BIOETHNIC CONSCRIPTION: Genes, Race, and Mexicana/o Ethnicity in Diabetes Research

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Diseases that involve complex gene–gene-environment interactions (e.g., asthma, diabetes, heart disease) are the next frontier for genetic medicine. The Mendelian gold rush of the previous single-gene model has given way to a vastly more complex scientific venture.[1] Researchers must now transform "the environment" and at risk "populations" into variables that fit biological analyses while keeping in mind the speculative futures of potential drug markets, public health concerns and careerist imperatives. Characterizing the social and cultural implications of complex disease genetics requires analyses that attend to the collaborative, transdisciplinary, and multisited networks of knowledge production.

In this article, I detail the use of ethnic and racially classified DNA in the search for genetic susceptibility for type 2 diabetes.[2] Drawing on ethnographic research in wet (bench) and dry (computer) labs, at a DNA sampling field office on the U.S.–Mexico border and at scientific conferences,[3] I examine how the complicated social and biological meanings of race and ethnicity simultaneously shape the biomedical production and representation of diabetes knowledge.[4] By following DNA from its point of origin, through the pathways of knowledge production and into medicoscientific representations of diabetes knowledge, I illustrate that researchers use ill-defined ethnic taxonomies to parse populations and social history to rationalize their categorical choices. This results in a nuanced reiteration of biological human variation that naturalizes (Yanagisako and Delaney 1995) Mexicana ethnicity to explain diabetes etiology. In a process I call "bioethnic conscription" the social identities and life conditions of DNA donors are grafted onto the biological explanations of disease causality.

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This is not a simplistic remaking of the biological races of man. Rather, bioethnic conscription is a practice with at least two modes, one descriptive and the other attributive. In the first mode, ethnic and racial, hereafter referred to as ethnoracial, classifications are used to pragmatically describe human groups. They are used, for instance, to report to readers of scientific publications from which human groups the biological samples and data sets were derived for the study. The naming of human groups is a matter of method. In the latter mode, ethnoracial labels do more than identify groups: The labels are used to attribute qualities to groups. Attribution modifies, often in a delimiting way, what it refers to. In grammar, for example, adjectives are attributive. Attribution easily leads to racialization. Thinking in terms of "bioethnic conscription" directs attention to both modes of classification and difference among human groups.

Bioethnic conscription, as described here, occurs at many stages of the scientific research process and operates independently of the intentions of human actors. Understanding the distinction between descriptive and attributive modes of classification is critical to understanding how bioethnic conscription actually happens, and consequently, how "race" operates—constructively and problematically—in the sciences.[5] In this article, I examine the conditions that make the slippage between description and attribution possible, instances in which it occurs, and, importantly, the scientific practices in which these two modes are contested.[6] The instances of bioethnic conscription examined here trouble the either–or binaries within the race–no race debate for biomedicine.

FINDING MEXICANA/O GENES

In 1996, a consortium of diverse researchers working on the genetics of type 2 diabetes formed an alliance to analyze the growing body of DNA samples acquired from diabetic affected individuals and their families.[7] The diabetes enterprise was formed because each researcher realized that, alone, their few samples could never reach the statistical significance required for genetic analyses of a complex disease like diabetes.[8] As several consortium members remarked, "Everyone had to fail a few times before coming to see the importance of joining the consortium." The academic, corporate, and state-funded alliance was composed of molecular, biological, computer, and clinical scientists from research sites in Europe, Japan, and the United States.[9]

In October 2000, the *Los Angeles Times, New York Times, Japan Times, Chicago Tribune, USA Today, Wall Street Journal,* and the *Times of London* all reported from *Nature Genetics* on a polygene candidate discovered for type 2 diabetes—a candidate my ethnographic interlocutors worked hard to find. A polygene is an observed genetic "effect" that is

the result of an interaction between two genes. One aspect of this research warrants explanation here.

A complex disorder like diabetes requires more than one gene to confer risk on a given individual. Finding genes by comparing affecteds with nonaffecteds is relatively easy when the culprit is a single gene, and when environmental factors are excluded from the analyses. This discovery of a polygene candidate for diabetes was newsworthy because it opened the doorway—both conceptually and methodologically—for polygenetic traits to be added to the genetics bandwagon (Fujimura 1996) of disease causation.[10] That is, until this *Nature Genetics* publication, the discovery of gene–gene interactions was thought to be too complicated. In addition to pointing toward a possible physiological pathway, the *Nature Genetics* piece offered conceptual and methodological avenues for subsequent investigators to pursue. In this case, the pursuit of the diabetes genes would be a collective one where data sets would be shared and findings discussed at regular meetings.

Of interest to this discussion are the ways scientists labeled their data sets. How do scientists use ethnoracially classified DNA? Do the labels change over time? Do standard labeling practices exist? How are race and ethnicity understood in this context? Most specifically, how is "Mexican" used in genetic epidemiology of type 2 diabetes? Ethnographers of science have long established that robust attention to the ways scientist actually practice the making of knowledge renders intelligible otherwise opaque sociocultural processes, products, and concepts. By attending ethnographically to the heterogeneous use of DNA labeled with ethnoracial terms, I was able to assess how race and ethnicity actually are crafted (Fujimura 1996) into these groundbreaking bioscientific findings.

As would be expected, the data sets collected and exchanged by members of the diabetes alliance are organized by geography and by population. They are also organized by sampling technique, by institution, and by the diagnostic criteria used to select research subjects. Racial and ethnic labels define the contents of vials of DNA, and also appear on shipping cartons and lab notebooks. The monikers include, "Native Americans," "whites," "African Americans," "Japanese Americans," and "Mexican Americans," with "Amish" and "Pima" as subset populations for whites and Native Americans, respectively. Yet the written use of these labels does not tell the whole story. In fact, in collaborative discussions among researchers the social meanings of these labels emerged. For example, while discussing a data set for inclusion in an experiment, one researcher interrupted a colleague who was suggesting that Puerto Rico might serve as a source of DNA that could be compared with Mexican American samples because of similar admixture frequencies. "No, the native populations on Puerto Rico were wiped out, so that [comparison won't work]," remarked the

researcher. The interrupting researcher challenged the apples to apples comparison by arguing for the biogenetic heterogeneity between Puerto Rican and Mexican Americans. What is of interest here is that the reference to genocide signals a more socially nuanced understanding of the categories "Puerto Rican" and "Mexican American."

To be sure, the objecting researcher was thinking about the Native American admixture of both Puerto Ricans and Mexican Americans, arguing that claims of homogeneity would require similar histories of interbreeding between all three groups, (see Montoya n.d.). And although such narratives are predicated on sophistic genetic standards for what counts as Mexican, Puerto Rican, and Native American, to dismiss this as an instance of simple racialization would miss the ways social histories are being deployed in this interaction. The objections were founded on the researcher's understanding of social, not biological difference. That is, that the biogenetic similarities between Puerto Ricans and Mexicans could not be justified because their social histories differ. It is important to recognize that in this instance only social history could differentiate the two groups. Otherwise, the researcher would have referred to a specific Puerto Rican marker as absent in the Mexican American group and visa versa. I return to this in a moment.

In another instance, a researcher referenced an aspect of social history to inform the selection of populations for research. At one meeting I attended, a guest lecturer suggested that the people of Haiti were the ideal ancestral proxy for African Americans because Haiti, the researcher argued, was a hub of the slave trade where many Africans remained. The researcher's suggestions that Haitians are more genetically pure than African Americans were followed by inconclusive talk about who was sampling in Haiti and how those samples might be secured for genetic analyses. However, unlike in the case of native Puerto Ricans, the construct of Haitian genetic purity, the geopolitical role of Haiti for the slave trade, and the utilitarian manner in which researchers discussed the acquisition of Haitian blood samples were never questioned. Still, as in the case against Puerto Rican DNA, it is sociohistorical knowledge that is deployed to argue for the genetic utility of Haitians. Haitian social history is conscripted into the genetic rationale for their use in research

Over the course of conducting participant-observation with scientists, there were other moments when anthropologically informed questions would rupture the otherwise "black boxes" of population labeling. Often after a meeting between scientists, I would pull one aside for a debriefing. Among the questions I would ask were queries about data sets and methods. Once, when I questioned the use of skin tone as a measure of ethnicity, one epidemiologist remarked, "Well, yes, skin tone is just one measurement among many that we use. We know the classification is not ideal, but we had to have some kind of replicable measurement." Another researcher remarked,

"Hey, these are the labels given to us by [the researchers doing the sampling]," after I asked why they classified the populations as they did.

