The Biologistical Construction of Race: `Admixture' Technology and the New Genetic Medicine

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ABSTRACT This paper presents an ethnographic case study of the use of race in two interconnected laboratories of medical genetics. Specifically, it examines how researchers committed to reducing health disparities in Latinos with asthma advance hypotheses and structure research to show that relative frequencies of genetic markers characterize commonly understood groupings of race. They do this first by unapologetically advancing the idea that peoples whom they take to be of the 'Old World', or 'Africans', 'Europeans', 'East Asians', and 'Native Americans', can serve as putatively pure reference populations against which genetic risk for common diseases such as asthma can be calculated for those in the 'New World'. Technologically, they deploy a tool called ancestry informative markers (AIMs), which are a collection of genetic sequence variants said to differ in present-day West Africans, East Asians, Europeans, and (ideally Pre-Columbian) Native Americans. I argue that this technology, compelling as it may be to a range of actors who span the political spectrum, is, at base, designed to bring about a correspondence of familiar ideas of race and supposed socially neutral DNA. This correspondence happens, in part, as the scientists in question often bracket the environment while privileging racialized genetic variance as the primary source of health disparities for common disease, in this case between Mexicans and Puerto Ricans with asthma. With their various collaborators, these scientists represent a growing movement within medical genetics to *re*-consider race and 'racial admixture' as biogenetically valid points of departure. Furthermore, many actors at the center of this ethnography focus on race as a function of their personal identity politics as scientists of color. This to say, they are driven not by racist notions of human difference, but by a commitment to reduce health disparities and to include 'their' communities in what they describe as the 'genetic revolution'.

Keywords admixture, AIMs, ancestry informative markers, asthma, genetics, race

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> The very word 'race' applies to a hypothetical past, or to a problematical future, not to the actual present … the only way to measure the genetic relationship of ethnic groups would be by ascertaining the quantitative values of their coefficients of common ancestry, which would be based entirely upon the statistical methods of probability theory. (*We Europeans* [Julian Huxley and Alfred Court Haddon, 1939: 114])

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> To me, the refusal to use race in medicine is political correctness gone awry. It's a lot of white researchers gone political. (Esteban Gonzàles Burchard, asthma geneticist at the University of California, San Francisco Lung Biology Center; field notes 2003)

The Molecularization of 'Admixture': A History of the Present

In 1949, the year before the first United Nations Educational Scientific and Cultural Organization (UNESCO) statement rallying against the race concept, Linus Pauling characterized sickle cell anemia as the first 'molecular disease' (Pauling et al., 1949). At the time, most experts and lay people considered sickle cell a 'black-race disorder'. Despite global good will and contrition for the violence perpetuated in the name of racial purification in Germany and elsewhere a few short years before, some North American scientists called the UNESCO statement an 'incautious affirmation' and claimed that sickle cell anemia in American blacks (who by definition, it was assumed, had white ancestry) was a perfect example of how 'race mixture can be disadvantageous in its racial effects' (Gates, 1952: 896). The then 'odd' observation that 'hybrids' (black Americans) seemed to have more sickle cell disease than their 'pure' (African) counterparts who had more sickle cell trait (which was actually mistaken for a milder form of the disease in many cases) gave immediate rise to theories that 'racial admixture' could affect disease risk and/or severity (Gates, 1952).[1] With Pauling's Nobel-winning observations came the first intellectual opening for the molecularization of race. Immediately with it came the idea that racialized ancestral mixing, or 'admixture', constituted increased risk of disease pathology. In what follows, I examine a present-day resurgence of the concept of human biological admixture as a factor in disease risk in some quarters of contemporary American medical genetics.

Biogenetic Race Revived

Pauling's discovery of the molecular nature of sickle cell anemia entered science at a crucial time in world history and in the history of humanistic thought about biological concepts of race. Although the first UNESCO race statement appeared shortly after the Nuremberg trials, when the publicity about Nazi crimes greatly tempered the previously facile acceptance of race as biogenetic in disciplines such as physical anthropology, a belief in the biology of race, nonetheless, persisted for many.[2] Accordingly, the second UNESCO statement on race, written mostly by physical anthropologists and geneticists, determined that 'race' as a biological anthropological concept indeed 'existed'.[3] The revised UNESCO statement drafters supported race as a scientific concept, but worried that it could be misused in society. They thus asserted that some vigilance would be required to insulate biological definitions of race from any 'social end – whether it be totalitarian or egalitarian' (Reardon, 2005: 31). During the meeting on the

second UNESCO statement there was much divergence of opinion regarding when and how race mattered, or even where to locate its contours. Some saw racial types as related to human 'subspecies' while others saw them as 'clines',[4] or patterns of trait frequency concentration and gradation in human populations around the globe.[5] How to talk about this clinal distribution and how to reconcile population trait frequencies with stark racial typologies proved to be an epistemological question that still yields no single answer. Herein lay an open terrain for many scientists to develop models, theories and, most recently, *products* that partition human groups and measure their similarities and differences.

In the half-century between the two UNESCO statements on race and the Human Genome Project, many biologists and geneticists have publicly declared the non-existence of race based on genetic sequence comparisons. In the year 2000 when the heads of both public and private genome mapping efforts unveiled their feat, they made a point of specifically speaking about racial genetic difference. On that millennial 26 June, the heads of both teams concurred that race was not a valid scientific category.[6] Some geneticists, however, saw such statements not only as incorrect but also as irresponsible since their own research suggested that society might benefit from the use of American racial taxa in medical genetics (Risch et al., 2002; Burchard et al., 2004), as well as in forensics (Devlin & Risch, 1992; Shriver et al., 1997). Independently and through collaborations with medical genetics labs, one of which is the central focus of this paper, certain of these actors have committed themselves to proving that race is indeed biogenetic. Some reduce it to probabilistic statements, such as: 'If you give me a DNA sample I could probably tell you what race it is.'[7] Yet, as will become clear shortly, the certainty of racial types, for those who make such claims, operates through logics and language of 'racial segmentation', which necessitates a constant referral back to conventional ideas of continental racial stocks, such as 'Africans, Europeans, and Native Americans'. Like the 1950s critics of UNESCO's first 'affirmation' that race was not biogenetic, certain present-day geneticists believe that the composite parts of 'racial admixture' can yield clues about disease severity, and the worst manifestations of health disparities (Burchard et al., 2003, 2005).[8]

In this paper I examine a contemporary instance of how a group of increasingly visible American physician-researchers and geneticists pursue questions of disease severity and racial difference while attempting to link the two at the level of the genome through a technology called ancestry informative markers (AIMs). As many social constructionists argue, race ascription has been a heedless endeavor to read physical traits off of bodies whose meaning and precision shifts depending on historical place, social contexts of power, and ever-changing legal concepts that have culturally defined racial difference (Omi & Winant, 1994: 88; Harris, 1995; Stoler, 1995: 27; Haney López, 1996: 203–08; Bowker & Star, 1999: 201). The point of this paper is to demonstrate how scientists come to correlate everyday broad American categories of racial groups with precise molecular and statistically significant notions of 'reading race in the DNA'. I argue that the methodology deployed

by my scientist-informants, and by some of their collaborators, is itself *designed* to bring about a *correspondence* of these two domains: (1) body traits made meaningful through conceptions of race (given certain social and political contexts) *and* (2) supposedly politically and socially neutral DNA. This correspondence comprises a biologistical construction of race in which certain raced US 'populations' ('Black'/African, 'White'/European, and 'Red'/Native American) and DNA markers with certain statistical frequencies in those populations are each posited as first principles to infer truths about the other. This is an instance that may, in part, be explained as 'the natural order' sustaining and being sustained by the 'social order' (Jasanoff, 2005: 275). This is to say, these scientists use a technology that selectively culls genomic sequence variants in peoples of 'Old World' populations and packages them as a tool to measure and demarcate race composition in those of the 'New'. This technique of selection, rooted in American definitions of human difference as these map onto continents, is then deployed to sustain the biological bases of racial ascription (a 'social' fact) and its power to bespeak genetic risk within racialized groups, or their 'admixed descendents'.

My analysis departs from other studies of co-produced phenomena, however, because the temporal dynamic of how AIMs are used is not about 'scientific ideas and practices and societal arrangements com[ing] into being *together*' (Reardon, 2005: 7; emphasis added). Rather, the 'races' in question have been around *a lot longer* than the recent enunciation of the genetic markers that now partially define and reinforce them.[9] Thus, an analysis of the persistence of race is needed, but so is a deeper understanding of the cultural contexts that make racialized genetics attractive to scientists who themselves claim racialized 'admixed' and 'minority' identities today.

Over the past few years, social scientists studying genetics and race have urged their colleagues to 'go to the very sites' of scientific production and 'document how [racial] categories are being constructed' anew (Reardon, 2005: 18; Duster, 2006a: 12). Following from this, it is as imperative that ethnographers also attempt to understand better scientists' motives for wanting to resuscitate such troubled categories. To this end, it is important for me to note how my informants' social experiences shape the tautological product of genetic racial admixture they use on a daily basis. In particular, one challenge these scientists have posed for themselves is to 'care' for their own disproportionately sick communities of 'racially admixed subjects' by recruiting and enrolling them in genetic research. A crucial aspect of their effort to reduce health disparities is a search for the biological component of these communities' mixed racial heritage. For several of my informants, this heritage is a point of biological difference that may contain clues about present-day health differences. Here it is many 'drops of blood' – rather than one – that now constitute the brown bodies in question. Today, Mexicans and Puerto Ricans in the US are assumed to be differentially constituted from African-Americans and Native Americans, based on their varying *amounts* of African, European, and Native (pre-Columbian) genetic ancestral contributions. Yet, contrary to

earlier American norms of hypo-descent, these mixed groups must remain conceptually separate, 'ethnically' and 'politically', from the referent groups that make them up. Today, Mexicans' and Puerto Ricans' African ancestries are deemed important for reasons that will become clear below, but they are rarely collapsed into a category of 'blackness'.[10] In fact, as one of the main researchers featured in this ethnography reminded himself and his team time and again, as of the 2000 census, Latinos surpassed African-Americans as the largest minority group in the US. Over the course of my fieldwork in his lab, I heard this feat by numbers repeated, as if to say that this researcher's 'community' needed and deserved the same kind of attention, political courtship, and scientific resources as one of the most historically 'important' and visible American minority groups.

I am presented with a renewed form of what George Marcus and Michael Fischer once dubbed a 'crisis of representation' when pursuing an ethnography of scientific practitioners who often self-present as members of a racial minority, considered as a political entity, *and* who believe that race is real as a biogenetic fact. For Marcus and Fischer, 'the crisis' was a 'postmodern', or 'post-paradigmatic' one, in which taken-for-granted truths and certainties on which a paradigm once rested were then problematized. The crisis I am faced with is a good old-fashioned modern one in which familiar paradigms about race resurface and my own anthropological subjects insist – in no uncertain terms – that they should be represented as believers in the contentious idea that there are three to four major human races. Additionally, in no way do they want the 'paradigm' of three to four races problematized, but they do want to be able to abstract and reorganize each part to be able to think in terms of new composite, 'admixed' wholes. Thus, their thought and research processes should be understood as sites where 'indeterminacy' *and* 'regularity', to use Marcus and Fischer's language, blur into one another as claims about *ethnos* abound in these labs and in the cultural field much more broadly. Such blurring allows this rearticulated racial typology of 'admixture' to work at seemingly opposite ends of the political spectrum (Marcus & Fischer, 1999: 8). *Ethnos* (aided by the use of AIMs) is a potentially oversimplified notion of ancestral racial background that is enlisted in racist concepts of human difference, such as white nationalist David Duke's claims about the right to a 'European-American' culture (Duke, 2007). *Ethnos* is simultaneously deployed to include underserved communities in American health disparities research (Risch et al., 2002; Burchard et al., 2005), or to map Americans' shared genetic background, despite common ideas of race difference in the American vernacular (Harmon, 2006). How to understand this?

