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Homogeneity and heterogeneity as situational properties: Producing – and moving beyond? – race in post-genomic science

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Abstract

In this article, we explore current thinking and practices around the logics of difference in gene–environment interaction research in the post-genomic era. We find that scientists conducting gene–environment interaction research continue to invoke well-worn notions of racial difference and diversity, but use them strategically to try to examine other kinds of etiologically significant differences among populations. Scientists do this by seeing populations not as inherently homogeneous or heterogeneous, but rather by actively working to produce homogeneity along some dimensions and heterogeneity along others in their study populations. Thus we argue that homogeneity and heterogeneity are situational properties – properties that scientists seek to achieve in their study populations, the available data, and other aspects of the research situation they are confronting, and then leverage to advance post-genomic science. Pointing to the

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situatedness of homogeneity and heterogeneity in gene–environment interaction research underscores the work that these properties do and the contingencies that shape decisions about research procedures. Through a focus on the situational production of homogeneity and heterogeneity more broadly, we find that gene–environment interaction research attempts to shift the logic of difference from solely racial terms as explanatory ends unto themselves, to racial and other dimensions of difference that may be important clues to the causes of complex diseases.

Keywords

ancestry; environment; ethnicity; gene–environment interaction research; genomics; heterogeneity; homogeneity; race

Introduction: post-genomic differences and complexities

In the decade since the completion of the Human Genome Project and its message of a universal humanity found in the 99.9% of genes that we share with one another, many observers have noted the rapidity with which post-genomic research has reverted to a focus on the logic and science of difference (e.g. Abu El-Haj, 2007; Bliss, 2012; Fullwiley, 2011; Koenig et al., 2008a; Lee, 2006; Montoya, 2011; Whitmarsh, 2008). They also have noted that a post-genomic logic of difference is distinctive in significant ways from what came before. Certainly the economic stakes are vastly higher, and the purported precision with which genomics can differentiate among individuals and groups sharing ancestry has the potential to re-naturalize racialized ways of understanding human biology (Koenig et al., 2008b).

At the same time, there has also been some impatience among health researchers themselves with what they perceive to be a myopic focus on racial difference in the science of disease etiology. Such critiques range in scope and content. There are those critiques that fairly evenhandedly argue that both genetic as well as other (social, cultural, environmental) differences by race should be examined for their contributions to disease etiology (e.g. Burchard et al., 2003; Jorde and Wooding, 2004; Rebbeck et al., 2006). Others acknowledge that genetic differences *may* exist but may not correspond to race, while other (social, cultural, environmental) kinds of differences by race *are known to* exist and to contribute to disease and therefore ought to be studied (e.g. Cooper et al., 2003; Shields et al., 2005). And finally, still others dismiss the etiologic significance of racial genetic differences altogether and instead forefront investigations in health research of other differences (e.g. Chaufan, 2007; Schwartz, 2001). Despite this range, however, these critiques collectively fault a kind of inertia in the science of racial differences in disease, comprised of an over-reliance on race as a seemingly self-evident and etiologically meaningful dimension of difference, and a reluctance to try to ascertain the factors that may account for variable disease incidence and outcomes. In the eyes of these scientists, prior logics of difference that essentially began and ended with race no longer serve. Instead, health researchers argue for a re-engagement with racial disparities in health, but one that seeks to *unpack* the factors that may give rise to those racial disparities, and to thereby *move beyond* an exclusive focus on race to other, more precisely defined, potential disease determinants (see also Bliss, 2012).

For some, post-genomic science offers the scientific possibility to fulfill these twin desires. In part, this is because it avoids simplistic claims of genetic determinism, embracing instead a seemingly newfound consensus about the combined genetic and environmental causation of disease. In this article, we examine the practices of scientists who profess a commitment to joint genetic and environmental causation, and the question of whether and how they are re-engaging with, unpacking, and/or moving beyond race in their studies of gene–environment interactions (GEIs). We find that our participants are influenced by broadly circulating ideas about the unfulfilled promise of etio-logic and genomic science to ascertain disease causation, and thereby motivated to mobilize and study homogeneity and heterogeneity of racial *and* other kinds. Rather than an exclusive attention to racial similarity and difference, scientists pointedly argue for attending to racial *and* other homogeneities and heterogeneities in dynamic relation to each other. That is, scientists conducting GEI research continue to invoke well-worn notions of racial difference and diversity, but use them strategically to try to examine other kinds of etiologically significant differences among populations. Scientists do this by seeing populations not as inherently homogeneous or heterogeneous – racially or otherwise – but rather by actively working to *produce* homogeneity along some dimensions in their study populations and heterogeneity along others. Thus, we argue that homogeneity and heterogeneity are *situational properties*: properties scientists seek to arbitrate and achieve in their study populations, the available data, and other aspects of the research situations they are confronting, and then leverage to advance post-genomic science.

In doing so, post-genomic researchers speak to two contrasting aspects of the contemporary logics of difference at work in post-genomic science. On the one hand, we see the *persistence* of race and ethnicity as a way of organizing thinking about populations, disease distribution, and practices of recruitment, data collection, and analysis. But in addition, we see the *fluidity* of race and ethnicity and their potential *malleability* into other notions of population similarities and differences that may be as or more meaningful for disease etiology. Our participants' research practices therefore show how race and ethnicity are not inevitable ways to define populations and/or their homogeneity and heterogeneity, but that similarity and difference as scientific objects and attributes are themselves situational: variously present or absent, invoked or produced, useful or not. Pointing to the situatedness of racial and other forms of homogeneity and heterogeneity[1] in GEI research underscores the work that these properties do and the contingencies that shape decisions about research procedures. Through a focus on the situational production and leveraging of homogeneity and heterogeneity more broadly, we point to how GEI research attempts to shift the logic of difference away from using racial terms as explanatory ends unto themselves, to using racial

