer." *International Journal of Oncology* 18:683-687; Bruning, PF, JMG Bonfret, PAH Van Nooyrd, AA Hart, M De Jong-Bakker, and WJ Noojen. 1992. "Insulin resistance and breast cancer risk." *International Journal of Cancer* 52:511-516; La Vecchia, C, E Negri, A Decarli, and S Frabceschi. 1997. "Diabetes mellitus and colorectal cancer risk." *Cancer Epidemiology and Biomarkers Prevention* 6:1007-1010; and Will, JC, DA Galuska, F Vinicor, and EE Calle. 1998. "Colorectal Cancer: another complication of diabetes mellitus?" *American Journal of Epidemiology* 147:816-825.

[9] For more on the proposed relationship between components of the metabolic syndrome and HIV infection, see Hadigan, C., J. B. Meigs, C. Corcoran, P. Rietschel, S. Piecuch, N. Basgoz, B. Davis, P. Sax, T. Stanley, P. W. F. Wilson, R. B. D'Agostino, and S. Grinspoon. 2001. "Metabolic abnormalities and cardiovascular disease risk factors in adults with human immunodeficiency virus infection and lipodystrophy." *Clinical Infectious Diseases* 32:130-139; Murata, H., P. W. Hruz, and M. Mueckler. 2000. "The mechanism of insulin resistance caused by HIV protease inhibitor therapy." *Journal of Biological Chemistry* 275:20251-20254; and Safrin, S.

and C. Grunfeld. 1999. "Fat distribution and metabolic changes in patients with HIV infection." *Aids* 13:2493-2505.

[10] For more on the proposed relationships between components of the metabolic syndrome and erectile dysfunction, see Bansal, T. C., A. T. Guay, J. Jacobson, B. O. Woods, and R. W. Nesto. 2005. "Incidence of metabolic syndrome and insulin resistance in a population with organic erectile dysfunction." *Journal of Sexual Medicine* 2:96-103; Guay, A. and J. Jacobson. 2007. "The relationship between testosterone levels, the metabolic syndrome (by two criteria), and insulin resistance in a population of men with organic erectile dysfunction." *Journal of Sexual Medicine* 4:1046-1055; Kaplan, S. A., A. G. Meehan, and A. Shah. 2006. "The age related decrease in testosterone is significantly exacerbated in obese men with the metabolic syndrome. What are the implications for the relatively high incidence of erectile dysfunction observed in these men?" *Journal of Urology* 176:1524-1527; Kupelian, V., R. Shabsigh, A. B. Araujo, A. B. O'Donnell, and J. B. McKinlay. 2006. "Erectile dysfunction as a predictor of the metabolic syndrome in aging men: Results from the Massachusetts Male Aging Study." *Journal of Urology* 176:222-226; and Makhsida, N., J. Shah, G. Yan, H. Fisch, and R. Shabsigh. 2005. "Hypogonadism and metabolic syndrome: Implications for testosterone therapy." *Journal of Urology* 174:827-834.

[11] A more extensive discussion of this topic can be found in part I of chapter three.

[12] A more extensive discussion of this topic can be found in chapter four. For more on the proposed relationships between the components of the metabolic syndrome and mental illness, see my analysis of schizophrenia and antipsychotics in chapter four and see also Bermudes, R. A., P. E. Keck, and J. A. Welge. 2006. "The

prevalence of the metabolic syndrome in psychiatric inpatients with primary psychotic and mood disorders." *Psychosomatics* 47:491-497, Heiskanen, T., L. Niskanen, R. Lyytikainen, P. I. Saarinen, and J. Hintikka. 2003. "Metabolic syndrome in patients with schizophrenia." *Journal of Clinical Psychiatry* 64:575-579; Letter, Harvard Mental Health. 2006. "Schizophrenia and the metabolic syndrome." *Harvard Mental Health Letter* 23:7-7; Remington, Gary. 2006. "Schizophrenia, Antipsychotics, and the Metabolic Syndrome: Is there a silver lining?" *American Journal of Psychiatry* 163:1132-1134; and Thakore, Jogin H. . 2005. "Metabolic syndrome and schizophrenia." *British Journal of Psychiatry* 186:455-456.

[13] I analyze the NCEP's definition of the metabolic syndrome in greater detail in part I of chapter three.