I argue that the productive powers (Foucault, 1980) of the AIMs technology to draw from and influence disparate fields of modern life lie in its ability to bring rhetorical, anthropological, and popular notions of human difference together to form a bio-logistical construction of race. This construction, statistically derived from genetic markers said to signal continental 'ancestry', safely avoids the politically charged historical baggage of the word 'race' itself (Kittles & Weiss, 2003). Thus, in some instances, AIMs

have emerged as a dispassionate research product that purportedly rises above subjective practices of racializing the phenotypes of others. Yet, in other instances, this technology can be reframed, quite passionately, as promising to discount racial purity (Harmon, 2006; Gates, 2007: 149–50).[11] To understand this range of interpretations, we must first examine the rhetorical tools that power them, as well as the geographic and genetic frames that circumscribe them.

The Black Box of Genetic Ancestry: Rhetorics of Geography and Race

> [R]hetorics work more on the model of contagion than communication or representation. (Richard Doyle, *On Beyond Living* [1997: 3])

Although genetic ancestry studies in the US have been carried out on 'Caucasian genes in American Negroes' since the 1950s (see Reed [1969] for a review), a group of population geneticists at the University of Texas at Houston attempted an 'admixture' analysis in Latinos for the first time in 1991. With the goal of pinpointing disease risk, these scientists set out to 'estimate the contribution of putative ancestral populations to the contemporary gene pool' in a study called the 'Origins of U.S. Hispanics–Implications for Diabetes' (Hanis et al., 1991: 618). The Texas researchers were thus the first to distinguish Mexicans and Puerto Ricans as having different bases of 'genetic ancestry' according to analyses from tests for a series of markers that had different frequencies in select European, Native American, and African groups as comparative referents. Mark Shriver, then a graduate student at Texas, later developed a much broader panel of markers, which are now known as AIMs. During my fieldwork, I visited Shriver's lab to observe their research and to interview him and others in his lab who work with AIMs in different populations. I was led to Shriver by Esteban Gonzáles Burchard, one of my main informants in the San Francisco Bay area. Burchard collaborated with Shriver in order to utilize Shriver's set of markers in his attempt to resolve differences between Mexicans and Puerto Ricans with asthma. In one of the Burchard group's publications that resulted from his fundamental query about 'which ancestry' was 'associated' with severe asthma, they determined that Mexican ancestry on average was: '3.4 ± 0.97% African, 44.9 ± 1.7% European, and 51.7 ± 1.7% Native American'. Puerto Ricans, on the other hand, were: '16.2 ± 1.6% African, 65.5 ± 2.2% European, and 18.3 ± 2.1% Native American' (Salari et al., 2005: 80).

These exceedingly precise admixture percentages – which, unlike ancestral correlations with disease, are rarely questioned by this research team or their statisticians – were generated by formulas that estimate the relative frequencies of, in this case, 44 AIMs of select Europeans, Africans, and Native Americans.[12] When developing these AIMs, Shriver identified the continental groups of interest, and then examined human DNA samples from public

genetic databases, such as *dbSNP* (<www.ncbi.nlm.nih.gov/projects/SNP/>), to identify a set of genetic markers that would distinguish between the groups. Taken together, all subjects in a defined group, for example 'Africans', were tested for the panel of alleles. Then, the researchers determined the frequency at which AIMs appeared in each group as a whole. Finally, they contrasted marker allelic frequencies between the three groups to come up with the percentages for each of the 'admixed' subjects' ancestries. This last step was possible only after researchers recorded the genotypes of interest for the given 'parental populations' and then used the computer software program STRUCTURE™ to determine the ancestry frequencies. When using the program, the researchers input the populations or 'ancestries' they wanted it to output. In the Burchard lab this number was invariably three.[13]

In the literature and on the commercial website[14] that advertises the AIMs test (as 174 markers), these sequence variants packaged as a technology are a classical case of an 'assembly of disorderly and unreliable allies' slowly turned into an organized, cohesive whole – a 'black box', as Latour (1987: 130–31) characterizes it. To '*un*-black box' this biologistical construction of race, let me begin by explaining how the assembly of AIMs has come to represent the three ancestral parental groups operative in Burchard's lab (African, European, and Native American). Most AIMs are found in at least two of these three populations, and most are found in all. Thus, it is not that these markers are 'population specific', as the inventor of the panel Burchard uses originally wrote (Shriver et al., 1997). Rather, they have been carefully purified, examined, and selected because their allelic frequencies differ by at least 30% (in earlier publications their differential could be up to 50% [Shriver et al., 1997]) between any two of the three populations in question (sometimes four, if researchers are interested in finding East Asian ancestry, which Burchard was not).

What, in Essence, are AIMs?

The markers themselves are DNA base-pair differences that are the source of genetic sequence variation. Some actually affect the coding of proteins associated with vivax malaria receptors (*FY*), freckling, skin and hair pigmentation (*MC1R* and *OCA2*), vitamin D binding (*GC1*), hormone catalysis and drug metabolism (*CYP19*), lipid regulation (*LPL*), muscle enzymes (*CKM*), and blood clotting (*F13B*). There are many more whose functions are either unknown or are not publicly accessible by consulting resources such as the *Online Mendelian Inheritance in Man* (OMIM) database (<www.ncbi.nlm.nih.gov/sites/entrez?db=omim>). Time and again, when I asked researchers in the various labs where I have conducted fieldwork (now four US academic sites) what these markers actually are, very few could provide an answer. Thus, in the larger cultural realm, many others who are utilizing this technology for 'recreational' or 'genealogical' reasons may not realize that single nucleotide polymorphisms (SNPs) such as these may differ in some groups, such as Africans and others, because of

different kinds of mutation events, gene flow, or environmental exposures and selection over time. It is important to highlight what kinds of 'data' these are as well as to have an understanding that AIM models 'assume that evolutionary pressures other than admixture have been insignificant' (Pfaff et al., 2004: 306).

Still, one might argue that the AIMs listed above are good representative markers for differentiating the human groups (African, European, Native American) of interest to admixture researchers since they were expressly culled for their rare status in the genome as minute points of relative difference between the sampled groups. Again, most of the original markers that constituted Shriver's original AIMs are located in genes that have some clear ecological and/or evolutionary function (variable malaria protection, vitamin D regulation based on exposure to sunlight, and, of course, melanin concentration). Clearly, these markers that are being used to assign 'shared ancestry' might just as easily be used to assign 'similar historical environmental exposure', which may or may not be the same thing as shared direct (and *unique*) continental origin or 'ancestry'. Thus, if the American cultural context was one that was less interested in 'race' and more interested in 'ecology', then the few AIMs that dramatically differ in selected Africans, Europeans, and Native Americans might be renamed, or at least *rethought*, as possible 'environmental exposure markers', with the 'exposure' bearing the broad-ranging effects of human history. Additionally, even for these most obvious, 'most informative', AIMs, the allelic frequency distributions are not as drastic as the test results (x% African, y% European, and so on, ancestry) imply. With few exceptions,[15] most alleles are neither present nor absent in *all* samples assigned to one population or another.[16]

On the subject of the actual ancestral populations, the scientists who have constructed this approach to race have done so by testing the markers in present-day populations, which, because of their global geographical locale, have been cast as 'Old World'.[17] Despite the rhetorical force of such language, these are modern-day peoples currently living in Africa (Cameroon, Central African Republic, Ghana, Nigeria, Sierra Leone), Europe (England, Germany, Hungary, Ireland, Lithuania, and Germany, and the Valencia and Basque regions of Spain), and 'Native America' (from the Dogrib, Navaho, Pima, Keres, Tiwa, Cheyenne, Aymara, Ketchua, Suruí, and Pehuenche, as well as seven unnamed groups from Central America).

The complexity of how both 'geography' and 'time' have born out human variation has been drastically simplified in what Karin Knorr Cetina (1999: 3) has termed the 'epistemic machinery' that makes the AIMs technology increasingly appealing to the wide range of lay, scientific and law enforcement clients who are now using it. While the girth of the globe has been flattened to a small area of West Africa, sporadic points in North and South America, and even sparser points of Europe, time has been collapsed into a world history that pivots on the year 1492 with Columbus's arrival in Latin America (Burchard et al., 2005:

TABLE 1
Seven of 44 AIMs' frequencies in the selected African, European, and Native American groups that are used to determine 'admixture' in 'New World' peoples*

Marker	Chromosome location	Allelic frequency in selected Africans	Allelic frequency in selected Europeans	Allelic frequency in selected Native Americans	Frequency difference between Africans and Europeans	Frequency difference between Africans and Native Americans	Frequency difference between Europeans and Native Americans
CKM	19q13.32	.15	.29	.86	.13	.71	.57
CYP19	15q21	.32	.29	.72	.03	.40	.43
GC1	4q13	.93	.41	.45	.52	.48	.04
FY-null	1q23.2	.99†	.00†	.00	.99	.99	.00
F13B	1q31.3	.70	.08	.03	.62	.67	.05
LPL	8p21.3	.97	.52	.45	.45	.52	.07
MC1R	16q24.3	.51	.14	.04	.37	.47	.09
OCA2	15q13.1	.14	.72	.48	.58	.34	.23

* For the full set of the 44 markers used by the Burchard lab and their frequencies, see Salari et al. (2005: 79). I chose these seven because they are well known and have easily recognizable identifiers. Thus their functions were easy to research on the OMIM database.
† The frequencies for *FY*-null between Europeans and Africans listed here are .99 for Africans and .00 for Europeans. These differ from the paper from which these values were taken since there appears to be an error that lists this frequency as .00 for Africans and .99 for Europeans; see Salari et al. (2005: 79).

