

# WHEN SOCIALLY DETERMINED CATEGORIES MAKE BIOLOGICAL REALITIES: UNDERSTANDING BLACK/WHITE HEALTH DISPARITIES IN THE U.S.\*

## *1. Introduction: When the Social Makes the Biological*

There remains a broad consensus that current folk racial categories—those categories usually used on surveys, recognized by the U.S. Office of Management and Budget (OMB), and used on census forms and by U.S. Federal Drug Administration (FDA)—do not correspond to meaningful biological categories. *Pace* some recent attempts to defend the use of folk racial categories as a proxy for ancestry and population-level genetic differences (see e.g., Risch et al. 2002), the links between folk racial groups and ancestral populations remain weak, in part because currently identified folk racial categories are historically contingent social constructs (see e.g., Smedley and Smedley 2005 and cites therein), and are in any event for the most part too internally heterogeneous with respect to ancestry to be useful as a proxy for population-level genetic differences (see Barr 2005, Root 2003, 2005; Bamshad et al. 2004 makes a similar point, but raises the possibility of important exceptions). But the broad failure of folk racial categories to correspond to biologically meaningful population-level genetic differences does not imply that there is no biological reality to folk racial categories; this conclusion would only hold if all important biological differences between populations were genetic. The social importance of folk racial categories for (nearly) every important aspect of social success in the U.S. creates a situation in which (at least some) folk racial categories are correlated with (and causally wrapped up in) important biological differences—for example, the long-standing health disparities between Black and White Americans.

Rather than standing as proxies for ancestry or population-level genetic differences, folk racial categories should themselves be recognized as key causal components of biological differences. That these categories are *social*

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—and that membership in a particular category is contingent upon the particular social/cultural milieu in which one finds oneself—does not imply that the categories are biologically meaningless. It *does* imply that there can be no straightforward causal pathway *from* biological facts *to* membership in a particular folk racial population—biology *doesn't* make 'race'. But 'race' might very well make biology—the causal arrow can point from a social category to the creation of biological differences between the populations identified. And indeed, the evidence is overwhelming that, at least in the U.S. context, race membership does just that.

Recognizing the ways in which biological differences are created by race (as a social category) is important for at least two reasons. The first is that it shows why race-based medicine, as a clinical practice, might have some success even if there are not population-level genetic differences between folk racial categories that cause the observed health differences. That is, even *if* for example particular drugs have systematically different effects on for example Black and White Americans, this *would not* itself provide unambiguous evidence for the existence of a genetic difference between the populations. Similarly, if race (as a social category) influences the etiology of diseases, the hope that individual genetic analysis will be able to supplant paying attention to race in medicine is misguided. If the health effects of race emerge from the social aspects of race rather than from genetic differences, genetic information will never be able to replace the folk-racial category in medicine. The second reason is that *even if* race-specific medicine succeeds in finding, e.g., particular drugs that work differentially on Black and White Americans, insofar as the health disparities between folk racial categories are the result of social differences (including of course racism), a purely medical approach to dealing with the health disparities should be seen as inadequate and indeed misguided. If racism is the root cause of, or even a substantial contributing factor to, the health disparities, medically treating the health disparities created by racism while ignoring the underlying cause should seem perverse.

## 2. *Biology (Still) Doesn't Make Race*

Until relatively recently, there existed a consensus in evolutionary biology and anthropology that, biologically, there were no human races. The evidence cited for this conclusion is that the vast majority of genetic

variation in the human species exists within, rather than between, the various 'races' identified on the basis of social categories. Because most genetic variation exists within folk racial categories, knowing someone's socially identified 'race' tells one very little about what alleles that person will have. That is, knowing someone's race doesn't tell you much about their genes. But some researchers have argued that despite the genetic heterogeneity of socially identified races, human races *do* exist in a biologically meaningful sense (see Rosenberg 2002, Risch et al 2002, see also Andreasen 1998). This conclusion is based on the fact that, when multiple different genetic markers are used, populations (albeit carefully chosen ones) can be split into groups that correspond (roughly) to major (historical) continental populations (Risch et al 2002, 3). Since these major continental groups (Africa, Europe, East Asia, the Americas, the Pacific Islands) can be mapped (albeit roughly) onto some of the folk racial categories currently recognized in contemporary western society, (African/Black, Caucasian, Asian, Native American, Pacific Islander), the idea is that genetic analysis has vindicated the biological meaningfulness of (some) contemporary folk racial categories. This conclusion is further supported, it is argued, by the fact that by testing many genetic markers, individuals can (often) be assigned "unambiguously" to one of these major groups, or to a particular admixture among the major groups.