Researchers, when directly queried, do not argue that the labels represent biological differences. Theirs is a pragmatic claim.[11] Although imperfect, the classificatory labels have coherence. Why are population data sets so important? The simple answer is that the more genetically homogeneous a population is, the easier it is to detect genetic variation that is statistically more likely to be related to a trait of interest. In trying to find diabetes genes, researchers must conduct case control comparisons; they must compare the genotypes of people with diabetes with those without diabetes. However, they need family data to decrease the likelihood that the nondiabetic is not also a carrier—that is, a person with the same genotype who has not (yet) manifested symptoms. Researchers compare the gene variants at a particular region of the chromosome or they compare genome wide scans to detect differences between diabetic and nondiabetic people. The differences between them are regarded to be the genetic contributions to disease susceptibility.

But this would not control for what is called population stratification, and here again the distinction between descriptive and attributive differences becomes evident. Good geneticists must work to ensure that any genetic differences they find between affected and nonaffected individuals are not simply attributable to the different evolutionary histories of the groups, an effect known as "population stratification." When, for example, geneticists compare so-called "whites" with "African Americans," they may find that an anonymous genetic marker occurs at a significantly higher frequency among diabetics in the "white" population. Call this marker SNP-W, because Single Nucleotide Polymorphisms (one random change in a string of DNA nucleotides) are a very useful tool in these kinds of investigations. SNP-W is probably not a gene, probably has no known function in the mechanisms of diabetes as currently understood, but may be very near an undiscovered gene that may play a role in the disease; its use is merely a descriptive one, to indicate that a small chromosomal region containing SNP-W is found, not always, but at a significantly higher frequency in diabetics among the population categorized as "white" than in nondiabetics of that population, or in any member of the "African American" population. So for example it would be a mistake to attribute diabetes to SNP-W, just as it would also be a mistake to attribute SNP-W to the members of the descriptive category "white," that is, defining "whites" by the biological fact of harboring SNP-W in their genomes. Moreover, this simply descriptive association between SNP-W and diabetes in the "white" population may be spurious, once population stratification is taken into account.

According to diabetes scientists (and some physical anthropologists), each population has stratified according to evolutionary histories that are different enough, one

from the other, to confound genetic analyses.[12] So SNP-W, to continue our example, may appear in more diabetics in the "white" population because it appears with greater frequency within the "white" population used for the study than it does within the "African American" population used as a comparison group for this study. The differences are not for any physiological reason, but simply because the random difference SNP-W has been passed along as part of the distinct reproductive history of the group of "whites" used for that study.

Thus, geneticists explain, by using population data sets like "white" and "African American," the likelihood that the observed genotypic differences between diabetics and nondiabetics are simply caused by different ancestries in the two populations is minimized, making meaningful variants easier to detect. But for this very important analysis of population stratification to work, researchers must stipulate robust criteria for the population categories "white" and "African American;" the difference lies in deploying them either descriptively, for pragmatic analysis, or attributively, for positing some essential difference. However, because ethnorace is an ill-defined biomedical construct that for diabetes genetic epidemiology operates at times descriptively and at others attributively, the robust criterion for population differences are nowhere to be found.[13]

The rationale for the use of these population categories is occasionally problematized by consortium members—on their own or through ethnographic interlocution. None of the scientists with whom I spoke openly argued that their use of population specific data reiterated biological distinctions between humans. Yet neither did they directly acknowledge the fundamental contributions of social and cultural factors to the labels used to identify the populations.[14] The scientist's social differentiation of Puerto Rican and Haitian samples as a means to evaluate their genetic value illustrates the simultaneous registers (social and biological) of bioethnic conscription in the diabetes enterprise. This is not to say that biological human variation is not real. Rather, the more challenging task is to explain how the biological and social registers of race and ethnicity are intertwined or conscripted in biomedical sciences of complex disease.

Some social scientists argue that the science of population variation and genetic medicine often involve what Lee et al. (2000:37) label a "naïve genetic determinism." That is, the minute genetic differences discernable with SNPs, patterns of single nucleotides (A,G,T,C), and other mutation analysis technologies are now used to explain patterns of disease between populations, which are in turn understood as the basis for biological differences between the populations themselves. The case of diabetes genetics research affords a more nuanced look at what is labeled genetic determinism. It is evident in diabetes research that SNPs and haplotypes, (an inherited pattern of

SNPs) are construed as de facto determinants of diabetes, although most diabetes researchers are reticent to make this claim. Further, as the discussions of Puerto Rico and Haiti reveal, genetic differences between ethnoracial groups are presumed to bear attributive significance as well, that is, they can be used to differentiate between the groups. However, the varied social and historical forces that shape Haiti and Puerto Rico, (slavery, colonialism, conquest, and genocide) do not get entirely lost in this process. How can we account for such apparent contradiction? To answer this, it is first necessary to briefly review the political contours of race and ethnicity within biomedical sciences and the epidemiological characterizations of type 2 diabetes.

RACE IN MEDICINE, SCIENCE, AND GENETICS

It is almost cliché to speak of race and ethnicity as unstable sociocultural formations subject to contestation (Gilroy 1991; Gregory and Sanjek 1994; Haney López 1996; Harrison 1995; Omi and Winant 1994). Increasingly, debates in the life and medical sciences invoke differing opinions on the biological and genetic basis of race. On one side of the debate are those who argue that distinct races do not exist (Gould 1981; Lewontin et al. 1972, 1984; Marks 1996, 1998; Templeton 1998). These "no-race" researchers contend that there is no biological justification for so-called racial groupings. Drawing on decades of population genetics research, they conclude time and again that variation between members of each so-called "racial group" exceeds variation found between groups. Arguing against biological notions of race, anthropologist, Jonathan Marks writes,

> Biological variation exists within the human species, and some of it is structured geographically. But this component of our biological variation is (1) very small relative to the total and (2) not patterned in such a way as to permit the formalization of a reasonably small number of natural "races." To the extent that we popularly identify such clusters, they are the result of cultural impositions of meaningful distinctions on nature, a classically anthropological example of a "folk taxonomy." [Marks 1998:4]

The "no-race" school of thought argues against those who claim that racial and ethnic classification is based on complex biological or geographical factors between and within different populations (Cavalli-Sforza, with Menozzi and Piazza 1994; Mitchell et al. 1993; Molnar 1998). Molnar, for instance, writes in *Human Variation: Races, Types, and Ethnic Groups,* "The term race or population is used to refer to that geographically and culturally determined collection of individuals who share a common gene pool" (1998:33). Vogel and Motulsky, in *Human Genetics: Problems and Approaches* write "A race is a large population of individuals who have a significant proportion of their

genes in common and can be distinguished from other races by their common gene pool. . . . Subdivision into three main races, Negroids, Mongoloids and Caucasoids, is accepted by practically all observers" (1997:610–611). Biological Anthropologist Cavalli-Sforza is more to the point, "a race is a group of individuals that we can recognize as biologically different from others" (2000:25).

In tournaments for academic authority, competing scientific arguments are normal (Latour 1987). For those interested in making race work as a scientifically valid concept there are at least four kinds of discussions. There are general discussions of the merits of race as a proxy for biological differences (Cavalli-Sforza 2000; Marks 1998; Mitchell et al. 1993; Molnar 1998). There are scholars who attempt to make a contribution to a methodological or theoretical problem with race or racial labels (Burchard 2003; Hahn with Mulinare and Teutsch 1992; Risch et al. 2002). For example, Hahn with Mulinare and Teutsch (1992) demonstrated the instability of racial labels, whereas, conversely, Risch and others (2002) argue for the statistical fit between self-reported ethnicity and genotype. There are also discussions of the consequences of racial thinking in medical or scientific practice for health policy and clinical care (Kahn 2004; Kaufman and Hall 2003; Schulman et al. 1999). Additionally, there are numerous assessments of the ethical implications and proper use of race and ethnicity in science and medicine (Chaturvedi 2001; Kaplan and Bennett 2003; Lee et al. 2001; *Nature Genetics* 2000; Schwartz 2001; Shields et al. 2005).[15] To make matters more complicated, discussions of race and ethnicity are plagued by the social semiotic nightmare that is the long and ever-changing list of labels and their referents. Within these ethical warnings, scholarly exchanges, and technical debates, it is often a combination of issues that are under consideration.[16]