TABLE 2
'Parental populations' samples used for the AIMs technology as shared with the author by technicians in the Burchard laboratory (the table presented here is an unaltered reproduction of the document given)

Continent	Sample size	Continent	Sample size
Africa		**North and South America**	
Nigeria, (Benin city)	100	Dogrib (Northwest Terr.)	70
Nigeria (Sokato city) [SIC]	46	Navaho (Southwest US)	37
Nigeria (Yoruba)	100	Pima (Southwest US)	35
Nigeria (Hausa)	120	Keres (Southwest US)	24
Nigeria (Kanuri)	100	Tiwa (Southwest US)	28
Nigeria (Bini)	100	Cheyenne (Southwest US)	33
Liberia (Kru)	80	Central America (7 groups)	300
Ghana (Akan)	100	Bolivia (Aymara)	70
Central African Republic, Bantu	49	Peru (Ketchua)	75
Sierra Leone (Temne)	98	Surui (Brazil)	23
Sierra Leone (Mende)	181	Pehuenche (Chilean	120
Camaroon [SIC]	150	plateau)	
Europe		**Asia**	
Spain (Valencia region)	90	Southern Chinese (Taiwan)	300
Spain (Basque region)	100	Northern Chinese	200
Irish (eastern coast)	90	Southern Chinese	300
England (London)	48	(Han and minority	
Germany	80	populations)	
Hungary	50	Japanese	600
Lithuania	50	Insular Southeast Asia	600

2161).[18] Although Burchard and colleagues acknowledge that 15th-century Spain consisted of 'Celts, Greeks, Romans, Sephardic Jews, Arabs, Gypsies, and other groups' (2005: 2161), their AIMs model permits them to gloss over this complexity with a general 'European' label. This 'Old' vs 'New' World terminology holds for 'African ancestry' as well. As a clear example of what social epidemiologist Nancy Krieger has termed the 'politics of time' (2005b: 2157), it is assumed that, for instance, present-day Yoruba and Mende people are 'older' than 'African-Americans', 'Puerto Ricans', and the more than a few 'whites' with 'African ancestry'.[19] In both instances it is taken for granted that those in the 'New World' with 'African' or 'European' ancestry, detected by the test, have actually *inherited* the ancestral (Yoruba, Mende, Valencian, and so on) genotypes denoted by AIMs. Other problematic assumptions, such as that Mexican Americans' and Puerto Ricans' ancestral populations are currently still in existence, are also in play. For instance, using 'putative' Native American 'parental source populations' overlooks an actual history of genocide of peoples who no doubt contributed to present-day populations in the Americas, but who no longer exist (Pfaff et al., 2004: 310–11). To complicate matters more, Mexicans often have more Amerindian heritage than the putative referent groups

FIGURE 1
The selected points on the four continents where people of interest to the Burchard laboratory were sampled

who are posited as their Native American ancestors. For political and historical reasons Native Americans need only possess 1/8 (12.5%) demonstrable Native American ancestry, whereas Mexicans may have considerably more (Kittles & Weiss, 2003: 48). Ideas of unique and direct ancestry derived from AIMs are indeed 'contagious', spreading more or less blindly due to their compelling nature, rather than strictly 'communicative', which would require that a degree of actual knowledge inheres in the passage of information (Doyle, 1997: 3).[20] Here is why: in addition to the sampling quandaries and assumptions outlined above, when alleles that have a high frequency in the specific reference groups tested (those labeled African, European, Native American in Table 2) appear in a client taking the test whose ancestors may *also* have possessed those alleles with a high frequency, the AIMs test reads that the client, himself or herself, has inherited the *specific referent ancestry* (African, European, or Native American) rather than say ancestry (or SNPs) from *other* still unsampled parts of the globe.

There is no better place to illustrate this last point than in the laboratory where the test was developed. In the summer of 2004, a French graduate student in the Penn State Anthropological Genomics Laboratory received an AIMs test result, which indicated that he possessed, what was to him, a surprising portion of 'Native American' ancestry. He was at first frustrated by what he understood to be a questionable result, and went to discuss this with his advisor, Mark Shriver, the test's architect. Shriver told

him that he was correct to be suspicious; indeed, this was an error of typology, and the test was 'probably picking up some Central Asian ancestry'. After thinking long and hard about the problem, while now embarking on his own research into European genetic diversity, the student told me:

> In the meantime, I bet a bunch of people in the US believe they have Native American ancestors based on a 10–15% Native American component, and they might not … Or believe they have 'black' ancestry when they have [a result of] 10–15% African, which could be due to Arab or North African ancestry, or even possibly Central Asian.

Thus, as the Frenchman learned, and hopes to redress, the test is set up to read race as we – *les Américains* – know it.

It should be clear by now that the very continents and peoples chosen for this product were selected due to their perceived proximity to what we in North America imagine race to be. Although the language of scientists who invented this panel of AIMs is now that of 'biogeographical ancestry', the conceptual configuration of human racial typology remains intact even though, as the vignette above illustrates, Shriver has the ability to employ a larger interpretive frame when pressed by a smart student. That said, the most frequent presentation of AIMs resembles that described in Shriver's 18 November 2004 patent application, which he filed with collaborator Tony Frudakis of *DNAPrint Genomics*. The language there is: 'AIMs [are] a method of inferring, with a predetermined level of confidence, Biogeographical ancestry, or BGA … which is the heritable component of "race".' The patent application goes on to say that AIMs can detect 'race' at several levels: it can distinguish Europeans from others, and second, with a 'finer' resolution, it can separate DNA into Indo-European, African, Asian, and Native American.[21] In other words, the assumed bounded groups on which AIMs draw (African, European, Native American, and Asian) correspond to American cultural ideas of race, which, in the case of many scientists, also ends up shaping where across the globe they collect the DNA of 'populations' (Serre & Pääbo, 2004: 1680; Ossorio, 2006: 281–82). Scientists' own understandings of race in their larger social contexts clearly informed their initial choices of who would be selected for the early testing of these variable genetic loci in search of population differences. This action, as a social response of sorts, now yields new correspondences of subsequent AIMs results that reveal 'ancestral' percentages when present-day American asthmatics and others are tested for variants of these markers.

The Study of Asthma in Latino Americans[22]

The Genetics of Asthma Laboratory lies in an historical brick tower of the University of San Francisco's (UCSF) General Hospital.[23] The asthma lab director, Esteban Gonzáles-Burchard, is a 'no-nonsense', highly combative, yet humane and affable, pulmonary 'physician-scientist' who easily shares a laugh or fishing story with a janitor in the corridor or the food vendor who

sells him his mid-day California roll. In lab meetings, between serious moments of discussing sequencing issues, Burchard could precipitously code-switch to street slang (to refer to an enzymatic solution the lab had prepared as 'home-brew', or, to talk about exploitative power dynamics in science as 'pimping'). Most people in the lab get the references – those who do not may simply let them pass, while others might ask for clarification, much as they would with an interesting p-value. Burchard refuses to talk and act like a researcher who is isolated from 'the real world'. Others in the lab appreciated his informality and told me that they also wanted to 'keep it real'. Burchard is known on campus for his work on the genetics of 'minority populations', 'the reality of race and genetics', as he puts it, as well as a fee-for-service DNA bank. One major use of the bank is to store his own research samples of DNA taken from more than 2000 Mexicans and Puerto Ricans, which comprise his Genetics of Asthma in Latino Americans (GALA) database for the study by the same name funded by the National Institutes of Health (NIH) and the Sandler Family Foundation's research program on asthma. The Sandler Foundation's mission is to fund 'highly original thinking from investigators willing to step away from their current areas of research and tackle the riddle of asthma … ' through 'innovation and risk'.[24]

The General Hospital, or the General as the locals call it, is a teaching hospital that serves the city's most indigent population. It is a primary landmark of San Francisco's Mission district, which has long been home to various immigrant populations. The Mission was largely Irish and German for much of the 20th century, and as these groups assimilated and moved to more prosperous locales other groups moved in. Today the Mission neighborhood is largely home to immigrants from Central and South America and, most important demographically, from Mexico.

Burchard was born and raised in the Mission. 'We were in the Hispanic ghetto,' he told me. He then added, 'and were right on the edge of the black ghetto. So that's where I grew up … I've always been keenly aware of race, ever since I was a child.' When asked about his research and academic life he often talks of being a 'product of Affirmative Action', and having minority sensibilities despite the fact that he was 'bi-racial', of 'white (French-Canadian) and Mexican' descent. Affirmative Action, he's proud to say, opened the doors to him of Stanford University, where he received his MD, as well as those of Harvard, where he was a fellow who specialized in pulmonary medicine and the genetics of asthma. Burchard's main patient recruiter (also an MD), the manager of his DNA bank, one of his bioinformaticians, and the project's data manager are also 'Latino', though of different origins spanning South and Central America from Argentina to Mexico. The other lab workers were all 'hand picked', as Burchard says, for their 'diverse backgrounds', which, for most, also happens to be 'humble'. They are as follows: (1) a woman from New Delhi who holds a post-doc; (2) a Japanese-American man who formerly worked for the Department of Justice DNA bank and who remains a technician; (3) a first-generation Cambodian-American geneticist whose family fled the Khmer

Rouge who does genotyping; (4) a Canadian 'Caucasian' woman with many years' work experience with genotyping, but who does not have a Bachelor's degree and whom Burchard calls his 'affirmative action' case; (5) an undergraduate computer programmer/pre-med student of Iranian origin; and (6) Burchard's friend and collaborator Dr A (who wishes to remain anonymous), who uses Burchard's lab for his projects in exchange for his statistical expertise.

In addition to his regular staff, Burchard takes in two minority high school students every summer through a local mentorship program that aims to increase the dismally low numbers of minorities in US science (see Campbell et al., 2000). Seventeen-year-old students, who have only heard about polymerase chain reaction and other laboratory technologies, are given the chance not only to learn techniques, but also to conduct research. During my stay Burchard hosted two young scientists. The first student in the program, whom Burchard described as 'scientifically brilliant, but socially awkward', was an African-American high school junior. The second, whom all agreed was 'perfectly social, but less driven', was a more mature Latina woman in the same year of school. Like all of the researchers and technicians in the lab, the students donated their blood for DNA extraction fairly early during their stay. As a sort of modern-day initiation into a family that would now share this blood and its DNA for tests and training purposes (so as not to waste precious DNA from patient samples), the students, like the others, began to watch their DNA appear fluorescently on gels for particular markers of interest to the lab. For a few hours a day, the students were chaperoned by the project manager, who talked them through writing the abstracts for their research projects and walked them through how to run gels carefully. Their pace was slow and, to their delight, it was often necessary for their mentors to leave them and pursue their normal rhythm of science in the lab: a frantic hustle replete with coach-like chants from Burchard (a former wrestler), aiming to put out three papers for review before the end of the summer. As the team often rushed around the shared office space discussing their latest results, Diego Rivera's *The Flower Carrier* hung on the wall (Figure 2). As Burchard told me:

> I have this [in the lab] to remind me of all of the people who have come before me, including my mother who was a migrant worker. … [I]t is a picture of a man on his knees. His wife is loading a very large basket of flowers on his back, much like you would load a mule. To me, this symbolizes the struggle that we have and continue to face.

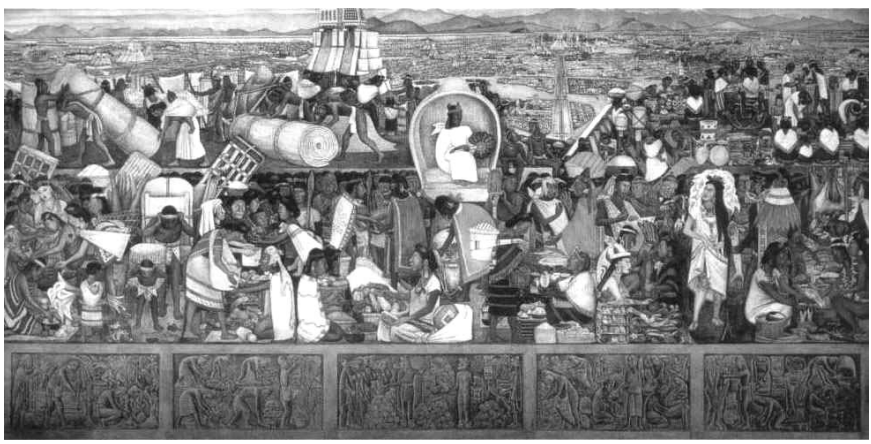
A few feet away, another reproduction of one of Rivera's murals (Figure 3) that contained more busy details of daily life than were legible at first glance displayed the caption: *La base de un gran futuro está en nuestro pasado* (*The Base of a Great Future Lies in our Past*). The original mural is part of a series reproducing 2000 years of Mexican history that Rivera was commissioned to paint for the Plutarco Elías Calles regime in 1929. Although Burchard simply 'got it from the California Lottery', the significance of the mural's placement, as the first thing one sees upon entering the space

FIGURE 2
***The Flower Carrier*, Diego Rivera, 1935. Courtesy of the San Francisco Museum of Modern Art**

where the analysis of DNA patterns takes place, is quite telling. The mural represents Tenochtitlán, the ancient Aztec capital, now Mexico City. The Aztecs were sophisticated mathematicians. Today they signify a distant source of modern Mexican origins that are presently rolled into the broader term 'Native American' for the marker panel.

