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# The Molecularization of Race: Institutionalizing Human Difference in Pharmacogenetics Practice

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In the contemporary United States an increasingly palpable scientific trend to individualize medicine has ushered in new debates on the biological basis of race. Medical researchers, consumers, and public science funding agencies are all grappling with how much importance to attribute to racial difference in drug and disease susceptibility. Many are furthermore trying to determine when, and at what level, racial differences influence, and are influenced by, biogenetic factors. As health disparities for common diseases are evermore characterized as disproportionately burdening African Americans, Native Americans and Mexican Americans, research on the genetics of race for health is increasingly framed as an anodyne ethical obligation. Those who take such stands argue that a 'colorblind approach to medicine will lead to a disservice of minorities' (Risch *et al.*, 2002, p. 11; Tate and Goldstein, 2004, p. S40) and 'will retard progress in biomedical research' (Burchard *et al.*, 2003, p. 1171).

Nonetheless, the difficulties of racial conceptual coherence abound when dealing with *Homosapiens* (Kittles and Weiss, 2003, p. 36; Lieberman *et al.*, 1992, p. 302). Many wonder how medicine can faithfully deploy a concept that has repeatedly been deemed genetically tenuous and scientifically arbitrary (Graves, 2001, pp. 182–185; Smedley, 1999, pp. 307–309). Today various genetic units of analysis allow researchers to focus on the *frequency* of traits (rather than dichotomous values) in some groups when compared to others. Will the observation of such frequencies, as seen in US 'Caucasians' as opposed to 'minorities' outdo and rewrite the former claims that race is not genetic (Lewontin, 1972; Livingstone, 1969)? Lastly, will institutionalizing race at the molecular level truly prove therapeutic for individuals, and also for society? Such questions constitute some of the most basic practical concerns for pharmacogenetics scientists as well as for those concerned with health disparities in the life and social sciences more broadly. An attentive reading of one key set of papers in these debates reveals that neither prize nor

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peril has an obvious monopoly on answering either question in stark black-and-white terms (see Burchard *et al.*, 2003; cf. Cooper *et al.*, 2003).

At stake is the possibility that aiming for health equity through the biological prism of race may paradoxically *re-inscribe* to the letter (to the As Ts Gs Cs of our genetic code) a belief in racial difference. The danger of such a prospect potentially includes a biological reification of health disparities—allowing for an eclipse of the social, medical market and ecological determinants of disease that social science health researchers suspect are at issue (Dressler *et al.*, 2005, pp. 235–236; Ossorio and Duster, 2005; Graves, 2001, p. 171; Krieger, 2000; Montoya, 2003, p. 96). Another concern is that medical and societal inequities of the racialized groups at the centre of much of today's research will simply increase with 'race-based medicine'. These are clearly vital issues.[1]

Here I shift the focus slightly to an equally important matter that has received surprisingly little attention: that is *how* do scientists today use race as a biogenetic entity? It is my view that social scientists must do more to understand the work of racial logics in the research settings—in this case, in two related pharmacogenetics laboratories—where calls to renew our regard for race in science originate.

## Race and the Genome

In the early 1990s the United States Department of Energy (DOE) and the National Institutes of Health (NIH) began a massive funding effort as part of the international Human Genome Initiative to map the genetic information found in each cell of the human body known as DNA. At the time, ideas on how this eventual knowledge would better the human condition seemed limitless. James Watson, who helped to discover the structure of DNA, was one of the many prominent figures who publicly announced that gene therapy would right the wrongs that genetic fate conferred on those with rare diseases. Another hopeful notion also prevailed in the early days. This was the thought that humans shared a common genetic make-up, or that anyone's genome would do for the map, irrespective of race. Nonetheless, J. Craig Venter, who led what became the privately funded arm of the American project, sequenced the DNA of a handful of people from 'diverse' US racial groups, consciously remarking: 'it would be fundamentally wrong to end up with five white men' (Shreeve, 2004, p. 220). And thus, his team mapped three women and two men who self-identified as African American, Asian, Caucasian, and Hispanic. This move also gave the genome mappers a sample, albeit small, from which to conclude that humans are all 99.9% the same at the level of their three billion base pairs of nucleotides (called adenine, thymine, guanine, and cytosine) that make up the genomic ensemble of our DNA. Both ideas—that research should focus on genes themselves as the targets of therapy and that race is not genetically salient—have proven more complicated than initially pronounced.

Today the value of what is now called the 'Genome Era' (Bonham *et al.*, 2005, p. 9) lies less in gene therapy than in new opportunities to tailor genetic profiles to the right pharmaceutical therapies, and vice versa. Such a value has furthermore incited a literal potential increase in pharmaceutical worth: a departure from the 'one-size fits all' to a 'many sizes' approach to drug prescription (National Institute for General Medical Sciences, 2000). In its first instance (the African American heart medication called BiDil), race-tailored therapy has dramatically increased cost for the racial group it claims to benefit

(Sankar and Kahn, 2005). Nonetheless, this focus on specific modes of bio-racial targeting in the US has ushered in a new era of self-proclaimed 'racially profiling' doctors and scientists who utilize racial categories as proxies for potential genetic differences in populations' health outcomes and drug responses (Burchard *et al.*, 2003; Satel, 2002). The timing of this trend dovetailed the Human Genome Project (HGP) and the pronouncements that race was not genetically valid as a scientific concept.

On other fronts, as the human genetic draft was prepped for publicity in the early summer of 2000, the NIH simultaneously launched the Pharmacogenetics Research Network (PGRN). With the PGRN, Homosapiens as 'knowing beings' set to rationalize the data (not yet knowledge) of human genomic variation for pharmacy. The PGRN would prove a less massive yet more concentrated effort than the HGP. Its purpose would be to exploit the 0.01% *minority* fraction of human genetic difference that complemented the seemingly overwhelming (but for those interested in variation, somewhat less interesting) 99.9%. In short, as race was minimized by the public and private genome mapping teams (Shreeve, 2004, pp. 219–220, 356), the quantitatively peripheral points of difference in the sequence data (the 0.01%) provided lineaments for its successful restoration. The same year that the HGP heads repudiated race as genetically significant, certain PGRN teams hypothesized its necessity for 'rational medicine'.[2]

Some of the means by which the initial values of genomic science that underscored human commonality have come undone at the level of laboratory research are the focus of this paper.

## Categories and Corollaries

In a 1986 book that she laments should have come much earlier in her career, *How Institutions Think*, British social anthropologist Mary Douglas attempts a bold outline of how the natural world functions as a rhetorical resource for social categorization. She writes that for essences to become real

> There needs to be an analogy by which the formal structure of a crucial set of social relations is found in the physical world, or in the supernatural world, or in eternity, or anywhere, so long as it is not seen as a socially contrived arrangement (Douglas, 1986, p. 48).

In other words, social structures are often maintained by tacit logics, which, in language and practice, draw from *assumed* orders to institute them as their visible referents. Douglas continues: 'When the analogy is applied back and forth from one set of social relations to another, and from these back to nature, its recurring formal structure becomes easily recognized and endowed with a self-validating truth' (1986, p. 48). Thus, when social category becomes rooted in natural corollary it seals itself in the rootstalk of an incontrovertible materiality, spawning various forms of similar, and often tautological, structural reproduction.[3]

In what follows, I argue that the human genome, deeply rooted in references to both the 'supernatural world' and 'eternity' (Shreeve, 2004, p. 124), not only produced a natural referent seen to embody our coded meaning, but, more importantly, it yielded a renewed 'physical world' referent for racial distinction in the United States today. This happened in part because the 0.01% of genetic difference in humans' three billion base

pairs translates to *30 million* genomic sites of change, or in genetics parlance, to 30 million Single Nucleotide Polymorphisms, or SNPs (pronounced 'snips').[4] SNPs refer to single base pair changes in DNA sequence. Where most people might have a stretch of ATGCCTTA in a genetic sequence, a few might have ATGCCTTG, or a single polymorphic change. Such potential polymorphisms were observed to happen at about every 1,000 base pairs, or DNA letters. Analysed alone, or taken together in blocks known as haplotypes, they are now the primary units of comparative genomics, and thus the focus of much pharmacogenetics.

It must be said, however, that by sheer numbers, the overwhelming presence of genomic points of sameness (compared to differences) might have razed previous taxonomies of race. Instead, the possibility of such a *tabula rasa* was precluded by habits and ways of reading socially understood racial difference, coupled with habits and ways of seeking points of variation as a basic practice in the life sciences (from zoology to molecular genetics). Hopeful that they were free of prejudice, with a goal to understand genetic variation in drug response to ameliorate human health, certain researchers of the PGRN instead structured their studies—from the collection of DNA to the organization, storage and analyses of that DNA—by none other than a literal tabula *raza*. Through both logics and practice, DNA frequency differences and race often emerged as the two primary units of analysis in the case study to be presented shortly. As such, each was routinely articulated through the other in tables, spreadsheets, and bioinformatic cells whereby racial delineation inspired a gaze of differentiation that conditioned scientific discovery.