[1]We use the terms homogeneity and heterogeneity in their colloquial and scientific sense, which have a number of binary, opposing meanings. At the most basic level, *homogeneity* refers to things deemed to be the same, identical, alike, and equal, and *heterogeneity* to things deemed different, distinct, unlike, and non-equivalent. Homogeneous things are therefore seen as consistent, uniform, and regular, while heterogeneous things are mixed, assorted, and diverse. But these definitions of homogeneity and heterogeneity also suggest that these qualities can be produced and constructed: homogeneity refers to that which can be harmonized, made to be uniform, and standardized, while heterogeneity refers to that which can be separated and distinguished. In the broad context of human populations – one of the central objects of gene–environment interaction (GEI) research and of this article – a *homogeneous population* is one that is believed to have, or that can be made to have, a uniform character, where all the constituents are of the same or similar nature, and are therefore recombinable and exchangeable. In contrast, a *heterogeneous population* is one that is believed to be, or that can be made to be, diverse in nature, composed of constituents of different and dissimilar kinds that are, and ought to remain, separate.

and other dimensions of difference as important clues to the causes of complex diseases. In the end, however, we argue that it remains an open question as to whether a GEI approach that is nested within a larger context of ongoing and accelerating genomic discovery can adequately unpack and move beyond race.

Situating our argument

Interest in the possible relationships among genetic and environmental causes of disease can be traced to mid–20th-century concerns about industrial pollutants and individual variability in susceptibility to disease (Shostak, 2003). Early research on gene-environment interactions, or 'ecogenetics', attempted to explain how specific genetic conditions affected individual responses to toxins (Brewer, 1971; Calabrese, 1984). The enormous growth of molecular biology and genetic testing technologies from the 1970s onward, however, led to entirely new techniques for thinking about and measuring how genetic variations and environmental conditions interact to produce susceptibility to disease (Puga et al., 1996, as cited in Shostak, 2003). In the 1990s, proponents of the integration of new knowledge from genetics and a public health emphasis on environmental determinants of disease urged geneticists and epidemiologists to set aside their epistemological commitments to disease causation as either environmental or genetic in favor of a new etiological paradigm of gene-environment interaction (Khoury et al., 2000). In this new paradigm, the focus is on genetic factors that confer more or less susceptibility, sensitivity, and/or responsiveness to environmental exposures, and on environmental factors that affect the expression and effects of genetic variations.

The dynamic tension and interplay between homogeneity and heterogeneity actually predates the post-genomic era, but examination of their use in scientific research is relatively scant. Taylor (2008, 2010) has examined the possibility of underlying heterogeneity in genetic and environmental determinants as a problem confronting geneticists in their quest to identify what makes conditions heritable. As his research suggests, reducing the underlying heterogeneity by studying more narrowly defined and less variable populations has been one common response. Scholars such as Navon (2011) and Kohli-Laven et al. (2011) have further observed that genomics continues to re-work clinical designations of persons and diseases, often leading to new understandings of human and disease heterogeneity. Homogeneity and heterogeneity have been long- standing objects of attention for epidemiologists and other health researchers as they consider study design, sampling, and methods; indeed, these qualities must be found in most any scientific study and are either implicitly or explicitly part of how scientists make sense of their data.[2] In our article, however, we analyze the meanings that GEI researchers invest in notions of heterogeneity and homogeneity as a means to track how they make sense of human diversity of different kinds.

Finally, there is of course a sizable literature on race, population differences, and the politics of group classification and identification in genomic research (e.g. Abu El-Haj, 2007; Bliss, 2012; Braun and Hammonds, 2008; Fujimura and Rajagopalan, 2011; Fullwiley, 2007,

[2]Our thanks to an anonymous reviewer for bringing this to our attention.

2008, 2011; Lee, 2006; Lee et al., 2008; Montoya, 2011; Reardon, 2005, 2009; Whitmarsh, 2008). In this literature, many (e.g. Abu El-Haj, 2007; Fujimura and Rajagopalan, 2011; Gannett, 2001; Reardon, 2005) have noted the widespread claims of a shift from typological notions of race to statistical notions of difference among populations (e.g. Cavalli-Sforza and Bodmer, 1971/1999; Dobzhansky, 1963; Lewontin, 1972). Such claims are enabled, in part, by human population genetics, and in particular, the notion of admixture, in which mixed genetic ancestry can purportedly be estimated to come from different ancestral populations characterized by continental origin. However, scholars informed by science and technology studies and critical race studies have contested the claim that these technoscientific developments have the potential to eclipse 'old' ideas about racial difference and hierarchy.

Gannett (2001), for example, asserts the 'ethical limits of population thinking', arguing that the replacement of races with populations does not and has not eliminated notions of race from scientists' thinking about populations, moderated their inclination to identify and measure group differences that incorporate cultural meanings of race, or precluded stereotypical and racist interpretations of statistical patterns. Fujimura and Rajagopalan (2011) explore the use of statistical genetics software that in theory allows scientists to identify and define population clusters without an a priori use or conceptualization of race. However, their concept of genome geography, referring to researchers' location of portions of the genome to specific geographic places and times, shows instead how notions of population, race, and genetic ancestry are brought together in ways that are difficult to disentangle.

Other scholars examining post-genomic science have found that researchers strategically and explicitly characterize populations as racial. Lee (2006), for example, finds that genetic biobanks naturalize race in their naming of groups whose DNA samples they store, and in so doing, render the body as the grounds in and from which racial difference can be read. 'Race-making through biobanking' (Lee, 2006: 458) is thus illustrative of an 'infrastructure of racialization' (Lee, 2005) that organizes post-genomic research on disease and pharmaceutical development.