[14] This data was accessed on IMS Health.com, a global leader in pharmaceutical industry information, on April 5, 2009 (http://www.imshealth.com/portal/site/imshealth/menuitem.a46c6d4df3db4b3d88f611019418c22a/?vgnextoid=841365272046e110VgnVCM100000ed152ca2RCRD)

[15] #1 Lipitor for cholesterol--74.8 million prescriptions; #4 Norvasc for hypertension and angina--38.3 million prescriptions; #5 Toprol-XL for hypertension 35 million; #7 Zocor for hypertension 29.6 million.

[16] In 1997, the Office of Management and Budget (OMB) provided the definitions of race and ethnicity that must be used in all biomedical and health policy research funded by the federal government (see Epstein, Steve. 2007. *Inclusion: the politics of difference in medical research*. Chicago: University of Chicago Press and Shields, Alexandra E., Michael Fortun, Evelyn M. Hammonds, Patricia A. King, Caryn

Lerman, Rayna Rapp, and Patrick F. Sullivan. 2005. "The Use of Race Variables in Genetic Studies of Complex Traits and the Goal of Reducing Health Disparities." *American Psychologist* 60:77-103.

[17] For such my analysis of this research, see chapter three.

[18] See Jones, David S. and Roy H. Perlis. 2006. "Pharmacogenetics, Race, and Psychiatry: Prospects and Challenges." *Harvard Review of Psychiatry* 14:92, Kahn, Jonathan T. . 2006. "Race, Pharmacogenomics and Marketing Putting BiDil in Context." *American Journal of Bioethics* 6:W1-W5, Lee, Sandra Soo-Jin. 2005. "Racializing Drug Design: Implications of Pharmacogenomics for Health Disparities." *American Journal of Public Health* 95:2133-2138.

[19] See my analysis in chapter four.

[20] In terms of Foucault's own scholarship, there are a few key sources for Foucault's ideas about genealogical historiography: Foucault, Michel. 1972. *The archaeology of knowledge and the discourse on language*. Translated by S. A. M. Smith. New York: Pantheon Press; Foucault, Michel. 1978. *The History of Sexuality, An Introduction: Volume I*. Translated by R. Hurley. New York: Vintage; Foucault, Michel. 1980. *Power/Knowledge: Selected Interviews and Other Writings, 1972-1977*, Edited by C. Gordon. Translated by C. Gordon, L. Marshall, J. Mepham, and K. Soper. New York: Pantheon Books; and Foucault, Michel. 2003. *The Essential Foucault*, Edited by P. Rabinow and N. Rose. New York: The New Press. There are too many secondary interpretations of the genealogical method to mention here, but I relied especially on the following: Davidson, Arnold I. 1986. "Archeaology, Genealogy, Ethics." in *Foucault: A Critical Reader*, edited by D. C. Hoy. Oxford: Basil Blackwell.; Mahon,

Michael. 1992. *Foucault's Nietzschean genealogy: truth, power, and the subject*. Albany: State University of New York Press.; Visker, Rudi. 1995. *Michel Foucault: genealogy as critique*. Translated by C. Turner. London: Verso.; Dean, Mitchell. 1994. *Critical and effective histories: Foucault's methods and historical sociology*. London: Routledge, Drefus, H.L. and Paul Rabinow. 1982. *Michel Foucault: Beyond structuralism and hermeneutics*. New York, NY: Harvester Wheatsheaf; May, Todd. 1993. *Between Genealogy and Epistemology: Psychology, Politics, and Knowledge in the Thought of Michel Foucault*: Pennsylvania State University Press.

[21] Scholars and theorists from a range of disciplines have attempted to craft a clear conception of what genealogy entails. For this discussion see Dean, Mitchell. 1994. *Critical and effective histories: Foucault's methods and historical sociology*. London: Routledge; Kendall, Gavin and Gary Wickham. 1999. *Using Foucault's Methods* Thousand Oaks, CA: Sage Publications; Lash, Scott. 1984. "Genealogy and the Body: Foucault/Deleuze/Nietzsche." *Theory, Culture, and Society* 2:1-17; Levy, Neil. 1998. "History as Struggle: Foucault's genealogy of genealogy." *History of the Human Sciences* 11:159-170; Mahon, Michael. 1992. *Foucault's Nietzschean genealogy: truth, power, and the subject*. Albany: State University of New York Press; May, Todd. 1993. *Between Genealogy and Epistemology: Psychology, Politics, and Knowledge in the Thought of Michel Foucault*: Pennsylvania State University Press; Meadmore, Daphne, Caroline Hatcher, and Eric McWilliam. 2000. "Getting tense about genealogy." *Qualitative Studies in Education* 13:463-476; Prado, C.G. 2000. *Starting with Foucault: An introduction to genealogy*. Boulder, CO: Westview Press; Sax, Ben. 1990. "On the Genealogical Method: Nietzsche and Foucault."