In what follows I will examine the place of race in two related scientific efforts funded by the PGRN. I argue that race, as an organizing principle, emerges as a natural choice and referent for categorizing humans for many working in this field. This happens not because of any explicit commitment to a political agenda or racism, but because the hopeful curiosity that often spurs contemporary research into raced groups is set within institutional formal structures that, 'through a back and forth', to use Douglas' terms, allow human variation its sense on multiple registers.

These registers map onto those described by sociologist Nikolas Rose as 'the framing of explanations at the molecular level', the use of artefacts fabricated there, as well as, and most importantly, 'a reorganization of the gaze of the life sciences, their institutions, procedures, instruments, spaces of operation and forms of capitalization' (2001, p. 13). Drawing upon Rose, I argue that this back and forth between DNA and its seemingly natural organization by societal descriptors of race works to *molecularize* race itself. This happens through practices of marked recruitment, storage, organization and reporting that rely on sorting DNA by US racial population differences for pharmacy.

During six months of ethnographic fieldwork with two groups of collaborating pharmacogenetic scientists, it became clear that DNA molecules, which at the time of the HGP were most often pronounced as racially 'human', are increasingly made to carry the self-reported US racial descriptor of their donors as they leave his or her body and enter the laboratory. The DNA is then analysed with the racial label attached for the duration of its life in the lab and beyond. As the actors I observed organized donor datasets, and thus findings garnered from that organization, by race, racial difference was tagged as a natural conveyor of human distinction within yet other formal structures. These include broader scientific studies, medical practice, and referenced knowledge databases that pick up this work. To this end, race not only became a substance discernable at the molecular level, but it became naturalized there through study designs structured by American racial

norms and the comparison of trait frequency differentials between DNA molecules in humans classed by the social forms those norms yield.

This way of seeing, or 'gaze of the life sciences' in question, has emerged alongside a recent reorganization of institutional procedures mandated by scientific funding agencies. The most important of these for medical genetics has been the National Institutes of Health.[5] In borrowing from Rose's idea of molecularization, the focus here lies less on forms of monetary capitalization [although detailed analyses of what Kaushik Sunder Rajan has termed 'biocapital' take up this aspect elsewhere (2006, pp. 163–167; cf., Kahn, 2006; Tallbear, forthcoming)] than on the reorganization of scientific *social* capital. As one pharmacogenetic scientist to be presented shortly affirms, it was 'politically incorrect' to use racial orderings in genetics a few short years ago. Today, researchers who are part of, or who are affiliated with, the projects described herein, are quite successfully publishing, building databases, and inspiring prolonged debates in major biomedical journals on their assertions that racial differences exist at the level of human genome biology (see Risch *et al.*, 2002; Burchard *et al.*, 2003, 2005).

## The Good Intentions of Health Institutions

> [M]embers of minority groups and their subpopulations must be included in all NIH-supported biomedical and behavioral research projects involving human subjects (...). NIH funding components will not award any grant, cooperative agreement, or contract or support any intramural project that does not comply with this policy (NIH, 2006).

As American sociologist Steve Epstein has shown, the inclusion of race and other points of difference, such as gender, sexuality, and age, was not a natural given with regard to the type of body that has historically been included in human medical and scientific research (2003, 2004; cf. Kahn, 2006, p. 1351). Through various forms of activism, and explicit intervention on the part of key women scientists at the NIH and African American members of congress, women and minorities lobbied for, and succeeded in obtaining, congressional legislation mandating that publicly funded research include members from these populations.

Part of such action was to contest that white men, traditional research subjects, could stand in for all of humanity. Their efforts resulted in the passage of the NIH 1993 Revitalization Act. Yet, as Epstein writes:

> The new emphasis on inclusion and the distrust of extrapolation across social categories were not without opponents (...). Concerns were (...) raised about the problematic business of defining medically meaningful racial and ethnic categories. In its implementation of the NIH Revitalization Act's directive concerning 'minorities', the NIH follow[ed] the path of other government agencies by adopting 'Statistical Directive No. 15' of the Office of Management and Budget (...). [And] Directive 15 specifies the racial and ethnic categories used in the census (Epstein, 2003, p. 183).

One key African American scientist, Otis Brawley, who from 1995 to 2001 was Assistant Director of the National Cancer Institute for Special Populations Research, and who

presently serves on the NIH Advisory Committee on Women's Health, argued in 1995 that the implementation of the 1993 act:

> may eventually do more harm than good for the minority populations that it hopes to benefit. The legislation's emphasis on potential racial differences fosters the racism that its creators want to abrogate by establishing government-sponsored research on the basis of the belief that there are significant biological differences among the races (Brawley, 1995, p. 293).

A small number of mostly African American researchers shared his views (Epstein, 2003, p. 184).

During initial discussions of how the PGRN would collect 'diverse' human subjects and categorize genetic difference, the ethos of the Revitalization Act was present, as was the reality that the PGRN was funded by the NIH, which, as part of the US Federal Government, would require the use of OMB categories.[6]

In an interview with Dr Rochelle Long, the NIH programme director of the PGRN, it became clear, however, that she and others were concerned about using race/ethnicity as a biologically based sorting technology for pharmacogentic discoveries on the part of PGRN scientists. Yet, in discussions amongst herself and specialists in the fields of genetics and pharmacy invited to 'advise' on the issue, they decided that it made sense to use these ascriptions with their imperfections, given that NIH grants and reporting were already structured around their importance.

The decision went forward despite the fact that, according to the Census Bureau, 'the Office of Management and Budget's standards for maintaining, collecting, and presenting data on race (. . .) generally reflect a social definition of race recognized in this country. They do not conform to any biological, anthropological or genetic criteria'.[7] Similarly in the NIH's own words: 'The categories in this [the OMB] classification are social–political constructs and should not be interpreted as being anthropological in nature' (US Department of Health and Human Service, Grant Application, PHS 398 part II, p. 38). Yet, at base, according to Long, the PGRN was powerless to challenge the monopoly that race and ethnicity descriptors already had on government reporting with regard to US population differences, regardless of what investigators were describing (in this case 'genetic criteria'). She understood that the OMB terms were meant to refer to 'social–political constructs'. Nonetheless, she thought that these realities hinted at ancestry, although they were not perfect indicators of heritage as it sheds light on biology and human genetic difference. In my conversations with Long it became clear that the continued use of race was justified by its previous use, as evinced in this interview excerpt.

> RL: It was agreed that when the information was collected for PharmGKB [the PGRN collective database], that the official OMB terms would be used. And this also relates to government-funded work in general. The terms were changing at the time. They were actually separating descriptions of race, if you will, from ethnicity. So now you could be white-Hispanic, black-Hispanic, you could be either Asian or Pacific Islander. While I always agreed that these were not the greatest terms for describing everything about somebody's heritage, they were at least consistent with the ways that [scientists] already had to recruit and report research

study enrolment to the NIH. And, they were valid for the purposes of the Office of Management and Budget. They had been running [discussion] groups and concluded that these are the standards that the government is going to use. So that's the way the data are reflected in the PharmGKB.
DF: Is there any talk at the NIH of revamping those categories? Has the mapping of certain [drug] phenotypes or research of the PGRN shed any light on how those categories might, perhaps, be too rigid?
RL: There is always talk about whether those categories are suitable for research purposes. After all, to call somebody 'Black' doesn't mean that they're 100% Black. On the other hand, there's only one category there. You either are, or, you are not. That's the way that categories are set up. It's not up to the NIH to change those categories. They're determined at a higher level.

## Race

As Geoffrey Bowker and Susan Leigh Star have shown in their exemplary work on classification, *Sorting Things Out*, race operates via an Aristotelian binary system of mutual exclusivities (i.e. white or black, but not both, 'you either are, or you are not') *and* a more abstract system of prototypical thinking. With regard to the latter, racial norms emerge from a range of socially resonant possibilities and practices that are co-produced amidst heterogeneous beliefs and cultural associations (Bowker and Star, 2000, pp. 61–63). Because of its existence on these multiple registers, race as a composition of 'legal' categories is often used without much reflection in many aspects of daily life. Yet, it can simultaneously provoke active scrutiny (or confusion) when cases of ambiguity emerge. This is especially true where social and political circumstances, individual biography, or race itself (when compared to other notions historically or geographically) do not comply with the categories (Aristotelian and prototypical) characteristic of specific societal contexts. Such disconnects can lead to experiences of cognitive dissonance where personal and lived experience are 'torqued' by classification and vice versa (Bowker and Star, 2000, p. 324).

As we will see in several of the interviews with scientists funded through the PGRN, there exists an expected ease and recognition that work to stabilize race as a type of Aristotelian classification where 'enough binary features are adduced to place any member of a population into one and only one class' when it comes to sorting genes (Bowker and Star, 2000, p. 62). Yet when these same researchers are asked to reflect on the use of racial categories for their science, they immediately point out inconsistencies in their own personal, local (US) experiences of race. This is a moment where the certainty of such classification in the lab could begin to lose its force. The fact that it does not attests to the power of socially understood and seemingly objective cultural norms, despite the fact that individuals themselves do not always find themselves in such understandings.