From this, Risch et al. (2002) conclude that self-identified race, acting as a proxy for the continent of ancestry, is likely to be useful in clinical medical contexts, as ancestry will point towards different genetic backgrounds. These genetic differences, they argue, might result in different disease prevalences, or in differing prevalences of particular variants of metabolic pathways, and hence in different average responses to various drugs. So more accurate risk assessments for particular illnesses, and better pharmaceutical practices, might result from taking self-identified race into account in clinical practice (Risch 2002, Holden 2003). While such "racial profiling" in medicine would be temporary on this view—the hope, according to Holden, is that "the advent of genomic medicine will obviate the need to grapple with race issues" (Holden 2003)—that doesn't make it any less important as an "interim strategy" (Goldstein, quoted by Holden 2003, 596).

There are at least two major problems with this line of reasoning. The first is that if self-identified race is to have genetic meaning relevant to clinical practice, the fact of population-level clustering is (nearly) irrele-

vant, as is the supposed ability to accurately assign individuals to these groups by testing multiple genetic markers. Recall that what differs between these populations is the *proportion* of individuals with one variant rather than another; nor are these differences in proportions particularly profound. Since most high-prevalence conditions that are thought to have a genetic component will be broadly multigenic (see Weiss 2008 for review), it seems unlikely that individuals picked out on the basis of self-identified race, or even ancestry, will be sufficiently similar to each other, and sufficiently different from people in other self-identified races, to share different genetic risk factors in a way meaningful to clinical practice (see Barr 2005 for discussion). As Krieger (2005) argues, while the particular alleles one has might well predict one's ancestry, one's ancestry cannot predict which alleles one has; since it is the latter that is important for the clinical practice of medicine, the genetic differences between populations cannot support the use of self-identified race in clinical practice.

There do exist relatively rare genes for which ancestry makes sense as a proxy for risk; the HbS allele associated with sickle-cell anemia is perhaps the most famous of these (other examples include the genes associated with Tay-Sachs disease in Ashkenazi Jews, thalassemia in various Mediterranean and other malarial regions). But the risk for carrying the HbS allele is associated *not* with one's folk racial category—the usual self-identity—nor even the *continent* of origin, but rather with the very specific area(s) of one's ancestry. As Root, for example, stresses, it is *not* that Blacks have a higher frequency of the HbS allele than Whites; rather it is that people whose ancestors are from areas with endemic malaria have a higher prevalence. Since this includes populations from both malarial regions of Europe and Africa, HbS risk cuts across both folk racial categories and the clusters identified by, e.g., Rosenberg et al. (See Root 2003, see also Pigliucci and Kaplan 2003). Here, Root argues, self-identified race should *not* be used as a proxy, at least in part because it is a poor one, overpredicting Black risk and underpredicting White risk.

The second major problem with the approach to rehabilitating a biological concept of race defended by for example Rosenberg et al. and Risch et al. is that it misses the point of denying the biological reality of races. What Rosenberg et al. have shown is that people identified as members of populations with ancestors in particular continents are more

genetically similar to each other than they are to people whose ancestors came from different continents; statistically, you are in fact more likely to share a particular allele with someone whose ancestors are from the same continent as yours than you are to share that allele with someone whose ancestors are not. Risch et al. make a similar point when they suggest that contemporary evidence tells against the hypothesis that humans mated randomly with respect to location (2002, 2–3). But no one has seriously suggested that human populations in general mate randomly with respect to distance, language, and culture, or that there is *no* population structure! That populations—especially those on different continents—would differ in the proportion of alleles was never in doubt. Rather, the claim against the traditional conception of human races is that human evolution has been marked by substantial gene flow between populations (see Pigliucci and Kaplan 2003, Root 2003, 1175); this is entirely compatible with there being different *degrees* of gene flow between different populations. And certainly, there will be, on average, less gene flow between populations on different continents than between populations on one continent. Paul Farber (2003), for examples, notes that this is why Dobzhansky insisted that there *were* in fact human races; since for Dobzhansky, a ‘race’ was any population that differed in the proportion of alleles from another population, Dobzhansky argued that the population structure in the human species basically guaranteed the existence of human biological races, *of this sort* (see also the 1962 exchange between Livingstone and Dobzhansky).

