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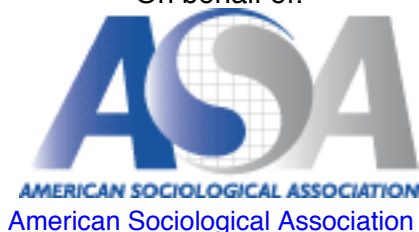
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The Genomic Challenge to the Social Construction of Race

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Abstract

Recent research on the human genome challenges the basic assumption that human races have no biological basis. In this article, we provide a theoretical synthesis that accepts the existence of genetic clusters consistent with certain racial classifications as well as the validity of the genomic research that has identified the clusters, without diminishing the social character of their context, meaning, production, or consequences. The first part of this article describes the social constructionist account of race as lacking biological reality, its main shortcomings, and our proposed solution: the concept of clinal classes. The second part discusses the character of the group differences that would be consistent with clinal classes and introduces the concept of genomic individualism, which extends an emerging model for understanding biosocial causation to include the genetic effects of ancestry. The third part develops the argument for a “bounded nature” reformulation of racial constructionism that reconceptualizes racial and ethnic categorization as the social perception of ancestry. The final part summarizes the article’s contributions and outlines implications for future research.

Keywords

Race, ethnicity, ancestry, genetics, genomics, social construction, sociology, racial formation

In *Racial Formation in the United States*, Michael Omi and Howard Winant (1994) observed that as a result of intellectual efforts since the start of the twentieth century and political efforts by racially defined groups, “we have now reached the point of fairly general agreement that race is not a biological given but rather a socially constructed way of differentiating human beings” (p. 65). In their original formulation, they advanced their theory of racial formation “to refer to the process by which social, economic, and political forces determine the content and importance of racial categories, and by which [those forces] are in turn shaped by racial meanings” (Omi and Winant 1986:61). Later they reconceptualized racial formation as “the sociohistorical process by which racial categories are created, inhabited, transformed, and destroyed” (Omi and Winant 1994:55), placing more emphasis on the construction of racial categories than on their social consequences.

Although the idea of race as a social construction predates Omi and Winant’s (1986) first edition by almost a century, their book has arguably become its dominant theoretical statement. This occurred in part

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because their theory successfully supplements (1) the social constructionist refutation of race as possessing any biological reality with (2) a detailed account of race as possessing a social reality independent of its entanglements with ethnicity, social class, and nation. Their theory of racial formation delineates the social processes particular to race, developing the idea of racial constructionism beyond the general approach of social constructionism, which “rejects any category that sets forward essential or core features as the unique property of a collective’s members [and treats] every collective [as] a social artifact—an entity molded, refabricated, and mobilized in accord with reigning cultural scripts and centers of power” (Cerulo 1997:387). Expansive in its conceptualization, the theory of racial formation has provided a unifying framework for myriad studies that document how geography, history, and politics shape the salience, meaning, social rules, and boundaries of racial categories (e.g., in sociology alone, see Almaguer 1994; Bonilla-Silva 1997; Kim 2008; Lie 2001; Saito 1998; Shiao 2005; Telles 2004). We suggest, however, that these many fine contributions are premised on, and unnecessarily burdened with, a conception of human biological variation that is out of step with recent advances in genetic research.

In our view, recent research on the human genome challenges the basic assumption that human races have no biological basis (Abraham 2006; Leroi 2005; Risch et al. 2002; Rosenberg et al. 2002; Wade 2006). The increasing power of computer assisted quantitative data analysis and the growing resolution of available genetic data has enabled quantitative geneticists to identify an empirical structure within human genetic variation that at a certain scale resembles the continentally based racial classifications of the U.S. federal government. Many proponents of these new advances, mostly nongeneticists, frame them as challenges to social constructionist theories of not only race but also gender, sexuality, and even other phenomena, such as ethnocentrism, violence, and child rearing (Hsu 2007; Khan 2007; Pinker 2002; Salier 2006). These challenges form a broad critique of what their proponents perceive to be a “pronurture” bias that dominates the mainstream of contemporary academia. They recognize this bias as rooted in an understandable reaction against the historical complicity of the biological sciences in justifying racism, sexism, and ethnocentric “chauvinism” (Pinker 2002:14), but they also regard this complicity and the need for its correction to be largely matters of the past. Instead, the proponents of the new advances argue that the time has come for a bolder examination of the possible biological basis of human variations that many academics assume to be entirely social in both origin and character. In other words, they argue that the time for fear of biological realities, even in relation to the thorny issues surrounding race, has passed.

Others, however, have criticized these arguments as constituting a new biological determinism (Duster 2005, 2006a; Social Science Research Council 2006; Weiss and Buchanan 2006). Specifically, these critics charge that this new intellectual current (1) exaggerates the degree of consensus among natural scientists about recent research (Dunklee, Reardon, and Wentworth 2006; Lewontin 2006; Morning 2009); (2) incorrectly presumes the absence of racial assumptions and beliefs among natural scientists and in scientific practice (Duster 2006a; Epstein 2008; Fullwiley 2008); (3) minimizes alternative explanations based on social, cultural, and historical factors (S. Lee, Mountain, and Koenig 2001); (4) overlooks the complexity of the biological mechanisms and biosocial interactions mediating gene expression and social process (Duster 2005; Fujimura, Duster, and Rajagopalan 2008; Hubbard 2006; Ossorio and Duster 2005); (5) glosses the cultural baggage associated with the idea of race and how it has distorted the study of human biological variation (Fausto-Sterling 2008; Goodman 2006); and (6) evidences a negligent disregard for the social reception of its pronouncements about the “reality” of racial biology (Cho and Sankar 2004; Kaufman 2006; Stevens 2006). In our view, these criticisms provide an effective response to the “less nurture” critique of the proponents, but they fail to provide a reformulation of racial constructionism that would be consistent with the new advances.