The landmark study by Hahn with Mulinare and Teutsch (1992) astutely pointed out that the fluidity of the U.S. folk taxonomies and their associated phenotypes make both unreliable for biomedical research. Further, Goodman (2001) clearly and succinctly highlights the conceptual and methodological inaccuracies in science that uses ethnoracial variables of all sorts. Yet the use of ethnoracial labels, data sets, and groups continues unabated, even among careful and socially thoughtful scientists. Debates about race and ethnicity cannot be reduced to a race—no-race binary proposition.[17] The issues cannot be resolved by an appeal to technical merits, to an ethical position de jure, or to methodological or moral claims of rectitude. Race and ethnicity are ideological, in Althusser's sense of the term (Althusser 1971), operating as a system of ideas and representations about human difference that—as shown here—work within and are sustained by both scientific and sociopolitical registers.[18]

Sociopolitical registers of race and ethnicity are conscripted into the production and consumption of biomedical knowledge about type 2 diabetes at multiple stages of

the research process. The life conditions of groups labeled with social terms (Mexicana/o, Puerto Rican, Haitian, European) are pressed into service of the biomedical explanation of disease. These are neither simplistic racist maneuvers by ignorant scientists nor purely racializing (Miles and Brown 1989) discourses that confer biological differences on social groups, although such maneuvers and discourses can certainly be found within the diabetes research enterprise. Understanding the practices and multiple stages at which social and the biological registers of ethnoracial human differences intertwine is therefore critical. What I call "bioethnic conscription" is a practice wherein the social conditions—past and present—of Mexicana/o and other groups' lives are folded into the biomedical registers of diabetes knowledge.

In the remainder of this article, I (1) present past and current theories of diabetes causation that inform researchers today, (2) explore the social and historical context of DNA sampling, (3) analyze the routine use of population DNA by diabetes researchers, and finally, (4) evaluate the representation of human difference in diabetes publications and pharmaceutical marketing materials. Examining these instances of bioethnic conscription illuminates the means by which the always already social constructs of ethnic differences are conscripted into biomedical discourse.[19]

RACIALIZED ETIOLOGIES OF DIABETES

Diabetes is not one disease but many. More than 90 percent of all diabetics have type 2 diabetes, which is characterized by elevated blood glucose triggered by a combination of poor insulin production, insulin resistance in skeletal muscle and lipid tissue, or both. Type 2 diabetes is also known as Non-Insulin-Dependent Diabetes because, unlike the rarer form of the disease, people with type 2 diabetes produce insulin and therefore seldom need therapeutic insulin at the initial onset of disease. Type 2 diabetes (hereafter, "diabetes"), like heart disease, hypertension and asthma, is referred to as a complex disease because its putative determinants lay in both environmental and biological domains. That is, diabetes is caused by a still-unknown combination of factors that include lifestyle, diet, physical activity, and an array of physiological triggers.

According to the U.S. Centers for Disease Control and Prevention (CDCP) diabetes is the seventh leading cause of death in the United States (CDCP 2000) and is frequently referred to as a public health crisis in government and academic publications as well as the popular press. The World Health Organization (WHO) has called diabetes an emerging epidemic with over 16 million people affected in the United States and millions more in the rapidly urbanizing southern hemisphere and China. According to the CDCP, by 2025, 270 million people worldwide will have diabetes.

The pervasiveness of the rhetoric of diabetes as a public health threat is evidenced in the September 2000 issue of *Newsweek* magazine (Adler and Kalb 2000). The cover story, entitled "An American Epidemic: Diabetes," details the rise of diabetes among Americans. It featured (in the online version) a photograph of Yolanda Benitez, a middle-aged woman with diabetes. The photo shows Benitez heating a tortilla on a comal. The front page text reads, "Something terrible was happening to Yolanda Benitez's eyes. They were being poisoned; the fragile capillaries of the retina attacked from within and were leaking blood."

The main health concerns for people with diabetes, as the story of Benitez illustrates, are the complications. According to the CDCP, diabetes is the leading cause of blindness in the United States among adults aged 20–74 years. In fact, the CDCP estimates that adding the costs of diabetes complications—eye disease ($470 million), kidney failure ($842 million), lower extremity amputations ($860 million)—to the costs of controlling glucose ($100 million) the costs of diabetes in the United States alone exceeds $2.7 billion annually (CDCP 2000).

Diabetes is also framed as an ethnoracial disease. In fact, it is hard to find a discussion, popular or scientific, about diabetes without a discussion of its differential impact on certain ethnic groups. A CDCP publication reads,

> The burden of disease is heavier among elderly Americans—more than 18% of adults over age 65 have diabetes—and certain racial and ethnic populations, including African Americans, Hispanics/Latinos, and American Indians and Alaska Natives. For example, American Indians and Alaska Natives are 2.8 times more likely to have diagnosed diabetes than non-Hispanic whites of similar age. [CDCP 2000:2]

Similarly, a U.S. Public Health Service (PHS) grant application submitted by one scientist I worked with begins its "Background and Significance" section with the differential prevalence patterns of diabetes in populations defined with ethnoracial taxa. It reads, "Type 2 Diabetes has an estimated prevalence of 6–8% in White populations, 10–12% in African Americans, and 15–20% in Mexican Americans." The proposal cites an array of scientific references for each population. Additionally, the *Newsweek* (Alder and Kalb 2000) story describes Benitez as a "representative victim" of diabetes, citing the usual ethnoracial statistics.

Why are ethnoracial differences part of the requisite narrative of diabetes in venues as diverse as government publications, scientific journals, grant applications, and the popular press? If we use population estimates to figure the total number of diabetics by ethnoracial group as defined by the U.S. Census Bureau, 6–8 percent of the U.S. white population is 18 million people.[20] In raw numerical terms, 18 million white diabetics

is four times greater than all African American diabetics, three times greater than all Hispanic diabetics of any ethnicity, and 37 times greater than the number of American Indians and Alaskan Native diabetics.[21] Overall there are 1.6 times as many white diabetics as African American, Hispanic, and Native American diabetics combined. The representation of diabetes as a disease that afflicts some ethnoracial groups more frequently or "harder" than others reflects the unequal weight given to the prevalence rates of diabetes over the raw incidence numbers. This representational slippage is emblematic of a series of practices, examined below, that configure people of color as diabetes prone. The CDCP and WHO report—accurately—that diabetes affects different populations at varying rates. Yet the numbers merely describe the patterns of increased incidence of disease in various populations, whereas the common reading of these different incident rates easily and frequently slides into a presumption of some biological difference.

Although the number of disease conditions for which the biomedical literature reports positive indications of genetic contributions increases weekly, diabetes has enjoyed a relatively long history of geneticized explanations. Medical geneticist James Neel's (1962) famous thrifty genotype hypothesis, for example, postulated that in the early stages of evolution those people who had a "quick insulin trigger" could rapidly convert sugar to fat in times of famine. Accordingly, peoples who have recently undergone a shift from hunter-gathering to a modern sedentary lifestyle (with concomitant energy dense food intake) are at increased risk of diabetes because they still carry genes that conferred this selective advantage. "The Coca Colonization" hypothesis (Zimmet 1997), as the thrifty genotype hypothesis is sometimes called, posits that recently "primitive" groups have undergone a "domestication of lifestyle" as they have moved to urban areas or lost their old way of life (Neel 1962, 1982; Zimmet 1982). According to this hypothesis, these populations have, over time, evolved genetic traits that could metabolically compensate for periods of food scarcity. Because such scarcity is no longer the norm, the theory contends, the phenotypic consequence of thrifty genes in combination with the abundance of food and sedentary lifestyle typical of contemporary urban living make for impaired metabolic regulation of glucose. In other words, diabetes is thought to result from a genetic anachronism.