Abu El-Haj (2007), using the example of admixture mapping technologies, also argues that claims about genomics undermining racist classifications are misplaced. Empirically speaking, admixture is measured using ancestry informative markers (AIMs), which are a set of genetic variations for a particular DNA sequence that appear in different frequencies in populations from different regions of the world. The use of AIMs compares an individual's polymorphisms at these markers with previously analyzed genomic reference sets from people whose ancestral history is supposedly fairly well known. AIMs are used to estimate the geographical origins of an individual's ancestors, typically expressed as proportions of one's ancestry that comes from different continental regions. Thus, Abu El-Haj points out that, by definition, AIMs and admixture presume race-mixing – here, she follows Michaels (2000) who says that to choose between 'pure' or 'hybrid' race is still to have already chosen race itself. In turn, AIMs are purported to provide biologically or historically useful information in calculating disease risks for a particular population.

Both Montoya (2007, 2011) and Whitmarsh (2008) examine post-genomic research in transnational contexts and attend to the ways in which race is invested with genetic, social, and other meanings. In examining race-based genetic research on asthma conducted in Barbados, Whitmarsh (2008) argues that while initially the biological, medical, geographical, and socially constructed were all simultaneously seen as defining or diagnostic of race, this capaciousness disappeared once results were found. When scientists had to adjudicate, for example, the representativeness of a population to stand in for other populations, or the generalizability of a set of findings, they then resorted to pragmatic and expedient efforts to measure 'race' precisely by admixture, for instance, or by presence of genetic variations. This use of biomedical technologies to provide extremely specific measurements of variable objects like race is what Whitmarsh refers to as hyper-diagnostics, a set of mutually reinforcing practices that knit together race, disease, and genetics, as well as nation-states, commerce, and moral logics about how to know a complex disease like asthma.

For his part, Montoya (2007, 2011) explores what he calls bioethnic conscription, the process by which social identities and life conditions of genetic research subjects are grafted onto biological explanations of disease causation. In a study of type 2 diabetes at the Texas–Mexico border, Mexicans and Mexican Americans were enrolled because they were seen as an ideal research population. While they were genetically admixed, they were seen as an ethnically homogeneous group; that is, the genetic admixture found in county residents was seen to be representative of that in the Mexican American population more generally (Montoya, 2011). Montoya argues that researchers thus produced a standardized admixed Mexican body. Even while researchers acknowledged the joint genetic and environmental determinants of diabetes, the end result is that

> [b]y deploying the ideology of admixture and hereditary disease etiology as the rationale for Mexicana/o sampling, researchers and DNA donation field staff construct research participants as genetic carriers in a stratified social order that places a premium on genetic purity.... By accepting the biogenetic risk narrative, affected people cede the sociocultural risk factors so central to disease. (Montoya, 2011: 103–104)

In Fullwiley's (2007, 2008) ethnography of two medical genetics laboratories, she finds, in line with Abu El-Haj (2007), that researchers used AIMs and the concomitant comparison of purportedly pure 'Old World' reference populations to 'New World' admixed populations to read race in DNA and revive race as biogenetically valid. Moreover, these researchers were explicitly motivated to do so, as minority scientists, by their moral commitment to intervene in health disparities. Bliss (2011, 2012) finds much the same in her study of elite genomic scientists, who exhibited what she calls a 'reflexive biosociality: [their] conscious effort to create analytics that contribute to a future they themselves want to live in' (Bliss, 2011: 1019). Even as they acknowledged the great heterogeneities within racial groups, these scientists attended to race in research participation as a means to ensure racial equality in research and drug development. In the shift 'from practicing colorblind science to race positive inquiry' (Bliss, 2012: 67), Bliss finds that elite scientists were no longer content to use 'race' as a catch-all proxy or black box variable for any number of unexamined,

unmeasured, and taken-for-granted factors (e.g. biological processes, diet, income, toxic exposures). Instead, they evinced a preference for and a commitment to measuring those previously un-interrogated factors themselves. This led them to carefully craft sampling taxonomies, enrolling populations within which, and so that, those previously unexamined factors could be explicitly measured. Bliss argues that within an 'anti-racist racialism', researchers strategically incorporate racial populations into genome science with the goals of deconstructing biological race and producing an inclusive genomic science.

However, Lee (2008) and Reardon (2009) complicate scientists' desire for genomic research to be more racially inclusive. Lee (2008) traces the emerging narratives of personal and scientific responsibility and courage to examine race, and to do so despite professional reputation and self-consciousness, against the perceived tyranny of political correctness. This discourse of 'racial realism', when leveraged in debates on the relevance of racial difference in health, can circumscribe our interpretations of that difference, and in turn, 'put in motion trajectories of inquiries that recapitulate ideas of distinct groups' (Lee, 2006: 460). Reardon (2009) assesses the current situation in genome science as one characterized by co-dependence: the dependence of scientists on subjects for participation and the dependence of research participants on scientists for genomic information to use for self-identification and access to care and services. In such a situation, argues Reardon, science conceived of as an autonomous realm cannot ensure freedom from domination by science or scientific experts, or freedom from the misuse of science. This is because democratic practices now play fundamental roles in constituting genomics, while genomic practices constitute what it means to be democratic. In this instance, according to Reardon, simply democratizing genomics through racial representation and participation does not lead self-evidently to anti-racist science.

In this situation of shifting concepts of and investments in race and genomic difference, then, it remains crucial to investigate how GEI scientists, on the ground, engage in research practices that influence and are shaped by conceptions of race, ethnicity, diversity, and population similarities and differences. Below, we describe the methods of a study in which we seek to analyze those very practices. We then detail our findings on how scientists conducting GEI research attend to homogeneity and heterogeneity as situational properties, their strategic efforts to produce those qualities in their research populations, and their subsequent leveraging of racial and other similarities and differences to reveal etiologic patterns that could advance post-genomic science.