But because gene flow is a matter of degree, the “clusters” that emerge from population genetic analysis will depend on the choices we make regarding what populations to use as well as when to stop searching for finer-grained analyses (see Weiss and Fullerton 2005 for discussion). For example, when techniques similar to those that generate the five major continental groups are applied to populations in Europe, one finds (not unexpectedly) that the chances of two people sharing an allele vary based on the physical proximity of those persons’ ancestors *and* the chances that those ancestors shared a language (and culture more broadly) (see Novembre et al. 2008). Hence, roughly speaking, these techniques can “reveal” the population structure that resulted from linguistic and cultural affiliations—e.g., sharing a country. And, given enough markers to test, Novembre et al.’s work reveals that many individuals will be able to be assigned (often “unambiguously”) to a particular country of origin. So, by

the standards Dobzhansky endorsed, there are therefore many human races *within Western Europe*; indeed, using these standards, there will be a very large number of "races" of various sizes, some nested neatly inside each other, some partially overlapping in more complex ways. At this point, it should be clear that these kinds of genetic analyses may well detect the kinds of population-level differences in allelic frequencies Dobzhansky called "races," but they cannot lend support to the more traditional conceptions of human populations comprising some privileged number of biologically distinct "races" (see e.g., Appiah 1996 and Hull 1998 for discussion).

### 3. *Causes of Black/White Health Disparities:*

#### *How Race Makes Biology*

By almost every measure, Black Americans have, on average, worse health and health-outcomes than do White Americans. On average, Black Americans have a life-expectancy that is about 6 years shorter than that of White Americans (see Krieger 2005); Black Americans are more likely to die at almost every age than are White Americans, from the first year of life (about 2.5 times as likely, see Williams and Collins 2004) to the 75-84 age group (about 20% more likely, see Williams 2005); Black Americans self-report worse health than White Americans (see Williams and Collins 2004); Black Americans are much more likely to suffer end-stage renal failure associated with diabetes, and have lower survival rates for most major cancers (see Sellers et al. 2006); etc. In all, if the Black-White disparities in health outcomes were eliminated, there would be between 65 and 100 thousand fewer deaths of Black Americans *per year* (see Williams and Collins 2004, Williams and Jackson 2005). These differences are reduced, but not eliminated, when one controls for SES (Social Economic Status) and education; even when SES and education are controlled for, if Black Americans had the same mortality profile as White Americans, there would *still* be some 38 thousand fewer deaths among Black Americans per year (see Mays et al. 2007, but see Nazroo 2003 for discussion of the difficulties involved in properly controlling for SES).

Neil Risch has argued that at least *some* of these differences in average health outcomes are likely the result of population-level genetic differences, and that for those reasons attention to population-level genetic differences can be important to clinical practice. He writes for example that:

The structure of human populations is relevant in various epidemiological contexts. As a result of variation in frequencies of both genetic and non-genetic risk factors, rates of disease and of such phenotypes as adverse drug response vary across populations. (Risch et al. 2002)

Similarly, David Goldstein, a geneticist at the University College, London is quoted by Holden (2003) as saying "If you say on average the difference between West Africans and Europeans is slight, that does not rule out a great many variants that influence how people respond to drugs."

These are not isolated or maverick views; the NIH has been quite explicit in its recommendation that health disparities be addressed through, at least in part, genetics research. Charles Rotimi, the head of the NIH Intramural Center for Genomics and Health Disparities notes that the center's priority "will be to understand how we can use the tools of genomics to address some of the issues we see with health disparities" (NIH News, 2008), and NIH Director Elias Zerhouni argues that the "center's genetic and genomic researchers and disease experts . . . will advance our understanding of health disparities for the benefit of minority groups and all Americans" (NIH News, 2008).