In a conversation with one of Burchard's collaborators, an African-American geneticist who also uses AIMs, I asked why, as 'minority scientists', they frame their analyses of ancestry and risk with racialized genetic

FIGURE 3
***The Great City of Tenochtitlán*, 1945, Diego Rivera. Courtesy of the National Palace, Mexico City**



categories, rather than attempting to isolate social conditions that lead to asthma, cancer, and the many other ailments that now constitute racial health disparities? Before assuring me that he does incorporate the social, he first corrected me by saying: 'I don't use the word "minority". I use African-American. I don't want to give up my identity to simply be seen as a "minority".' I then asked what he made of the fact that Burchard uses the term? He replied, 'Esteban's nation building'. This phrase was meant to underscore the idea that Latinos were the fastest growing group in the US and that research on health disparities that disproportionately affect them is grossly underdeveloped. Burchard's genomic research 'empire' was arming to address both realities.

The murals, the explicit 'peopling' of this lab, and the reference to science as 'nation building' were some of the first indications to me of what these researchers held dear. How these signs translated into genetic studies would prove to be a more complicated matter.

The Public and the Political

Burchard forcefully helped to generate debates on the place of race in science and medicine by co-authoring two papers in 2002 and 2003 on the importance of race in genetics. Both papers advance the idea that humans can easily be categorized into groups based upon patterns in their DNA, and that such groups fall along socially understood racial lines. The first paper, by Burchard and his close colleagues Neil Risch and Elad Ziv, was a much-cited (and heavily debated) piece called 'Categorizations of Humans in Biomedical Research: Genes, Race, and Disease', published in the online journal *Genome Biology* (Risch et al., 2002). A year later, Burchard and Ziv co-authored the second piece with Risch and several other authors, entitled 'The Importance of Race and Ethnic Background in Biomedical Research and Clinical Practice' in the *New England Journal of Medicine* (*NEJM*) (Burchard et al., 2003). These publications placed Burchard and his colleagues at the center of an increasingly voluble conversation on the place and purpose of race in genetic research and medical practice.[25] The now-persistent question in both fields concerning 'Is there a genetic basis underlying what was previously thought to be merely "skin deep"?' (Brower, 2002) gets answered with a resounding 'yes' by Burchard and colleagues. Moreover, they frame their position as 'political', but not 'ideological'. Burchard's team presents its science, which is based on genetic markers and high-precision statistics, as *non*-ideological, which is to say, strictly *materially* informed. Dr A hoped to clarify any misgivings by saying:

> the eugenics movement was about ideology. Those scientists twisted science to advance their racist cause. We want to rely on good science and *hard data* to include minorities in medicine, not to advance an unjust agenda. We realize that one group could become stigmatized if we find the asthma gene to be associated with them, or their ancestry, but we also believe that it's important to a larger number of people to find those genes.

Burchard is highly aware of 'the ideological' as it relates to, and draws support from, his own research. When the *Genome Biology* and *NEJM* papers were published (again, arguing that one could 'categorize humans' into races based on genetic differences) he and his collaborators received hundreds of emails. As Burchard told me in an August 2003 interview:

> **EGB:** We got praise and criticism from, I would say, four different groups. On the praise side, we got praise from people who were really interested in studying minority populations and who really believed that we were on the right path. That was group A of praise. And, group B of praise was [from] the white supremacists [who said] 'Right on! You're confirming what we believed all along.' And the argument there is, you know, 'Yes, only a single base pair is required to cause cystic fibrosis, sickle cell anemia, Tay-Sachs, well, it's probably only a single base pair that's required to cause violent behavior in blacks, criminal behavior in blacks, cheapness in Jews.'
>
> **DF:** Are these white supremacist scientists? Do you mean to say that white supremacist groups are sitting around reading *Genome Biology*? Do you know who these people were?
>
> **EGB:** One of them – well they probably wouldn't identify themselves as white supremacists, but one of them is trying to link intelligence – well looks at head size and intelligence. And he has published papers –
>
> **DF:** So he's a scientist?
>
> **EGB:** Yeah. He's published a paper showing that blacks have the smallest heads, and Asians have the biggest, and that was correlated with – I don't know if you know about that?
>
> **DF:** 19th-century science.
>
> **EGB:** No, it was *recent*.
>
> **DF:** … I mean ideas carried over from that era.
>
> **EGB:** Oh, yeah. And then, on the David Duke site, their research, or what they talk about – I'm not sure that they do research – is very similar to what we said in the *Genome Biology* paper. And that's where we found out that we were referenced on their website.
>
> **DF:** Oh really …
>
> **EGB:** Then on the other side, on the critique side, I got letters [after] the *New England Journal* paper [was published] from what appeared to be African-American scientists saying that we were supporting the effort to put down the black man – or that we were supporting 'the conspiracy' – that's what it was – to wipe out the black race with HIV. [One of the population-specific alleles mentioned in the paper was a *CCR5* mutation that protects some Europeans against the virus.]
>
> **DF:** So that was [from] a scientist as well?

EGB: Yeah, I assume [so], from the tone of his email. He was scientifically savvy. And *then* we got critiques by email and in public saying that there is no [biological] basis to race and so forth. That's fine.

DF: And so did you answer any of these people?

EGB: No.

DF: No.

EGB: The first, well, the African-American scientist – and it's all presumptive that he's African-American – I did answer [him] and then I realized where he was going with it and I just stopped responding. I got about a hundred emails and Neil [Risch] got about 200.

DF: Wow. So you obviously couldn't go through all of those.

EGB: Oh, we went through them! But we weren't going to *respond* to any of them. And we also didn't respond to the letters to the editor in the *New England Journal*.

DF: So, if you *were* to make a general response, what would you say about your intention of doing this research?

EGB: So, I think as we stand today, we don't know the answer.

DF: To …?

EGB: To whether or not there is a biologic basis to race. We don't know. And if we're proven wrong, that's fine. But what many people have argued for is the cessation of this sort of research. My point is that it's premature, and we would be negligent in our responsibility as physician-scientists if we were simply to close the door on this sort of research simply to bow down to people with political interests.

DF: OK.

EGB: I *believe*, though, that there are biological differences between races. As a physician, I see different presentations of disease. I see different responses to medications. In my own hands, in my own lab, I'm seeing genetic differences between populations. It seems obvious to *me*. And I think it's our responsibility as physician-scientists, as *members* of this population, and as taxpayers who are funding the NIH [National Institutes of Health], that we *require* the NIH to study minority populations.

Burchard's duty to his 'own' community, as a form of care and ethics, is clear. And, in many ways, this form of governmentality, for him, trumps the 'political issues' and strange alliances of support surrounding his research.

Four years after their controversial papers were published, the 9 March 2007 issue of the San Francisco-based newsweekly *The Asian Week* ran a story on race and medical genetics entitled 'First: Do No Harm?' The story included an interview with Burchard that repeated some of the themes he

had covered in his conversations with me a few years earlier. In the same no-nonsense tone, he publicly told the weekly that he gets unexpected praise and criticism from both racists and anti-racists:

> The sociologists are afraid that one group will use this sort of information to try to subjugate another group … That's the fear. I mean, David Duke [former Grand Wizard of the Ku Klux Klan] probably loves the kind of research we do because it seems to play right into his supremacist views. (Picture, 2007)

One of the first discussions Burchard and I had was about how David Duke and other 'white supremacists' wrote to him and his collaborators, as indicated in the above transcript. On 10 March 2007, the day after the *Asian Week* article appeared, David Duke posted an open letter to Burchard on his website, <davidduke.com>. The former Klan leader first corrected Burchard's choice of identifiers for him before commending him for his work on the 'reality of race'. He wrote:

> I am not a supremacist … [but] I do believe that different population groups have different characteristics in important areas that reflect everything from disease rates to tolerance for medications – even psychological characteristics and tendencies. I do believe that your work and others who show real biological differences between races is important. You show that race is real, not a societal construct or some sort of conspiracy theory. (Duke, 2007)

The question is how do Burchard and others show that race is real and not a societal construct? Do they succeed in bracketing the societal in favor of the biological, or is the biologistical use of statistics informed by, and infused with, societal constructions of race?

From Hypothesis to Publication: Differentiating Black versus White Asthma

While still a fellow at Harvard, Burchard worked on the interleukin-4 (*IL-4*) gene, located on chromosome 5q31 – an area thought to be involved in asthma, based on linkage studies.[26] Under the guidance of Jeffrey Drazen, recent editor-in-chief of the *NEJM* (and who was editor when Burchard's controversial paper appeared there), Burchard undertook a project to show that a novel SNP in the *IL-4* promoter region was differentially associated with severe asthma in whites and African-Americans.[27] In the following excerpt, Burchard recalls the discovery, but in a strangely incomplete way. When recounting his findings on the dynamics of race and the correlation with asthma severity, he neglected to mention that the strong association between the genetic variant and asthma severity was actually *only* observed in the 'Caucasian' samples. He then explains how this work, without this important detail, would come to bear on his nascent research career.

> **EGB:** [At Harvard] I knew that working under Jeff it was an all or none phenomenon, I was either going to be mediocre or I was going to be outstanding.

> I was working 18 to 20 hours a day, at least 4 to 5 days a week just so that we could get this project finished. … We were looking at a genetic variation in a gene called interluekin-4, cytokine IL-4, and we wanted to see if it was associated with asthma severity. And Jeff had a sample of patients that I worked with – about 700 patients, about 130 of which were African-American and the rest were Caucasian. And what we found was a very strong association with a genetic variation of the gene and asthma severity, so if you inherited this mutation you're more likely to have asthma, or severe asthma, than not. The interesting thing was that [the] mutation was about 40 per cent more common in African-Americans than it was in Whites.
>
> **DF:** Hmm.
>
> **EGB:** And that was impressive. An impressive difference – the reason being is that asthma mortality and asthma severity is much more severe in African-Americans than it is in whites. At the same time the *Centers for Disease Control* published an abstract at a meeting that Jeff had taken me to in which they looked at mortality rates for Hispanics in the United States and they demonstrated that Hispanics in the Northeast, or the East coast, had a mortality rate from asthma that was three to nine times higher than Hispanics from the west, the Southwest or the Northwest.
>
> **DF:** Wow.
>
> **EGB:** And being Hispanic and having lived on both coasts I knew that what they were *really* seeing was a difference between Puerto Ricans and Mexicans. From my own data I knew that this genetic variant was more common in African-Americans and I also knew that Puerto Ricans had more African genes than Mexicans. … At that time I proposed the GALA study, the Genetics of Asthma in Latino Americans study, and it got funded right away.