In this article, we provide a theoretical synthesis that accepts the existence of genetic clusters consistent with certain racial classifications as well as the validity of the genomic research that has identified the clusters, without diminishing the social character of their context, meaning, production, or consequences. We argue that the recent research in genetics demonstrates that certain racial, and also ethnic, categories have a biological basis in statistically discernible clusters of alleles rather than in the traditional notions of

human races as arising from categorically distinct ancestries or as possessing categorically unique essences (Marks 2006; Spickard 1992). The first part of this article describes the social constructionist account of race as lacking biological reality, its main shortcomings, and our proposed solution: the concept of clinal classes. In brief, clinal classes are the lumps in otherwise continuous genetic variation, similar to social classes in otherwise continuous economic variation. The second part discusses the character of the group differences that would be consistent with clinal classes and introduces the concept of genomic individualism, which extends an emerging model for understanding biosocial causation (Freeze 2008) to include the genetic effects of ancestry. The third part develops the argument for a bounded-nature reformulation of racial constructionism that reconceptualizes racial and ethnic categorization as the social perception of ancestry. The final part summarizes the article's contributions and outlines implications for future research.

THE PROBLEMS OF RACIAL SOCIAL CONSTRUCTIONISM AND THE SOLUTION OF CLINAL CLASSES

We introduce our main conceptual intervention of clinal classes by first summarizing the current biological foundation of the theory of race as a social construction. We then explain its shortcomings especially in contrast with recent research on the human genome. Third, we formally define our solution of clinal classes and further discuss recent genetic research to explain how they provide a biological basis for racial and ethnic categories. In brief, we recommend the removal of obsolete claims about human biology from racial constructionism.

Humanistic and social science scholarship on race/ethnicity generally assumes that race has, at most, a superficial biological reality. This assumption is supported by the often-cited research of Harvard geneticist Richard Lewontin comparing the degree of human genetic variation that exists within groups with the variation that exists between groups. He found that while more variation on any given gene exists between "classically defined" racial categories than between national or linguistic groups within the same race (10 percent vs. 5 percent), the largest fraction of human variation, 85 percent, exists within the local populations (Lewontin 1972, 2006). In other words, there is more genetic difference between French and Vietnamese than between French and Germans, but both differences are dwarfed by the variation among the French alone.

In addition, his contemporaries in what we might now term modern biology found that there was more genetic variation within the continent of Africa than in the rest of the globe combined (Cavalli-Sforza, Menossi, and Piazza 1994; Diamond 1994). These findings substantiated the "out-of-Africa" thesis that *Homo sapiens* evolved on that continent, diverging from chimpanzees roughly 5 million years ago, before a subset departed, roughly 50,000 years ago, and ultimately populated the remaining continents. The resulting populations lived, and mated, in relative isolation until the start of mass migrations associated with the rise of world systems in the past 2,000 years (Abu-Lughod 1989). Thus the rise of the morphological differences (e.g., in skin color, hair form, facial features, etc.) that are associated with racial and ethnic categories is relatively recent in the evolutionary history of the human species.

According to modern biology, this history accounts for (1) Lewontin's finding that interracial genetic variation is greater than intraracial variation yet much smaller than intraethnic variation; (2) the nonconcordance of racial categories with individual psychology, behavior, or capabilities; and (3) the nonconcordance of racial categories with even internal biology, for example, lactose tolerance and antimalarial resistance. Furthermore, anthropologists observed that morphological differences among humans do not "jump" from category to category but are instead clinal, meaning that they show incremental change correlated with distance (Goodman 2006; Marks 1995, 2006; Ossorio and Duster 2005). *Clinal variation*, or *clines*, refers to the gradual changes in genetic or phenotypic variation within a species over a geographic area. Therefore, human populations appear dramatically different from each other only when the geographically intermediate populations have been excluded from consideration (Graves 2004; S. Lee 2005). In sum, modern biology refutes the traditional conception of races as biological categories that are "discrete, non-overlapping, discontinuous, [and defined by] racial genes" (Sesardic 2010:147) by arguing that

human populations are instead characterized primarily by genetic similarity due to millions of years of evolution in Africa and secondarily by clinal variation due to only thousands of years of relative isolation across the Earth.

Relying on modern biology's refutation of racial categories, contemporary social theory conceptualizes race as a purely social construct that is both maintained and contested through a sociohistorical process that involves politics and other social institutions (Omi and Winant 1994; Saito 1998; Shiao 2005) and that results in historically and nationally different racial classification systems (Almaguer 1994; Lie 2001; Telles 2004). However, there are several shortcomings to this refutation, especially in light of recent genetic research. The first and core problem is the reduction of a biological basis for racial/ethnic categories to the existence of categorical differences between groups. As the philosopher Neven Sesardic (2010) has noted in his critique of racial constructionism, this criterion for a biological basis is "so unrealistically demanding that . . . even the species concept would fail to pass muster" (p. 147). Arguably, the origin of the essentialist criterion for biological differences lies less in actual science than in its use in the historical justifications for the categorical exclusion of nonwhites from political, economic, social, and cultural citizenship in the United States. By contrast, biological science does not require the white supremacist belief in species-level, much less greater, differences between human subspecies.

Second, it is now possible to statistically distinguish human subspecies that overlap on many biological characteristics. The methods for genetic research have advanced considerably since the original research of Lewontin and his contemporaries, for whom DNA sequencing was not an available technology. Kittles and Weiss's (2003) historical overview of these methods evokes the scope of their progression:

Genetic studies of group and regional variation began . . . with the discovery in 1901 by Karl Landsteiner of the ABO blood group system. . . . In subsequent decades, various blood types and assorted Mendelian traits were added to the repertoire, but the genetic floodgates opened in the 1950s with the addition of protein electrophoretic polymorphisms and the hypervariable human leukocyte antigens (HLA) system. Subsequently, the data were augmented by restriction fragment length polymorphisms (RFLPs), mitochondrial DNA (mtDNA) sequence variation, to today's outpouring of single nucleotide polymorphisms (SNPs), tandem repeats (e.g. microsatellites), distributed repeats (e.g., Alu elements), and the like. (p. 36)

In addition to the advent and continuing advancement of DNA sequencing, the increasing speed of computer microprocessors has magnified the scale of genetic analysis, expanding the resolution and power of genetic analysis from the isolated examination of individual genes to the simultaneous examination of an ever-increasing number of alleles or variants at given DNA loci or locations. Whereas modern biology was largely limited to examining one-dimensional distributions of a limited number of genes, that is, the protein-coding sequences of DNA, current science can examine high-dimensional correlations among distributions of base pairs irrespective of their known functions, including the so-called junk DNA, which constitutes over 98 percent of the human genome. Indeed, 80 percent of the human genome, including much of the noncoding DNA, shows signs of being expressed (Pennisi 2007).