Neel's thesis is predominantly an environmental hypothesis. That is, it is the different evolutionary environments of certain groups that confer present day risk. His published statements evince an uncanny reflexive modesty. For example, in revisiting his (Neel 1962) hypothesis 20 years later, Neel concludes that, "although incorrect in (physiological) detail [i.e., that there is a quick insulin trigger], it may have been correct in (evolutionary) principle [i.e., that there are discernable genetic differences among

populations]" (1982:284). Neel concludes his revision with the following cautionary invitation:

> All these speculations may be utterly demolished the moment the precise etiologies of NIDDM [Non-Insulin-Dependent Diabetes Mellitus] become known. Until that time, however, devising fanciful hypotheses based on evolutionary principles offers an intellectual sweepstakes in which I invite you all to join. [Neel 1982:290]

In perhaps his last written statement on the thrifty genotype hypothesis, Neel writes that there is "no support to the notion that high frequency of NIDDM in reservation Amerindians might be due simply to an ethnic predisposition—rather, it must predominantly reflect lifestyle changes" (Neel 1999:S3). In spite of this, many genetic epidemiologists argue that genetic differences explain rates of diabetes between different populations. For example, drawing on research with Mexicanos/as, one diabetes consortium member writes, "there is strong evidence that Mexican Americans living in the barrio have considerably more Native Amerindian genetic admixture and as a result may have higher genetic susceptibility to diabetes" (Stern 1999:S67). "It smells and tastes like a thrifty gene in terms of its metabolic function," remarked one molecular biologist interested in the protein implicated in a genetic study of diabetes.

Genetic explanations for diabetes vie with conventional epidemiological analyses that rely on the predictive capacity of social and environmental data. For example, in a critique of the thrifty genotype hypothesis, social epidemiologist Robyn McDermott (1998) shows how the conflation of genes and race within this paradigm elides the consideration of the social determinants of the disease. McDermott concludes,

> Diabetes in Aborigines has been defined, by non-Aboriginal scientists, simply as a problem of "race" and "genes" in a changing environment. Race becomes a biological entity and an independent risk factor, reified over and over again in repeated studies of disease which take no account of socioeconomic status, history or culture. [1998:1193]

Interrogating the thrifty genotype hypothesis as biological determinism, McDermott counters with evidence from numerous studies that strongly suggest sociological explanations for diabetes. For example the "Dutch famine" (Ravelli et al. 1998), forced labor, exile and starvation of the Nauru during World War II (Hales and Barker 1992), maternal deprivation, and poor childhood nutrition (McCance et al. 1994; Pettitt et al. 1993) followed by overnutrition and obesity in the Pima all result in higher than expected rates of diabetes and other diseases.

Hales and Barker propose a "thrifty phenotype " hypothesis of diabetes arguing "poor nutrition in fetal and early infant life are detrimental to the development and function of the Beta cells" (1992:600), especially when coupled with childhood food deprivation and later caloric excesses. Although the specific causal mechanisms are still being investigated, the principles behind the fetal origins hypothesis, later life conditions and health outcomes have proven to be robust (Leon 2004; Rasmussen 2001). Such studies offer support to an extensive body of research that demonstrates the physiological effects of poverty, unequal access to health care, discriminatory experiences and social inequality for predicting low birth weight, poor fetal and postnatal nutrition, as well as complex diseases like heart disease, diabetes, and hypertension (Auerbach and Krimgold 2001; Cooper and David 1986; Krieger 1999; Wilkinson 1992; Williams and Collins 1995).

The epistemological differences between those who rely on genetics and those who analyze social and environmental data can not be resolved by alternate hypotheses, however. They are not sufficiently explained by appeals to improved DNA data sets, nor should these competing explanations be written off as simply the disease research imaginary that has resulted from the hype around the Human Genome Project(s).[22] In fact, both kinds of analysts appeal to prevention and etiological explanation as the central motive for their work. For example, geneticists who use DNA from populations with high prevalence rates of diabetes speak of causality and prevention as the primary motivation of their work. "Our work will benefit the cohort born today," explained one geneticist. "Look, we're doing the easy part. The difficult task will be identifying the physiological pathways [for which new drug therapies could be designed]," remarked another. One researcher commented that he simply wanted to understand the biology of disease in humans.

My purpose here is not to offer an alternative causation hypothesis or to critique the epistemological shortcomings of genetics or epidemiology (see DiGiacomo 1999; Frankenberg 1993; Garro 1995; Hahn 1995). What I can provide is an ethnographically informed critique that carefully examines the logics that orient and the practices that ground these debates as they play out in diabetes genetic epidemiological research. To this end, I turn to the circulation of racialized concepts about diabetic groups as they appear in DNA data-set discussions, publications of a genetic discovery, and drug promotional campaigns.

SOCIAL HISTORY OF MEXICANA/O DNA

The primary DNA data set used by diabetes researchers in the consortium I studied is derived from samples from Mexicanas/os from Sun County, Texas. Sun County is one of the poorest regions in the country. The U.S. Census Bureau estimates that half

of Sun County's population lives in poverty and estimates that nearly 60 percent of the county's children live in poverty (U.S. Census Bureau 2000). Sun County is known as one of the roughest drug portals on the U.S.–Mexico border. It is also known for its close multigenerational families and an indifference to the formalities of border crossings, a reflection of the persistent tenuousness of the U.S. nation-state along the border and elsewhere (Acuña 1981; Almaguer 1994; De Genova 1998; Montejano 1997).[23]

Sun County's unemployment rate, which hovers around 30 percent, has been precipitated by historical shifts in agricultural labor practices, shifts that are deeply imbricated in the formation of the U.S. frontier, in the economic development of the U.S. Southwest and in the stratified race relations between Anglos and Mexicans in the region. In particular, over the past 50 years, shifts to the pesticide laden, mechanized production practices of corporate agribusiness (Montejano 1997) have led to an influx of farmworker families into the urbanized areas of Sun County. Presaging the current refrains about Mexicana/o laborers, one Sun County land development agent, Taylor, remarked in 1931, "The white people won't do the work and live as the Mexicans do on beans and tortillas and in one room shacks," (Montejano 1997:199). Similar rationale, but with kinder, gentler rhetoric, is used to justify current efforts for a new worker amnesty or bracero program.

Anthropology and the subfield of medical anthropology have long offered useful approaches to phenomena that are biologically and socioculturally entwined, such as race, health, disease, evolution, acculturation, adaptation, and more recently biology and medical knowledges (Singer 1998). Further, diabetes has long captured the attention of researchers interested in the relationship between lived conditions and the disease (Garro 1995; Szathmary 1990, 1994; Rock 2003, 2005). This assessment of the use of Mexicana/o DNA used by researchers to find biogenetic susceptibilities for diabetes reveals the epistemological shortcomings of reducing diabetes to a biogenetic condition somehow linked to specific population ancestries but devoid of data on lived conditions. Ironically, the use of data sets shows the fundamentally sociocultural bases of biomedical knowledge of chronic disease even when biogenetic explanations are sought.[24]

Considering then the conditions of poverty, dispossession and capital dislocation, Sun County diabetes etiology displays a glaring sociological register.[25] Access to health care is difficult, insurance a luxury. There are no health clubs, public swimming pools, or bike paths. As one Mexicana diabetic remarked, "Es que, people are always saying that we should exercise more. But where are we supposed to do that? The roads aren't safe to walk on, snakes and dogs are everywhere, and for much of the time it is too hot to be outside." In this light, diabetes is a profoundly social condition for Sun County

residents—a condition brought about by forced migration from rural to urban settings, by an overdetermined reliance on certain foods or foods with limited nutritional value, by the restrictions of physical movement required of underpaid wage labor and the undercapitalized infrastructure that accompanies it, and by unequal access to health care.[26]

The first-order instance of the ways Sun County's political history and the current conditions of life in the *colonias* are being conscripted into a biological discourse occurs through the identification and selection of the population to be studied. By the mid-1970s, Sun County had caught the attention of epidemiologists who documented that of Texas's 254 Counties, Sun County had the highest rates of diabetes. By 1983, the *American Journal of Epidemiology* reported that Sun County "Hispanics" were three to five times as likely to have diabetes as the general U.S. population. Even the researcher who gathered the early epidemiological data and continues to collect DNA and provide free screening to Sun County residents admits that poor access to health care is the primary cause of diabetes in Sun County.

Nonetheless, "evidence" for the genetics of diabetes risk is mounting, often at the expense of understanding the social context and determinants of the disease. Biogenetic views tend to trump sociological views in the diabetes research imaginary of consortium members. However, the genetic epidemiologists who make up part of the diabetes consortium are not ignorant of the effects of proper diet and adequate exercise. "Take away the television and the automobile and diabetes would all but disappear," quipped the head of one lab. Neither are researchers unsympathetic to those who suffer from social inequality in the United States. Their career and intellectual interests lie in genetic explanations of diabetes, which, as I aim to show in this discussion, involves folding political and economic social relationships into biomedical discourse. In fact, the case of diabetes genetic epidemiology illustrates how, in spite of the sympathies of diabetes scientists, arrangements of racial inequality in the United States find their way into diabetes research publications and drug company promotional campaigns. To illustrate this phenomenon further, I present two tales from the field, one dealing with the naming of a publication article, the other with the marketing of a diabetes drug.