But the view that this health disparity is the result of the differences in the genetic background of Black and White Americans fails to account for key elements of the available data. Aside from the obvious problems of the genetic heterogeneity of socially identified "races" (noted above) and the (likely) multigenic nature of most common diseases with a genetic component, the experiences of recent immigrants and the available cross-cultural comparisons are worth attending to. There is a profound difference in expected health and health-outcomes between native-born African-Americans in the U.S. and Black immigrants to the U.S. (whether from sub-Saharan Africa or other locations); recent immigrants have much lower rates of hypertension, heart disease, etc., and this difference is not eliminated when SES or education is controlled for, nor does it seem likely to be a selection effect (e.g., it does not seem likely that the effect is due to immigrants self-selecting for health) (see Williams 2005, Singh and Miller 2004; see also Nazroo 2003). Similarly, many of the health disparities between Black and White Americans are not reproduced across different national and cultural contexts; native born Black Americans have very different (and usually worse) average health outcomes than Blacks of

similar SES who are born in other (rich, western) countries, and Black and White populations in other countries often do not have remarkably different average health outcomes, once SES is controlled for (see Sankar et al. 2004 and cites therein, Cooper et al. 2005). If the different genetic background of Black and White Americans were responsible for the different health-outcomes, this would be mysterious.

None of this demonstrates that there are no average genetic differences between Black and White Americans that influence the average health outcomes; while the above is suggestive, there might yet be differences that are of interest to epidemiological research. However, first, it is worth noting, as de Melo-Martin and Intemann (2007) make clear, that *even if* there were significant genetic differences between e.g., Black and White Americans that caused different health outcomes, there is no effective way of either discovering that fact or of ameliorating it without addressing the current social and economic inequalities. Since most of the diseases that contribute to the health disparities are thought to be the result of complex gene-environment interactions, it is unlikely that the results of genetic research will be useful if undertaken without addressing the environmental differences associated with folk racial categories in the U.S. (see also Sankar et al. 2004). Second, even if it were possible to generate accurate data using such epidemiological research in the current social climate, it is unlikely that such epidemiological research would provide evidence of robust enough differences to support improved individual clinical decision making, and unlikely that such research would point towards useful ways of ameliorating such differences, without, again, first addressing the social inequalities that for example influence access to health care.

But even if there are no genetic differences that generate racial differences in health outcomes (or indeed, even if there are such differences), racial differences might yet be responsible for generating important biological differences, namely, those differences in health outcomes associated with membership in a particular racial category in a particular social context. After all, the most profound Black-White differences in the U.S.—differences in income, wealth, education, residential patterns, etc.—are *caused by* those very same folk racial categories (and/or are the historical legacies of those categories). These profound differences between folk racial categories do not reflect biological differences; they reflect a long history of racism and racialist thinking in the U.S. (See for



example Omi and Winant 1989, Appiah and Gutmann 1998, Nazroo 2003, Williams and Collins 2004). Nor do these folk racial categories themselves reflect biological differences—one is not labeled as “Black” in the U.S. because of the particular pattern of alleles one happens to have, nor even because of what continent one’s ancestors came from, but rather on the basis of superficial phenotypic characteristics (skin color primary among them) and social history. And being labeled “Black” has profound implications for how one will be treated, as well as for one’s life-prospects. Given this, it is not surprising that there is increasingly strong evidence pointing towards the experiences of racism itself, along with the structural inequalities associated with race, being the major cause of the Black-White health disparities in the U.S.

On this view, we can understand why controlling for SES and education reduces the health disparities between Black and White Americans but does not eliminate them. Because Black Americans are also systematically disadvantaged with respect to SES and education, and because SES and education are associated with health outcomes in the U.S., controlling for these reduces the disparities. However, controlling for SES and education does not eliminate the disparities because racism and the effects of racism extend beyond those standard measures of one’s life-prospects. Black and White Americans living at the same SES and with the same level of education yet have, on average, very different life experiences. Some of these are obvious and easy to recognize, such as the remarkable pattern of residential segregation that persists in the U.S. (and which was actively created and encouraged by U.S. government policies, through at least the 1950s; see Kaplan and Valls 2007, Williams and Collins 2004). Others, such as the (daily low-level and occasionally more dramatic) direct experiences of racism, are harder to quantify, but no less profound.