Sesardic (2010:149) provides a useful figure, replicated here, that illustrates what is now called "Lewontin's fallacy," or the mistake of reducing the validity of genetic classification to the average degree of variation on individual genes instead of also considering their correlation (Edwards 2003). In Figure 1, there is no way to divide either the y -axis or the x -axis in the graph so that all of the triangles are either above or to the right of all of the squares, respectively. In addition, there is more variation on the y -axis among the triangles or among the squares than there is between the triangles and squares; the same is true on the x -axis. Lewontin's analysis of averaging the degrees of variation on individual genes amounts to trying to separate the triangles and squares by drawing either horizontal lines or vertical lines through the graph. To consider the correlation between y and x , one would instead try to draw diagonal lines, with which, as the figure illustrates, it is possible to separate the triangles and squares. To be clear, there are no

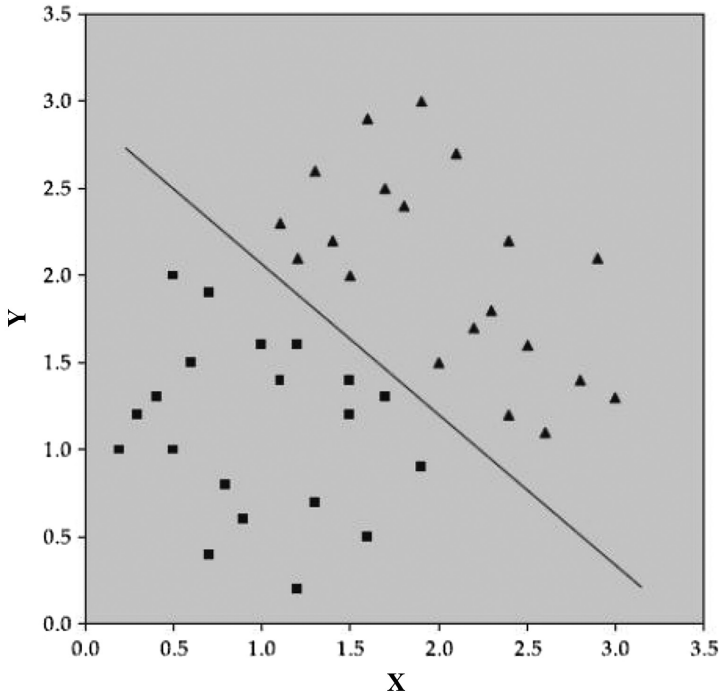


Figure 1. Clusters emerging in a two-dimensional perspective
 Source: Reprinted by permission from Sesardic (2010:149).

two DNA loci whose correlation would separate any two human populations; instead, geneticists have found that roughly 60 to 150 randomly selected loci are needed to separate individuals into genetic clusters that are homologous to continentally based racial categories, that is, the equivalent of a 60- to 150-plus-dimensional “graph” (Rosenberg et al. 2005). Nevertheless, as Risch et al. (2002) contend, “genetic differentiation is greatest when defined on a continental basis . . . irrespective of the type of genetic markers employed, be they classical systems, restriction fragment length polymorphism (RFLPs), microsatellites, or single nucleotide polymorphisms (SNPs)” (p. 3).

Third, membership in these statistically identified genetic clusters appears to be concordant with at least one individual characteristic: racial/ethnic self-identification (Guo et al. 2010; Risch et al. 2002; Rosenberg et al. 2002; Tang et al. 2005). More precisely, individuals’ sampling locations and/or responses to survey questions about their racial/ethnic ancestry are homologous with the statistical clusters that emerge from treating individual genomes as points in high-dimensional space and calculating their Euclidean distances from each other (Hsu 2007). Specifically, the rate of “discordance” between cluster assignment and self-reported racial/ethnic ancestry appears to be less than 1 percent, albeit with the revealing exception of ancestries that developed at intercontinental boundaries, for example, Ethiopians, or that formed during the rise of world systems in the past two millennia, for example, Hispanics and African Americans. In fact, Sesardic (2010) pointedly notes that in one major study, the rate of discordance between chromosomal and self-reported sex was larger than that for genetic cluster and self-reported race/ethnicity.

On the one hand, recent genetic research has confirmed the primary findings of modern biology that humans have overwhelming genetic similarities with each other regardless of their membership in racial or ethnic categories (Kittles and Weiss 2003) and that humans evolved in Africa before populating the globe through more recent migrations (Wells 2002). On the other hand, recent research has also found that human genetic variation is composed of not only clines but also clusters that are homologous to racial and

ethnic categories. We believe these findings strongly suggest replacing the refutation of biology in racial constructionism with a version of the feminist distinction between biological sex and socially constructed gender (Rubin 1975).

Accordingly, we propose to conceptualize the biological counterpart to socially constructed race/ethnicity in terms of clinal classes, as analogous to social classes in the long-standing debate within social stratification research over whether variations in individual economic status are discrete or continuous; that is, do social classes exist? Just as Kalleberg, Reskin, and Hudson (2000) argue that economic status is continuous but statistically lumpy, we argue that human genetic variation is clinal but statistically clustered (Rosenberg et al. 2005).¹ Rhetorically, the concept of clinal classes provides a way to conceptualize the lumpy structure of human genetic variation beyond the often categorical connotations of race and the similarly categorical meaninglessness associated with clines. Relative to clines, clinal classes are a complementary measurement of ancestry in terms of how both physical geography and mating restrictions have produced clusters in human genetic variation with direct consequences for individual morphology and psychology and second-generation consequences for social processes, such as racial formation and stratification, which potentially feed back on morphology, psychology, mating practices, and social geography.