Methods

Our study began in 2010, investigating the conceptions and uses of race and ethnicity in GEI research on the etiology of complex diseases. In particular, we were concerned to gather data on scientists' actual research practices – including how and why specific study designs, samples, variables and measures, measurement tools, and analytic procedures are selected and implemented – and how these practices have changed over the course of a study. We recruited US-based genomic scientists currently conducting major GEI studies of heart disease, type 2 diabetes, and cancer etiology. We ascertained that these studies could be defined as GEI research by examining publications, study aims, and scientists' own

descriptions of their studies. Studies also had to include at least one non-white population and be supported by US federal funding.

All of the 32 participants whose interview data were analyzed for this article are affiliated with either US academic institutions or the US National Institutes of Health. GEI research is an inherently multidisciplinary field, and we therefore asked our participants to characterize the work that they do, rather than to locate themselves within a particular discipline.[3] In the course of answering this question, our participants did report a range of disciplinary affiliations (numbers total greater than 32 because many participants listed more than one affiliation): 'epidemiology' (n = 9) or some specific field of epidemiology (n = 6); 'genetic epidemiology'[4] (n = 10); 'molecular epidemiology' (n = 3); and 'genetics' (n = 7). Three other disciplinary affiliations were mentioned by just one participant each (e.g. 'physician' or 'cancer researcher'), and an additional two scientists did not name any disciplines or subfields. In this article we refer our study participants more generically as 'GEI researchers' or 'GEI scientists'. Moreover, given the relatively small numbers of participants within specified subfields, we do not make claims about the potential connections between identification with a discipline and the kinds of research practices they engage in. However, when presenting quotes from participants, we have included their disciplinary affiliations as they identified them.[5]

To date, we have conducted 53 in-depth interviews with 32 scientists (21 of these interviews were follow-up interviews conducted on average 1 year after the first interview). We have also conducted over 200 hours of observations and additional informal interviews at nine scientific conferences where findings from GEI research were presented. All interviews were transcribed verbatim, while observation data consisted of field-notes along with selected verbatim transcriptions of podium presentations and question-and-answer sessions.

All data were uploaded to the qualitative data analysis software ATLAS.ti. Initial codes were first generated inductively through a collaborative reading and analysis of a subset of interviews and then finalized through successive waves of coding into approximately 80 categories and codes. While some of our participants used the terms 'homogeneity' and 'heterogeneity' (or 'homogeneous' or 'heterogeneous') in their interviews with us, they also used a variety of other related terms, such as variation, variable/variability, subgroups, similar/ity, and different/difference. While none of our codes explicitly tagged participants' uses of these terms, we had a number of codes that related to the larger themes of homogeneity and heterogeneity. The ATLAS.ti query tool was used to extract data tagged with those particular codes and intersections of those codes (for example, the code 'study design', and the intersection of codes 'race/ethnicity' × 'recruitment'). We wrote, circulated,

[3]Following Abu El-Haj's (2007: 284, n2) call, we limited our study and this analysis not to post-genomic science as a single field or domain, but to those who conduct gene–environment interaction research. However, for reasons we explain in our 'Methods' section, it is difficult to discern the different perspectives of different disciplines, as she suggests analyses should do.

[4]We acknowledge here Montoya's (2011) description of genetic epidemiology as Janus-faced, where *genetic* epidemiology refers to 'the use of populations to understand the genetics of disease', and genetic *epidemiology* refers to 'the use of genetics to understand disease in populations' (p. 58). We also saw this difference in emphasis among our participants.

[5]To protect the identity of our participants, we have elected at various points to delete specific details about study design or samples, or the disease conditions they study.

and revised memos on these queries in order to generate our findings on homogeneity and heterogeneity.

Homogeneity and heterogeneity as situational properties

We argue that homogeneity and heterogeneity are *situational properties* – properties that scientists seek to arbitrate and achieve by working on their study populations, the available data and other aspects of the research situation they are confronting, and which they then leverage to advance post-genomic science. Why 'situational'? With this term, we make several related points. First, much of the focus on homogeneity and heterogeneity is along racial and ethnic lines. Race and ethnicity continue to be 'a prominent "search tool"' (Koenig et al., 2008b: 3) for post-genomic GEI research, a conventional set of dimensions by which scientists organize what is known about disease risks and incidence to reveal potentially informative patterns of disease. Our scientist-participants either seek out or aim to produce research situations characterized by racial and ethnic homogeneity or heterogeneity.

Next, the situational presence or production of racial similarities and differences in disease risk and incidence are then used to strategically examine *other* differences that may be important to complex disease etiology. Many leveraged sameness and difference along commonly used racial and ethnic categories to *see and think through, with, and about* various *other* kinds of homogeneity and heterogeneity, such as the prevalence of genetic variants, disease subtypes, clinical sites, health-related behaviors, and environmental conditions. That is, homogeneity and heterogeneity within and among these categories served as tools that revealed other, potentially etiologically important population variations.

Thus, scientists regard populations not as inherently homogeneous or heterogeneous (racially or otherwise), but as homogeneous or heterogeneous enough for some purposes and not for others, depending upon the situation and the purpose at hand. Here, we invoke Clarke and Fujimura's (1992) assertion that perspectives on what constitute the 'right tools for the job' – that is, notions of 'tools', 'jobs', and 'rightness' – are all situationally constructed. In our context, racial homogeneity and heterogeneity are 'jobs' in that they are problems to be tackled and properties to be produced, and they are also 'tools' that do work for scientists. These then are the two scientific situations in which racial homogeneity and heterogeneity were deemed useful for the conduct of post-genomic science and the advancement of knowledge about disease etiology and disparities. We describe each of these in turn below.