International studies in philosophy 22:129-141; Shiner, Larry. 1982. "Reading Foucault: Anti-Method and the Genealogy of Power-Knowledge." *History and Theory* 21:382-398; and Visker, Rudi. 1995. *Michel Foucault: genealogy as critique*. Translated by C. Turner. London: Verso..

[22] These are known as the prescriptive effects of the jurisdiction of power and the effects of the veridiction of truth, respectively.

[23] Foucault euphemistically calls this network of power/knowledge relationships the "hazardous play of dominations" (Foucault 2003[1971]: 357). In his thinking, this play of dominations manifests itself in the rituals and practices of bodies and laws and regulations that impose various rights and obligations on bodies (Foucault, Michel. 2003[1971]. "Nietzsche, Genealogy, History." Pp. 351-369 in *The Essential Foucault* edited by P. Rabinow and N. Rose. New York: The New Press.)

[24] For the articulation of discourse analysis I use in this study, see (Clarke, Adele E. 2005. *Situational Analysis: Grounded theory after the postmodern turn*. Thousand Oaks: Sage Publications.)

[25] Clinical pharmacology is the branch of biomedicine that studies the intended and unintended effects of drugs on the body.

[26] There is a voluminous body of knowledge about the history, philosophy, and politics of race in science over the past 20 years. Some outstanding sources on these issues are: Duster, Troy. 2003 [1990]. *Backdoor to Eugenics*. New York: Routledge; Graves, Jospeh L. Jr. 2001. *The Emporer's New Clothes: Biological Theories of Race at the Millenium*. Brunswick, NJ: Rutgers University Press; Harding, Sandra. 1993. "The 'Racial' Economy of Science: Toward a Democratic Future." Bloomington:

Indiana University Press; Paul, Diane. 1998. *The Politics of Heredity: Essays on Eugenics, Biomedicine, and the Nature-Nurture Debate*. Albany: SUNY Press; Stephan, Nancy Leys. 1982. *The idea of race in science*. Hamden, CT: Archon Books; and Zack, Naomi. 2002. *Philosophy of Science and Race*. London: Routledge.

[27] This is the shift that historian Elazar Barkan famously described as the retreat of scientific racism which refers to physical anthropology's adoption of the concepts, methods and theories of population genetics (Barkan, E. 1992. *The Retreat of Scientific Racism: Changing Concepts of Race in Britain and the United States between the World Wars*)

[28] In a more lyrical way, Goldberg explains "the "primitive" is the romantic fabrication of and longing for an original human subjectivity, pristine in its representation" (Goldberg 2002: 202). For more on scientific discourses of primitivism, see Haraway, Donna. 1989. *Primate Visions: Gender, Race, and Nature in the World of Modern Science*. New York: Routledge.

[29] At least since Omi and Winant, contemporary critical race theorists have recognized the centrality of the state to racial formation (see Goldberg, David Theo. 2002. *The Racial State*. Malden, MA: Blackwell Publishers; Omi, Michael and Howard Winant. 1994. *Racial Formation in the United States*. New York: Routledge; and Stevens, Jacqueline. 2003. "Racial Meanings and Scientific Methods: Changing Policies for NIH-Sponsored Publications Reporting Human Variation." *Journal of Health Politics, Policy and Law* 28:1033-1087.)

[30] The 1790 census asked five questions: the number of free white males over 16 years old, free white males under 16, free white females, other, and number of slaves.

[31] I discuss these specific regulations in chapter three in the context of the metabolic syndrome.

[32] The Food and Drug Administration enforces similar guidelines regarding the inclusion of women and racial minorities in drug clinical trials. I discuss these regulatory guidelines in chapter four.

[33] Several other scholars have examined the use of race and ethnicity in pharmacological research and development. See Duster, Troy. 2005. "Race and Reification in Science." *Science* 307:1050-1051; Gannett, Lisa. 2005. "Group Categories in Pharmacogenetics Research." *Philosophy of Science* 72:1232-1247; Lee, Sandra Soo-Jin. 2005. "Racializing Drug Design: Implications of Pharmacogenomics for Health Disparities." *American Journal of Public Health* 95:2133-2138; and Sankar, Pamela and Jonathan Kahn. 2005. "BiDil: Race Medicine Or Race Marketing?" *Health Affairs*: 54-55.

[34] I explore this dynamic of biopower in chapter four, when I examine prescription drugs as a means of extracting both knowledge and profit from bodies.