Relative to the traditional conception of races refuted by modern biology, clinal classes possess a biological reality more consistent with recent genetic research: statistical, scalable, and not deterministic. As already noted, the concept of subspecies, human or otherwise, does not require categorical differences between populations. Instead, clinal classes assume a common evolutionary history, possess extensive genetic similarities, and coexist with clinal variations both within each class and across classes.²

Not only do clinal classes possess a statistical character, but they are also identified through still-improving statistical procedures. The primary tools for identifying population structure have been, in order of emergence, (1) comparisons of predefined populations, (2) the Bayesian clustering approach of the program STRUCTURE developed by Pritchard, Stephens, and Donnelly (2000), and (3) new variations on the classical technique of principal components analysis (Paschou et al. 2007). Although social studies of science continue to criticize the circular research design of the first method (Bolnick 2008; Duster 2006b; Marks 2006), the second and third methods have been preferred among academic researchers for over a decade (Risch et al. 2002; Rosenberg et al. 2002). The newer tools allow researchers to treat their genomic samples as containing "cryptic" population structures, that is, ignoring information about ethnic affiliations and ancestry (Wilson et al. 2001); to use randomly selected DNA loci to identify clusters; and then to select the loci that best separate the clusters to serve as ancestry informative markers (AIMs) that are more valid than the "loaded" AIMs produced from the comparison of predefined populations. In brief, these tools amount to a race-neutral approach of "letting the data define the groups rather than starting with predefined categories" (Fausto-Sterling 2008:660).

Additionally, these methods do not require the assignment of individual genomes to single clusters even during the stage of cluster identification; instead, they simultaneously estimate each individual's degree of admixture from each cluster. To be clear, these estimates of individual admixture are based on calculating probabilities, not inventorying ancestors. However, as in other forms of quantitative analysis, researchers can combine individual estimates into more reliable aggregate statistics, such as a mean 17 percent "white" admixture among African Americans and a mean 39 percent "Native American" admixture among South-west Hispanics (Risch et al. 2002).

Second, clinal classes are empirically scalable measurements of ancestry, in contrast with the inherently ad hoc answers that traditional conceptions of racial biology give to the question of "how many groups exist?" The program STRUCTURE requires researchers to specify the number of clusters (K) to be identified, requiring researchers to repeat runs of the program across different K and to evaluate those runs with statistical estimates of confidence and reliability. In fact, focusing on intracontinental variation in Africa (Tishkoff et al. 2009), East Asia (Jung et al. 2010), and Europe (Drineas, Lewis, and Paschou 2010; Novembre et al. 2009; Tian, Gregersen, and Seldin 2008), researchers have identified many clusters below the scale of continents. Thus far, regardless of the number of clusters selected, typically, $K = 1$ to $K = 14$,

Rosenberg et al.'s (2002) observation has held true: "Each increase in K split one of the clusters obtained with the previous value" (p. 2382). Therefore, rather than comprising competing classifications, genetic clusters identified at different Ks are hierarchically related to each other.

Unlike the traditional conception of race, the concept of clinal class does not require a fixed number of classifications, as the number of classes depends on the attempted resolution for detecting population structure, independently of the degree of clustering that divides the classes (Bamshad et al. 2003). Put another way, a clinal class is like a watershed, "the area of land where all of the precipitation drains to a common water body . . . determined by the shape of the land around them" (Network of Oregon Watershed Councils 2011), wherein both can be defined at the scale of a large classification, such as the Mississippi River watershed or Africa, or at the scale of a subclassification, such as the Missouri watershed or the southern African Koesan. Like watersheds, clinal classes are nested within and divided from each other in ways that reflect their emergence in physically bounded geographies.³ To assess the possibility that genetic clusters are artifacts of unevenly sampled clines, Rosenberg et al. (2005) constructed a measure of "clusteredness," or the probability of assigning an individual genome to a single cluster versus equal assignment to all clusters. They find that far from serving as crude measurements of clines, the clusters indeed result from "small discontinuous jumps in genetic distance-across oceans, the Himalayas, and the Sahara" (Rosenberg et al. 2005:668).

Last, although clinal classes provide a biological basis for racial and ethnic categories, they do not determine their social consequences, much less the broader sociology of race/ethnicity, that is, the "process by which social, economic, and political forces determine the content and importance of racial categories, and by which [those forces] are in turn shaped by racial meanings" (Omi and Winant 1986:61), or even the process by which select clinal classes, and not others, become defined as salient racial/ethnic categories. Instead, clinal classes simply summarize a potential input to the social definition of racial/ethnic categories: the geographic distribution of humans in genetic watersheds over the past 50,000 years.

In sum, individuals who share cluster membership do not all share any characteristics that are absent from all individuals outside the cluster in question. Instead, the population of the cluster possesses a unique combination of genetic characteristics, and individual members vary in their possession of any given characteristic. Furthermore, these characteristics are primarily markers of ancestry rather than genes, much less Mendelian traits. Nevertheless, their clustering demonstrates the incompleteness of the position that human genetic variation is solely clinal and therefore provides no empirical basis for racial and ethnic categories. Having advanced an alternative to the assumption of biological nonreality in racial constructionism, we now turn to refining its position that the social, cultural, and historical bases of race are the sole determinants of group differences.

GROUP DIFFERENCES AND THEIR IMPLICATIONS FOR BIOSOCIAL CAUSATION

In this section, we explain the character of the partially heritable group differences that would be consistent with clinal classes by first clarifying the public statements about group differences made by two proponents of recent genetic research. Next, building on Jeremy Freese's (2008) recent model of biosocial causation, we add the concept of genomic individualism to introduce the genetic effects of ancestry. Third, we describe the specific individual characteristics that might show average differences between clinal classes and discuss a common procedure in genetic research that supports the plausibility of their existence. In brief, we recommend that sociologists incorporate the possibility of nondeterministic genetic effects into their theories of race and ethnicity.

In 2006, *New York Times* science reporter Nicholas Wade and Harvard psychologist Stephen Pinker made comments about recent genetic research that illustrate the difficulty of describing the implications for group differences without lapsing into a language of categorical differences.