Burchard continues:

> When I got involved in this project, it was like – analogous to falling in love. I couldn't sleep, I'd work all night, all I'd think about [was] what I was doing … five years later it is still driving me.

When reading the actual published paper on Burchard's *IL-4* study, one is beset with confusion around the question of race and asthma severity. First, a brief overview of the publication is in order. As with most studies on asthma, the operative measurement of severity here is a decrease in lung function, or low forced expiratory volume (FEV_1).[28] Concerning the *IL-4* promoter region and the 'races' of African-Americans and whites, some subset of each group indeed possessed differing allelic frequencies of the 'wild type' (usually considered the common type) and the mutation in question. The nucleotide (DNA 'letter' ATGC) considered to be normal at this position in sequence is C (cytosine), and the mutant consisted of a change from a C to a T (thymine). Four per cent of white asthmatics possessed two copies of the T allele at that locus (were homozygous for the mutant gene), while 30% of African-American asthmatics had the TT genotype. However, the paper went on to say, 'the overall levels of airway obstruction were similar in

the two groups despite these allele frequencies' (Burchard et al., 1999: 922). In other words, the genotype patterns differed, but the principal 'phenotype' (symptom expression) was the same for the two asthmatic groups, and hence could not be wholly ascribed to this genetic difference. Nevertheless, Burchard et al. still insisted on the 'significance' of the *IL-4* promoter region's 'differing racial frequencies'. Finally, they found that the TT genotype was associated with severe asthma, but *only in whites*. Although African-Americans possessed the allele almost eight times as often, it was *not* associated with severe asthma in this group.

On other matters, Burchard et al.'s reporting of a 'striking genetic difference' was further characterized by a salient omission of the prevalence of these alleles in the general 'African-American' and 'white' populations. Most statisticians would want to know if the pattern of allelic frequency difference observed in this study was the same for the general (healthy) population of whites and blacks. If, in general, whites had TT at a rate of 4%, then there would be no association between the prevalence of the allele and asthma in the affected asthmatics. (In other words, if non-asthmatic whites had TT at a rate of 4%, then there would be no basis to assert this association.) Furthermore, if non-asthmatic blacks had TT eight times more than non-asthmatic whites, then there would be no striking genotypic difference with regard to asthma either. Almost strangely, no such crucial information was given.

Again, one is left wondering why the main point that Burchard retained from his time at Harvard and from this study was that *blacks had the gene for severe asthma*, now, '40 per cent more often than whites', when there was zero correlation between the *IL-4* allelic change and asthma severity in the African-American subset of the study population. Finally, according to Burchard et al.'s paper, their genetic data in the *IL-4* study explained only 0.6% of the observed variance in FEV_1, or less than 1%. To put these oversights in context, it is critical to understand that Burchard was 'driven' by a personal need – passion even – to solve a maddening problem that he witnessed in the social realm, that Puerto Ricans were much sicker than Mexicans with the *same* disease. For this committed physician-researcher, his observations and experiences were fundamentally informative. It was race as biology, not environmental or social issues, that was at stake. This is to say, even though his first real study of race and genetics did not in fact show that African-Americans had a genetic basis for asthma *severity*, he retained that fact, because the genotype frequencies themselves did differ by race (despite the lack of an association in blacks, and despite adequate sampling of blacks [1999: 921]). It was simply the difference as he recollected it that proved significant for his burgeoning theory about African admixture and asthma severity.

Visions of 'Confounding' and 'Cause': African Ancestry and Asthma Severity

It was just a few short years after this work at Harvard that Burchard established his own lab largely on the hypothesis that black/white biological distinctions were driving the differences in asthma in the groups that truly

interested him, Puerto Ricans and Mexicans. These are two US populations that he felt often were overlooked in medical studies on complex diseases, in part because they were not of 'one race', but were rather, as he puts it, 'admixed', representing 'different admixtures of three major racial groups' (Burchard et al., 2005: 2163). Yet for Burchard, his colleagues, and many other contemporary genetics labs using this or similar techniques, 'admixture' could be an advantage – or a *tool* for mapping disease genes. Such an idea runs counter to the rationale that 'homogenous' populations, such as that of Iceland, are inherently better for gene mapping. They write:

> Recently admixed populations such as African-Americans and Latino ethnic groups are known to have areas of LD (linkage disequilibrium) that can extend over large chromosomal regions due to allele frequency differences between the ancestral populations. This increased LD among admixed populations can facilitate mapping complex traits in an approach generally referred to as admixture mapping.

A potential complication of studying admixed populations is the possibility of spurious association due to population stratification. If the risk of disease varies with ancestry proportions, this will create associations of disease with genotypes at any locus where allele frequencies differ between ancestral populations. Thus, admixture mapping first requires appropriate adjustment for population stratification (Salari et al., 2005: 77).

Through multiple and ongoing collaborations with Mark Shriver, the Burchard lab scientists have reconstructed Shriver's AIMs technology and have created their own 'home-kit' technology, which they use for two stated goals. The first purpose, simply put, is to separate the 'European', 'Native American', and 'African' parts of their Mexican and Puerto Rican samples' genomes, which then could be associated with phenotypes of asthma severity. By narrowing down the genome 'to chromosomes made up of segments with ancestry from different subpopulations' (McKeigue, 2005: 1), they hope to find asthma genes, and to do so with much less labor than with other linkage methods. The second stated purpose is to develop a method that will allow scientists to compare apples and oranges, or cases and controls, without letting the fact that they are both small round fruits define the result. In other words they are looking for the gene that makes the orange an orange (and not an apple), *not* what makes it round or fruit-like (traits they both share). In their terms, they want to control for the possibility of a spurious association between an asthma candidate gene in the cases versus the controls. Such potential spurious associations constitute what geneticists call 'confounding'.

Time and again these researchers used AIMs at every level of their analysis, which often confounded their own understanding of what they were attempting to do: Were they looking for genetic causes of asthma severity through 'ancestry' in any one analysis, or, were they attempting to control for confounding (in the technical sense defined above) in their asthmatic cases versus controls? They were doing both, and in doing both

they had trouble conceptually teasing out 'racial admixture', as conveyed by AIMs, as a cause for severe asthma, in and of itself, from simply using these markers to control for spurious associations that might present themselves as a function of shared genetic heritage. These two ways of thinking about the role of AIMs in defining causation, on the one hand, and isolating 'noise' to localize causation, on the other, collapsed into each other. A common refrain of Burchard, and of many members of his lab, was: 'the more African ancestry one possesses, the more severe their asthma'. Again, citing the Centers for Disease Control (CDC) prevalence levels, Puerto Ricans had more severe asthma than Mexicans, and, like African-Americans, they also had more African ancestry. Yet, it was never emphasized that Puerto Ricans also had more 'European' ancestry than Mexicans as well (according to Burchard's own bar graphs that were part of his regularly referred-to PowerPoint presentation). Although the team knew that it was not African-Americans who contributed African ancestry to Puerto Ricans, their formulation of Puerto Rican asthma severity made use of black Americans' asthma epidemiology, rather than that of any specific group in Africa. By the logic of some admixture mapping theory, one *ancestral* group in question should have a high prevalence of the trait or disease found in the 'admixed' group (McKeigue, 1997, 1998, 2005). This was the Burchard lab's stated rationale for assuming that 'the cause' of disease could lie in chromosomal segments contributed through particular ancestries; this was the appeal of using 'admixed' populations to find risk alleles. Yet, there was never any discussion in the lab of the prevalence of asthma in Africa or, more specifically, in Puerto Ricans' African 'parental' source populations. Race served to fill in such blanks.

When his own data eventually overturned his theory about blackness and Puerto Ricans, Burchard, 'surprised and confused', suddenly distanced himself from the idea that he was looking to associate ancestry and the genetic causes of disease severity. Instead he now highlighted that he was mostly interested in using such associations to control for confounding variables. In fact, these two processes are linked, both conceptually and methodologically, as admixture mapping requires controlling for the many loci which are not linked to the disease trait in question, even though they are linked to the ancestry in question (McKeigue, 2005: 1). In the following excerpt, I quote at length from a transcript I took manually while sitting in on a conference call with Burchard, the AIMs project post-doc and one other researcher, all of whom spoke with Dr A, a physician and statistician, on the first day that they undertook a full analysis of their genotyping data. During the conversation, Dr A takes a very careful approach to prepare Burchard for an unexpected result. He begins by slowly describing the p-values of different measures for comparing AIMs markers in Puerto Rican asthmatic cases and healthy controls.

> **Dr A:** For the chi-squared versus the delta values all we're really saying is that the markers are different between the two groups [Puerto Rican cases and Puerto Rican controls]. Now we can do individual estimates as well,

but what I've just seen [in doing the group analysis] is that, in the cases, the mean European ancestry is higher than the mean African ancestry. I think we can go ahead with this. We have a story to tell.

EGB: What is the story?

Dr A: The story is that European admixture is higher in the Puerto Rican asthmatics cases than in the controls.

EGB: [long sigh] … [silence]

Dr A: I don't see a problem with this – except for the fact that it goes against the initial hypothesis.

EGB: You mean that blacks were to blame [joking, looking at me] … Ok, Ok. The only problem is that whites have a lower prevalence of asthma than blacks.

Dr A: I understand, I understand, but this is our data. This is what our data is showing us. You just have to couch it [the fact that African-Americans have a higher morbidity and mortality]. You could say that this is something specific to Puerto Ricans. It could be something in terms of the way these people were referred to treatment, it could have to do with recruitment in clinics … that the Puerto Ricans who were seen have more white ancestry than the controls who were collected. [pause]… So what do you guys think?

[silence]

Dr A: … about what I just said?

EGB: I'm not sure. I'm trying to graph it to get a picture of it. [Burchard is at his computer, with his PowerPoint presentation open looking at the individual admixture bar graph for Puerto Rican cases and controls.]

Dr A: The bottom line is that there is a reasonable chance that we may have significance. Let me call you guys back in a few minutes. I'm going to see if this is significant.

Group: OK.

[During this time. Burchard looks at me shaking his head. He smiles, lowers his head, and resorts to humor again.]

EGB: So we can't blame the blacks? [laughs slightly]

DF: [I laugh with him] … In talking to [a colleague in a related lab], he didn't think that you guys would find that Puerto Ricans' African ancestry would be causal and Dr A was skeptical as well, but you've always thought that you would.

EGB: Hold on. I didn't– in 1996 and 1997 I thought that way, but as things have progressed in the research we've all started leaning toward–

[Dr A calls back]

Dr A: So, it's significant. The p-value is [0].0014. So it's statistically significant. What we're seeing is high European ancestry correlated with high risk in Puerto Ricans. African ancestry is correlated with *low* risk and the Native American ancestry is correlated with nothing.

EGB: So this is like the Knowler paper [on European ancestry increasing diabetes risk in Pima Indians].

Dr A: Well I don't know. [Hesitates] … I think we have something very interesting here.

EGB: I guess what bothers me is that we didn't find confounding [ancestry association] in the Mexicans and I thought that we would.