WHAT'S IN A NAME?

Compelling examples of how social valences of Mexicana/o ethnicity are conscripted into biological registers occurred when findings from the polygene "discovery"—describer earlier—were under review for publication. The quantitative geneticist who endured me the most during my fieldwork and whom I will call "Nora" was neither ignorant of the social history of donor populations nor immune

to the realities of U.S. race relations. She asked me to lunch one day because she was concerned with the way Mexican Americans were being represented in her work. That week her computer simulations suggested that a particular combination of genetic material related to Mexicano Native American admixture increased the carriers' susceptibility to diabetes.[27] She wondered aloud to me if someone might use her findings in a way that could negatively impact Mexican Americans. I asked her if she was worried about possible objections—mine or some unspecified reader bent on feeding an explicitly racist agenda. "I wouldn't want to do anything that could be used by someone to negatively portray the Mexican Americans who donated their time and DNA for our analyses," she clarified. She meant her DNA sources. The following day, Nora's principal collaborator and office mate, whom I call "Gary," addressed the issue at his lab meeting. "You know, we should be careful. These things are sensitive. . . . It could be construed as an argument for anti-miscegenation and such," Gary remarked.

About six months before the *Nature Genetics* piece was published, my field note–taking had been interrupted by groans of disgust from both Nora and Gary. Both of them, who shared an office, had just simultaneously read an e-mail from the editor at *Nature Genetics* who had been assigned to their submission. "We've had trouble getting published even though we've replicated our finding in a sample of Mexican Americans," Nora explained. Their anger palpable, Nora and Gary talked over each other to explain to me how their original paper had been population neutral; it had not labeled in any way the population from whom the DNA had come. *Nature Genetics* had told them that because the DNA only came from Mexican Americans, the title needed to reflect this fact. Nora and Gary were incensed that their work was being ghettoized in this way. "We do human genetics, for christ sake," Gary grumbled. For a human geneticist, "human" was equivalent to "universal." "They are treating millions of Mexican Americans and the millions more Mexicans like the Amish, some small population isolate!" Two collaborators, a Swedish and a German researcher, had confirmed the findings in their own population samples. The polygene was also found in these European populations, albeit at a lower frequency.

"And now," Nora said with sarcastic emphasis on the "now," "we are told that because our work has been confirmed in two white populations, that we can take the 'Mexican American' out of the title of our paper." Gary's and Nora's displeasure was accompanied by a sense of relief that one of the impediments to publication had been overcome and publication was nearer. However, their objection to the title change was an ethical one. Gary and Nora inferred that *Nature Genetics* had allowed them to take "Mexican American" out of their title based on the "confirmation" studies because in the eyes of the journal's editors what had been a Mexican American genetic condition had been transformed into a universal human condition. Nora and Gary were clear that

the editorial coercion was wrong and that their work needed no such confirmation. Further, their last and most biting objection to the editor's titling requirement was that if it only affects Mexican Americans, then it is not fit to publish in *Nature Genetics* unmarked by race and ethnicity. But if it is a genetic condition found in "whites," that makes it a human gene and thus worth publishing. "It's not replication they wanted, it's confirmation," Gary said cynically.

In this last complaint, Nora and Gary were clearly unloading some of their frustrations at the delays in publication. Past experience with article and grant reviewers had not always been pleasant and several consortium members commented that competition has become downright nasty at times. However, that Nora and Gary marked the irony of the editor's concern, with its implicit denigration of Mexican Americans as unfit for stand-alone genetic evidence, speaks to Nora's and Gary's recognition of the political and social implications of scientific knowledge production. Not only was the titling requirement an insult to them as professionals but it also referenced the wider context of race relations in the United States. Their disagreement with the editor marked an instance in which scientists worked to counter biomedical claims wherein certain bodies count more than others, where knowledge about so called "white" bodies holds more scientific weight and thus sells more journals, and where subtly pernicious notions of Mexicans as a racial human subspecies persist.

The same month that Gary and Nora informed me of their exchange with *Nature Genetics,* the journal published an editorial explaining a new policy on the use of race in their publication. Citing the American Anthropological Association's statement on race, *Nature Genetics* noted that "race" is not a scientific term and that it is commonly used with poorly defined lay terminology. The editorial states that, as a consequence, *Nature Genetics* would henceforth "require that authors explain why they make use of particular ethnic groups or populations, and how classification was achieved" (2000:98). In Gary's and Nora's article, estimates of population structure were broadly specified in genetic frequency language and Sun County was mentioned as a relatively homogeneous population. Other populations used in their paper (Pima, German, Zapoteca, Bosnia) were "specified" to a similarly vague degree. Furthermore, the justification for the use of population DNA was implied and the reasons for using these specific populations were not detailed. Hence, I am not arguing that Gary and Nora are actively situating (Haraway 1988) their knowledge claims. Rather, I am offering an instance in which the artifice of the social and biological divide is being actively worked on in the production of a biomedical claim. That is, this was a moment in knowledge production in which both the sociocultural and the biological bases of human variation were at stake, a moment easily missed when cultural analysts begin their work expecting to find simplistic racialized genetic determinisms.

For these scientists, the quibbling over the words *replication* versus *confirmation* is not trivial: Replication requires reproducibility of a finding whereas confirmation in this case required broader application of the finding to another human group. Wrangling over the two is about the struggle over what counts as scientific knowledge. The ethical and political struggle over the title of Gary and Nora's article is an instance of knowledge production that is cognizant of subjugated positions and the responsibility of science. Nora's concern about the characterization of Mexican Americans seemed to be operating with what Donna Haraway describes as a "power sensitive conversation" (1988:591) and with a well-formed sense of the responsibilities of positioning. It is a form of knowledge making that does not blindly rely on the "god trick," the force of objectivity, but rather on the strategic positioning of the scientist, of their work product, and of the lives of the people whose donated DNA made the knowledge work possible. In other words, Nora and Gary were deliberate in their representation of Mexican Americans and paid special attention to the consequences of the knowledge they produced. Reflecting on the potential miscegenation messages that could be read into his and Nora's piece, Gary remarked, "We only included what was absolutely necessary [in our article]." Gary was referring to their exclusion of any specific reference to genetic admixture of DNA donors even though it is the alleged mixing of "Native American and Caucasian" genes that make the polygene possible in the first place (see Montoya n.d.).[28]

The labeling controversy demonstrates the complex difficulties of bioethnic taxonomies in genomic sciences, taxonomies that conscript both the natural and the social registers. Not only did Nora and Gary's protests actively disrupt the editorial practices that stratify (biosocially) "whites" and "Mexicano" but their protestations also signaled their politicized engagement as genetic scientists with the contested boundaries of what counts as human difference, and who counts as "human."[29] This is a story often missed by the strict "no-race" school of thought adherents for whom any use of racial labels is simplistically portrayed as cut-and-dried racist science. Still, Gary's and Nora's disavowal of the need for "white" confirmation studies makes this case not less but more illustrative of the process of bioethnic conscription. Had Mexicana ethnicity truly been a "black biological box," there would have been no need to contest the editorial requirements that "Mexicano" be part of the title. Gary and Nora would have simply acquiesced to the editorial requirement and left intact the sociocultural implications—that Mexicanos are some kind of human subspecies.

FROM SCIENCE TO MEDICINE

Bioethnic conscription occurs outside of the laboratory and in other contexts. In the case that follows, I move from scientific representation of Mexicano ethnicity

FIGURE 1. **The image conveys the multiple meanings of cutting blood sugars, harvesting sugar cane, and the social relationships of plantation economies. The advert enlists the ethnic body as a market for the drug. The image thus conscripts the social conditions of sugar consumption and health inequalities into the biophysiological condition of high blood sugar. Copyright 1999, Hoechst Marion Roussel, Inc. Photo by Michael Montoya.**

to the biomedical representation of ethnicity more generally in a particular diabetes drug promotional campaign.[30] While attending three annual scientific meetings of the American Diabetes Association (ADA), I could not escape noticing the marketing and promotional tactics of dozens of companies. Like most scientific conferences, corporate logos, products, and "drug reps" abound at the ADA meetings. Amidst the visual bombardment of product placement that nearly envelops the conference venue, the ad campaign for Amaryl® stood out (see Figure 1).[31]

The Amaryl® ad appeared on the back of *Diabetes,* the American Diabetes Association's Annual Meeting Program and Abstract book. Its colorful full-page artwork depicted a man wielding a sickle midstalk on a sugar cane. Behind him, purple mountains accentuated by white cumulus clouds starkly contrasted with the orange sunset and thick cane field in the foreground. The block-relief artwork, more cartoon style than realist, showed this lone caneworker filling the clapboard bed of a trailer with downed canes headed for processing. The worker had been busy. Hillsides of cleaved cane stubs marked the midground landscape, between the distant mountains and the stacks of cane in the trailer.