The fact that different races or ethnic groups tend to excel at different sports—Africans at track, Chinese at ping pong, Europeans at weightlifting—is not proof in itself of any genetic component but just a starting point that hints at possible genes to look for. (Wade 2006:197)

What I predict will become the dangerous idea of the next decade [is] that groups of people may differ genetically in their average talents and temperaments. (Pinker 2006:3)

Their comments suggest that genetic researchers will soon find genes for racial performance in different sports and a genetic basis for group differences in ability and personality. On the surface, they appear to predict the scientific confirmation of traditional notions of racial biology. However, in the context of the authors' respective arguments, they actually predict that geneticists will find morphological and psychological differences that are neither categorical nor self-explanatory. Wade (2006) prefaces his comment by suggesting that contemporary international sports events are practically necessary for observing the slight differences between racial/ethnic groups. Using the example of height, he makes the analogy that

most members of a population are of average height, very few are of dwarf or giant stature. . . . The difference [between groups] might hardly be noticeable in comparing average members of each population. But if you hold a competition for the ten tallest people . . . in this case it is the extreme, not the average, that is being compared. (Wade 2006:197)

In brief, the outcomes of sporting events in the more egalitarian present magnify the small differences in the mostly overlapping distributions of individual ability.

By comparison, Pinker's (2006) use of the word *average* in both his above comment and its broader context is more precise: "Group differences, when they exist, pertain to the average or variance of a statistical distribution, rather than to individual men and women" (p. 3). Nevertheless, he admits that "the underlying fear, that reports of group differences will fuel bigotry, is not groundless" (Pinker 2006:3). In his influential book popularizing "the new sciences of human nature" (Pinker 2002:xi), he argues that discrimination premised on empirical, even biological, group differences still violates the principle of equality that "condemns judging an individual according to the average traits of certain groups to which the individual belongs" (p. 145). Put another way, what makes the idea of genetically based group differences "dangerous" is the tendency to exaggerate their significance and interpret them in ways that simply confirm traditional racial attitudes.

For these proponents of recent genetic research, the critical distinction between their conception of group differences and traditional notions of racial biology is that the unit of genetic influence is not the racial or ethnic group but, rather, the individual. In our terms, their racial/ethnic groups have genetic significance when they are homologous to clinal classes, membership in which is associated with an increased likelihood of inheriting the combination of alleles that were more prevalent in particular ancestral geographies. For Wade, therefore, there is a high probability both that scientists will identify clinal classes homologous to contemporary West Africa and that the distinguishing clusters might include alleles that influence the distribution of athletic abilities advantageous for sprinting such that a higher-than-average percentage of class members would show an extreme level of sprinting ability. Similarly, Pinker's comments suggest both that scientists will identify clinal classes homologous to contemporary groups and that the distinguishing clusters might include alleles that influence the distribution of psychological traits, again, such that a higher-than-average percentage of class members would show extreme levels of certain cognitive characteristics. However, for a given individual who identifies with a racial/ethnic category homologous with the relevant clinal class, for example, West Africa, there would be no guarantee of inheriting the alleles influencing the relevant characteristics, for example, athletic abilities, much less of becoming part of the percentage that demonstrates extreme performances, for example, in sprinting competitions. In brief, the character of group differences is that clinal classes may vary in the frequency of certain alleles, which means in turn that every clinal class likely includes individuals with every allele variant, albeit in

different proportions. We call this nondeterministic influence of genetic ancestry *genomic individualism*: the strict mediation of ancestral genomes by individual genomes.

Our concept extends backward in time Jeremy Freese's (2008) foundational concept of the phenotypic bottleneck, "the strict mediation of genetic causes by the phenotype" (p. S13). In Freese's counterfactualist evaluation of the role of genes in sociological explanations, he distinguishes the "whole genome effect" of switching out an individual's entire genome for an alternative genome from the "specific gene effect" of switching out a small part of an individual's genome for some alternative part. Focusing on the whole genome allows him to focus on the implications of heritability in aggregate rather than the heritability of specific characteristics. He posits,

The total effect of the whole genome includes all causal paths from $G_i \rightarrow P_i \rightarrow E_i \rightarrow y_i$, including whatever complicated chains of sequences might unfold over time of environmental effects on phenotype ($E_i \rightarrow P_i$) and phenotype effects on environments ($P_i \rightarrow E_i$). (Freese 2008:S9)

In this model, G_i is a vector comprising "the genome of person i at biographical time $t = 0$ (i.e. 'conception') with originating environment $E_{i, t=0}$ " (Freese 2008:S6).

P_i refers to "the phenotypic characteristics of person i ," by which he means "the relatively restrictive sense of characteristics that are embodied [or] materially realized as part of the organism, [i.e., their] height, personality, and skills [but not] their earnings, marital status, and spouse's educational attainment" (Freese 2008:S8).

E_i refers to the environmental characteristics of person i , which he subdivides into (1) actions, including recurring behaviors; (2) actors' internal states or psychology; and (3) actors' immediate circumstances conceptualized as not only locations in social structure, for example, earnings, marriages, and family resources, but also the results of earlier iterations of individual actions evoking social responses that are at least partially exogenous.

Last, y_i refers to the phenotypic or environmental characteristics of person i that serve as the explanandum or phenomenon to be explained in a given causal explanation. As suggested above, these explanations are actually paths in a broader causal cycle that moves from $t = 0$ toward $t =$ death of person i . Accordingly, components of P_i and E_i take turns serving as y_i , with the result that P_i and E_i are reciprocally and iteratively reconstructed over biographical time.

This model of biosocial causation highlights how genetic effects occur solely through the body and thus cause y_i only insofar as they explain (1) the original phenotype, that is, at time $t = 0$, and (2) changes to those embodied characteristics over time in response to changes in their immediate circumstances. The bottleneck is that the effect of G_i on y_i can be estimated but cannot be explained without a narrative that articulates the role of phenotypes in mediating genetic effects.

To this model, we add another vector and a second bottleneck: $A_i \rightarrow G_i \rightarrow P_i \rightarrow E_i \rightarrow y_i$, where the new vector A_i refers to the genetic ancestry of person i , that is, the genomes of person i 's ancestors, which represent the "menu" of alleles from which G_i is selected through inheritance. Thus far, the model does not mention the geographic structure of ancestry, much less distinguish between the clinal claims of modern biology and our clinal class amendment. We connect A_i to geography by defining genetic ancestry as the sum of the genomes of ancestors that lived in specific places in specific historical times: $A_i = \sum \sum A_{ph}$, where p refers to place and h refers to historical time. The counterfactual conception of the effect of genetic ancestry would therefore involve the hypothetical reassignment of some A_{ph} within A_i and comparing the distribution of actual, observed G^a to informed expectations about the distribution of G that would have been observed had the reassignment of some A_{ph} within A_i occurred instead, that is, G^c . In the language of whole and specific effects, these hypothetical reassignments are thus "specific ancestry effects," referring to the reassignment of a small part of A_i , rather than a "whole ancestry effect," which would amount to a transspecies reassignment.