Dr A: … I think we need to do this with individual estimates and see if we can make these p-values go away [verification]. I think it will hold, and if it does we could start re-writing the paper and have it in the mail within a few weeks.[29] I guess this is all very surprising and confusing …

As one might expect, this was not the end of the story, but rather a new beginning. It was this first report of their genotyping results that, in the research team's view, unhitched African ancestry from an imagined causal agent. As Burchard saw the clear lines of his racial theory challenged by the data, he assured me that everyone had already begun to shift their thinking away from the African ancestry hypothesis. Yet, in talking to all of the researchers involved in this specific project during the following days, it was evident that they were just as 'surprised and confused' as Dr A figured Burchard was during the initial call. Until this exchange, there was a near 'social consolidation' (Fleck, 1935 [1979]: 47) among the group that African ancestry mattered for asthma severity. There were only two polite dissenters on the issue from the outset, both of whom gave Burchard the benefit of the doubt, while believing that 'something' would come of the inquiry.

In our discussions of the results, Burchard explained the implications for admixture mapping by saying:

> it's like freshly mixed paint. You can see the different colors for a while, until they are too mixed up to see any more. We think that the lines will be clear for ten generations [referring to the chromosomal parts that they call 'European', 'Native American', and 'African']. Ten generations, that's recently admixed. After that it's too mixed up to see anything.

When I asked what he thought about his first results – that European, not African ancestry seems to be the 'cause' of severe asthma – he replied, 'it's interesting. We're not quite sure yet what's going on. But we were never actually out to see which ancestry is to blame [association with ancestry]. It's about controlling for confounding [bracketing or holding constant spurious associations with ancestry]'. In the published paper – solely about ancestral inferred risk – many more iterations of analysis produced the

result that European ancestry was 'associated' with various severe asthma phenotypes in Mexicans (to blame), but *not* in Puerto Ricans (Salari et al., 2005). In the latter population, African ancestry was associated with a *beneficial* response to the frontline asthma medication, albuterol. Both findings went against Burchard's initial hypotheses. His revision of the terms and the causal arrows, however, was in no way a total paradigm shift. The racial triad still prevails in the final publication, which continues to assume that asthma severity can be linked with one of three groups: European, African, or Native American.

The Making of Latino Genetic Difference: Correspondences

> [T]he care of the self always takes shape within definite and distinct networks or groups, with combinations of the cultic [and] the therapeutic … [T]he care of the self is expressed and appears in this splitting into, or rather this belonging to a sect or group. If you like, you cannot take care of the self in the realm and form of the universal. The care of the self cannot appear and, above all, cannot be practiced simply by virtue of being human as such, just by belonging to the human community, although this membership is very important. It can only be practiced within the group, and within the group in its distinctive character. (Foucault, 2005 [1982]: 117)

In a mode of scientific nation building, largely based in identity politics, Burchard's lab constructs their studies with the following logic, or methodology. First, they comb the literature for promising studies on SNPs or haplotypes related to asthma severity or drug response.[30] Second, they determine if a study in question, and the SNPs found, was done by 'white researchers on white patients' (and in most cases such studies have been). And finally, they attempt to perform the research in question in their lab, on 'their populations' (Mexicans and Puerto Ricans). The logic often goes like this: if they find the polymorphisms in 'their populations' that had been found in the reference population (that is, in whites), then it is most likely that these SNPs will be found in Mexicans, because Mexicans are, by the researchers' reckoning, 'less African', which initially was understood as 'more European' than Puerto Ricans.

One example of the Burchard lab testing 'their Latino population' for markers that seemed to indicate an effect in 'whites' concerned a result found in the year 2000 on the beta 2 adrenergic receptor (*β2AR*). The original study was led by Connie Drysdale of the University of Cincinnati and *Genaissance Pharmaceuticals*. The Drysdale team found thirteen SNPs in the *β2AR* gene when '23 Caucasians, 19 African-Americans, 20 Asians, and 15 Hispanic-Latinos' were examined. All were healthy. Drawing upon what was discovered in this small 'repository', Drysdale and colleagues then examined a cohort of '121 Caucasian patients with asthma' to 'determine whether haplotypes of the *β2AR* gene were associated with an [increased or decreased] bronchodilatory response to the agonist albuterol'

(Drysdale et al., 2000: 10485). The research question was clearly pharmacogenomic in nature in that it aimed to learn something about how to tailor albuterol treatments based on genetic profiles. The published paper also claimed that there were 'ethnic' differences in SNP frequencies related to how asthmatics responded to this drug. Yet, Drysdale and colleagues did not have large enough samples from non-white groups to support a rigorous analysis. In addition, none of the SNPs found by the Drysdale team demonstrated any association with the albuterol drug response when examined alone. They noticed, however, that the 13 SNPs found 'were organized into only twelve haplotypes out of the theoretically possible 8,192'. Such limited haplotype diversity was coupled with the fact that two of the SNP combinations dramatically differed from the rest. They wrote: 'Based on *in vivo* data, it appears that haplotype 4 is associated with depressed responsiveness and haplotype 2 with increased responsiveness' (Drysdale et al., 2000: 10487). This original study, which examined a 'Caucasian' population, even though the variants were originally isolated in an 'ethnically' mixed group, merely tried to generate some hypotheses about ethnic difference in albuterol response based on 12 haplotypes. The prospect of finding such associations motivated Burchard to look at the same haplotypes in his population in a more 'robust' way (with more samples and, most importantly, 'ethnic' ones).

During one of the first *β2AR* data runs in the Burchard lab, each haplotype found in Puerto Ricans was also found in Mexicans, and vice versa. This was not what Burchard expected to see. At the same time that the GALA set was undergoing genotyping, Burchard and collaborators in San Francisco, Mexico City, and San Juan, Puerto Rico, were working on a paper based on their clinical observations. Their paper documented 'unique and important' findings that Puerto Ricans and Mexicans had 'differential' responses to albuterol, as well as other phenotypic differences (Burchard et al., 2004: 390). After an initial analysis of the genetic data, when one of Burchard's post-docs and Dr A noticed that the Puerto Ricans with severe asthma possessed a certain haplotype, they also noticed that the Mexicans had the haplotype at the same frequency. The post-doc assumed it was a mistake, and said: 'but in the clinical paper Esteban says that the Puerto Ricans are more severe and that they don't respond as well. We should be seeing some genetic difference in the two groups.' Dr A, still slightly wary of the simple, direct racial hypothesis, replied: 'I wouldn't buy land just yet.'

Meanwhile, the team was waiting for more genotypes to arrive from the lab of their collaborators at Harvard. The data would arrive in batches and upon arrival the new data would be added to the database and analyzed. During one of these waiting periods, two researchers assigned to working on this gene were mulling over the available data. For days they would 'play with it', meaning that they would examine the sequences for patterns of association using two different software programs, the Transmission Disequilibrium Test (TDT) and the Family Based Association Test (FBAT). They focused on three of the various manipulations that can be

made with these programs. When I asked about this process, one of the younger researchers who was on her way to graduate school confided:

> Well, if there is an association that we know is there, but it's not very strong, then we could manipulate the data to make it stronger. These genotypes are specific in Caucasians and we know that they are different in minority groups. So we want to make that difference stand out, which needs to be done, or else science will never change. People will just keep looking at Caucasian genes.

When I asked her if 'trying to make the data stronger', even when done to make science and medicine more inclusive, could compromise the truth of their findings, she responded:

> I see DNA like something that changes depending how you look at it and who's looking at it. It's like the idea of light in physics. The question is always is it a particle or a wave? Some look at it one way and say, 'it's a particle'. Others, look at it another way and say, 'it's a wave'. There's this philosopher – I can't remember his name – that said 'our world is shaped by the way we look at it'. I think a lot of science operates this way. … Truth is something else. I'm not sure if it's attainable. We can get precision – we can have 95% correct observations.

There are scientifically legitimate reasons to analyze datasets using many different statistical tests and to focus only on the tests that yield statistically significant p-values. Such an approach should not necessarily be perceived as 'manipulating the data' in some pernicious way. Such reasons have to do with finding which test better organizes, or groups, patterns of SNPs found in families with severe asthma compared with those found in controls. Perhaps because the researcher who was interviewed had not yet attended graduate school, and had not yet taken enough statistics to fully understand those reasons, she relied on an ethos that she felt was socially acceptable, and more importantly, operative in the lab.

At one point in the *β2AR* analysis, the researchers began to see a pattern in the Mexicans' DNA that seemed to diverge drastically from the Puerto Ricans' data. To their dismay, however, the event did not persist, and they eventually ascribed it to genotyping errors. After they caught the error, and had nearly completed the genotyping, Burchard and his team located Drysdale's haplotypes 2 and 4 in both groups. The 'severe' haplotype 4, which was associated with decreased responsiveness to the medication in question, was found with the same frequency in Mexicans and Puerto Ricans, but so was the 'good one', haplotype 2.[31] In other words, the expected stark genetic difference did not materialize. Nonetheless, the researchers found a way to make their findings cohere with their vision of how the Mexican vs Puerto Rican DNA should behave. Given their results, they did not so much 'manipulate the data' as adjust their framework, slightly. They let the DNA, as they say, 'tell its story' but on their terms. At this point, 'ethnicity' – the fact of being Puerto Rican versus Mexican – came to matter more than 'the genes' that each possessed, since the same

genetic base pairs (as they existed across the board) were now interpreted to be the 'same genes' but 'doing different things depending on ethnicity', according to Burchard.

This last point could have prompted a radical reinterpretation of their data, as well as a crucial theoretical shift away from a simple inquiry that aimed to link genes with race to one that linked race with recent (versus generational) immigrant status, environment, ecology and biology (which includes but is not limited to genetics or allelic frequency distributions in their bodily and population contexts). Bioethicists, sociologists (Ossorio & Duster, 2005) and epidemiologists (Cooper et al., 2000, 2002; Cruickshank et al., 2001; Krieger, 2001, 2005b; Krieger & Smith, 2004; Wright et al., 2005) have repeatedly conducted, and called for further, research into how 'race' as a social and political phenomenon produces sick biological outcomes. These outcomes are not 'natural' in the sense that they are caused by something called nature that yields specific and immutable conditions. Instead, they result from the variable ways that social position and racial status in American society become embodied (Abraham, 1993; Krieger, 2001, 2005a), and where disease symptoms are 'socially significant signs' (Scheper-Hughes & Lock, 1987, 1991).

The levels of detail involved in genetic expression (Lock, 2005), much less these larger issues, were not the focus of this lab. What might mitigate or instigate differential phenotypic expression (for example, of severe asthma) in Puerto Ricans and Mexicans, was simply, 'ethnicity' and, alternately, 'race' (their percentages of 'African' vs 'European' 'admixture'). Indeed, Burchard's group would later write:

> our results demonstrate that Puerto Ricans with asthma have an ethnic-specific genetic predisposition to more severe asthma. Specifically, our study of *β2AR* polymorphisms shows that the Arg16 allele is significantly associated with asthma severity and bronchodilator responsiveness in Puerto Rican but not in Mexican subjects with asthma. (Choudhry et al., 2005: 568)

Like the 'good' and 'bad' haplotypes discussed above, the allele in question was present in both groups *at the same rate*. Yet given these researchers' 'drive' to resolve their community's asthma health disparity through genetics, which they based on the black/white differentials they witnessed in the US, it was not necessarily the genetic change per se that was at issue. Rather, it was the 'ethnic-specific genetic predisposition', Puerto Rican versus Mexican 'admixture' composition, that mattered.