Indeed, it seems increasingly likely that the stresses associated with the experiences of racism (including residential segregation) might account for many of the health disparities that remain when SES and education are controlled for. These stresses extend from “active” discriminatory behavior (different treatment in shops, in the work place, in social situations, by the police including while driving, etc) to such stresses as the internalization of culturally dominant stereotypes (see e.g., Mays et al. 2007, 211 for discussion of one such possibility). More generally, Steele and Aronson’s work on “stereotype threat” points towards the ways in which internalized

stereotypes can influence, e.g., educational achievement in profound ways through relatively subtle mechanisms (see e.g., Steele and Aronson 1995, 2004). Tellingly, one of the proposed mechanisms through which stereotype threat works is stress, including raised blood pressure (see e.g., Osborne 2007, Blascovich et al. 2001).

The effect of these combinations of race-based stressors is to create what has been referred to as a high "allostatic load" (see Mays et al. 2007, Hogue and Bremner 2005, Fausto-Sterling 2004). Allostatic load has been defined as the "cumulative biological burden exacted on the body through attempts to adapt to life's demands" (Seeman et al. 2001), and a high allostatic load affects health through a variety of biological pathways, some of which are still poorly understood. But various measures of allostatic load (including perceived discrimination and racism, both chronic and acute) are associated with for example increased risk of hypertension and heart disease (see for example Din-Dzietham et al. 2003, Davis et al. 2005; see also Williams et al. 2003, Mays et al. 2007), increased risk of preterm birth and low birth weight (Giscombé and Lobel 2005; Hogue and Bremner 2005; see also Mays et al. 2007), and poor self-reported health (Borrell et al. 2006). Some of these, for example low birth weight and cardiovascular disease, are further associated with intergenerational effects; having been born preterm is associated not only with increased infant mortality and increased morbidity throughout life (Giscombé and Lobel 2005; Hogue and Bremner 2005), but with low birth weight and increased cardiovascular risk in subsequent generations as well (see Drake and Walker 2004).

In addition to factors directly attributable to the stresses of racism (high allostatic load), other "preventable" risk factors (tobacco use, alcohol use) are also associated with the stresses of racism, with the experience of racism increasing both the likelihood of using e.g., tobacco and the likelihood and severity of the health impacts of such use (Mays et al. 2007, Shields et al. 2005). So while "controlling" for e.g., tobacco use in studies of heart disease is common, doing so may in fact underestimate the effects of racism on Black Americans, if part of the effect of racism is on the likelihood and the style of tobacco use. Further, given that differences in SES and education are themselves the result of racism and the historical legacy of racism, it seems clear that controlling for the effects of SES and education will overcorrect and remove much of the significant disparities that are in fact caused by racism.

The upshot of this is that there *are* in fact important biological differences associated with folk racial categories in the U.S., but the causal arrow points not from biology to race, but rather from race to biology. Insofar as racism forms a long-standing and ubiquitous part of life in the U.S., folk racial categories will themselves reliably create biological differences. Indeed, these biological differences will be robust and will be reliably recreated to the extent that race persists as a major organizing concept in the U.S., and to the extent that racial differences in income, wealth, etc., are reliably recreated across generations. And in some cases, the very biological differences in health outcomes will have cross-generational effects, and be at least partially recreated even in the absence of the original environmental stressors (see Drake and Walker 2004 and cites therein).

This should serve to remind us that while differences in genotypes are one way in which phenotypic differences between populations can be reliably recreated across generations, they are not the *only* such way. Other pathways, not involving differences in genes, can just as reliably lead to different phenotypes, and be recreated across generations with roughly equal robustness (for more on ways in which development is reliably influenced by nongenetic factors, see for example Odling-Smee, Laland, and Feldman 2003, Oyama, Griffiths, and Gray 2001, and cites therein; Fausto-Sterling makes a similar point to this regarding the recreation of racial differences in the U.S. in her (2004) and (2008)). Finding biological differences between populations, even differences that are reliably recreated across generations, does *not* guarantee that there are genetic differences between the populations that *cause* the observed differences.

#### 4. *Ameliorating the Health Disparity:*

##### *Race-Specific Medicine or Reparations?*

Once it is accepted that race, as a social category, creates disease risks through racism itself, it becomes clear that race can be a medically useful category even if it is not a good proxy for population-level genetic differences. Race, as a socially defined, historically contingent way of categorizing humans, has its effects not via population level genetic differences, but by the very same social mechanisms that maintain race as an organizational category in contemporary society.