Although the possible counterfactual comparisons include historical and historical-spatial reassignments, the relevant comparison here involves the spatial-only reassignment from p^a , the actual, observed

place where person i 's ancestors lived, to p^c , the desired comparison place at the same historical time, that is, comparing $A_{p(a),h}$ and $A_{p(c),h}$. In the clines-only version of geographic ancestry, the change in A_{ph} , specifically, $A_{p(a),h} - A_{p(c),h}$, is a function of the linear distance D between p^a and p^c , whereas in the clines-plus-clinal classes version that we propose, the change in A_{ph} is a function of not only the linear distance D but also the nonlinear distance R between the respective genetic watersheds surrounding p^a and p^c , where the scale of the watersheds depends on the desired resolution for genetic structure, indexed by the number of watersheds K , to use the STRUCTURE notation. In the latter model, when p^a and p^c are in the same watershed or clinal class, then $R = 0$, and the change in A_{ph} becomes a purely clinal function.

This conception has several implications. First, both clines and clinal classes have potential genetic effects; therefore, the difference between a purely clinal and an also classed measurement of ancestry is not a difference between meaningless and meaningful genetic variation but rather two forms of potentially significant variation. Second, the effect of A_i on y_i , regardless of whether variations in A are only clinal, $A_{p(a),h} - A_{p(c),h} = f(D^{a:c})$, or also include clinal classes, $A_{p(a),h} - A_{p(c),h} = f(D^{a:c}, R^{a:c})$, occurs solely through the individual genome, that is, A_i causes y_i only insofar as it explains G_i , hence the second bottleneck of genomic individualism. Third, the difference between clinal class and statistical cluster is that the class is a theoretical concept, positing the existence of genetic watersheds, with relationships to other concepts, like clines and racial/ethnic categories, whereas the cluster is the output of applying quantitative procedures, for example, STRUCTURE's Boolean analysis, to genetic data. Fourth, although the identification of statistical clusters confirms the existence of clinal classes and provides estimates of admixture and thereby also estimates of $A_i \rightarrow G_i$, it does not provide an estimate of $G_i \rightarrow P_i$. In other words, the existence of clinal classes does not by itself confirm any specific morphological or psychological differences in P_i . Therefore, extending the phenotypic bottleneck, the effect of A_i on y_i can be estimated but cannot be explained without a narrative that articulates the roles of both phenotypes and individual genomes in mediating genetic effects.

Regarding the role of phenotypes in mediating the effects of genomes on individuals, Freese (2008:S11) provides a table indicating that the scope of individual outcomes for which researchers have identified substantial heritabilities has become extremely large. That said, this list of 50 outcomes includes both characteristics that fulfill his definition of phenotype (e.g., social skills) and other characteristics that are phrased in ways that qualify as social responses to phenotypes (e.g., occupational attainment). More importantly, we observe that these partly heritable characteristics indicate three primary dimensions of phenotypes—morphology, personality, and cognitive ability—plus a residual set of arguably secondary characteristics that combine a primary dimension with a specific environmental component (e.g., political party affiliation as a combination of personality and political opportunity structure). Within Freese's table, we conceive of age at first intercourse, athletic activities, diabetes, eating breakfast, homosexuality, gender identity disorder, low birth weight, and obesity as primarily indicating the morphological dimension. We classify as primarily indicating the personality dimension the characteristics of aggressive behavior, agreeableness, altruism, antisocial behavior, behavioral inhibition, conditionability, conscientiousness, coping styles, depression, extraversion, impulsivity, leadership emergence, loneliness, neuroticism, openness to experience, parenting behavior, perfectionism, self-esteem, sensation seeking, and social skills. We classify in the cognitive ability dimension the eponymous characteristic of cognitive ability along with the secondary but associated characteristics of college plans, educational attainment, occupational attainment, reading books, and school performance. Last, we classify in the residual set of secondary characteristics abortion attitudes, alcoholism, astrology attitudes, church attendance, criminal behavior, death penalty support, divorce, earnings, electoral participation, illicit drug use, immigration attitudes, modern art acceptance, patriotism, political party affiliation, religious fundamentalism, and smoking.

In brief, Freese (2008) argues that researchers have established ubiquitous partial heritability for many individual characteristics and that social scientists' "default expectation [regarding purely social explanations] should be biased estimates" (p. S19). Furthermore, he suggests that sociologists should regard themselves as fortunate if unmeasured genetic effects prove to be either independent additive causes to

purely social explanations or distal causes mediated by measured social variables rather than confounding causes of major independent and dependent variables. Last, he observes that given the nature of these characteristics, their challenge to sociology is “really less a challenge from biology [than] from psychology” (Freese 2008:S14).

Regarding the role of genomes in mediating the effects of ancestry on these largely psychological characteristics, we admit that presently there is an absence of direct evidence for clinal class differences in the distribution of individual outcomes with substantial heritability. Even if researchers document correlations between clinal classes and individual characteristics, these would not illuminate the within-case process (Brady and Collier 2004) or the general sequence of social events or processes (Gross 2009) that connect a specific G_x with a specific y_x . Instead, following Freese (2008), we argue that these potential ancestral effects should be regarded as conceptual “*placeholder[s]*,” indicating our lack of understanding of what about [genetic ancestry] is responsible for the unresolved effects [emphasis ours]” (p. S14). It is an empirical question whether the structure of human genetic variation includes only markers of ancestry or also material that shows signs of being expressed. However, there is a common component of quantitative genetic research that makes their existence plausible.