In the end, their emphasis on 'ethnic-specific genetic predisposition' proved to be a safe presentation of the issues. Today they continue to look for underlying ancestral/raced genetic causes, but they have also begun to change course and to incorporate into their analysis social and environmental variables that might be associated with 'ancestry' and 'ethnicity' (Choudhry et al., 2006; Peralta et al., 2006). Yet, despite their recent inclusion of non-genetic factors, at the outset of many of their studies, as well as later, it was clear that their focus was fundamentally *not* on the environment, nor was it on social determinants of health. As they wrote in 2005, 'By

design, neither environmental nor cultural differences were a primary focus of the GALA (the Genetics of Asthma in Latino Americans) Study, and therefore, could be confounders' (Choudhry et al., 2005: 568). Today they are taking these 'confounders' a bit more seriously in part because the genetics of AIMs have not borne out the clear story that they had imagined.

Race on the Bus Today

On my way to the lab one spring San Francisco morning, a curious thing happened that drove home the utter messiness of the 'environment' in which these scientists work; an 'environment' in which the public also lives, and in which I must finally situate my own narrative of their research to sketch a conclusion that is in no way a 'final reading' (Crapanzano, 1986: 51). After having boarded the bus at 24th Street and Mission, I noticed a wheelchair-bound black man approaching the door. He had waited for everyone to embark before situating himself at the vehicle's entryway, where he then engaged the driver in conversation. A few minutes later the driver lowered the wheelchair ramp to allow the man on. Once aboard, the man and the driver buckled his chair to the bus wall. The man sat in the space designated, reserved for 'the pregnant, elderly and handicapped'. In the back of the bus, another transport employee, also a black man, who had recently gone off duty and was visibly tired, yelled to the front of the bus over the crowd: 'you should've gott'n on first, and you wouldn't be takin' up everybody's time!' The man in the wheelchair told him to mind his own business. The off-duty bus driver nevertheless continued his rant:

'You takin' up my time. You and yo' wheelchair.'

At this point the wheelchair-bound man looked back, pointed his voice toward the anonymous yeller and said: 'You just sayin' that 'cause ya black!'

The Mission, as stated earlier, is largely Hispanic, but also quite racially diverse. That morning, however, there were virtually no black people on the bus, other than me and the two men now engaged in this strange verbal row. A silence overcame the other passengers, who were mostly Latinas accompanied by their children and who tried not to make eye contact with anyone.

'I ain't black!', the man in the back cried back. 'Black? Look at me! I'm Choctaw!'

'You black! Ya hear me? Black!' the wheel-chaired man retorted.

'I'm Choctaw. I know m'a roots … !'

At this point a white man – thin, with a sallow face indicative of near starvation, or perhaps chronic amphetamine use – clad in death rock black, came alive. 'Hey brother, you Choctaw? I'm Choctaw too!'

Then the white man in black got up from where he was sitting and joined his new 'Indian' brother a few seats back. After establishing their respective places of origin and shared roots, both 'Indians', one visibly white, the other black, continued to taunt the sole black man now on the bus.

As I arrived at the lab, where race was neatly labeled on DNA plates and vials, while it completely structured the databases as well as the vehicular language of the lab scientists, I wondered about the porous relationship of science and society. Why were this morning's observations on race, on the

bus and in the lab, so starkly different? Or were they? The men on the bus, who freely, and messily, donned new races and identities at their convenience, on the one hand, had no place in the orderly lab environment where the quest for racial precision reigned. On the other hand, however, if confronted with the bus drama, which empirically attests to peoples' own visions of their complex identities and histories of mixing in contemporary America, my scientist-informants might claim to have the right tools to resolve the dispute. Those who use AIMs believe that this technology could tell if the black off-duty bus driver was really 'Native American', and, if so, they would then set out to find, what percentage of him was 'Indian'. What if – as is the case, according to the AIMs science – his ancestry resembled that of many black Americans, with 'Native American' ancestry on the order of 2%, 'European' at about 20%, with the remainder said to be 78% 'African' (Shriver et al., 2003: 391)? Would such a small 'Native' proportion (perhaps Choctaw) simply take on the symbolic power that 'one drop' has historically enjoyed, and order this man's identity toward the most minute 'minority' fraction to substantiate his claim? Or, would such a 'drop' cease to matter if the man on the bus read the disclaimer on the *DNAPrint Genomics* website for AIMs, which says that this test's standard error for individuals tested with 174 AIMs is anywhere between 1.5 and 15%? Consequently such small percentages may *in fact* be, in the website's words, '*artificial ancestry*'.[32]

Given such options, new needs and invented demands, how do we distill the artificial from the real in today's world? Or, more to the point, how is it that notions of race today are being rearticulated to include such scientific precision in a search for one's 'deep roots' even though critics might point out limitations to the science all the while conceding its appeal (Duster, 2006b)? Increasingly, 'old' social identities bear 'new' specificity and definition through biological markers, such as AIMs (Rabinow, 1992: 245; Lock, 1993: 39), especially when contested histories mix with claims to privileged positions for cultural, political and economic resources (Abu El-Haj, 2004; Brodwin, 2005; Koerner, 2005). My point is that how one integrates biomarkers of ancestry, artificial or not, with assumptions about race and one's own sense of group belonging inherently relies on their perceptions of cultural difference as well as affinity. The reason that the bus altercation initially seemed so strange has everything to do with the fact that such incidents of spontaneous racial denial and swapping are, despite narratives of 'passing', quite rare. Neither of these men was 'passing'. One may have been looking to dissociate himself from a source of his annoyance while the other may have been seeking affinity. Dissociation and affinity, difference and similarity, have been with us for perhaps as long as our genes and our ideas of human groups.

Conclusion

This paper has provided an ethnographic account of how racial thinking persists in a contemporary American laboratory focused on complex problems of health disparities in asthma. One of my objectives has been

to show how new genetic technologies that link geography and 'ancestry' do not necessarily depart from older notions of 'race'. Although scientific attention to geographical place and population migrations over the past 500 years hints at an inclusion of the complexities of human variation and migration history that researchers need to take into account, the AIMs model proves static as soon as one questions the nature of the 'parental' populations that the model treats as fixed points of departure. Culturally, the idea of 'admixture' again makes sense in contemporary America. Admixture is an old idea, as the concept of African-American mixture with whites influenced some scientists' thinking about sickle cell anemia severity at various points during the past century (Tapper, 1999: chapter 2), while various markers were tested in 'American Negroes' to determine their real status as 'biracial hybrids' (Reed, 1969: 762). Today, admixture theories rely less on a notion of hypo-descent to solidify one's identity than on a notion of racialized genomic fractions, or parts, that constitute potential disease risk. The ways that admixture is thought about at present more closely resembles the use made of it by European scientists in the 1930s. In *We Europeans* Julian Huxley and A.C. Haddon (1939: 49–50) discussed traits of 'the Dinaric type', 'the Mediterranean', 'the Alpine', 'the Nordic', and 'the Jew' among others. Using these types, they purported to identify a statistically significant prevalence of traits found in people living in various geographical regions within Europe. Today, by North American standards, these varied Europeans of the 'Old World' would all simply be labeled 'Caucasian'.[33]

For most Americans today the 'Dinaric type' means little. Instead, conceptions of 'African ancestry' or 'Native American ancestry', and calculations of their percentages, have made their way into consequential discourses about tribal affiliation, criminal culpability, disease risk, pharmacological susceptibility, and personal narratives about identity and race, while the technology that supports their use has become a teaching tool in some university departments, such as African-American studies and sociology.[34] There is much that is compelling about AIMs. In a healthcare system with data collected mostly from whites, the intentions of scientists like Burchard to 'include' (Epstein, 2007) groups that are thought to be 'hard to study', for both social and biological reasons, are well placed. As I have shown, Burchard made it his personal mission to include minorities in genetic studies because he believed they have the civic right as US taxpayers to 'be included in the genetic revolution'. His fear of his community's exclusion from the revolution meets a reality on the ground where many minority groups distrust institutionalized science, producing a situation where some, including colleagues on Burchard's own faculty, may think that a member from a minority group would be a safer, trusted person to shepherd his community into research on biological difference at the level of 'minority DNA'. As Burchard told his mentees time and again, 'if it feels this good being used, I don't mind being used'.

Surely there are differential disease outcomes and other health disparities between US racial groups that will be observed time and again until they are

redressed. As several of Burchard's and colleagues' investigations revealed, phenotypic differences may or may not correspond to genetic patterns that occur at different rates in comparisons among demarcated groups. My point has been to interrogate the nature of these differences and to probe how they are situated within logics of disparate races – races teased apart and triangulated as distinct points of humanity through genetic markers. I have asked whether the genetic differences found in this lab's studies were in fact racial by 'nature' – or, by cultural, scientific practice?

In 1935 philosopher, sociologist, and historian of science Ludwik Fleck distinguished between what he called 'passive elements' – the categories of 'real', 'objective' and 'true' in science – and 'active' elements that belong to the realm of 'cultural history'. Fleck conceded that the imagined line between passive and active elements was fuzzy at best, and that the associations, or *knowledge*, they yielded for science could only come about through their 'inevitable' unification (Fleck, 1979 [1935]: 10). At present, the biologistical construct of race, in which DNA markers and American racial taxa have been brought into correspondence, takes Fleck's concept of unification, itself an 'admixture', a step further. As concerns the AIMs technology and social notions of race today, there is no a priori distinction. The passive and the active, the 'real' and the 'cultural', are of the same species – of the same 'race'. As a model, AIMs may be effective for programmed purposes, and these purposes change depending on social, historical, and medical context. The jury is still out on whether or not admixture mapping will yield incontrovertible discoveries of actual disease genes.[35] If it does, such a feat will result from the use of this system of markers as a linkage tool – as the chosen AIMs may serve as chromosomal landmarks linked with traits of interest – more than any direct association with race. That AIMs test results are thought of in racial terms, and that the markers collected for this technology were triangulated to reflect race in North America, is a mutable trait of the science that will continue to correspond to aspects of researchers' cultural and political lives that are currently *(un)*seen within the technology itself.

Notes

This paper is part of a larger research project and has therefore benefited from many conversations with more people than I can name here. I am especially grateful to all of the scientific practitioners who took the time to explain the intricacies of their work and for enrolling in this research project. I also thank them for their commitment to ongoing conversations on these important matters. An early version of this essay was presented at the Institute for Advanced Study in Princeton in November of 2004, the Departments of African-American Studies and Sociology at Yale in the Race, Health and Medicine Lecture Series in 2004, at the Stanford Center for Humanities in May 2005, and in the Harvard Anthropology Department in 2006. I thank the members around the table at all events for their insights. I would also like to acknowledge the helpful comments of colleagues who have read more recent versions and/or have engaged me in discussions around various points. These include Kjell Doksum, Troy Duster, Joan Fujimura, Rebecca Herzig, Jonathan Kahn, Jay Kaufman, Arthur Kleinman, Nancy Krieger, Michael Montoya, Alondra Nelson, Paul Rabinow, Rayna Rapp, Helen Tilley and four anonymous reviewers. This research was funded by the National Science Foundation under Grant no. 0208100.