The maker of Amaryl® is Aventis Pharmaceuticals. The pharmaceutical division's principal area is prescription drugs, contributing about three fourths (13.9 billion

Euros) of total Aventis SA sales, with the remainder coming from Aventis's agricultural, animal nutrition, and other life sciences products. It is difficult to say what Aventis Pharmaceuticals spent on this one ad campaign. However, the Amaryl® brand permeated ADA's conference materials for 1998, 1999, and 2000.[32] The caneworker image appeared on the back of the program and abstract book for the conferences. It was one of only 16 color ads in the 550-page volume. It also appeared on glossy brochures and T-shirts, and in numerous journals. The Amaryl® name and its chemical name *glimepiride* were branded to the backs of the conference name tags, exhibition hall ID cards and numerous other items. This ubiquity could explain the overwhelming success of this ad, as measured by product recognition studies referenced by Aventis employees I spoke with at the meetings.

The Amaryl® ad shifts the meaning between the biological need addressed by the drug—its ability to reduce blood glucose levels—and the bodies the ad depicts while capitalizing on the social semiotic cachet that the image carries. For example, stylistically the ad evokes the popular Mexican protest art of the Taller de Grafica Popular. Its relief print motif could have just as easily been employed to protest monocrop agriculture or indentured labor.[33] The advertisement's vibrant orange, yellow, brown, and green evoked the palette made popular by the artist Diego Rivera. Recalling the "thrifty genotype," often referred to as the "Coca Colonization" hypothesis, the setting was clearly a plantation, somewhere in Latin America or the Caribbean, with a lone brown-skinned laborer who works to feed our appetite for sugar. It was both an image of a place and of a relationship in which, in anthropologist Sidney Mintz's words:

> Europeans . . . imported vast numbers of people in chains from elsewhere to work. These [people] would be, if not slaves, then men who sold their labor because they had nothing else to sell; who would probably produce things of which they were not the principal consumers; who would consume things they had not produced, and in the process earn profit for others elsewhere. [Mintz 1985: xxiv]

"It's cutting sugars," the Aventis Product Manager for Amaryl® assured me after I queried him about the ad campaign, its history and the meaning of the image. Standing in the middle of the Amaryl® exhibit in the ADA Scientific Meeting's "product theater," we examined the five-foot tall depiction of the caneworker scene like two art critics at the Getty. I was curious, I told him, if the ad was intended to link people of color to their product. In my mind, the images in the Amaryl® ad work to link the (formerly) colonized and enslaved to Amaryl® . The tactic powerfully installs African Americans, Latinos, Indians, and Asians into the role of the market for Amaryl® . In the process,

the social history of Caribbean plantation labor is conscripted into an ideal biochemical target population.

"Marcus," as I will call Amaryl's product manager, denied the connection. In fact, he interrupted my question with an anxious defense of the ad's potential messages. "I've only heard of three negative comments since we started this ad," he said. Mine, that it could be read as a reference to an emerging market of ethnoracial patients; one that he himself suggested, that the caneworker could be seen as subservient; and a third "negative" interpretation, "that the image is inaccurate because you've got to burn sugar cane before harvest." Marcus's own interpretation—that an image of a caneworker could be seen negatively as subservient—is surely the most critical of these alternative readings: In my query, I had not implied that an "emerging market" reading was "negative." In fact, as several annual industry marketing conferences prove, the "emerging market" trope is a commonly accepted demographic truism. That is, to succeed, businesses must now appeal to the ethnic market.

As I looked further into Aventis and Amaryl®, I found Marcus's denial that there was any ethnoracial intent behind Amaryl's ad campaign at variance with other evidence. On November 10, 2000, some months after my meeting with Marcus, an Aventis press release read, "Amaryl® tablets provide significant reductions of HbA1c and fasting plasma glucose (blood sugars) in Mexican Americans with Type 2 Diabetes." The release noted that "10.6 percent of Mexican Americans are diabetic, that Mexican Americans are twice as likely to develop diabetes as whites and that since 1990 there has been an alarming 38 percent increase in the prevalence of diabetes among this population." It goes on to document a randomized trial that shows that Amaryl® outperforms diet and exercise in lowering blood glucose. Additionally, Aventis was a featured company in a March 2002 conference on marketing to multicultural audiences.[34] Because each product line is tied to stock valuations, bonuses, departmental expansion, and advancement up the corporate ladder, Marcus's denials seem all the more puzzling.

Amaryl® presents Aventis with a problem. In the wake of a new generation of products, how can the market for Amaryl® be sustained? As the press release implies, one apparent strategy is to link the drug to Mexican American diabetics. Yet Amaryl® cannot be directly marketed to Mexican Americans; the text of those ads would not be socially or politically tolerated because it would necessitate a thinly veiled stereotype of the entire Mexican American market segment as diabetic. Neither would such an approach be scientifically defensible because, as Goodman (2001) illustrates, the studies would have to argue that their definition of Mexican Americans is biologically accurate, and further that there is a connection between their biologically meaningful definition of "Mexican" and the disease.[35]

Instead, Amaryl® is aggressively marketed to physicians deploying what could be called cross-cultural signposts to reinforce repeatedly the connection between Amaryl® and its intended consumers. For example, Aventis, like many drug companies, sponsored a party during the 2000 ADA meetings. The slick ad quality invitation depicted a chameleon on a yucca blossom with the words, "Come show your true colors at the coolest party in town. Arriba, baby!" On the inside of the foldout invite were the Aventis logo and party particulars, and the sign-off that reads, "Don't Miss It—It's Muy Caliente!!"

In this case, Aventis' cross-cultural marketing linguistically reinforces the political and economic relationships between Anglos and Mexicanos in the U.S. Southwest. Linguist Jane Hill, in her 1993 *Hasta La Vista Baby: Anglo Spanish in the American Southwest,* points out that the jocular, ironic, and parodic register of overt Anglicizations of Spanish language, like that which appears in the Aventis promotional materials, forms an important part of the subordination of Spanish speaking people. Anglos' predominant borrowing of Spanish in boldly parodic registers "distances utterers from the voice which issues from their mouths (and texts), and serves to denigrate the source of that voice, constructing this source as ridiculous and contemptible" (Hill 1993:149–150). If the public use of Spanish language is mostly Anglicized in a vulgar way, as in the bold parodic nature of "Hasta La Vista" or "Arriba Baby," then the ethnic insult behind this linguistic practice is all too apparent.[36] Mistakes in the use of English are markers of social stratification, if not ridicule and discrimination. Overt flaunting of this bold parodic Anglicization reinforces the notion that Anglos can get away with linguistic mistakes in ways not open to Spanish speaking groups.[37] In this instance, the stratification between Anglos and Mexicanos is a consequence of the conscription of bioethnic groups as appropriate targets for a specific biochemical therapy.

The Amaryl® ad came out prior to the ADA meetings I attended. Further, the meetings where the party invitation was used were held in San Antonio, Texas. To infer intentionality to my critical assessment would misunderstand the workings of bioethnic conscription. The site-specific marketing gimmicks Aventis used during the ADA meetings reproduce the long-standing association of diabetes with ethnoracially defined populations. After all, the connection of diabetes with ethnic peoples is as at least as old as Neel's 1962 thrifty genotype hypothesis. Marketers and the graphic designers they employ are well trained in the arts of persuasion. Social, clinical, visual, and scientific information and symbols must all work together to be effective.[38] As one drug marketing director explained, "Our job is to translate clinical information into product promotional materials." Although Amaryl® may be effective in lowering the blood sugars of "non-white" diabetics, the implication that it is more effective than when used by other ethnic groups is a specious one.