## The Labs

In early March of 2003 I began a six-month fieldwork stay at the University of California, San Francisco's (UCSF) Department of Biopharmaceutical Sciences whose chair, Kathleen Giacomini, has a four-year grant as part of the PGRN to conduct a study on

the Pharmacogenetics of Cell Membrane Transporters, or PMT.[8] Cell membrane transporters are vital to understanding the first and last phases of drug distribution since these are proteins that sit on the outside or inside of cells that determine what chemical substance gets taken up, and in what quantities (influx), and what gets expelled when necessary (efflux). Any drug—toxin or medication to which we are exposed—must interact with transporters before they can be metabolized by enzymes [those entities that have been the focus of pharmacogenetics since the 1950s (see Hedgecoe, 2004, pp. 9–10 for a short history)]. The PMT project is vast with over 52 transporters and their genetic variants being characterized, tested in cell systems, and finally, investigators hope, challenged by the drugs they interact with in animals or humans.

At the same time that I started studying PMT I began fieldwork in another UCSF lab, headed by a physician-researcher of asthma genetics, Dr Esteban Gonzáles Burchard. Burchard has built his career on researching what he terms 'racial admixture' (Burchard *et al.*, 2005, p. 7) in Latino Americans after amassing a database of 2,000 Mexicans and Puerto Ricans from the US, Mexico City, and San Juan. In addition, during the second year of the PGRN he obtained a subsidiary grant under the PMT project to collect and bank a repository of 500 individuals' DNA from 'ethnically diverse populations'. The latter constitutes the phase II dataset for PMT (the phase I dataset will be described shortly). The DNA for phase II was collected locally in the San Francisco Bay Area as the researchers wanted to call back subjects after initial genotyping and analysis in order to conduct clinical studies on them. Burchard employed recruiters from his own lab to recruit PMT subjects.

## A Natural Course of Evolution: A Brief History of the PMT Project

In 1997 a graduate student at the UCSF programme in biopharmaceutical sciences cloned the first human organic cation transporter (OCT 1) from liver tissue (Zhang *et al.*, 1997a).[9] The rat version of protein had been previously cloned, and even though it and the human OCT 1 were found to be 78% identical, the discovery of the human protein was important for understanding its involvement in the absorption, distribution, and elimination of various pharmaceuticals. These span a wide array of therapeutic interventions, including antihistamines, skeletal muscle relaxants, antiarrythmics, and ß-adrenoceptor blocking agents (Zhang *et al.*, 1998, p. 431).

OCT 1 is also thought to interact with N-methyl-4-phenylpyridinium (MPP+ for short), a neurotoxin found in the drug 'ecstasy', which is also believed to play a role in the development of Parkinson's disease. Later that same year, this star student went on to clone a novel variant of the OCT 1 protein found in the rat, which was virtually identical to that previously found, with the exception that it had a 104 base pair deletion (Zhang *et al.*, 1997b). This tissue, obtained from a healthy enough mutant rat living somewhere in the world long enough to have its DNA sampled, got the team actively thinking about *human* differences in such proteins. The same protein in the pig, the mouse and the rabbit were also cloned shortly after the rat.

In the years to follow, Giacomini and collaborators became increasingly interested in questions of comparative SNPs and their pharmaceutical consequences across these species, and within the human. A veritable example of Douglas' description of the back and forth between physical world referents and social structure, the lab's inquiries into the human species were informed by mammalian forms of life in 'nature' to look at

different forms of life in society. The latter would consist of US racialized groups, or African Americans, Caucasians, Asians, and Mexicans. Simultaneously, the question of genetic function with regard to drug uptake, distribution, and elimination was gaining ground at the NIH. The PGRN requests for application (RFA) at the NIH was developed the following year in 1998.

## Race as Organizing Principle

Before submitting her PGRN proposal to the NIH, with the OCT 1 story in the backdrop, Giacomini, with collaborators in the neurological sciences at UCSF, purchased human genomic DNA from the Coriell Institutes for Medical Research Cell Repository. Coriell is a tissue bank run by NIH's National Institute of General Medical Sciences (NIGMS) that is utilized by scientists worldwide to purchase human lymphoblast cells as well as DNA. The UCSF researchers genotyped this DNA for polymorphisms in two neurotransmitters: the serotonin transporter (SERT) and the vesicular monoamine transporter (VMAT2). The team indeed found 'interesting polymorphisms' that affect the function of these two proteins, but they were ultimately frustrated by their findings because the DNA available from Coriell at the time was, in their opinion, lacking crucial information. Not the DNA itself, but rather the way that it was packaged: it was simply and generally 'human'—even though it was procured from 'different' US racial/ethnic groups.[10] The problem, as the researchers put it, was that these groups remained 'ethnically unidentified'. The various groups included in the panel did, however, possess a code associated with their 'ethnicity', whereby each sample from the same group had a similar key. The researchers noticed that certain polymorphisms happened only in some groups, and that frequencies for other variants were not evenly distributed between all. For the scientists, this meant that these prize polymorphic differences could not be fully interpreted because as Giacomini said: 'It was a waste, because we had no control for association studies. We wouldn't know who to compare the polymorphic samples to!'

### *Institutionalizing American Monikers of Race at the Level of Research DNA*

Founded in 1953 by Dr Lewis L. Coriell, the Coriell Institute was initially interested in using cell cultures to study viral diseases. In the 1970s the Institute won two contracts from the NIGMS and the National Institute of Aging (NIA) to establish and maintain what have become the world's largest cell repositories for the study of genetic and aging-related diseases. The contracts for these repositories have been renewed ever since.

In Coriell's early days, up until 2000, although some panels did give national or ethnic information, the most common naming of DNA had to do with the disease or health phenotype that the donor possessed. Researchers interested in questions of 'human variation' were thus given a panel of 'European American', 'African American', 'Mexican American', 'Native American', and 'Asian American' cell cultures wherein each sample, although coded by 'ethnicity', which was itself masked, contained no other information whether medical, phenotypic, or ethnic/racial. This panel was called the DNA Polymorphism Discovery Resource Panel, or PDR.[11] It was this PDR panel that Giacomini and collaborators genotyped and found differences in with regard to SERT and VMAT2 to their frustration. PDR still exists, yet it has dramatically fallen out of favour with scientists interested in pharmacogenetics funded by the PGRN. Today

Coriell offers Human Variation panels that are 'ethnically' identified, in part because various researchers convinced the NIH that knowledge of each DNA sample's race was necessary (Lee, 2003).[12]

In recounting how the Coriell Institute decided to collect DNA that had racial and ethnic specific information, Giacomini herself theorized that it had something to do with 'the changing political climate in the US, where people were less bound by pressures to be politically correct'. She also added that conversations that she and other PGRN researchers had with NIGMS as their new initiative got underway undoubtedly played a part. Granted $3.2 million a year, for four years, Giacomini was one of the first to be given PGRN funds in the first round of awards in the year 2000. In the first year the team genotyped 24 membrane transporters in the (then) new racially labelled 'Human Variation Panels' from Coriell. These consisted of '100 Caucasian Americans' (51 males/49 females), '100 African Americans' (17 males and 83 females) and '30 Asians', of which 10 were Southeast Asians (five males and five females), 10 were Japanese (four males and six females), and 10 were Chinese (gender unattainable since this panel [HD02] is now obsolete). They also obtained '10 Mexican' samples (three males and seven females) and 'seven Pacific Islanders' (four males and three females). These panel sizes were clearly uneven, but, at the time, the researchers were content to have such clearly marked populations in order to genotype the membrane transporters by race/ethnicity.

### *The Tabula raza*

Today, and since PMT's inception, all of their genotyped data are formatted for a shared database wherein each of the now 52 plus transporters from sets I and II, as well as their many variants, are organized by gene and then by race. The hypertext on their intranet database allows the viewer/researcher to click on the transporter's name, which then takes them to a list of options, two of which are the most useful for understanding the centrality of race for the project. The first allows the researcher to click on a graphic of the coding sequence, a reconstruction of all the exons of the gene in question (Figure 1).[13] Each exon is marked by colours that indicate what kind of changes—non-synonymous, synonymous, insertion, or deletion—that genotyping has revealed it to contain.[14] Each one, furthermore, has a link with its position number beneath it. The viewer can then click on the exon number link to get detailed information about what kind of change occurs in that coding sequence.

That link then opens onto a page that consists of a series of columns reproduced here for one transporter gene (Figure 2). (I have left this gene unnamed and unlinked to chromosomal position for confidentiality purposes).