[35] Biological theories of race, produced as part of the disciplinary knowledges of biopower, were mobilized to rationalize the expansionism and exploitation that accompanied colonialism and slavery. As I discussed in the critical race theory framework, scientific racism operated by deploying scientific justifications for racial conquest and domination.

[36] For more on the links between biological theory, eugenics, and race see Duster, Troy. 2003 [1990]. *Backdoor to Eugenics*. New York: Routledge, Graves, Jospeh L. Jr. 2001. *The Emperor's New Clothes: Biological Theories of Race at the Millenium*.

Brunswick, NJ: Rutgers University Press, Paul, Diane. 1998. *The Politics of Heredity: Essays on Eugenics, Biomedicine, and the Nature-Nurture Debate*. Albany: SUNY Press, Weingart, Peter. 1998. "The Thin Line between Eugenics and Preventive Medicine." in *Xenophobia in Germany and the United States*, edited by N. Finzsch and D. Schirmer. Cambridge: Cambridge University Press, Zuberi, Tukufu. 2001. *Thicker than Blood: An Essay on How Racial Statistics Lie*. Minneapolis: University of Minnesota.

[37] The analysis of emergence situates the emergence of a practice or discourse within in a broader network of institutionally based power/knowledge relationships. For more on the genealogical analysis of emergence, please reference my discussion in chapter one.

[38] I have selected the year of 1956 as a way to mark the publication of the research of Jean Vague, a French physician whose work on metabolism is considered by many metabolic syndrome scientists to be foundational to the new field. I discuss Dr. Vague's work in several points throughout the chapter.

[39] Researchers at the Metabolic Syndrome Institute, a web-based organization of biomedical researchers whose primary goal is to promulgate the idea of the metabolic syndrome, attribute the concept to Dr. Vague. Several prominent metabolic syndrome researchers belong this group, including Dr. Scott Grundy (http://www.metabolic-syndrome-institute.org/medical_information/history/#lien_a accessed December 20, 2006.). Indeed many others note the centrality of Dr. Vague's thought, but rarely to they explore his paper, which I do here.

[40] The Islets of Langerhans are a part of the pancreas that is responsible for insulin production.

[41] The National Heart Institute is the institutional precursor to the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health (NIH).

[42] The Framingham study sample consisted of 5,209 'white' men and women (30-62) living in Framingham MA; (1971) 5,124 of their children and spouses, and then their grandchildren in 2005.

[43] (www.cdc.gov/nchs/about/major/nhis/hisdesc.htm accessed on October 23, 2006).

[44] See my discussion of these population heart studies later in this section.

[45] (http://www2.merriam-webster.com/cgi-bin/mwmednlm) accessed on March 5, 2009 at 4:25pm.

[46] In 1993, this construct gets revived by Descovich and colleagues in a book edited by Crepaldi himself (Descovich, G.C., B. Benassi, V. Canelli, S. D'Addato, G. De Simone, and A. Dormi. 1993. "An epidemic view of the plurimetabolic syndrome." in *Diabetes, Obesity, and Hyperlipidemia: The plurimetabolic syndrome*, edited by G. Crepaldi, A. Tiengo, and E. Manzato. Amsterdam: Elsever Science.)

[47] The Banting Lecture is published annually in the journal Diabetes, which is the flagship journal of the American Diabetes Association. As of August 19, 2008, Reaven's published lecture had been cited 5,953 times.

[48] In the 1980s, three groups of researchers created three new techniques for measuring insulin resistance. DeFronzo and colleagues developed the "euglycemic hyperinsulinemic clamp technique" in 1983, and it still is the gold-standard procedure to measure insulin resistance (Defronzo, Ralph A., Eleuterio Ferrannini, and Veikko

Koivisto. 1983. "New concepts in the pathogenesis and treatment of noninsulin-dependent diabetes mellitus." *The American Journal of Medicine* 74:52-81.) Other noteworthy techniques include the "homeostasis model assessment-insulin resistance index" (Matthews, DR, JP Hosker, AS Rudenski, BA Naylor, DF Treacher, and RC. Turner. 1985. "Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man." *Diabetologia* 28:412-419.) and the "oral glucose tolerance test" (Belfiore, Francesco, Silvia Iannello, and Giovanni Volpicelli. 1998. "Insulin Sensitivity Indices Calculated from Basal and OGTT-Induced Insulin, Glucose, and FFA Levels." *Molecular Genetics and Metabolism* 63:134-141.)