The way the social is conscripted into the biological in making Amaryl® an ethnic drug is far more racializing than the *Nature Genetics* article dispute described earlier. That is, the stratification that is the signature of making a social group into a biological race requires the conflation of race with biology. As Kahn aptly demonstrates with antihypertensive BiDil, the making of an ethnoracially specific drug "transmutes health disparities rooted in social and economic inequality into mere health 'differences' rooted in biology," (2005:125). This is the pernicious side of bioethnic conscription, for it can work both to problematize racializations and produce them, to describe differences between human groups and to attribute differences to them.

CONCLUSION: CONSCRIPTING BIOETHNICITY

Evaluating Nora and Gary's work alongside the Amaryl® ad campaign, what are we to make of the simultaneous sensitivity to racial inequality and use of racial labels within the diabetes research and drug development apparatus? Nora's mindfulness of the possible negative impact of her work on her Mexican American donors, and her (and Gary's) objections to the implications of the *Nature Genetics* title dispute, present an anthropological problem of the first order. If the task is to assemble and make intelligible the multiple material and semiotic processes through which scientific knowledge is made and works, then the specific practices through which the social and biological are coproduced need to be acknowledged. In process, it is also important to note how scientists themselves struggle to understand, articulate, and respect human difference—often in discursive contexts springloaded with either—or propositions of the "right way" to think. Race, especially in the United States, provokes these kinds of binaries with particular force.

The modest proposition of this article is that a move from either—or to both—and comprehension of the ways ethnorace is deployed in genetic epidemiological and related biomedical guises would be both empirically and politically sound. The term *bioethnic* is empirically descriptive of the social differences that constitute DNA classified with the labels forged of one's family history, geography, class position, political subjectivities, and cultural affinities. Furthermore, the term *conscription* accurately portrays the active means by which race and ethnicity are transformed into bioethnicity, sometimes more "bio" and sometimes more "ethnic."

Bioethnic conscription so conceived extends what Rabinow (1991) calls *biosociality*. Biosociality is a phenomenon whereby biological and genetic discourse becomes part of a person's identity, at times intentionally so (Taussig with Heath and Rapp 2003). The naming dispute between *Nature Genetics* and Nora and Gary marks an occasion in which instead of biology informing notions of self, the social narratives of colonialism, Eugenics, slavery, and conquest—as embedded in population constructs,

code switching, and imagery—inform the portrayal and selection of the biological units of analyses for diabetes research. However, more than an inversion of biosociality, from the biological informing the social to the social informing the biological, bioethnic conscription is an effect of the multiple levels of analyses that compose complex disease gene research. In this case, Mexican racialized DNA "actants" (Latour 1987) operate as material semiotic actors (Haraway 1997). That is, the biogenetic material is so infused with social meaning that those who handle it inevitably reattach (conscript) the sociohistorical context of its production as it travels from the DNA collection site to the laboratory to the journals to clinical practices and back again.

In other words, it matters where the DNA came from. In the laboratories, there was never a mention of the labels used for the units of analyses. It only came up when (1) the populations were being evaluated as potential donors, (2) Nora and Gary were struggling to get a particular kind of knowledge published, and (3) certainly when the wide world of physicians and their patients were being enlisted as allies in the diabetes enterprise. In fact, as we see in the case of Amaryl® and the press Nora and Gary's "discovery" received, the farther away the bioethnic knowledge circulated from the labs, the more it reflected its donors and thus the more encumbered it became with meanings forged—although never entirely—out of racialized social differences.

The differential pattern of diseases between and among human populations is an important topic for research. To dismiss variations in disease incidence and prevalence between human groups because it threatens to biologize race and thus offends our long-standing political battle against biological determinism is a mistake. I locate myself as an ally to my ethnographic interlocutors—however, as one with different epistemological premises and political commitments. The different morbidity patterns for diabetes might well be researched as consequences of the different environmental (life) conditions of groups whose ability to mate with other groups has been socially, geographically, legally, and often violently constrained. More succinctly, epidemiological differences between racially classified groups "might be a proxy for discriminatory experiences, diet or other environmental factors" (*Nature Genetics* 2000:98). Such research could account for the local biologies (cf. Lock 1994) of those who live, work, and die along the U.S.–Mexico border. At minimum, these factors should be investigated, step-by-step, along with genetic ones.

The concept of bioethnic conscription, instantiated here in the case of diabetes genetics, challenges the anthropological distinction between race and ethnicity, complicates characterizations of race in science, and shows how the social and political context of ethnic identity is variably expressed at each stage of biomedical research and development. From this vantage point, diabetes science becomes intelligible as an instance of a racial project (Omi and Winant 1994) that sustains social inequalities

through the circulation of racialized genetic material and through the representations of ethnic populations. However, because the impact of Nora and Gary's careful attention to the social consequences of their scientific representations is unknown, it cannot be said that the diabetes genetic enterprise is overdetermined by the stratified social orders of race and ethnicity in the United States and is thus simply reiterative of biological (R)ace.[39] We have seen that bioethnicity can be conscripted in both productive and reproductive ways. It is a practice that is "both–and," not "either–or."

The case of the diabetes genetics enterprise can certainly support arguments that critique the use of "race" as a variable in epidemiology, medicine, and social scientific quantitative analyses (Hahn 1992; Hahn with Mulinare and Teutsch 1992; Kreiger and Bassett 1993; Zuberi 2001). But such critiques, although vital in the efforts to call attention to the social relations hidden in racial statistics, do little to explain why in spite of repeated reminders, scholars and scholarly journals consistently deploy ethnicity as a static attribute or as a biological category. Understanding this mechanism requires a broader analytic frame in which the context of the production of scientific knowledge is analyzed along with the content.[40]

ABSTRACT

This article is an examination of academic, corporate, and state-funded alliance of molecular, biological, computer, and clinical scientists who are conducting research into the genetic epidemiology of type 2 diabetes. Because type 2 diabetes affects human groups differently, researchers use ethnic and racial taxonomies to parse populations and social history to rationalize their categorical choices. In a process termed "bioethnic conscription," the social identities and life conditions of DNA donors are grafted into the biological explanations of human difference and disease causality in both objectionable and constructive ways. Bioethnic conscription is presented as an ethnographically sound alternative to the either–or proposition of the (R)ace–no race debate within biomedicine and anthropology.

Keywords: science, race, epidemiology, genetics, U.S.–Mexico border, diabetes

NOTES

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1. The early days of the human genome project was flush with the scientific race to find the single genes with determinant functions related to a disease or its treatment. Being the first in this competition meant a patent and subsequent royalties for downstream use of the knowledge. See Fortun (2003), Kevles and Hood (1992), Fujimura (1996), Sunder Rajan (2006) and Rabinow (1997) for analyses of the intersection between genetic discovery and corporate development.

2. Type 2 diabetes is also known as Non-Insulin-Dependent Diabetes (NIDDM) because, unlike those affected by the more rare form of the disease, people with type 2 diabetes produce insulin and therefore seldom need therapeutic insulin in the initial onset of disease to control hyperglycemia. Some 90 percent or more of all diabetics have type 2 diabetes, which is characterized by elevated blood glucose thought to be caused by a combination of poor insulin production and insulin resistance in skeletal muscle and lipid tissue.
3. Over a period of 21 months of field research from 1998–2000, I conducted research at the University of Chicago School of Medicine, at scientific conferences and at a genetic epidemiology field office on the U.S.–Mexico border. Blending empirical research methodology from anthropology and the social studies of science, I followed a cluster of researchers in their labs, field offices, conferences, and other venues of knowledge production. I followed DNA data sets through the pathways of research in Mexico, Texas, Chicago, and the United Kingdom. I detailed conversations, both formal and casual, noting the labels and other discussions that surround population specific DNA and the data sets created from it. I conducted over 20 interviews with diabetes researchers from public, private, and corporate institutions; attended six national scientific conferences; and observed over 80 formal and informal meetings between collaborating investigators. Additionally, I analyzed manuscripts, conference presentations, journal club reports, grant proposals, laboratory materials, newspaper articles, and drug company promotional materials to document the ways populations are represented for an array of audiences.
4. I keep race and ethnicity somewhat bundled because they are used together in the medicoscientific practices under discussion. It is this bundle that I seek to elucidate in this article.
5. See Duster (2003) for an analysis of the different uses and representations of populations, groups, and individuals in genetic analyses.
6. My thanks to Mike Fortun for pushing me on this point.
7. Some of the 53 members include University of Chicago, University of Texas, several branches of the NIH, American Diabetes Association, Harvard University affiliate Joslin Diabetes Center, DeCode Genetics Inc., and the then–Glaxo Wellcome, Inc.
8. Many researchers had a small number of samples; some had used different measures to ascertain diabetes, whereas still others had used differing markers to genetically characterize their subjects. All of these nonstandardized practices made combining data sets and replication of a finding nearly impossible.
9. For a history and analysis of the mutual benefits inherent in the cooperation between academic and drug researchers, see Swann (1988).
10. The terms *discover* and *cause* reference the usage by the scientists under investigation. This project will critically engage and show the consequences of the genetic determinism this usage implies (see Hubbard 1990; Hubbard and Wald 1993; Lewontin et al. 1984).
11. For a discussion of the pragmatic uses of race by forensic anthropologists, see Smay and Armelagos (2000).
12. See Cartmill (1998) on the use of race concepts by physical anthropologists.
13. It should be noted that scientists are not explicitly searching for ethnic specific alleles. Rather, they are looking for shared regions of nucleotides that are statistically associated with diabetes.
14. One need only look at the labels "African American" and "Mexican American" to understand that these taxons are social. There is nothing biological about the slave trade or the Spanish conquest without which neither label would exist.
15. There have been hundreds of publications on race and ethnicity in scholarly journals in field as diverse as medicine, biosciences, law, anthropology, and in newspapers and popular magazines. Authors referenced here are just a small sample.
16. Notable exceptions occur in cases in which the social origins of race and racialization are not taken for granted (e.g., Duster 1990, 2001, 2003; Lee et al. 2001).
17. See Reardon (2001, 2005) for a discussion of the coproduction of race in the U.S. Human Genome Diversity Project.
18. Duster (2003) astutely argues that whenever "race" is used as a stratifying research variable it is at once biological and social. Duster's is an argument very similar to the one presented here.