Working on race: the situational production of racial homogeneity and heterogeneity in study populations

One major way in which we saw the situational invocation and production of racial and ethnic homogeneity and heterogeneity was in the recruitment, enrollment, and consideration of study populations. As such, our data align with Epstein's (2007, 2008) observation of the rise of 'recruitmentology', an emerging science that evaluates 'the efficacy of various social, cultural, psychological, technological, and economic means of convincing people ... that they want to become, and remain, human subjects' (Epstein, 2007: 182–183).

Recruitmentology involves both intellectual and practical work aimed at recruiting and retaining study participants, especially those from hard-to-recruit populations. In our study, while scientists alluded to the challenges of recruiting for diverse study populations, they did not address the 'science' of recruitment that Epstein notes has produced the convergence of institutional policy and framing to entice participants into research. Instead, our participants described multiple situations in which they made ad hoc, on-the-ground, decidedly not evidence-driven decisions as to how to characterize the individuals they sought and managed to recruit, as well as decisions about how to manipulate, post hoc, their study samples in order to achieve sufficient levels of homogeneity and heterogeneity. In some situations, racial self-identification was seen as a 'good enough' tool for designating racially homogeneous populations for study, while in other situations, such modes of categorization were perceived to lead to faulty assumptions of sameness. In still other situations, racial homogeneity by self-identification was balanced against racial heterogeneity defined in terms of genetic admixture.[6] Throughout, researchers perceived racial and ethnic homogeneity or heterogeneity not as inherent to specific populations, but rather as properties that were situationally present or absent, and as properties to be worked upon and produced. In this sense, the racial and ethnic homogeneity and heterogeneity of populations are construed as 'jobs' to be accomplished.

For example, one investigator told us about his motivation to initiate a prostate cancer study among African American families:

> All of the populations that were being studied – this was across several different groups – were Caucasian. Why? Because it was easy to collect those families.... So in the U.S. there were huge numbers of Caucasian families collected and some African American families collected, but not enough to be able to really say why was there more prostate cancer in African American men.... [We] said, 'This is ridiculous. We know there are huge health disparities. And no one is making an effort to find out why. We have got to start a study that is going to enroll large numbers of African American men and their families and try and figure this out.' And that's what we did.... [We] went out and found terrific mostly African American clinicians around the country. Put out a contract. These guys got together. They designed their study. They collected these families from all across the country and were phenomenally successful in enrolling African American families so that we finally have a decent number. (Interview 08, statistical geneticist/genetic epidemiologist)

In this example, African Americans are seen as relatively unproblematic in terms of their homogeneity as a categorical group. In order to maximize recruitment and sample size, African Americans were considered – at least in this situation – to be homogeneous enough for the purposes of studying prostate cancer disparities.

[6]To be clear, we do not ourselves equate racial homogeneity or heterogeneity with genetic admixture. However, many of our participants frequently assumed or stated that genetic admixture was one dimension along which racial sameness and difference could be assessed. Thus, we include those instances in our discussion of how scientists work on racial homogeneity and heterogeneity.

In contrast, another scientist told us of the difficulties in enrolling subjects for a case-control study of another type of cancer[7] among Hispanics. In a case-control study design, researchers must find statistically sufficient numbers of cases, or individuals who have the disease in question, as well as individuals from the same population who do not have the disease, to serve as controls. For this participant, however, the requirement that cases and controls come from the 'same' population proved to be particularly complicated to ascertain and in turn challenging to fulfill:

> We had major complexities with our Latino populations, because ... how you recruit the cases versus how we recruited the controls was not really parallel.... The [cancer] cases we get through a population registry. [For the controls] we tried doing random-digit dialing. But because they're a lower frequency in the population, it just was very prohibitive to find them that way. And we tried sort of [using claims] files ... it still was very, very, very difficult to identify Hispanic controls. So we ended up doing community recruitment. And that turns out the people who we were recruiting through the community ... had different Hispanic ancestry than the population-based cases did. So the population-based cases tended to be more sort of multigenerational Hispanic or from Mexico, whereas the community-based controls tended to be Central American ancestry. So that, of course, threw a huge monkey wrench into a lot of our analyses, as well, because the Central American Hispanics had much more Amerindian admixture than the cases did, who had much more Caucasian admixture.... We did do a second series where ... we used a [different strategy to recruit] for both the cases and the controls for the Hispanics ... since we weren't able to really recruit adequate numbers through other population sources. (Interview 07, genetic epidemiologist)

In this example, the nature of Hispanics as 'a population' is of explicit concern to the scientist who explains having to match cases and controls for admixture. In contrast to the prostate cancer study among African Americans described above, in which self-identification was deemed sufficient to define the study population, here, genetic heterogeneity among Hispanics due to their systematically variable admixture precluded their treatment as a singular population or category.

And a third example shows how in still other situations, scientists seek not to produce a racially homogeneous or heterogeneous population, but rather one in which racial homogeneity and heterogeneity are balanced with one another in order to advance post-genomic science. This participant described a global consortium bringing together studies of African American–descent and African-descent men:

> There are [many] different members or studies that are involved in this multi-center consortium. They're all a little bit different. Most of them are case-control studies, there're cohort studies .... As you can imagine, finding a sufficiently large sample of African-American or African-descent men to do this kind of work is difficult in any one center.... We have centers in [a number of cities in Africa].... We really went to people who had existing studies or were in the position to develop studies quickly

[7]See note 5.