When geneticists seek to identify the specific alleles associated with certain medical conditions, they compare “cases” or samples that evidence the condition in question with “control” samples that do not evidence the condition. To avoid spurious findings, geneticists control for population stratification, which “refers to differences in allele frequencies between cases and controls due to systematic differences in ancestry rather than association of genes with disease” (Freedman et al. 2004:388). Without controlling for population stratification, case-control studies risk mistaking alleles that are simply markers for ancestry for the alleles that are actually associated with medical conditions, because the frequency of the causal alleles empirically varies across different ancestries. In other words, researchers routinely control for group differences in the frequency of the alleles that they seek to identify as genetic causes (Pritchard and Rosenberg 1999). Indeed, the first step in controlling for population stratification is to identify the ancestral groups that potentially vary on the alleles in question, and for accomplishing this step, the use of STRUCTURE remains a respected method (Price et al. 2010). To be clear, it remains an empirical question whether there is ancestral variation within those individual characteristics that are of particular interest to social scientists. In sum, the research documenting ubiquitous partial heritability supports the existence of nondeterministic genetic effects, whereas the practice of controlling for population stratification suggests that those genetic effects include group differences in the frequency of alleles associated with the heritability of morphological, personality, and cognitive characteristics among individuals. We now turn to reformulating racial constructionism to accommodate clinal classes, ubiquitous partial heritability, and the possibility of clinal class differences.

TOWARD A BOUNDED-NATURE ALTERNATIVE TO PURE CONSTRUCTIONISM

In this section, we outline our bounded-nature reformulation of racial constructionism by first distinguishing the levels of social process that are more and less directly affected by genetic effects. We then propose to conceptualize racial and ethnic categorization as the social perception of biological ancestry, which in turn influences the mating restrictions that maintain clinal classes. Last, we clarify the relationships between clinal classes and racial/ethnic categories by outlining distinct modes of biosocial causation illustrated with reinterpretations of recent research in the sociology of race/ethnicity. In brief, we recommend that sociologists theoretically embed the genetic effects of ancestry in causal processes that include but extend beyond persons.

Genomic individualism suggests the level of social process that is most directly affected by genetic effects. Following Arthur Stinchcombe’s (2005) conception of causal units, that is, the units of analysis that are “sufficiently unified, sufficiently bounded, to turn causes into effects” (p. 149), we observe that genetic ancestry directly affects those social explanations that rely exclusively on persons as causal units in contrast

with explanations that rely on variations in situations, networks, organizations, and the social processes of cultural production, distribution, and salience. That said, genetic ancestry may have important implications for the latter explanations when they rely on persons either as units of observation, typically as dependent variables, or as mechanisms for the causation attributed to the higher-level units. For example, select persons perform the roles defined in situations, direct the flow of information in networks, establish the rules in organizations, and produce, share, interpret, and use symbols in cultural processes. In sum, social explanations that rely on persons as their causal units are directly vulnerable to the criticism of missing unmeasured genetic effects, whereas explanations that also, or solely, rely on situations, networks, organizations, and cultural processes are less vulnerable.

Even at the person level, sociologists have demonstrated that racial/ethnic categories matter for social psychology in ways that cannot be reduced to group differences in morphology, personality, and cognitive ability. In particular, persons can possess different attitudes about particular racial/ethnic categories, that is, group stereotypes, which can become salient in interactions between differently categorized persons and thereby create situations of not simply interpersonal but intergroup relations. According to Pinker (2002), psychologists have found that persons “have no trouble overriding a stereotype when they have good information about an individual” (p. 204) so long as these persons have had sufficient contact with the other individual’s group and also belong themselves to a group that is not in conflict with the other group. Put another way, under certain conditions, persons’ attitudes about different groups can be accurate generalizations about group difference that do not raise the salience of race and ethnicity in interpersonal interactions. Unfortunately, two of the most persistent conditions for racial/ethnic relations in the United States have been asymmetric contact between groups and racial status hierarchy.

According to the contact hypothesis, prejudices about an out-group are more likely to flourish within an in-group when the in-group is a numerical majority with little regular contact with the out-group. In these situations, the direct contact that might dispel, or at least give nuance to, stereotypes is less frequent than the vicarious or indirect contact that the majority has with the out-group through media representations or in-group stories (Kanter 1972; B. Lee, Farrell, and Link 2004; Tuan 1998). In addition, according to status theory, prejudices about an out-group are more likely to flourish within an in-group when the groups are perceived to be unequal in their respective resources (Ridgeway 1991). In these situations, “the mere existence of racial inequality may buttress the very stereotypes that are used to explain and justify racial inequality in the first place” (Brezina and Winder 2003:403). Furthermore, because challenges to inequality by an out-group have the potential of eliminating that inequality, they also tend to reveal prejudices among the in-group based on its acceptance of the preexisting inequality as legitimate. In sum, the prevailing realities of primarily indirect exposure and persistently unequal resources undermine the conditions for overriding generalizations and thereby suggest the likelihood that persons will consciously and/or unconsciously exaggerate average group differences, including those found in genetic research (Kahn 2003; S. Lee et al. 2001).

In addition, racial and ethnic relations involve not only contemporary attitudes about in- versus out-groups and higher- versus lower-status groups but also a history of state interventions that explicitly marginalized nonwhites and privileged whites until recent decades (Glenn 2002; Haney-Lopez 1998; Lipsitz 2006; Oliver and Shapiro 1997; Takaki 1979, 1994). This political history of, in broad strokes, Native American genocide and removal, African enslavement, Latino conquest, Asian exclusion, and white citizenship is the distal source of the cultural stereotypes that circulate in the absence of sufficient contact and naturalize the inequalities between specific groups. Indeed, Omi and Winant (1986, 1994) have argued that in the post-civil rights era, the racial state remains the primary arena for the contestation, continuing development, and persistence of racial ideology, or what Shiao and Tuan (2008) refer to as racial culture “the totality of racial attitudes, assumptions, and beliefs within the national mainstream culture” (p. 260). As a result, persons perceive their own and other groups not only by (1) internalizing “objective social divisions” whether as accurate group generalizations or exaggerated group differences in their immediate environments but also by (2) associating the groups with cultural representations that “transform mere

likeness into categorical identity,” cognitively escalating perceived subspecies differences into species-level differences (Martin 2000:195, 227). When this perception occurs at the person level, its effects may interact with, and be potentially confounded with, genetic effects; however, when this perception occurs among persons differentially situated by virtue of situations, networks, organizations, or cultural processes, its effects involve additional processes less likely to be confounded with genetic effects.