So for example, “race-specific” drugs and taking self-identified race into account in clinical practice *might* make sense, despite self-identified

race *not* being a good predictor of the presence of particular alleles. This reveals the mistake in Gannett's (2005) claim that, for example, a patient's self-reported or inferred race "matter only if there are sequences of DNA involved in the metabolism of the particular drug the clinician is contemplating prescribing that exhibit minimal overlap in their distribution between" regularly recognized folk racial groups (p. 1242). Gannett is certainly right that this is unlikely to be the case. But it is plausible that drugs that work on different causal pathways might have systematically different effects in Black and White Americans *because* the causal pathways responsible for disease states in Black and White Americans are sufficiently different, despite there not being genetic differences that influence (for example) the metabolism of the drugs in question. It may be, for example, that the dominant causes of heart disease in self-identified Black Americans are different from the dominant causes of heart disease in self-identified White Americans, because, for example, high allostatic load is causally associated with the former and not that latter. That is, race itself, as a social category, may well be a direct cause of the differences in the frequencies of different etiologies of diseases in Black and White Americans, and hence be reliably associated with the differential success of treatment regimes.

So while there is not yet good evidence that drugs that work on different causal pathways have systematically different effects in Black and White Americans (claims surrounding BiDil notwithstanding; see Kahn 2005, Sankar and Kahn 2005, Kahn and Sankar 2006), the possibility of such effects does not depend on whether there are sufficiently large *genetic* differences between self-identified Black and White Americans. Different disease etiologies might respond to different treatment protocols, and there is gathering evidence that, for example, heart disease and hypertension have different causal etiologies in Black and White Americans. Again, the high allostatic load of Black Americans, suffered throughout life, is thought to cause particular kinds of ongoing stresses (what Nazroo 2003 refers to as "weathering") that result in (statistically) early deaths; it is possible that White Americans who suffer from "the same" kinds of heart disease in fact come to have those conditions via very different pathways.

If the above is correct, then the hope that once genetic sequencing becomes cheap enough, race will fall out of individual medical decisions, to

be replaced with the more accurate genetic assessment of the individual, is misguided (see Root 2003, 1180; see also Lee 2005, Gannett 2003, Shields et al. 2005). Even if genetic testing of individuals yields some insights into drug choices and risk factors, folk racial categories will continue to contain information not encoded in genes—for example, how one is viewed in society, how one is likely treated on average, and the like. And if it is differences in how one is treated or viewed in society that make for different causal pathways in disease states, then genetics is *not* the right place to look.

For these reasons, taking self-identified race into account in medical decision making might make sense locally, given that self-identified race *is* a good predictor of one's experience with racial discrimination, prejudice, and racism more generally. The objection that to do so reifies race is, in essence, correct—self-identified race is *real* (it is socially contingent, but no less causally powerful for that) and, if the above is correct, it is also biological. But to be biological is not to be genetic, nor does biology make race. Race is biological because racism (and more generally a society organized by race) has profound biological effects.

But even if race *is* biological in this sense, it is still worth considering whether race-based medicine—the search for particular drugs or treatments that are particularly effective on say Black Americans—is the right direction in which to look for ameliorating the large and long-standing health disparities between Black and White Americans. Here, even if it proves *possible* to develop, e.g., drugs that more effectively treat the diseases that are caused by racism and the racial divisions in the U.S., the real problems—continued racism, massive residential segregation (and with it educational segregation and systematic differences in investment in infrastructure, etc.), income and wealth disparities, etc.—would remain, and would very likely continue to work against improved health for Black Americans. Fausto-Sterling suggests that there are good reasons to suspect that these kinds of “low-level” treatments will likely *fail* to have meaningful medical benefits even for most individual patients because (in part) such interventions cannot address the causes and ongoing effects of high allostatic loads (2004 esp. 28–30). But even if such treatments were marginally more successful for individual patients identified on the basis of race, it would remain perverse to treat the diseases caused by racism and the historical legacies of racist policies, but to leave those legacies in place.

In the end, it is likely that Williams and Collins (2004) are correct when they suggest that seriously addressing the health disparities between Black and White Americans will require programs to “rebuild the physical and economic infrastructure of disadvantaged Black communities” (994) (see also Williams and Jackson 2005, de Melo-Martin and Intemann 2007). Recognizing the profound biological effects that socially identified, historically contingent racial categories have might be one way to encourage calls for such programs to be taken seriously.

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