> or immediately.... But it's really difficult ... getting anything done in Africa is incredibly difficult.... You take absolutely whatever you can get. And you can't be too picky about what that is.... This large consortium ... is all men who self-identify as being of African descent. So these are African Americans, Africans, or Caribbean, African-Caribbean men. And so they are all self-identified. (Interview 04, cancer researcher)

This participant went on to argue that by involving studies that enrolled simply on the basis of self-identification, the result was a consortium-wide sample that included wide variability along a number of other dimensions, including ethnic, geographic, and environmental differences. This then enabled them to examine associations between disease outcomes and, for example, socioeconomic status, stress, diet, and proportion of African or European ancestry. In this instance, then, recruiting for racial homogeneity along a fairly loose criterion – self-identification – allowed the compilation of a pool of participants who were not only heterogeneous in terms of genetic or biogeographic ancestry but also heterogeneous in other factors of potential etiologic significance.

Thus racial homogeneity and heterogeneity are not seen as singular properties that are inherently present or absent in any given population, but instead as situational properties that are arbitrated and produced according to the scientific contingencies and needs of the particular moment and study. To accomplish this, scientists report having to spend a tremendous amount of time and energy; highlighting the situational nature of population homogeneity and heterogeneity underscores the work involved in their production. In the prostate cancer study, this work involved identifying strategically placed clinicians with access to desired populations, cultivating working relationships with them, modifying recruitment and data-collection protocols to work in various clinical contexts, and forging connections to existing studies through which other families could become study participants. In the cancer study among Hispanics, researchers had to pilot various strategies for recruiting controls for a case-control study and identify and implement alternatives to failed strategies. This is, as one scientist told us plaintively, a ‘nightmare’:

> The more you start to study something, the more the complexities you see in it.... In thinking about doing multiethnic studies, you really have to think.... It's a learning process, right? And it is very, very, very, very challenging to really design a really good multiethnic study, I think, for all of those reasons that I just laid out.... It's one of those things where it is not as clear as ... we had hoped that it would be.... It's been pretty daunting.... The recruitment issues are really serious.... Just the reality of how difficult it is, is really serious. (Interview 07, genetic epidemiologist)

Yet this work of producing situationally appropriate levels of population homogeneity and heterogeneity was considered extremely important for understanding complex disease etiology. For example, scientists we interviewed and observed described how they controlled and adjusted for genetic heterogeneity within their populations. This work ensured that associations found between disease outcomes and genetic variations could be properly attributed to those genetic loci, and not to systematic differences in allele frequencies between populations due to different ancestry (population stratification, in genetic parlance). Genetically homogeneous populations are widely perceived as producing

'clean' and 'beautiful' results, while heterogeneous populations are characterized as 'mongrels' and 'mutts', providing 'dirty' data or data with too much 'noise'. One participant used the computer science phrase 'garbage in, garbage out' as a cautionary metaphor: using inappropriately heterogeneous populations ('garbage in') without cleaning up the data would yield useless results ('garbage out'). This whole lexicon indicates the extent to which scientists are still drawing on notions of populations as imbued with properties of purity or contamination, notions that harken back to well-worn ideas about racial homogeneity and heterogeneity. Yet, at the same time, with their focus on the quality, clarity, and credibility of the results yielded by study populations, scientists also gesture to their quest to homogenize human populations along racial and ethnic dimensions *so that* they can explore *other* factors relevant to disease (e.g. environmental conditions, exposures, etc.). In short, they seek to produce the kinds of populations that can *do* and *say* something useful about complex disease etiology, and it is to this point we turn to next.

Unpacking race: the situational use of race to produce other forms of homogeneity and heterogeneity

We also see racial homogeneity and heterogeneity strategically invoked and leveraged in the hopes of unpacking what are seen as crude, simplistic, and imprecise understandings of race. Our participants mobilize claims of racial and ethnic homogeneity or heterogeneity as a means to uncover other shared or distinctive factors that shape disease distribution. That is, they compare racially and ethnically defined populations exhibiting different epidemiologic patterns as a first cut to reveal differences that help interpret, explain, and otherwise unpack those patterns. In this sense, then, racial homogeneity and heterogeneity are situational properties that act as 'tools' that can do work for post-genomic etio-logic science.

In discussions of cancer disparities, for example, we repeatedly witnessed situations in which investigators walked carefully through a set of observations to support their claim that different racial populations may in fact be experiencing different forms of a particular cancer. We observed this most frequently for breast cancer but also for prostate cancer, leukemia, and other cancers. In one example, a conference presenter explained that the widening gap in breast cancer incidence and mortality between African American and White women could be due to the advent of tamoxifen therapy, which lowered rates among white women who have more endocrine receptor(ER)-positive tumors, but not among African American women who have more ER-negative tumors. Another participant in our study mentioned the same epidemiologic pattern of more aggressive breast cancer being seen among African American women, and raised the question, 'Are they Black–White differences or are they cancer subtype differences?' Here, Black–White differences provoke scientific questions about population variations in cancer, and 'cancer subtype differences' are seen as potentially distinct from 'Black–White differences' rather than being invariably correlated and conflated.

Thus, racial difference directs attention to the possibility of significant tumor type differences underlying racial disparities in breast cancer mortality. Because racial difference only imperfectly lines up with disease type, they are not seen as equivalent or interchangeable distinctions. Rather, tumor type is seen as a potentially more precise

characterization of difference. That is, while categorization and comparison by race persists as a frequent starting point for this kind of disease research, in many situations it no longer serves as a terminal analysis (Shim, 2014) wherein an analysis by race and ethnicity is seen as a sufficient explanation unto itself, given that racial differences are to be expected and thus require no additional investigation. Neither simply color-blind nor purposefully race-conscious (Bliss, 2012), and discontented with using racial classifications as 'a necessary evil' and 'good enough' variables (Montoya, 2011: 61), scientists in our study use racial and ethnic categories to *both* gain a sense of those underlying differences *and in order to* move beyond those very categories. Ultimately, the goal for them is to find other, more precise and specific, and therefore scientifically 'actionable' dimensions – that is, those that lend themselves to some intervention – along which study populations can be characterized and differentiated. In this way, racial and ethnic homogeneity and heterogeneity are not scientific endpoints in and of themselves, but rather do work in the service of etiologic discovery.