[49] In this simple schema, three points are awarded if your fasting glucose is greater than 11, or your glucose at two hours into the Glucose Tolerance Test is greater than 140; fasting triglyceride level is greater than 200; fasting HDL-cholesterol levels is lower than 35; blood pressure is greater than 145/90. You earn one point if your weight check reveals you are more than 15 pounds overweight; family has a history of heart disease, high blood pressure (hypertension) or diabetes; lifestyle is characterized by physical inactivity in both work and leisure hours. Your risk of having a heart attack triggered by syndrome X can be low (0-4 points), moderate (5-8 points), high (9-12), and very high (13 or more). (Adapted from Reaven, Strom, and Fox 2000: 68).

[50] (http://www.icd9data.com/2009/Volume1/240-279/270-279/277/277.7.htm) retrieved on February 11, 2009.

[51] Recall from chapter one that a second methodological strategy was to use the ISI Web of Science cited citations index to conduct citation counts on the published documents I found on the metabolic syndrome and race to determine the extent to which a particular document has traveled and gained scientific currency throughout biomedicine.

[52] I discuss AstraZeneca again in chapter four because they are the producers of Crestor, a cholesterol lowering medication that has been studied in populations classified with the metabolic syndrome.

[53] The proinflammatory state refers to elevated levels of C-reactive protein, another biochemical that has been associated with the metabolic syndrome.

[54] See the forthcoming section titled "The New Special Populations".

[55] (http://www.nlm.nih.gov/medlineplus/metabolicsyndrome.html) accessed on February 13, 2009 at 10:23 am.

[56] (http://www.nhlbi.nih.gov/health/dci/Diseases/ms/ms_whatis.html) accessed on May 4, 2009 at 4:30pm.

[57] See Figures 3.2 and 3.3.

[58] Several other studies illustrate the general argument presented here. See, for example: *MESA* (Multiethnic Study of Athlerosclerosis)—Bild DE, Bluemke DA, Burke GL, Detrano R, Diez Roux AV, Folsom AR, Greenland P, Jr. Jacob DR, Kronmal R, Liu K, Nelson JC, O'Leary D, Saad MF, Shea S, Szklo M, and Tracy RP. 2002. "Multi-ethnic study of atherosclerosis: objectives and design." *American Journal of Epidemiology* 156:871-881; and *IRAS* (Insulin Resistance and Atherosclerosis Study) Festa, Andreas, Ralph D'Agostino, Jr, George Howard, Leena

Mykkanen, Russell P. Tracy, and Steven M. Haffner. 2000. "Chronic Subclinical Inflammation as Part of the Insulin Resistance Syndrome: The Insulin Resistance Atherosclerosis Study (IRAS)." *Circulation* 102:42-47.

[59] I will discuss the theory of genetic admixture in the final part of section two, The New Special Populations Research. (www.clinicaltrials.gov/ct/show/NCT00005146) retrieved on February 13, 2009.

[60] (www.clinicaltrials.gov/ct/show/NCT00005146) retrieved on February 13, 2009.

[61] (http://www.cardia.dopm.uab.edu/lad_info.htm) accessed on February 16, 2009 at 2:08pm.

[62] (http://www.cscc.unc.edu/aric/) accessed on February 16, 2009 at 2:51 pm.

[63] (http://jhs.jsums.edu/jhsinfo/) accessed on February 16, 2009 at 3:23 pm.

[64] (http://www.nhlbi.nih.gov/about/jackson/2ndpg.htm) accessed on February 16, 2009 at 2:59 pm.

[65] These four studies are not exhaustive of the population studies that incorporate measurements of the metabolic syndrome in racially categorized groups. The Multiethnic Study of Atherosclerosis and the National Health and Nutrition Examination Study are two other studies that meet these criteria.

[66] According to an NIDDK website on the special role the Pima have played in government biomedical research on diabetes, “This cooperative search between the Pima Indians and the NIH began in 1963 when the NIDDK (then called the National Institute of Arthritis, Diabetes and Digestive and Kidney Diseases), made a survey of rheumatoid arthritis among the Pimas and the Blackfeet of Montana. They discovered an extremely high rate of diabetes among the Pima Indians. Two years later, the

Institute, the Indian Health Service, and the Pima community set out to find some answers to this mystery" (http://diabetes.niddk.nih.gov/dm/pubs/pima/pathfind/pathfind.htm) accessed on February 16, 2009.

[67] In chapter five, I briefly discuss how ideas about family intersect with race and ethnicity in the context of colorblindness, but I wanted to note their historical deployment here by Reaven.