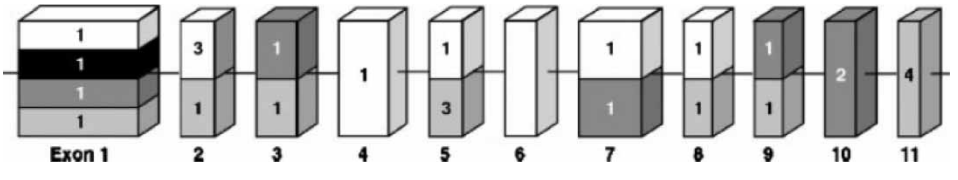


**Figure 1.** A graphic used to represent the exons of a transporter gene, with various colours representing the type of genetic change found there in the sampled population. *Source*: Fujita *et al.* (2006, p. 31). Courtesy of Wiley InterScience, Journal of Pharmaceutical Sciences.

| Exon | SNP # | Exon Position | Nucleotide Change | Amino Acid Position | Amino Acid Change | Total Freq. | AA Freq | CA Freq | AS Freq | ME Freq | PA Freq |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | n=494 | n=200 | n=200 | n=60 | n=20 | n=14 |
| 1 | 1 | (-38) | C ⇒T | - | - | **0.023** n=472 | 0.005 n=190 | **0.053** n=188 | 0.000 n=60 | 0.000 n=20 | 0.000 n=14 |
| 1 | 2 | (-23) | C ⇒T | - | - | **0.119** n=472 | **0.053** n=190 | **0.218** n=188 | **0.050** n=60 | **0.100** n=20 | 0.000 n=14 |
| 1 | 3 | 9 | C ⇒T | 3 | Synony-mous change | **0.042** n=474 | **0.105** n=190 | 0.000 n=190 | 0.000 n=60 | 0.000 n=20 | 0.000 n=14 |
| 1 | 4 | 38 | C ⇒G | 13 | Ser ⇒ Cys | 0.004 n=474 | **0.011** n=190 | 0.000 n=190 | 0.000 n=60 | 0.000 n=20 | 0.000 n=14 |

**Figure 2.** Tabula of genetic difference and race. The first exon of an unnamed transporter gene of interest to PMT as submitted, displayed, and consulted on their intranet database. All transporters and their exons were presented in this manner. Gene changes with a frequency of 0.010 or higher were detailed in bold-faced red. N = the number of chromosomes in a sample set (each person has two chromosomes for each locus).

The purpose of reproducing the structure of this graphic is to illustrate the ordering system of DNA changes as these intersect with race. In a careful system of tabulation, each row highlighted both in the following order:

1) the position of the exon number in the gene sequence;
2) the order of the SNP;
3) the position where it occurred;
4) the nucleotide change;
5) the amino acid position in the protein;
6) the amino acid change brought on by the nucleotide change (and whether or not there was one); and
7) the racial distribution of the change.

Racial variation consumes half of the page. The first row lists the total frequency of the genetic change in question in all groups combined. Then there follow five rows named 'AA' (African American), 'CA' (Caucasian), 'AS' (Asian), 'ME' (Mexican) and 'PA' (Pacific Islander, which was dropped at a later stage) wherein the variant's frequency is listed in each racial group. Gene changes that occurred at a frequency of more than 1% in any one group were highlighted in red.

The second salient, and often consulted, option on the database was a topographical model of the two-dimensional protein structure of the transporter gene in question, constituted by its amino acids (Figure 3). Each amino acid that underwent a change was colour-coded, whereby various colours depicted the type of change that took place

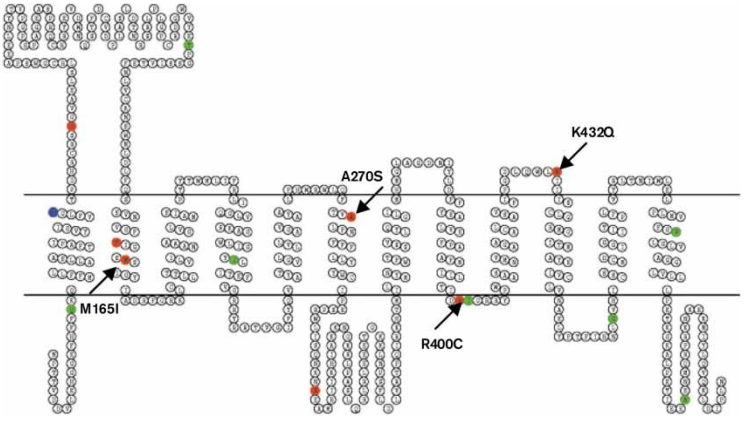


**Figure 3.** Topical representation of a transporter protein. Where there are amino acid changes of difference, an online viewer from the team can click on these and then be taken to a screen with the information featured in Figure 2. *Source*: Leabman *et al.* (2002, p. 399). Courtesy of Lippincott Williams & Wilkins, Pharmacogenetics and Genomics Journal.

(non-synonymous, synonymous, insertion or deletion). If one clicked on the coloured (changed) amino acids, they would then be taken to the link discussed above (Figure 2), which, again, gave the breakdown and frequency of the variant in question for each racial group.

*New Needs*

During the first year and a half of the study, the PMT team was dedicated to genotyping and learning how the set I genes (typed in the Coriell panel) actually functioned in cellular models, i.e. in frog eggs as well as in immortalized cancer cells used for such experiments. They continued, however, to compile a second list of genes of interest to the scientific community. At the same time, PMT scientists began to imagine possibilities of doing human clinical trials regarding the transporter genetic variants that had since been characterized and tested in frog eggs in the lab.

Now the team began to place more importance on the fact that the Coriell Human Variation panels were unevenly matched when one compared the number of samples from each race. Furthermore, for the purposes of clinical studies, the people who donated that DNA were almost as mysterious to the researchers as those in the PDR panel. With the exception of race, which was actually 'self-reported', as the researchers bemoaned on numerous occasions, the samples were anonymous and could never be called back for clinical trials if found to have polymorphisms of interest. In an effort to get beyond what was perceived as a potential research impasse, those at the helm of PMT decided to call on Esteban Gonzáles Burchard to help them build a resource of racialized research subjects. Together they wrote a supplemental grant to the PGRN, as part of PMT, and were funded to build a genetic database specifically for PMT called the Study of Pharmacogenetics in Ethnically Diverse Groups, or SOPHIE.

Once again, learning from the limitations of their previous DNA panels, the researchers now emphasized that the point of SOPHIE was to enlist people from various US

racial groups who lived locally and who would consent not only to give their DNA, but who would also be willing to be called back for future studies. The major difference, however, between the two datasets was that Burchard was in many ways much more diligent than the NIGMS Coriell tissue sample-takers. Coriell's 'quality' of race identification is, as mentioned above, strictly based on 'self-report'. Burchard and his PMT collaborators were determined to collect, in his words, more 'racially pure' DNA. His method was to automatically exclude anyone who reported racial mixing in their genealogies for the past three generations. In theory, subjects were thoroughly screened to rule out such a possibility, while the donor, their parents, and their grandparents all had to identify with the ethnic/racial category in question. For the Chinese and the Mexicans it was stressed that a subject's grandparents had to also be from 'the homeland'.

Those recruited for SOPHIE were selected because the 'socially contrived arrangement' of their perceived 'physical world' purity was naturalized through a tautological assumption that their self-reported pedigree of 'the same race' traduced biogenetic racial sameness. That this was still 'self-report', and subject to an individual's perception of how they should self-classify, which has changed over time, especially for African Americans, seemed less important to Burchard who considered it 'pure enough'.

In an interview with Dr Giacomini, the place of race in PMT as the major organizing principle becomes clear. Other questions, such as age and gender, which surely impress upon how one distributes, transports, and metabolizes drugs, were included in initial discussions of the study design, but ultimately abandoned.

> DF: Was there any discussion when organizing SOPHIE to design the study in any way other than race as a phenotype? . . . Given some people's hesitancy in using race in science, was there any discussion about not using it?
> KG: Well, we were thinking of a few things at the moment of SOPHIE. First, did we want normal, healthy volunteers? Or did we want a disease population? So that was one thing. Secondly, what about age? Did we want to include pediatric patients? Or, even, elderly patients? So we had to make a number of calls. So then, also race. Which race[s]? . . . We were pretty clear that we wanted European Americans, African Americans, Asian Americans—and defining that was a little bit difficult. Also [defining] Mexican Americans. [But] those were the four groups that we knew that we wanted.

## Purity and DNA: Allelic Frequency Differences 'Out of Place'

When SOPHIE was finally compiled, the DNA extracted, and stored, researchers from PMT began to take transporter genes with variants that altered protein function found in Coriell and genotype them in the SOPHIE cohort for eventual *in vivo* trials. One such gene was the premier OCT 1 that had been cloned in the lab in 1997. The student who cloned OCT 1 from liver tissue had long graduated, and in the years since, a new student had become interested in OCT 1 (now known as the major hepatic, or liver, transporter). This graduate student was also an MD and became specifically interested in the transporter's targeted activity in the elimination of the neurotoxin MPP+, as well as the role of OCT 1 in the development of Parkinson's disease.

PMT scientists found human OCT 1 to have 15 protein-altering variants in the Coriell panels. Five of the variants reduced or completely eliminated function. Three of the five

had been screened previously in a 'European' population of 57 people with Parkinson's disease (Kerb *et al.*, 2002; cited in Shu *et al.*, 2003, p. 5903). Taking from this work, and expanding upon it, PMT researchers were able to obtain a Panel of 470 Parkinson's patients (95% of whom were 'Caucasian') from the Parkinson's Institute in Sunnyvale California, in the San Francisco Bay Area.

The researchers in question hoped to compare the Sunnyvale Parkinson donors' DNA against 'a healthy control' in order to isolate a variant associated with the disease. Taking the values that the lab had for these variants in the 'Caucasian' Coriell panel, the student heading the study soon discovered that the Coriell panel (Caucasian and, according to what the team deduced from Coriell's location, 'most likely from New Jersey') differed from the Sunnyvale samples regarding the frequency of one key transporter variant. The variant in question was G465R (normally a Glysine at amino acid position 465, changed now to an aRginine). The G465R change makes for a non-functional OCT 1 protein. The student then decided to genotype the SOPHIE 'Caucasian' panel with hopes that he would find the same variant to be able to conduct a further, informative, study with live humans in SOPHIE who had consented to be called back.