A second example of leveraging racial homogeneity and heterogeneity as a move to consider factors other than race was accounting for geographic variations in disease incidence. Participants took racially homogeneous populations and compared populations of the same race residing in different areas to reveal other etiologically significant forms of heterogeneity. In one case, a scientist explained,

> You can have populations which basically have no diabetes and then change their environment – put them in a obesogenic or ... diabetogenic diet context, with no physical activity, lots more exposure to refined sugar, fast food, whatever – and then diabetes becomes rampant. We see that historically: Native American populations, Pacific Islanders in particular, but also larger populations, like Japanese and other Asian populations.... When populations move from one context to another, they invariably adapt – whether it's 10 years or two generations, or whatever – they invariably adapt [to] the disease rates of where they moved to rather than where they came from.... People in Chinatown in San Francisco ... [even] if they marry other people from China and have kids, and ... their parents and grandparents all come from China, whatever, they still take on the disease patterns of the U.S., not of where they came from. (Interview 03, epidemiologist)

In this case, homogeneity along racial lines was initially leveraged in order to reveal heterogeneity in environmental contexts as a means to uncover factors *other than* race that are etiologically significant. Scientists were thus able to mobilize racial homogeneity and environmental heterogeneity situationally – holding race 'constant' and then observing differences across geography – to make claims about disease causation that were more precise, complex, and somewhat, or at least potentially, less deterministic.

Another set of examples we encountered of the situational leveraging of homogeneity and heterogeneity revolved around recruiting for racial and ethnic homogeneity and studying within-group heterogeneity. One interviewee told us his group is recruiting for an African American–only study population in order to study the effects of socioeconomic and environmental factors on one type of cancer. Their rationale was that:

> There may be a little broader ... exposure gradient, being African Americans are sort of maybe a wider social group ... more at the lower end of the economic spectrum and still some of the higher end.... You might benefit by studying them to look at sort of the environmental factors as well.... If you're interested in dietary factors, there would be different preferences in different populations ... you could try to quantitate [*sic*] better how much that influences the risk of [] cancer.... Some of those other factors ... might be more informative in this population. (Interview 20, geneticist/epidemiologist[8])

And at another conference, one of the principal investigators of a study of Hispanics characterized the objectives as

> [F]irst of all, to understand the prevalence of risk factors, or protective factors, of cardiovascular disease and pulmonary disease in Hispanics and Latinos of different backgrounds, not just one particular Hispanic group or one particular region of the nation ... according to national or background of the groups, [and] in addition, comparisons across genders and age groups.... As diverse as Hispanics are, abroad and here, once they come to the United States, all of them receive the denomination 'Hispanic' or 'Latino'. So all of a sudden it's like their roots and their backgrounds are erased, and then all of them are put in the same category.... These are differences that need to be acknowledged and need to be incorporated into the analysis of what is happening to the Hispanic/Latino population in general.

This presenter went on to enumerate other distinctions that may affect their health risks, from access to care in countries of origin, why and how they came to the United States, their political views, and their local health systems.

Yet another participant was conducting a GEI study of one type of cancer among Latinos, Caucasians, African Americans, and Asian Americans. This heterogeneous sample was both the result of no one racial or ethnic group being large enough to support its own study, and also an attempt to leverage racial and ethnic heterogeneity to reveal environmental exposures that may be important for cancer risk as well as possible determinants of the uneven rates of that type of cancer seen among different populations. But in order to do so, he told us that what they needed was 'an army of people to actually collect information about exposures, and infections, and other impacts to put together with the genetics':

> We've been assessing environment extremely intensively to the extent where we actually vacuum the homes and analyze chemicals in the dust. We get all the codes on the cans of stuff people leave under their sink.... Also nicotine and cotinine ... from maternal smoking or side-stream smoke from fathers or other family members.... So every person in the study has had a personal face-to-face interview ... and they had an assessment of diet.... There's also infections ... vaccinations.... Hydrocarbons from sources such as traffic ... where people live in proximity to traffic and how the wind blows. And pesticides ... any kind of diagnostic or therapeutic radiation. (Interview 02, molecular epidemiologist)

[8]This participant specifically self-identified as both a geneticist and an epidemiologist, though not as a genetic epidemiologist.

In this case, then, this scientist begins again with racial and ethnic heterogeneity, in the form of an extremely diverse study sample, but used that as a point of departure to then consider a laundry list of other dietary, environmental, and social exposures. Among this list are exposures known to disproportionately affect one racial or ethnic group (e.g. pesticide exposure among Hispanics) as well as those that are suspected to confer risk for certain types of cancer. This scientist described how the prevalence of these potential determinants were compared across racial and ethnic groups, which of these differences were found to be associated with higher risk for the kind of cancer under study and which exposures were found not to differ appreciably between groups. Thus, these study data were used to account for *differences* in risk seen *between* racial groups represented in the sample, but also some *similarities across* race and ethnicity as well as *differences* seen *within* a group. Thus, this participant initially leverages the situational presence of racial and ethnic heterogeneity, and seeks to produce data pointing to other forms of homogeneity and heterogeneity.