19. This study has been profoundly influenced by Duster's 1990 critique of the eugenic consequences of the medicoracialization of sickle cell anemia. See also Tapper (1995, 1998).
20. Cognizant that "The Master's Tools Will Never Dismantle the Master's House," (Lorde 1984), I use social taxonomies strategically. I document their use and here note the fundamentally social origins of the categories. However, as Durkheim and Mauss observed, "Every classification implies a hierarchical order" (1963:8). It is the process of ordering that this project interrogates. Thus, for the time being, I leave the explicit interrogation of each category largely undone.
21. Population estimates came from the U.S. Census Bureau. U.S. diabetic estimates were derived by multiplying the total population of whites by the prevalence estimates in my informant—collaborator's Public Health Service grant application. Native Americans and Hispanics were estimated as 20 percent, equal to the highest in the 15–20 percent range of Mexican Americans, African Americans as 12 percent. Asians and Pacific Islanders were not calculated for this example.
22. I refer to a "disease research imaginary" to reference the constellation of popular, biomedical and scientific representations of the causes and cures of disease in genetic terms.
23. Prior to September 11, 2001, residents of Sun County crossed back and forth sometimes several times daily.
24. Shields et al. (2005:86–88) demonstrate the methodological predisposition for conflating social history with race in genetic research. The present discussion seeks to explain the cultural processes and conditions of such methodologics.
25. See Myers 1999 for a photojournalistic essay on South Texas *colonias.*
26. Wal-Mart is the largest indoor air conditioned space within a 60 mile radius of the DNA sampling field office.
27. The search for candidate genes for type 2 diabetes began with the "discovery" of a single gene NIDDM1 on Chromosome 2 that correlated with increased susceptibility in Mexican Americans. The polygene reported in October 2001 is the effect of NIDDM1 in combination with a gene on Chr. 15, CAPN10. It is actually a combination that requires haplotype heterozygosity at both sites to confer the increased susceptibility. The admixture of Mexican Americans makes the heterozygosity more prevalent in Mexican Americans than in Europeans. Homozygotes for either of the specific NIDDM1 or CAPN10 haplotypes display no increased risk. Increased risk was determined through simulation. Since publication in 2001, the finding has had difficulty being replicated by other researchers eventually requiring a meta-analytical review of all attempts to achieve replication. In spite of these difficulties, the analytical (computational) methods developed for the polygene research occasion numerous speaking invitations for the investigators.
28. It is also possible that Gary knows the unstable foundation of a claim that relies on biogenetic differentiation between human groups as they are currently labeled.
29. See Harding (1998) for an analysis of Eurocentricity in science.
30. Although an emergent topic for social research, ethnographers are still developing our methodological and theoretical frames for analyses of pharmaceuticals. As Van der Geest (1996) observed, anthropologists have by and large focused on the buying and consuming of drugs in so-called underdeveloped nations. Exceptions include scholars who evaluate the social, cultural, and political contexts and consequences of the interarticulation of corporate and state power vis-à-vis pharmaceutical products (e.g., Dumit 2004; Farmer 1997, 1999; Hayden 2003; Koch 2000; Lakoff 2004).
31. Taken as a monotherapeutic or in combination with other drugs, Amaryl® —like all sulfonyreas—helps those people with diabetes whose pancreas still produce some insulin. Most people with type 2 diabetes suffer from elevated blood glucose because the insulin their pancreas produces is resisted by their body. Insulin is a hormone released by beta cells in the islets of langerhans of the pancreas. Its function is to control blood sugar levels. Insulin regulates glucose transport into cells so they can produce energy or store glucose for later. As food gets digested and transformed into simple sugars, the pancreas is stimulated to release insulin to be used by the muscles as energy, thus preventing hyperglycemia (high glucose levels). Diminished insulin uptake results in hyperglycemia, elevated blood glucose levels. Amaryl: glimipiride oral sulfonyrea agents (OSAs)

lower blood sugar levels by stimulating the pancreas—some argue detrimentally so—to release additional insulin.

32. I attempted to get figures from both the Aventis representatives and the ADA product theater officials. In both instances, trade secrets and negotiated rates were cited as reasons they could not disclose any dollar figures.

33. Rivera was supportive of the Taller de Grafica Popular (TGP) in spite of political disagreements among artists at the time. Several TGP members worked with him on some of his murals (see Herzog 2000). My thanks to Melanie Herzog at Edgewood College for connecting the Amaryl® advertisement's visual style to the broader context of art in Latin America.

34. Frank Clyburn, Ethnic Marketing Manager for Aventis, shared the billing with representatives from GlaxoSmithKline, Novo Nordisk and Kang and Lee Advertising on a panel entitled "How to Gain a Share of the Pharmaceutical Marketing Dollar?" The panel was held during the 3rd Annual Multicultural Pharmaceutical Marketing, Media, and Public Relations conference (www.srinstitute.com).

35. See for example Duster (2001) and Kahn (2004) regarding BiDil, a drug marketed as especially efficacious for African Americans. See also Goodman (2001) on racializations in antacid marketing claims.

36. Imagine a U.S. English reversal in which the word *y'all* was consistently overemphasized to stress a social position of those who use this colloquialism. English speakers from England can surely think of numerous examples of linguistic subordination.

37. See Bourdieu (1984:255) on the ways intentionally breaking linguistic rules serves to reinforce the social position of dominant groups while denigrating those who try to use language correctly.

38. See Scott with Stanford and Thompson (2004) for a recent analysis of the effects of pharmaceutical advertisements on physicians.

39. Race is a "fundamental dimension of social organization and cultural meanings in the U.S." (Omi and Winant 1994:viii), occurring through the linkages between social institutions and representation. Scientific institutions and related representations are not exceptions to this process. Insisting that race is an overtly political phenomenon does not attend to the mundane processes of the production of the cultural meanings of difference like those observed in biomedical milieu, however. This discussion has attempted to explain the mechanisms whereby ethnorace, those identities forged of one's family history, geography, class position, political subjectivities, and cultural affinities, gets repeatedly reintroduced into bioscientific milieu.

40. *Editor's Note*: *Cultural Anthropology* has published a number of other articles on the "conscription" of biology and culture, on genetics, and on race and ethnicity. These include Julia E. Liss (1998), "Diasporic Identities: The Science and Politics of Race in the Work of Franz Boas and W. E. B. Du Bois, 1894–1919"; Robert Boyd and Peter J. Richerson (1987), "The Evolution of Ethnic Markers"; Phyllis Pease Chock (1987), "The Irony of Stereotypes: Toward an Anthropology of Ethnicity"; Corinne P. Hayden (1995), "Gender, Genetics, and Generation: Reformulating Biology in Lesbian Kinship"; and Karen-Sue Taussig (2004), "Bovine Abominations: Genetic Culture and Politics in the Netherlands."

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