[68] The International Society on Hypertension in Blacks (ISHIB), Inc, publishes *Ethnicity & Disease*.

[69] I will talk more about the published research of members of the AALCC in Chapter Four. See Clark, L. T. and F. El-Atat. 2007. "Metabolic syndrome in African Americans: Implications for preventing coronary heart disease." *Clinical Cardiology* 30:161-164; Ferdinand, KC, LT Clark, KE Watson, RC Neal, CD Brown, BW Kong, BO Barnes, WR Cox, FJ Zieve, J Ycas, PT Sager, and A Gold. 2006. "Comparison of efficacy and safety of rosuvastatin versus atorvastatin in African-American patients in a six-week randomized trial." *American Journal of Cardiology* 97:229-235; Grundy, Scott M., James I. Cleeman, C. Noel Bairey Merz, H. Bryan Brewer, Jr., Luther T. Clark, Donald B. Hunninghake, Richard C. Pasternak, Sidney C. Smith, Jr., and Neil J. Stone. 2004. "Implications of Recent Clinical Trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines." *Circulation* 110:227-239; and Smith, Sidney C, Stephen R Daniels, Miguel A Quinones, Shiriki K Kumanyika, Luther T Clark, Richard S Cooper, Elijah Saunders, Elizabeth Ofili, and Eduardo J Sanchez. 2005. "Discovering the full spectrum of cardiovascular disease: Minority

Health Summit 2003: report of the Obesity, Metabolic Syndrome, and Hypertension Writing Group." *Circulation* 111:e134-e139.

[70] Recall that the San Antonio Heart Study was designed to assess the degree of genetic admixture as well.

[71] I draw these highlights from several published articles and reports on these technologies. Sarafidis, Panteleimon A. and Peter M. Nilsson. 2006. "The metabolic syndrome: a glance at its history." *Journal of Hypertension* 24:621-626. National High Blood Pressure Education Program, NHBPEP and Lung National Heart, and Blood Institute, NHLBI. 2002. "Summary Report: Working Meeting on Blood Pressure Measurement." National Institutes of Health, Bethesda, MD. Kuczmarski, Robert J and Katherine M Flegal. 2000. "Criteria for definition of overweight in transition: background and recommendations for the United States." *American Journal of Clinical Nutrition* 72:1074-1081.

[72] Recall from chapter one that the analysis of descent traces the multiple sites of knowledge production by documenting the actual research instruments, procedures, and practices used in the study of the body.

[73] There are other candidate drugs that could be examined here as well. The metabolic syndrome is increasingly used to refer to new drug targets, or new laboratory markers that reflect the efficacy of drug therapies. See Giugliano, Dario, Antonio Ceriello, and Katherine Esposito. 2008. "Are there specific treatments for the metabolic syndrome?" *American Journal of Clinical Nutrition* 87:8-11; Grundy, Scott M., James I. Cleeman, C. Noel Bairey Merz, H. Bryan Brewer, Jr., Luther T. Clark, Donald B. Hunninghake, Richard C. Pasternak, Sidney C. Smith, Jr., and Neil J.

Stone. 2004. "Implications of Recent Clinical Trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines." *Circulation* 110:227-239; Grundy, Scott M. T. . 2006. "Drug therapy of the metabolic syndrome minimizing the emerging crisis in polypharmacy." *Nature Reviews Drug Discovery* 5:295-315; Jacobson, T. A., C. C. Case, S. Roberts, A. Buckley, K. M. Murtaugh, J. C. Y. Sung, D. Gause, C. Varas, and C. M. T. "Characteristics of U. S. adults with the metabolic syndrome and therapeutic implications." *Diabetes, Obesity & Metabolism* 6:353; and Lesko, L. J. and Aj T. Atkinson. 2001. "Use Of Biomarkers in Development, Surrogate Endpoints In Drug Regulatory Decision Making: Criteria, Validation Strategies." *Annual Review of Pharmacology & Toxicology* 41:347-66.

[74] The case of antiretroviral drugs that treat HIV infection could have also been analyzed here. Many HIV drugs have metabolic side effects and the metabolic syndrome is also being deployed to describe these effects. See Hadigan, C., J. B. Meigs, C. Corcoran, P. Rietschel, S. Piecuch, N. Basgoz, B. Davis, P. Sax, T. Stanley, P. W. F. Wilson, R. B. D'Agostino, and S. Grinspoon. 2001. "Metabolic abnormalities and cardiovascular disease risk factors in adults with human immunodeficiency virus infection and lipodystrophy." *Clinical Infectious Diseases* 32:130-139; Murata, H., P. W. Hruz, and M. Mueckler. 2000. "The mechanism of insulin resistance caused by HIV protease inhibitor therapy." *Journal of Biological Chemistry* 275:20251-20254; and Safrin, S. and C. Grunfeld. 1999. "Fat distribution and metabolic changes in patients with HIV infection." *Aids* 13:2493-2505.