The student and the lab were surprised to find that SOPHIE 'Caucasians' and Coriell 'Caucasians', who were both healthy, differed *more* than the Coriell versus the Sunnyvale Parkinson's disease population for the variant in question. The student then added that this difference was furthermore 'statistically significant'. The student represented the situation in his lab book as shown in Figure 4.

For the G465R variant, the value of a 4% frequency in Coriell Caucasians vs. the value of a 0.4% frequency for SOPHIE Caucasians was highlighted as something warranting attention.

Upon hearing the results, Giacomini and the team wondered what went wrong. Her first reaction was to rule out error and re-genotype. Her next thought was that perhaps the difference was in fact 'a population stratification issue', or that these were different populations, with different mixes and genetic heritage. They theorized that if that was the case, then it must have been the Coriell DNA to have this characteristic. In various conversations the lab researchers faulted the Coriell samples for not being as 'pure' as SOPHIE, since SOPHIE 'Caucasians went back three generations'. Yet neither dataset compilers actually asked about European specific ancestry, such as Welsh, German, or Scot. In this regard, Coriell and SOPHIE were more alike than different. Still, Giacomini wondered about the Coriell Caucasians' purity by positing that the Coriell dataset might actually be 'contaminated'—by which she meant mixed.

| OCT 1 VARIANT | Parkinson's Disease | Coreill Sample | SOPHIE Sample |
|---|---|---|---|
| R61C | 8.6 % | 7.2% | 7.2% |
| G465R | 2.4% | **4.0%** | **0.4%** |
| G401S | 2.1% | 1.1% | 2.8% |

**Figure 4.** Allelic percentages of Coriell and SOPHIE Caucasians for OCT 1 variants

> KG: One of the students in my laboratory wanted to determine whether there was increased frequency of non-functional OCT 1 alleles in Parkinson's disease and then compare it to either Coriell or SOPHIE. In the PD study sample that we obtained, out of something like 470 samples, 450 are Caucasian. So Caucasian is what we needed. And, our non-functional variants in the Coriell sample are in the Caucasian samples, mostly. We decided to look at SOPHIE [too]. And we found a discrepancy between one of the variants. There were three variants that we were screening for. All three were either non-functional or [had] reduced function. One of the variants—no two of them agreed. Coriell had the same frequency as SOPHIE [for those two], but one of the variants did not agree in terms of its frequency in Caucasians between SOPHIE and Coriell. . . . The number one thing that I'm going to rule out is: is there an artefact? The student used a different sequencing method, comparing his to that done at the genomics core facility here at UCSF. He was not comparing his to his . . . The student is going to try to reconcile this [or at least rule out the possibility of it being an artefact]. Then, I did try and find out the ethnic stratification of the Caucasian DNA that we collected here in SOPHIE as well as in Coriell, but that's not easy to get. They're just 'European-Americans'. So whether in fact the ones from Coriell came from Ireland or Finland, and ours are all from Italy and Spain, I don't know. . . . One of the main differences between Coriell and SOPHIE is the way that they were collected [self-report vs. family history of identifying as that group for three generations]. I would say we should take a close look at this because people may not want to be using Coriell if it is contaminated.

This was the first sign of a discrepancy between the two datasets, one collected by NIH, the other collected by Burchard and his recruitment team for PMT. When the student did have the two datasets re-sequenced, with a newer and less error-prone technology, the frequency discrepancy for G465R remained. Other elements of 'Caucasian', as well as 'African American', discrepancies were on the horizon.

### *Black(s) and White(s)*

A few short months later, I witnessed an illustrative example of the extent to which notions of racial purity characterized SOPHIE, while the Coriell samples were increasingly seen as 'questionably' pure. This instance also demonstrates the degree to which 'African Americans' and 'Caucasians' were not only seen as different, but were furthermore taken to be two poles of a supposed physical world arrangement, that may characterize many patterns in nature, but that, by most accounts, runs counter to accepted ideas of human genetic diversity: each was perceived as the other's '*opposite* race'.

At issue was a second organic cation transporter, OCT 2, which is primarily found in the kidney, rather than the liver. In this case, another of Giacomini's previous graduate students, who went on to become a key postdoctoral researcher for PMT, looked at variants in OCT 2 in both Coriell and SOPHIE. She not only looked at single changes, but also constructed these into haplotypes (multiple genetic loci inherited together). She first-authored a paper a year earlier in 2002, based on 247 samples from Coriell (Caucasians, African Americans, Asian Americans, Mexican Americans and Pacific Islanders), which detailed 'the ethnic-specific distribution of these haplotypes'

(Leabman *et al.*, 2002, p. 398). Based on the genotypes that she found in the Coriell dataset, she identified two common haplotypes that were seen in close to 60% of the Coriell samples. In the published paper these were called haplotype *1 and haplotype *2A (see Figure 5).

Once the SOPHIE database was complete, the postdoc proposed to do a study with 'real SOPHIE people' who could be called back wherein she would examine the possibility that this transporter could prove a valuable drug target for the anti-diabetic agent *metformin*. Several PMT researchers combed the SOPHIE genotyping data to identify possible study candidates who possessed OCT 2 variants. While this initial phase of recruitment was happening, the postdoc began looking at the SOPHIE DNA samples to compare the OCT 2 frequencies with those of Coriell. As soon as the data came back, the postdoc noticed that the haplotype frequencies had not remained static, as she had assumed they would. This change in frequency distribution was perceived as alarming with regard to several haplotypes, including the most common. Upon seeing the results, she confided:

> Something's weird in OCT 2. Some of the haplotype frequencies have completely changed to the opposite race. Ones that were African American are now Caucasian, and ones that were mostly Caucasian are now African American. We've already seen discrepancies between Coriell and SOPHIE for OCT 1. Maybe this is the same thing. Maybe some of those Coriell people were not really African Americans, or, Caucasians. Maybe they were mixed, because Coriell was just based on self-report.

When I inquired further, one of the haplotypes involved was one shared amongst all groups, haplotype *1. The researcher had nonetheless noted that the majority of haplotypes retained their earlier patterns. In going back to the published paper, where haplotype *1 was concerned, I saw that the frequencies differed (slightly) between African Americans and Caucasians, yet it was in no way a majority African American or Caucasian pattern.

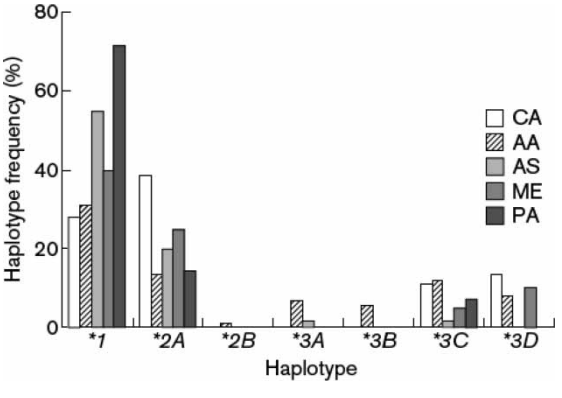


**Figure 5.** Frequencies for several OCT 2 haplotypes and their racial/ethnic coding as presented by Leabman *et al.* (2002, p. 400). Haplotype *1, wherein 'Caucasians' and 'African Americans' both have frequencies between 25 and 30%, is the haplotype under discussion here. *Source*: Leabman *et al.* (2002). Courtesy of Lippincott Williams & Wilkins, Pharmacogenetics and Genomics Journal.

Both Asians and Pacific Islanders possessed the haplotype nearly twice as often as either blacks or whites. More to the point, however, African Americans and Caucasians shared this haplotype *at nearly the same frequency.* From the bar graph, which denotes frequency in units of 20, one can deduce that these two groups hovered around 30% for haplotype *1.

That the Caucasians in SOPHIE surpassed the African American frequency for this haplotype, when the 'referent' frequencies in Coriell were so close, was what led this researcher to say that the haplotype frequencies 'switched to the opposite race'. Such a statement reaffirms various social scientists' observations that 'Black' has long been at antipodes with 'White' (and vice versa) at various levels of American racial understanding (Allen, 1997; Guterl, 2001, pp. 3–4; Tapper, 1999, Chapter 2; Wailoo, 1996, p. 308). More to the point, however, in each of these instances the team perceived frequency changes to somehow be 'matter out of place', which implied both an assumed order, or pattern, and 'a contravention of that order' (Douglas, 1966/2002, p. 44). As Mary Douglas reminds us, where there is 'contamination', there is a system of purity. Yet, these groups of humans, like many others around the globe, not only have long histories of mixing, but have also experienced varying degrees of cultural unifying forces once in the 'New World'.