1. In a chapter of *In the Blood* entitled 'An Anthropathology of the "American Negro"', anthropologist Melbourne Tapper (1999) examines the prominent view of 1950s sickle cell anemia researchers who upon learning of sickle cell trait rates in Africa (compared with the US) rushed to implicate 'race mixing' as the culprit for the seemingly higher rates of the disease in the 'American Negro'. Tapper cites researcher A.B Raper, whose words succinctly exemplify the imagined role of admixture: 'It is therefore … a possibility that some factor imported by marriage with white persons, is especially liable to bring out the haemolytic aspect of the disease, while the anomaly remains a harmless one in the communities in which it originated' (Raper, cited in Tapper, 1999: 41). The full scope of this logic is wonderfully documented by Tapper, who analyzes studies conducted in North America, the Caribbean, and Africa that permitted researchers their stance that racial admixture led to higher and more severe sickle cell incidence in the US.
2. In *The Retreat of Scientific Racism*, Elazar Barkan argues that, within circles of biologists in Britain and anthropologists in the US, race began its conceptual decline during the interwar period, and not after. He argues that, by the time of Nuremberg, race in science had long been repudiated and that the trials merely disseminated to a larger public what was already taking place in scientific thinking. I would argue that this idea suffers from a lack of nuance, as he writes: 'By 1950, while no consensus had been reached [regarding the concept of race], racism had been refuted' (Barkan, 1992: 342). Also see note 3.
3. As the rapporteur of the second UNESCO session on race, biologist L.C. Dunn, stated:

   > We were careful to avoid saying that, because races were all variable and many of them graded into each other, therefore races did not exist. The physical anthropologist and the man in the street both know that races exist; the former from the scientifically recognizable and measurable congeries of traits which he uses in classifying the varieties of man; the latter from the immediate evidence of his senses when he sees an African, a European, an Asiatic, and an American Indian together. (Dunn, in Montagu, 1972 [1952]: 140)

4. The term 'cline' was first introduced by Julian Huxley (1938: 219), who wrote: 'Some special term seems desirable to direct attention to variation within groups, and I propose the word *cline*, meaning a gradation in measurable characters.'
5. See Jennifer Reardon's *Race to the Finish* (2005: 32–41) for an in-depth discussion of the points of difference proposed by various authors of the second UNESCO statement, as well as for an account of the political contexts and consequences of different contributors' stances.
6. Francis Collins has since revised his statement to concede that 'it is not *strictly* true that race or ethnicity has no biological connection' (Collins, 2004: S13, emphasis added).
7. In the larger ethnographic project from which this paper comes, I have witnessed some scientists consciously avoid using the term 'race' in favor of 'ancestry'. For instance, one inventor of the AIMs technology, Mark Shriver, prefers 'biogeographical ancestry', or BGA, as part of this widespread move. In an April 2004 interview, Shriver confided that his earlier terminology of 'race', and also of 'ethnic affiliation' (Shriver et al., 1997), 'was a poor choice of terms'. He then continued, 'but you know it was a stage in my development and the development of the field. [The paper that uses the language of "race" and "ethnic affiliation"] is a great paper, actually, if I do say so myself. It's good science.'
8. Specifically, according to one set of researchers, 'Because Latino populations represent different admixtures of three major racial groups, it may be possible to begin to unravel some of the differences in disease incidence and outcomes through modern genetic techniques and a variety of epidemiological study designs' (Burchard et al., 2005: 2163–64).

9. Where the production of novel race measuring tools and products is concerned, processes of 'co-production' (Fujimura, 1996: 18; Fujimura & Chou, 1994; Jasanoff, 1996: 397; 2005; Reardon, 2005) are helpful but not sufficient to describe the dynamic that I am calling correspondence. Race is not brought into being *alongside* the scientific technology described here in a process of 'simultaneity', as Reardon has argued was the case for 'human genetic diversity' (2005: 6). Rather, the theoretical assumptions embedded in technologies of race and race apportionment have long preceded the present-day genetic tools and products that I detail in this account, which I would also argue was the case for how older notions of human difference got revitalized – as they remained largely unrevised – by the Human Genome Diversity Project. In short, we need to be careful not to lump everything into a co-production framework that implies co-emergence. What I describe here comes closer to processes that detail '*mechanisms of articulation* of the "bio" and the "social"' that allow emergent manifestations of biosociality' (Sunder Rajan, 2006: 159; italics in the original; also see Rabinow, 1992).
10. For a larger discussion of how 'admixture' relies on racial identities that remain separate from the 'one drop' that partially defines them, see Abu El-Haj (2007: 288).
11. Anthropologist Paul Rabinow (2008: 90–98) explores the genealogy of how scientific knowledge came to seen as a dispassionate enterprise, and argues that such an assumption represents a near impossible empirical reality, especially given the 'vehement contemporary' situation of much genomic science.
12. Clearly, however, 'precise' does not necessarily translate to 'correct'. As historical critic Mary Poovey (1998: xii) writes: 'even numbers are interpretive, for they embody theoretical assumptions about what should be counted, how one should understand material reality, and how quantification contributes to systematic knowledge about the world'. With regard to the potential fluidity of race based on the new precision of ancestry genetics, Sandra Soo-jin Lee et al. (2001: 52–53) write:

    > Reductionist research that locates ethnic identity in genetic variation confounds the notion of malleable identity. The implication of such research is that self-identity may be supplanted by a genetically based identification of individuals and groups. The result of such a shift in which identity is no longer a product of self-definition, but rather, a scientific ascription, has serious implications for how race and ethnicity will be conceived. Critical to this shift in identity politics is the explanatory power of genetic discourse in its appearance and 'allure of specificity' in classifying individual identity.

    On the notion of the power and the 'allure of specificity' see Conrad (1999: 228).
13. For a detailed analysis about this software program and researchers' assumptions about race when deploying it, see Bolnick (2008).
14. The private company for which Mark Shriver is a consultant and important shareholder is called *DNAPrint Genomics*. The commercial website is *dnaprint.com*.
15. The most utilized exception is a variant of the Duffy marker (*FY*), which codes for a protein receptor on red blood cells that facilitates vivax malaria parasitic infection. Most West Africans who are resistant have another variant, *FY*-null.
16. Because this system of markers is the basis for patent applications on the part of its inventors, not all of the SNPs are openly discussed. The genes that are listed here appear in these scientists' publications, and are indeed AIMs markers; yet, in some instances what is considered the 'normal variant' (consensus sequence) is not used as an AIMs marker while in other cases it is. The generic names (ontologies) are used here, since it is impossible to know to which of the exact variants the ontology listed by the researchers refers.
17. Ludwik Fleck observed 'that the social character inherent in the very nature of scientific activity is not without substantive consequences: 'Words which formerly were simple terms become slogans … [and] this completely alters their socio-cognitive value' (Fleck, 1935 [1979]: 43). It is clear that the language used in concert with AIMs

imbues them with powers and connotations that allow a belief in their structure of comparisons between the 'Old World' and the 'New'.

18. Ten generations is the point at which admixture is thought to become indiscernible. Therefore the present moment is seen by these researchers as a fleeting one, and with it may go their ability to locate asthma genes.
19. Krieger writes:

> As emphasized in recent scholarship, choice of time scale – often shaped by unconscious belief as well as conscious design – can exert profound effects on scientific analysis. This is because the framing of scientific questions depends heavily on assumptions, usually more implicit than explicit, regarding the appropriate time frame, level and scale of analysis. (Krieger, 2005b: 2157)

20. As noted earlier, most researchers who worked on AIMs did not know what these markers were. When asked, those doing both genotyping and analysis 'had no idea' when I inquired about the possible evolutionary histories and biological functions associated with AIMs, or how the markers worked in tandem as a technology. They simply 'knew' that they worked to differentiate ancestral, or 'racial' origins.
21. US Patent No. 20040229231 (filed 18 November 2004), 'Compositions and Methods for inferring Ancestry': 7.
22. All informants except for one gave consent to use their real names for this research. I sought Institutional Review Board (IRB) approval to use an 'attribution statement' that would allow me to use real names if subjects agreed since many of the scientists presented here will be publicly recognizable to readers who follow these issues. When possible, I have, nevertheless, avoided using names regardless of consent to do so. IRB approval for this research was granted by New York University.
23. It has since moved to the new Mission Bay campus.
24. See <www.sandlerresearch.org/>.
25. As of 11 July 2007, according to the *Web of Science*, the *NEJM* paper has been cited 184 times in just 3 years and the *Genome Biology* paper, which is not picked up by the *Web of Science,* is listed as 'highly accessed' on *Genome Biology*'s website.
26. *IL-4* is a gene involved in various aspects of immune system regulation. Variants of it have been associated with different asthma phenotypes as well as autoimmune diseases.
27. The promoter region is a combination of short sequence elements to which the enzyme RNA polymerase binds in order to initiate the transcription of a gene (one step in gene expression).
28. The measure used, FEV_1, is part of a measurement called spirometry. It is the volume of breath exhaled during the first second of a forced expiratory maneuver started from the level of the patient's total lung capacity. The patient's score is measured against a set of 'predicted' reference values. In this case, severe asthma was described as an FEV_1 less than 50% of the predicted score for their age, gender, race, and height. As Lundy Braun's excellent history of these measures reveals, FEV, along with forced vital capacity (FVC), was introduced into US medicine as a racialized technology and was initially used as an anthropometric tool to assess the fitness of white versus black soldiers at the end of the civil war: see Braun (2005).
29. They already had a near-complete draft of the paper on the issue written before they had their complete results. In the paper, African ancestry was associated with severe asthma in Puerto Ricans. When I asked Dr A why, and how, they could produce a near-complete draft before the data was in, he simply responded: 'because we're impatient'. When talking with other researchers, many have said that they at least write up their methods sections and introductions before the full story is known.
30. A haplotype is a pattern of alleles linked on a single chromosome.
31. In the eventual publication of these data, the Burchard lab focuses not on Drysdale's haplotype 2, but rather on a specific coding SNP. The SNP in question, again, has

similar frequencies in both Mexican and Puerto Rican asthmatics (see Choudhry et al., 2005).

32. For the *DNAPrint Genomics* account of 'Accuracy and Precision', see: <www.ancestrybydna.com/welcome/productsandservices/ancestrybydna/accuracyandprecision/> (accessed 13 January 2008).
33. In a concise historical review of why and how the term 'Caucasian' became the preferred racial descriptor of Europeans, starting with Johann Friedrich Blumenbach, Nancy Krieger writes: 'In brief, the region of the Caucasus (located between the Black Sea and the Caspian Sea, abutting Europe, Asia and the Middle East) provided a "safe" place on which to project back a common European Ancestry without getting embroiled in volatile nationalist politics.' It also was thought to be the place where Zeus seduced Europa, where Prometheus was bound, where Noah's ark landed, and where Blumenbach found what he considered the 'most beautiful race of men [and women]' (Krieger, 2005b: 2156).
34. For details on AIMs testing for Black Seminoles' claims to tribal access, see Koerner (2005), for a comprehensive analysis of tribal testing see Tallbear (2008); for two instances of forensic uses of AIMs, see Wade (2003) and Willing (2005), see also Ossorio (2006) for a critical analysis. Concerning the use of AIMs in race apportionment, theories on disease risk and pharmacological susceptibility, see Burchard et al. (2005) and Choudhry et al. (2005); for more on accounts of both race and identity quests, see Kaplan (2003), and the WABC (2003) program 'America in Black and White: A Question of Identity', and the WGBH (2006) program, 'African-American Lives'. Two examples of AIMs testing in university classrooms are featured in Daly (2005) and in WGBH (2006)
35. Recently admixture mapping models have, through linkage, helped researchers locate areas of the genome that indicate prostate cancer risk, while no gene has yet been implicated, see Freedman et al. (2006).

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