These cases illustrate how scientists see racial homogeneity and heterogeneity as situationally important, because leveraging these properties allow them to investigate factors associated with, yet potentially distinguishable from, race that could prove important to disease causation. Initially leveraging racial homogeneity or heterogeneity as an investigative starting point led researchers to other dimensions of heterogeneity (e.g. cancer subtypes, ‘diet contexts’, socioeconomic gradient, and ‘environmental factors’) that attempt to move beyond the very categorical racial differences that were initially employed. By taking advantage of situations in which racial sameness or difference was understood to be present, our participants were not using identity categories solely as ends unto themselves, but as tools to *see and think through, with, and about* various kinds of homogeneity and heterogeneity. Thus, scientists leverage homogeneity and heterogeneity strategically and dynamically to illuminate some new knowledge about the racial patterns of disease etiology. In this sense, properties of homogeneity and heterogeneity were seen and worked upon as situational properties – both in the sense of being useful properties found to exist in specific situations that could be leveraged and that could do work for producing post-genomic knowledge about disease risk, as well as properties to be achieved and produced in situations so that they could be leveraged.

Conclusion: beyond race?

In sum, scientists conducting GEI research strategically produce and leverage sameness and difference in individuals and populations in ways that allow them to ask certain questions about the etiology of complex diseases. Answering those questions relies in part on having homogeneous enough *and* heterogeneous enough study samples of sufficient size along different dimensions to make comparisons scientifically credible and worthwhile. Our participants therefore engage in a great deal of social and scientific work to *produce* the right mix of homogeneity along some dimensions and heterogeneity along others, depending on their specific research projects’ demands and circumstances. They also *leverage* homogeneity and heterogeneity to produce useful knowledge about disease etiology. Thus, we see scientists constructing homogeneity and heterogeneity as situationally and alternately valuable qualities, which they use to navigate a landscape of scientific, fiscal, and logistical

challenges, while maintaining a focus on making useful contributions to etiologic knowledge.

GEI scientists' vision of homogeneity and heterogeneity as tools and situational properties in constant relation to one another reveals when and where scientists are attempting to move beyond the simple inclusion of race as a self-evidently significant and meaningful determinant of disease. By closely attending to their lines of thinking about and practices to elucidate relative combinations of sameness and difference, we can see how homogeneity and heterogeneity are negotiated within the work of post-genomic GEI research in several continuous yet also potentially new ways.

On the one hand, the lens of race still remains a prominent means of distinguishing sameness from difference, and is quite often thought of in terms of the homogeneity of populations. That is, GEI scientists require that individuals be 'like enough' to enable them to be considered as one population, for recruitment purposes, in building consortia, and allowing for data sharing across studies. Racial categories therefore remain foundational to how study populations are conceived and are not always relinquished even when other differences are identified as more salient etiologically.

On the other hand, our participants often used race as a segue to thinking about other kinds of differences – for example, differences in behaviors, diet, and so on. Their considerations of homogeneity and heterogeneity now also include diseases, behaviors, and exposures in ways that both implicate and move beyond race and ethnicity. That is, GEI researchers use racial and ethnic homogeneity and heterogeneity as initial wedges to open and peer inside various black boxes and etiologic puzzles involving the mechanisms of disease, the nature and consequences of environmental exposures, the potential influence of genetic variations, and the underlying causes of health disparities. At the same time, researchers described racial and ethnic categories as themselves needing revision in light of new appreciations of – and technoscientific abilities to parse – heterogeneity, diversity, specificity, and complexity. Scientists continually tack back and forth between demands for adequacy and precision – in populations, group labels, disease classifications, identification of genetic variants, and measurements of environmental exposures.

We opened our article with the common observation that although much of the fanfare around the completion of the Human Genome Project focused on the presumed universality of the human genome, post-genomic science quickly circled back to the investigation of difference. At the same time, some researchers had been engaging in a critique of the cultural and scientific inertia within the study of race and health, and of a perceived myopia that racial difference in and of itself was sufficiently explanatory for disparities in disease incidence and distribution. In particular, with the emergence of a consensus on the joint genetic and environmental causes of disease, the circumstances seem ripe to both unpack the significance of race for etiology and to move beyond it. This rhetoric suffused many of our interviews with GEI researchers. At the same time, however, our findings on the production and leveraging of racial homogeneity and heterogeneity lead us to be fairly circumspect about whether post-genomic science is moving beyond race. On the one hand, our data show the persistence of race as a lens to see and produce robust data and science, but on the other

hand, they also simultaneously reveal a certain amount of fluidity immanent in the use of race to point to *other* homogeneities and heterogeneities of potential etiologic import. Our article thus underscores both the self-evident nature of race as a way to define and distinguish study populations, and yet also reveals its situated meanings and utility: post-genomic scientists sometimes see race as illuminating etiologically significant differences, and other times as obfuscating those very differences.

Our project centers on research that explicitly aims to examine GEIs because we saw this part of the post-genomic landscape as a fertile site to track whether and how scientists produce, leverage, and move beyond race. In the face of complex disease causation, to what extent does the now-common discourse, that scientists must account for genetic, biological, behavioral, and environmental determinants, apply to race? We argue that it is not apparent or inevitable that GEI research will push post-genomic science beyond race. Research on complex diseases with complex, multiple, interacting causes requires the 'right' tools to investigate that complexity. As our article shows, scientists conducting GEI research run into multiple social and technical constraints that shape the kinds of decisions they can make, what samples or measures they can cobble together, and how they do their science. Racial homogeneity and heterogeneity remain important tools in negotiating these constraints. As a society, as some of our participants have pointed out, we have invested in learning how to manage some kinds of complexity and not others, depending on our thinking and priorities about what kinds of complexities matter and can feasibly be studied. The post-genomic era has promoted ever-growing research infrastructures that promise to produce hyper-precise data on genetic heterogeneity, while the nuances, specificities, and heterogeneities in environmental exposures, social class, and resource inequalities remain under-investigated (Krieger, 2013; Montoya, 2011; Shostak, 2013). Thus, while the situatedness of homogeneity and heterogeneity hypothetically opens the door to more capacious thinking about the influence of race on health, the current situations in which post-genomic scientists find themselves can instead reinforce well-worn notions and uses of race.

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