In the first case cited above, the researchers make clear their assumptions about Caucasian stability and consistency. Yet, the coherence of the term 'Caucasian', a relatively new notion in the United States, emerged in the 1920s when the idea of 'so-called minor divisions of humanity' from Europe were 'willingly erased' and an encompassing scientific idea of whiteness (enshrined in the term 'Caucasian') took hold (Jacobson, 1998, p. 96). It was with the *Johnson–Reed Act* of 1924, grossly restricting European immigrants deemed unfit, that anxieties over inferior white races entering into the US began to be muted, and thus, when the economic and ideological stakes that kept such racial distinctions between Whites alive (such as Irish, Slav, or, Greek) also began to dissipate. The catchall term 'Caucasian', a political response to creating a shared identity amongst those desired by the US government (and allowed in) at a certain historical moment, today may divert scientists from recognizing important mutations that affect members of this culturally defined group at the level of their genome biology.[15] Researchers in the Giacomini lab admitted, when asked, that 'Whites', like all humans, surely possessed genetic variation when compared amongst themselves.

In the second case, the assumptions characterizing the first case are operative, since the Coriell Caucasians and the SOPHIE Caucasians again proved 'inconsistent' with each other. Additionally, the African American story of discrepancy warrants its own analysis. The 'coherence' of African American-ness at a biological level should indeed inspire pause (given that Africa as a whole contains the most human genetic diversity on the planet). African American cultural identity, based on self-report or going back three generations, is largely grounded in a shared experience hailing from a history of forced migration and chattel slavery in the US. Yet, the genetic diversity of the people about whom that history refers is often grossly simplified, if not overlooked, by many today.

Historians and anthropologists of the transatlantic slave trade emphasize the distinct elements of African cultures, languages and ethnicities that were not only extant in the New World, but that, moreover, helped to facilitate the very success of the slave trade in Africa, as African governments, merchants and others were involved in the capture and sale to Europeans of other Africans they considered 'aliens' or 'outsiders'. This happened for both economic and political reasons (see Eltis, 2000, p. 226, Chapter 9;

Greene, 2002, pp. 1018–1019; Lovejoy, 2000, Chapter 8; Thornton, 1998, Chapter 4; cf. Herskovits, 1936). The first African Americans, as an 'ethnically heterogeneous aggregate of individuals' (Mintz and Price, 1992, p. 14), created unifying social structures and cultural practices once in the New World. All the while, slaving patterns and economic demands often favoured ethnic and cultural retentions of certain groups over others in many places (Gomez, 1998; Eltis, 2000, pp. 250–257). Furthermore, the very notion of African identity, rooted in reference to the African subcontinent, is a relatively recent invention (Eltis, 2000, p. 230; Mudimbe, 1988, p. 1), as is the idea of an encompassing black identity with roots in a place called Africa for those in the United States (Gomez, 1998, Chapter 1). This brief historical context may shed some light on why an assumption of African American homogeneity might prove problematic in analysing genetic datasets that do not produce identical results.[16]

When asked to reflect upon a clear definition of the term race, the researcher responsible for the OCT 2 study no longer reasons about racial distinction in such stark terms when it might be applied to her own life. In fact, she had two relationships to race: one for science and one for society. It might be that her own thinking on race itself mirrors the dyadic arrangement of two poles of difference that she unconsciously referenced in her astonishment about the haplotype frequencies switching to the opposite race. In an interview I ask:

> DF: How would you define race?
> Postdoc: That's a good question. I mean I—from my limited knowledge—and you know I'm still learning so much about population genetics and how variation differs between groups—but I do feel as though there are ethnic specific SNPs. Because it seems as though people—when people do these large scale genomic screens, they see these frequencies in the groups. One SNP will only pop up in one group, and another SNP will pop up only in another group. So I do think that there's some genetic basis for ethnicity or race. In terms of defining a given race based on SNPs or genetic variation, I don't think we know enough yet to do that. But from what I've seen, I do think that there are genetic differences, or more global genetic differences between—and maybe it's just sub populations. I don't know—I don't really *know* what defines a race or an ethnicity, and I get confused between 'what is a race?' and 'what is an ethnicity?' So ...
> DF: So you're going on what you've seen. You've seen SNPs appear in what have been defined as different groups?
> PD: Yeah. That's what people report. ... I don't know because I don't know how they are defining their races and ethnicities, but I do think that it's not just chance. ...
> DF: Before you got into SNP mapping, how would you have defined race?
> PD: I think before I got into all of this, I would have thought, Oh 'where are your parents from?' And you know, my dad came from India. I actually grew up in a very mixed background. My dad grew up in India until he was 25, and my mom is part Czechoslovakian, so I'm of very mixed descent. So, if someone had two parents and they were second generation, and the parents came from China, I would say oh, they are Chinese. I mean that was my simple kind of definition of somebody's race or descent. But I think of it much differently now ...
> DF: As a mixed person of Czechoslovakian, Indian ...
> PD: And everything else! [laughs].

> DF: Do you feel that the racial categories that we use, for example in the United States, Black, White, Asian, and Hispanic, etcetera are too simplistic? Would you expand these if you could?
> PD: It always—as a child it always bothered me. I hated it on tests—you know, when you took your SAT test, or when you apply for college, they ask you to check off this box: are you Asian, Caucasian, African American, or Other? You know, and I didn't feel like putting Other. You know, because Other just seems like, 'gosh, I don't belong to anything'. It kind of makes you feel—I just thought it was weird. I never liked the fact—no. I didn't like the fact that you had to define yourself as one race. (. . .) So I guess it depends on the situation. I guess in social type things, I don't even want to have to think about it, if that makes any sense? . . . So, I have two extremes: when I'm doing my genetic type of research, I want things very well defined, and in a social setting I don't even want to think about it. I don't know if that makes any sense [laughs]?

In Douglas' conception of how we rely on nature to make sense of the social, we seem to have come a long way from explicitly basing social facts on near figures of nature embodied in the complex practice of adopting totems detailed by anthropologists such as Claude Lévi-Strauss (1962/1966). Yet, as with the peoples generalized in the *Savage Mind*, today in the lab 'the mind passes from empirical diversity to conceptual simplicity and then from conceptual simplicity to meaningful synthesis' (Lévi-Strauss, 1962/1966, p. 131). Contrary to the traditional Science and Technology Studies (STS) idea that science and society are 'co-produced', while key practitioners in this field show how that may be (Fujimura, 1996, p. 18; Jasanoff, 1996, p. 397; Reardon, 2005, p. 6), the scientists I encountered on the PMT project often relied on the cultural moieties of 'in the lab' versus 'in the world'. In so doing, they sometimes bracketed the reality of 'the social' to meaningfully synthesize knowledge for their 'science'. Anthropologists and sociologists have now evolved from the structuralism of Lévi-Strauss' day toward practice theory where individual agents have some say in shaping their worlds, ideas, symbols and signifiers (Bourdieu, 1977/1993, p. 97; Ferme, 2001, p. 20; Ortner, 2006). However, with regard to certain structuring structures such aspects of one's agency partially dissolve into the 'back and forth' described by Douglas. In this case the researcher confided that she strived to be anything but 'Other'.

The researcher ultimately became 'Caucasian'. She still admits, however, that such a category imposition on her whole person was 'weird'. Her options of race denomination—on the SAT, on the US census, and now in her lab—were vexingly unaccommodating. Lévi-Strauss may have been too hopeful for human universals. Such an 'experience-near' of weirdness seemingly had little bearing on how this researcher viewed and identified the human DNA that passed through her hands, her pipette, her lab notebook, and her computer graphs on a daily basis. That human product was by contrast unequivocally 'co-produced'. It was conceptualized as 'African American' versus 'Caucasian', in reference to the racialized bodies from which it was taken locally, and socially. In turn, the frequency gradients between these marked groups assumed a molecular corroboration that their social ascriptions traduced genetic difference.

Regardless of their adherence to the *tabula raza* in the lab, other researchers also quickly acknowledged that racial purity was hard to come by in the social world. Like the postdoc working on OCT 2, the OCT 1 researcher who found the Coriell and the

Sunnyvale Parkinson's Caucasians to be more alike than those of SOPHIE and Coriell, also assumed an ethnic classification distinct from his parents. His parents (and grandparents) from rural China were counted as 'minorities' in contradistinction to the Han majority group. He, on the other hand, was Han. He explained that this understanding of himself as Han had to do with his education and urban life style. He admitted that it was 'very confusing'. Before coming to America, he worked on pharmacogenomic variants among certain of China's 55 minority groups. He told me that the genomic differences he saw in the peasant Chinese he studied versus the Han had to do with 'diet and environment, more than race'. He laughed slightly, as if entertained, when I asked if he thought that race in the US could be just as fluid. 'Maybe', he offered, with no follow-up.

### Self-identified Latino Scientists and their Recruitment Dilemmas

In the Burchard lab, where the SOPHIE recruiters mainly resided, it was clear that these researchers also mostly held notions of 'Caucasians' and 'African Americans' static and immutable for research purposes. That said, however, specific tacit assumptions in the recruitment process were not lost on them. This is evident in one recruiter's musings on the category of Caucasian in a conversation about how it took nearly two years to recruit the African American, Chinese American, and Mexican American samples for SOPHIE, but only six months to recruit the Caucasians.

> R1: . . . And [recruitment for] the Caucasians, guess what? It took us six months to finish.
> DF: Six months?
> R1: They were the easiest group to recruit, and doesn't that all make sense? They are the ones studied the most often, the ones who are most involved, and most aware perhaps, you know? So that tells me a lot. It tells me a lot about conducting research and how difficult it is to get the other populations.