[75] On July 15, 2002, the Diabetes Mellitus Interagency Coordinating Committee, held a meeting at the main campus of the National Institutes of Health in Bethesda,

Maryland to discuss "Macrovascular Disease and Diabetes: Translation Issues." The committee brought together representatives from various the Institutes of Health, the Centers for Disease Control, academic biomedicine, and the pharmaceutical industry to "determine the means and methods for translating the current scientific data from clinical trials and epidemiological studies to diabetes patients and the general public" (DMICC 2002:1). The committee's responsibilities are to tell Americans about "an increased relative risk of cardiovascular disease (CVD) for those individuals who have been diagnosed with diabetes or pre-diabetes" (DMICC 2002:1) and to propose ways that the government, biomedicine, and the pharmaceutical industry might work together to help in the translation effort.

[76] Haraway uses the term in the following manner. She writes, "Software sufficiently powerful to revolutionize how computers are used—that is, how further hybrids of human and nonhumans take shape and act—are, unfortunately, called, killer applications. Comparable only to the importance of the word-processor and spreadsheet software, Mosaic-like browsers are likely to be such "killer applications" that reconfigure practice in an immense array of domains. Mosaic was about the power to make hypertext and hypergrahic connections of the sort that produce the global subject of technoscience as a potent form of historical, contingent, specific human nature at the end of the millennium. Contesting how such subjects and hybrids are put together and taken apart is a critical feminist technoscientific practice" (Haraway 1997: 126).

[77] #1 Lipitor for cholesterol--74.8 million prescriptions; #4 Norvasc for hypertension and angina--38.3 million prescriptions; #5 Toprol-XL for hypertension 35 million; #7 Zocor for hypertension 29.6 million.

[78] These FDA guidelines advocate the use of the Office of Management and Budget racial categories I discussed earlier in chapter two.

[79] (http://www.nlm.nih.gov/medlineplus/ency/article/00685.htm accessed on August 23, 2008 at 11:26am).

[80] (www.fda.gov/fsn accessed on August 22, 2008—Show #28, June 2004)

[81] Data collected from Appendix A, Table A-2 of this document.

[82] The term "statins" refers to a class of drugs that are hydroxylmethl glutaryl coenzyme A (HMG CoA) reductase inhibitors.

[83] The six statins in order of their FDA approval are: Mevacor (lovastatin), Lescol (fluvastatin), Lipitor (atoravastatin), Zocor (simvastatin), Pravachol (pravastatin), and Crestor (rosuvastatin).

[84] LDL, or low-density lipoprotein, is the so called bad cholesterol that has long been considered a risk factor for heart disease and stroke.

[85](www.astrazeneca-us.com/modules/PRMS/display.asp?id-591959 accessed on October 23, 2006). The Galaxy Program has included over 50,000 research subjects in 50 nations.

[86] The ARIES (2) The STARSHIP Study (Study Assessing Rosuvastatin in Hispanic Population) Lloret, R, J Ycas, M Stein, and SM Haffner. 2006. "Comparison of rosuvastatin versus atorvastin in Hispanic-Americans with hypercholesterolemia."

American Journal of Cardiology 98:768-773. and (3) The IRIS Study (Investigation of Rosuvastatin in South Asian Subjects).

[87] ARIES was conducted from March 2002-December 2003 at 76 academic and clinical research centers in the United States.

[88] Research subjects were recruited from the practices of physicians who participated in the study and all had been on either diet or drug therapy for high cholesterol during the previous three months. The physician sample (n=401) was drawn from a larger pool of practicing doctors who represented the top 26% of statin prescribers who worked under the auspices of IMS Health (based in Westport, CT). These doctors were responsible for 55% of prescriptions for lipid-lowering drugs in 2002. The doctors included in the sample may be what the authors call "enthusiasts" who may manage lipids more aggressively than average.

[89] The investigators considered simvastatin and atoravastin "high-efficacy statins."

[90] This is my estimate complied from these sources. Direct costs include the costs of physicians and other professionals, hospital and nursing home services, the cost of medications, home health care and other medical goods. In direct costs refer to lost economic productivity due to premature disease and death.