She then continues.

> R1: So, um, the other thing with Caucasians, though, is that it's [sic] anybody, basically. . . . There was really no set limit in terms of their background.
> DF: Did you ask about the four grandparents and all of that?
> R1: Yes, but there was no set protocol for them. They could've been Italian, German, Jewish (. . .) from anywhere.
> DF: And were they?
> R1: Yeah. I think parents and grandparents . . . a lot of these folks, I think their parents were born in the US, their grandparents may have been born in the US, maybe one grandparent was born somewhere else, we did get a variety of that (. . .) but there was really no tough eligibility criteria for the Caucasians, whereas for the other groups they [grandparents] had to be *Chinese*, from *China*, and *Mexican*, from *Mexico.*
> DF: Did you ever discuss that [with the team]?
> R1: . . . I don't know if it was a big issue for [the PIs] because ultimately what we want to see is more [on] the ethnic groups and how drug metabolism works for them, maybe in comparison to the Caucasians, but I think they've been studied so

> much that it wasn't a big deal. It wasn't a big issue for anybody. It was just like, 'get them and get them in'.

Later in the interview, when I posed a question about how she defined race in her work and other aspects of life, it became clear that, when probed, the SOPHIE recruiters had insightful observations about how race, especially what they perceived to be their own 'Latino race', was anything but immutable.

> DF: How do you define race?
> R1: Wow [pause]. That's a good one. (. . .) I think that there're many definitions out there. Race—people see it more as geographical, being from a certain geographic location. (. . .) And within our own community, like if you go to one area of our country, you see diversity within our own race, within our own geographical location of Mexico. And I think that's phenomenal. I don't know if we can really answer the question of what is race because we're just so diverse (. . .) within our own *race* of Latinos, if you want to call it like that, there's such diversity, you can just see it in skin colour, and eye colour and everyone's coming from different regions (. . .) you go to Vera Cruz, there are *black* people in Vera Cruz.

In an interview with a second recruiter, his discussion of the recruitment process also provides insight into the social forces that, in part, became the unquestioned contours of the *tabula raza* once SOPHIE was compiled. He refers to the method of amassing samples for SOPHIE as one of 'recruiting in crops'. This was the first instance that DNA, while still in donor's bodies, was targeted through a racialized gaze before it was even extracted. In a discussion about recruitment resources that were lacking he offers:

> R2: It would be ideal to have a (. . .) staff that was available year around of all the ethnic groups so that we're not recruiting in crops. Because what happened in the SOPHIE project is that we were essentially recruiting in specific ethnic groups just because at the time we had people of that ethnicity on staff [temporarily] to recruit [people of their same race/ethnicity] and so we had to take advantage of that, because those [students] wouldn't be around. Some of them were just here for the summer. Others were here on a part-time basis.

The notion of 'seasonality' here is important. Such cultivation was especially true for the African American DNA, since, as I was told, the lab had to rely on African American medical students who could usually only work summers. Black young researchers pursuing degrees and careers in the biological sciences were much harder to come by than were those of other groups.[17]

Later, when specifically asked how he views race for this work, but also in everyday life, like the first recruiter, the second recruiter's response includes processes other than DNA and biological purity:

> R2: I think race is essentially very culturally specific. . . . I don't think that there's a great definition for race other than culturally specific, culturally specific, oh, what's the word I'm looking for?—ideas and thoughts that a person can identify with when in their racial group. So, for me, specifically in a Latino racial group, as a South

> American Argentine–Columbian, I would say that my background is very different than a Mexican American who is also considered Latino. … And that differs dramatically in … food, music … everything across the board and I think that's not being addressed [in the US]. We just lump everyone [Latino] together here.

Despite the difficulties they saw in delimiting race by biology, and vice versa, as both recruiters detailed other processes, they each planted US raced groups in a seedbed of 'natural' rhetorical references. The first recruiter links the complexity of Mexican racial difference to geography, to 'the land' where diverse racial types and features could be found in specific locations. This is the only case where the physical world reference employed by the researchers I observed in fact worked to potentially uproot the order of race naturalness on US ground. For her, the blacks of *Vera Cruz* were still 'Mexican', of the 'Latino Race', as she ventured to call it.

The second recruiter, like the postdoc, raised in a 'mixed' background, saw his own identity as more complex than allowed by US categories. Yet his conception of the different types of humanity to be ensiled for SOPHIE was quite telling. In his description of the recruitment process, he refers to the targeted harvesting of DNA—where lab workers would be responsible for collecting their own race's DNA—as recruiting in crops. Although he feels limited by the seasonality of the process (and hopes to find a way to be more productive year round), the idea of targeted tilling remains.

Despite their porous definitions, references to cultural practices, geography and intra Latino diversity (even as both referred to this as a 'racial' group in part), both recruiters remained deeply committed to the design strategy of SOPHIE. This is evident in the following assertion by the second recruiter.

> R2: I know there is debate … and there are pros and cons on the issue, but I'm just of the belief that [race] is very important. … I think [PMT] is a perfect opportunity to look at very specific racial groups and see how they differ with the same medication. It's something that I don't think has been done, and needs to be done. And, there will be differences. I'm sure of it.

Indeed, with regard to the OCT 2 *1 haplotype 'switching to the opposite race', this same recruiter commented: 'It really might be because of the different recruitment processes. Coriell isn't as reliable as SOPHIE because we went back three generations, for both African Americans and Caucasians. Both could actually be mixed in Coriell'.

## Conclusion

In this paper I have detailed how a team of scientists invested in understanding pharmacogenetic differences in US racial groups maintains ideas of racial homogeneity and inter-group distinction with regard to genetic variation. Through practices of recruiting, organizing, storing, and comparing human DNA by US race categories mandated by the Office of Management and Budget and the National Institutes of Health, US racial distinction is conserved in the laboratory, with little open discussion about the possible bias it brings to thinking about genetic results and their significance.[18] Through a back and forth, in making analogies to nature to root social processes in the physical world, the societal tendency to comprehend Americans in OMB terms provides an initial

framework for the *molecularization* of race. Study design and logics based on animal species' genetic homology, as well as individual scientists' beliefs in the naturalness of colour lines deemed important in US society, further powers the logic of the *tabula raza* encountered herein.

As Douglas reminds us, the scientist who classifies 'according to known and visible institutions, saves himself the trouble of justifying the classification' (1986, p. 94). I have attempted to open up, to in fact *trouble*, the space compressed in such instances through ethnographic inquiry and candid questioning of how scientists involved in this project understood their work, utilized race, and to what end. Such queries, be they ethnographic or otherwise, may help us get beyond the polemics of whether or not race is genetic—towards better understanding how it *becomes* genetic, or molecularized.

It is clear that many are quite confused about scientific uses of race, including certain scientists themselves. Rather than rely solely on personal definitions offered up by practitioners of pharmacogenetics, I have sought to understand the propinquity of social categories to natural corollaries. 'Nature', the use of animal species' pharmacogenetic differences to structure certain scientists' thinking about human races, as well as references to perceived natural symmetry and 'opposites' with regard to Blacks and Whites, looms large in the scientific (social) space of the lab. This fact, perhaps not unlike the tendency to acknowledge that the social experience of race was more complex than their lab practices allowed, was second nature for many.

In this ethnographic account about one of the first funded forays into personalized medicine, each researcher expressed degrees of wonder or frustration that the anaemic OMB categories could not accommodate them as 'Han, as opposed to "minority", Chinese', 'Indian–Czechoslovakian', 'Mexican as a race' and 'Argentine–Columbian–Latino'. Yet, for the time being, they suspended their social experiences and the inscrutability of the categories to comply with larger societal scientific practices of using them to compare US groups. This is perhaps where the wider social environment of superiors, funding agencies, and US institutional definitions exert an obvious power. Even when these young researchers' data were 'inconsistent', potentially providing a space to rethink the assumed group homogeneity inherent in their categorizations, they most often reacted against that data. Social scientists of genomics have demonstrated the ways in which techniques that focus on genetic loci, such as SNPs, simultaneously render race protean (with certain manipulations) and stable (with others) (Duster, 2001, pp. 218–221; Haraway, 1994). Yet, somehow *despite* their wider social (both local and global) environments, these scientists took the protean to be errors, or contamination, and struggled to fix the race boundaries that were jolted by the appearance of 'inconsistent' DNA frequencies.

In closing, I find it useful to offer another insight from Douglas. She writes:

> However much they try to insulate their work, scientists are never completely free of their own contemporary society's pressures, which are necessary for the creative effort. Scientific theory is the result of the struggle between classifications being developed for professional purposes by a group of scientists and the classifications [operating] in a wider social environment. Both are emotionally charged (Douglas, 1986, p. 56).

This framing departs from the seemingly closed relationship described earlier where rhetorical resources of nature in many respects totally capture the social structure.

Clearly, elements of the social draw their validity from ideas in nature, but scientific production can still remain quite distant from various social experiences, and vice versa. The potential creative effort, in this case, lies in the struggle that researchers in the US face when using race as described by OMB categories, and the possibility that they might begin to trust their knowledge gleaned from 'social type things', and to in fact 'think about it'.

Individual scientists' experiences could provide an aperture to rethink race in the context of racialized medicine. Yet such a prospect can only take place in a back-and-forth partially mobilized by these scientists, if they find an acceptable venue to talk through feelings of being limited by the categories and by the public governmental institutions that fund them. There is little dissent at the head of the PGRN on the idea that our racial descriptors are 'social political constructs' that do not capture human genetic variation most appropriately and that the use of race in genetic studies requires more hard reflection.

## Coda

*'The audacity of hope' for better concepts beyond present categories*

Indeed, as this article goes to press, there is some evidence that funding by the US government into links between race, genomics and medicine may soon prompt pointed discussions about American scientists' deployable racial terminology. In early August of 2006 US Senator Barack Obama, of mixed background (mother from Kansas, father from Kenya), a heritage not so unlike that of many PMT scientists, proposed the *Genomics and Personalized Medicine Act of 2006.* The bill calls for $150 million a year for research into 'Race, Genomics, and Health'. The junior senator, who regularly extends overtures to the US public to look beyond divisive lines of colour, capital, and most recently political party—in essence categories—has drafted a comprehensive proposal that highlights one practical dimension of his plan for racial democracy in the American body politic.

In a piece of legislation that offers industry incentives such as shared access to a proposed public biobank as well as a tax break to individuals or businesses that develop pharmacogenomic tests, Obama's bill most notably calls for a conscious examination of the current terms of racial reporting and genetic research into racialized groups. Specifically, it states that:

> not later than 1 year after the date of enactment of this Act, the [secretarial committee] shall prepare, with public input, and publish trans-agency guidance regarding: (A) An appropriate definition for race and for ethnicity for use in genomic research and programs operated or supported by the federal government, and (B) Guiding ethics, principles, and protocols for the inclusion and designation of racial and ethnic populations in genomics research and programs operated and supported by the Federal Government (US Senate, 2006, pp. 22–23).

Furthermore, the bill's 'general points' on this topic include increasing knowledge about the 'interaction between genetics and the environment, and the influence of such interaction on the causality and treatment of diseases common in racial and ethic populations'. This injunction is immediately followed by a call to better understand the 'way in which

molecular genetic screening, diagnostics and treatments may be used to improve the health and health care of racial and ethnic minority populations' (p. 22). It also calls for legislation on drug 'labeling specific to racial and ethnic groups' (p. 36). Clearly certain goals of this proposed bill entrain some of the concerns I outlined in the opening passage with regard to the possible reification of race. For Obama, the ethical obligation to pursue health disparities now mixes with new social capital and the political will to earmark monetary capital to speak about racial differences in health outcomes. Yet these good intentions, in practice, could lead researchers on quests for racial purity similar to those of the scientists presented here—a quest that often resulted in idiosyncratic US human 'universals', or the antimonies of 'Caucasian' and 'African American' DNA.

As I have shown in the section of this paper that treats US government institutional uses of race, it may not prove so audacious to pursue such matters if the social categories, already dissolved into assumed biogenetic bases of race for many, continue to perform such double duty. Recall that the head of the NIH Pharmacogenetics Research Network, Dr Rochelle Long, admitted that race as we know it is not the 'greatest term for describing (. . .) heritage'. Like the examples encountered in PMT, many pharmacogenetic differences in drug response only affect a small percentage of any given population no matter how it is divided. As we saw with the distribution of reduced function variants for both OCT 1 and OCT 2, never are whole populations affected. Most often only small proportions of them are (Tate and Goldstein, 2004, pp. S36–S37), and rarely does it make genetic sense to racialize the results.

Minorities, like all Americans, may benefit from more knowledge on the sequence variants they possess. Yet as humans mostly share these variants they may not profit medically or socially from science that aligns their identities with increased frequency differences of DNA polymorphisms, such as 'African American haplotypes', constructed in contradistinction to those of 'Caucasians'. On the other hand, when important mutations are found in one population, and in one population only, researchers (and the population in question) stand much to gain by furthering our understanding of genetic precision and its import in context. That context is both within and without the body, and will include information on why and how a mutation event makes sense physiologically, evolutionarily, environmentally and historically, rather than strictly 'racially'. Obama, like Dr Long, suspects that, at present, *appropriate* definitions of human difference for genetic research are still wanting. Obama, unlike Long, however may have the power, as she says, to help 'determine [them] from a higher level'.

Time will tell if the *Genomics and Personalized Medicine Act of 2006* will indeed move us to new ground in both the social and physical worlds, i.e. toward better health. With a reorganization of the institutions concerned with racial categorization—assuming that together policy makers and their publics define a scientifically robust framework for researching genetic susceptibility—the molecularization of difference may indeed move us beyond the *tabula raza* to a less emotionally charged slate. Only then will the *meaningfulness* of genomic sites of difference outvie the mere fact that human genetic variation quite naturally exists.

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## Notes

[1]For more on where this debate is taking place, see the American Social Science Research Council's website entitled 'Is Race Real?' available at: http://raceandgenomics.ssrc.org/ (accessed 14 October 2006).

[2]The term 'rational medicine' is used interchangeably with pharmacogenetics. For more on this idea and the 'dictate' that race be used to guide rational medicine, see Evans and Relling (1999, p. 488).

[3]In Douglas' framing, the emphasis is put on the work that 'nature' does for 'social' categories, but surely this process is not so clear cut, as both realms resource the physical world as well as social and cultural forms in various ways.

[4]See Duster (2001) for a detailed analysis of how much public research began to switch its focus from genetic similarity to one of difference at the turn of the last century.

[5]In addition to the NIH, the Food and Drug Administration has also begun requesting that researchers include data on 'minority populations'. This is most evident in their issuance of the Food and Drug Modernization Act of 1997, which directed that the Secretary of Health and Human Services, in consultation with the Director of the National Institutes of Health, and with representatives of the drug manufacturing industry, review and develop guidance on the inclusion of minorities and specific 'populations' in clinical trials (see Kahn, forthcoming, 2007).

[6]For a description of the categories see: http://www.whitehouse.gov/omb/fedreg/directive_15.html (accessed 19 June 2006).

[7]See http://www.census.gov/Press-Release/www/2001/raceqandas.html (accessed 21 June 2006).

[8]Due to either their high profiles, or to the extreme specificity of their research, lead scientists' real names are used, while consent and permission to do so were obtained through an IRB approved consent form and attribution statement, which was signed by participants.

[9]Organic cations, by definition, are organic molecules that possess a positive charge with regard to physiological pH. Organic anions, also important in the world of transporter genetics, are negatively charged molecules.

[10]The terms 'race' and 'ethnicity' are used in tandem in most American government scientific purposes. I use the terms interchangeably as most of the scientists encountered for this study use both, often switching in a given conversation, to refer to the same thing.

[11]For more detail on this first panel, see the Coriell Institute for Medical Research DNA Discovery Resource Panel, available at: http://locus.umdnj.edu/nigms/products/pdr.html (accessed 22 June 2006).

[12]For the Human Variation Collection of NIGMS Repositories see http://locus.umdnj.edu/nigms/cells/humdiv.html. The most recent Human Variation panels available to researchers through Coriell are the Han Chinese and Mexican Americans of Los Angeles panels, collections which were funded by PGRN, and both of which emphasize the single ancestry of samples going back for three generations. See

http://locus.umdnj.edu/nigms/nigms_cgi/panel.cgi?id=2&query=HDPHAN and http://locus.umdnj.edu/nigms/nigms_cgi/panel.cgi?id=2&query=HDPHISP (accessed 22 June 2006).

[13]An exon is a segment of a gene that is present in the final functional transcript (messenger RNA) from that gene. It is any non-intron section of the coding sequence of a gene; together the exons constitute the mRNA and are translated into protein.

[14]Not all base pair changes bring on amino acid changes that lead to protein alteration. Amino acids are coded for by DNA based on coding sequences that always have more than one version. For instance, six different codes, each consisting of three base pairs, can code for leucine, two of which are UUA, and UUG. Following from this, if a SNP was found in a spot on the genome that normally coded for leucine, a number of things could happen. If in translation the normal UUG sequence was instead UUA, then this change would be called 'synonymous', since the amino acid leucine would be left unchanged. If, however, the G in this spot changed to a C then the amino acid produced would be phenylalanine, which is altogether different, or 'non-synonymous'. Insertions and deletions are insertions or deletions of coding material, which alter the number of amino acids made, and thus alter the protein.

[15]For more on the social production of whiteness in the US, see Allen (1997, Chapter 13), Guterl (2001, Chapter 2), Hale (1998), Roediger (2005) and Wray (2006).

[16]This tendency to reduce within group diversity to overarching labels is not limited to biologists. Much 'ethnicity theory' in sociology also overlooks the complexities in the *cultural* heterogeneity of both black and white Americans (see Omi and Winant for a discussion, 1994, pp. 20–23).

[17]For an informative overview of some of the reasons that the US academy has so few minorities in math and science fields see Campbell *et al.* (2000).

[18]For a technologically different, but ideologically similar, event in racial molecularization see Hannah Landecker's historical account of racialization and the HeLa cell line (2000).

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