[91] See my discussion of racial essentialism and naturalization in chapter two under framework of critical race theory.

[92] See chapter three for more on these genetic theories of race and the syndrome.

[93] The history and philosophy of race in science is an interdisciplinary body of scholarship from epidemiology, sociology, biology, philosophy, legal studies, and anthropology. Some contemporary voices from critical race theory on the issues of

race in science and medicine are: Cooper, Richard S., Jay S. Kaufman, and Ryk Ward. 2003. "Race and Genomics." *New England Journal of Medicine* 348:1166-1175; Duster, Troy. 2003a. "Buried Alive: The Concept of Race in Science." Pp. 258-277 in *Genetic Nature / Culture: Anthropology and Science Beyond the Two-Culture Divide*, edited by A. H. Goodman, D. Heath, and M. S. Lindee. Berkeley and London: University of California Press; Duster, Troy. 2005. "Race and Reification in Science." *Science* 307:1050-1051; Fausto-Sterling, Anne. 2004. "Refashioning Race: DNA and the Politics of Health Care." *differences: A Journal of Feminist Cultural Studies* 15:1-37; Gannett, L. 2001. "Racism and human genome diversity research: The ethical limits of "population thinking."" *Philosophy of Science* 68:S479-S492; Gannett, L. 2004. "The biological reification of race." *British Journal for the Philosophy of Science* 55:323-345; Ossorio, Pilar and Troy Duster. 2005. "Race and Genetics: Controversies in Biomedical, Behavioral, and Forensic Sciences." *American Psychologist* 60:115; Rabinow, Paul and Nikolas Rose. 2003. "Some Thoughts on Biopower Today." in *Vital Politics: Health, Medicine, and Bioeconomics into the Twenty First Century*. London School of Economics; Smedley, Audrey and Brian D. Smedley. 2005. "Race as Biology Is Fiction, Racism as a Social Problem Is Real." *American Psychologist* 60:16-26.

[94] See Feder, Ellen K. 2004. "Race, Biopower, and The Dangerous Individual." *Radical Philosophy Review*; McWhorter, Ladelle. 2004. "Sex, Race, and Biopower: A Foucauldian Geneaology." *Hypatia* 19:38-62; Rai, A. S. 2004. "Of monsters - Biopower, terrorism and excess in genealogies of monstrosity." *Cultural Studies* 18:538-570; and Stoler, Laura Ann. 1995. *Race and the Education of Desire:*

Foucault's History of Sexuality and the Colonial Order of Things. Durham: Duke University Press.

[95] For several examples of this scholarship in the field of science and technology studies, see Briggs, C. L. 2005. "Communicability, racial discourse, and disease." *Annual Review of Anthropology* 34:269-291; Franklin, Sarah and Margaret M. Lock. 2003. "Remaking Life & Death: Toward an Anthropology of the Biosciences." in *School of American Research Advanced Seminar Series*; Haraway, Donna J. 1997. *Modest_Witness@Second_Millennium. FemaleMan_Meets_OncoMouse*. New York, NY: Routledge; Melbourne, Tapper. 1995. "Interrogating Bodies: Medico-Racial Knowledge, Politics, and the Study of a Disease." *Comparative Studies in Society and History* 37:76-93; Orr, Jackie. 2006. *Panic Diaries: A Genealogy of Panic Disorder*. Durham: Duke University Press; Reardon, Jennifer. 2005. *Race to the Finish: Identity and governance in an age of genomics*. Princeton, NJ: Princeton University Press; Rose, Nikolas. 2001. "The Politics of Life Itself." *Theory, Culture, and Society* 18:1-13; and Shim, Janet K. 2000. "Bio-power and Racial, Class, and Gender Formation in Biomedical Knowledge Production." *Research in the Sociology of Health Care* 17:175-195.

[96] I compiled these prevalence data from the third (1999-2002) National Health and Nutrition Examination Survey. NHANES III (1988-94) is the seventh in a series of government epidemiological surveys designed to provide national estimates of the health and nutritional status of the civilian non-institutionalized population of the U.S. The NHANES III included about 40,000 participants, including, for the first time, an oversampling of African Americans and Mexican Americans.

[97] See my discussion of the challenges of defining genealogy in chapter two. Scholars and theorists from a range of disciplines, including Foucault himself, have struggled to craft a clear procedure for what a genealogy ought to entails.

[98] (www.cdc.gov/nchs/about/major/nhis/hisdesc.htm accessed on October 23, 2006).

[99] See Table 3.6.

[100] See Table 2.1.

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