Accordingly, we propose to conceptualize racial and ethnic categorization as a social perception of biological ancestry in contrast with the measurement of ancestry that clines and clinal classes provide. Interpersonal interactions occur not only as expressions of persons’ morphological, personality, and cognitive characteristics but also in response to their perceptions of each other, including each other’s ancestries and attitudes about those ancestries, which in turn depend on the immediate context for intergroup contact and inequality and also the broader context of historical-political culture. As a social perception, racial/ethnic classification can be accurate in terms of making categories that are homologous to clinal classes but be “inaccurate” when associating the categories with (1) putative group characteristics rooted in historic cultural representations or (2) group characteristics overgeneralized to particular individuals, including characteristics based on empirical group averages. And vice versa, racial/ethnic classifications may be accurately associated with empirical group averages but not be homologous to verifiable clinal classes when the perceived ancestries have no biological basis.

Similarly, changes in the relations between racial/ethnic groups may alter the political context for, the cultural context of, and social experiences associated with group membership, with no direct influence on genetic ancestry. The main exception would be changes in those intergroup relations that influence mating restrictions, as these have the potential to affect the persistence of the homologous clinal classes. These restrictions include the definition of natural or endogamous partners, their spatial availability, the relative preferences for exogamous alternatives, and the preferences of family and other significant third parties (Shiao and Tuan 2008). Indeed, it is a testament to the persistently nonrandom, that is, social, character of human mating that the vast majority of contemporary persons “know,” that is, self-identify with, their genetic ancestry, that is, whether their ancestors lived in sub-Saharan Africa, western Eurasia, the Pacific Islands, eastern Eurasia, or the Americas 50,000 to 2,000 years ago.

We now turn to the question of how to theoretically integrate person-level genetic effects and social perceptions of ancestry into sociological explanations of racial/ethnic phenomena. In brief, we propose to “bound” the effects of genetic ancestry within the processes of the social construction of race. We cannot provide a complete reformulation of racial constructionism but instead offer an initial integration of biosocial causation into existing social explanations from four major publications in the sociology of race/ethnicity; these studies examine racial stratification (Conley 1999), ethnic assimilation (Alba and Nee 2003), racial attitudes (Bobo and Tuan 2006), and racial/ethnic interactions (J. Lee 2002). Specifically, we classified four modes of biosocial causation, reinterpreted four publications’ empirical explanations in terms of Stinchcombe’s (2005) causal units, located their person-level units, and considered the biosocial modes to which those person-level units might be vulnerable.

On the basis of a synthesis of selected typologies for gene-environment interplay (Boardman et al. 2008; Freese 2008; Perrin and Lee 2007), we classify the range of modes for biosocial causation as (1) social or genetic determination, (2) social and genetic contributions, (3) social-genetic exaggeration, and (4) biosocial causal chains. The first three modes are the primary types, while the fourth classifies empirical combinations of the first three. In the end, we inferred three placeholders for potential genetic effects centered in (1) the personality dimension of characteristics (ethnocentrism), (2) a combination of the personality and cognitive dimensions (strategic action), and (3) the morphological dimension (visible ancestry).

Social or Genetic Determination and the Case of Racial Attitudes

Individual phenotypes, actions, and internal states may have either entirely social or entirely genetic causes, from (1) simple social or simple genetic causes to (2) complex interactions among exclusively social or

exclusively genetic variables (Perrin and Lee 2007). In these cases, the other domain of causes, genetic or social variables, respectively, may appear to have a relationship with an outcome that is actually spurious, because the real cause, social or genetic, determines both the other cause and the outcome of interest. Most explanations in sociology, and all four of our cases of racial/ethnic sociology, seek to follow the mode of social determination. Our review of the four cases finds that none is vulnerable to the mode of genetic determination for the simple reason that every case's explanation relies on causal units beyond the person level. Similarly, we also find that no case qualifies in its entirety as social determination, because every explanation relies on social perceptions of ancestries that are homologous with verified clinal classes. The case that arguably comes the closest is the study of racial attitudes (Bobo and Tuan 2006).

Since the civil rights movement, research on racial attitudes has focused on the principle-implementation gap within the public opinions of white Americans toward nonwhites and race-related public policies. Lawrence Bobo and Mia Tuan's (2006) *Prejudice in Politics* conducts a survey of Wisconsin residents to evaluate competing explanations of the policy attitudes of whites about the demands of the Lac Courte Oreilles Chippewa Indians that the U.S. government honor their specified treaty rights to fish, hunt, and gather on ceded tribal territory. In Stinchcombe's terms, their conclusions are that historical events produced cultural stereotypes and a status hierarchy of whites over Indians that remained latent until a network of white persons perceived organized Indian activism as a political threat and successfully mobilized a broader network of whites with a sense of group competition. Although the study's causal units include networks and cultural processes, its main units of analysis are persons, making it particularly vulnerable to confounding by unmeasured genetic influence. We suggest the effect in question to be a partially heritable tendency for ethnic nepotism or in-group favoritism centered on a more basic tendency to distrust nonkin (Van den Berghe 1987) and that this tendency could be a cause of both white attitudes about Indians (E_i) and white attitudes about treaty rights (V_i). However, Bobo and Tuan also measured antiblack attitudes to evaluate whether a generalized out-group animus characterized white opposition to treaty rights and found instead that white opposition depended on group-specific attitudes. Whereas a generalized ethnocentrism might qualify as a secondary phenotypic characteristic that combines personality tendencies to trust kin with a local definition of in-group, we believe that the situational distrust of specific groups has a qualitatively more social character. By contrast, the basic distinction between Indians and blacks as separate groups is homologous with actual clinal classes representing biological ancestry in the Americas and Africa, respectively.

Social and Genetic Contributions as Illustrated in Ethnic Assimilation and Racial Stratification

Both social and genetic factors may contribute to individual outcomes in significantly nonspurious ways. On the one hand, social factors may provide a "social push" to genetic variations such that both have independent and additive effects (Boardman et al. 2008; Freese 2008). On the other hand, they may interact at the person level such that either (a) genetic variation moderates the effect of social variables or (b) social variation moderates genetic expression. Freese (2008) describes the first form as "the stickiness of the self in response to events over time" (p. S23), wherein G_i is a cause of heterogeneity in the effect of some social variable on an outcome. Perrin and Lee (2007) describe the second form as the canonical view of gene-environment interaction, wherein the environment represented by both social and other biological variables moderates the genetic potential of an organism. Boardman et al. (2008) further divide these environmental moderations into subtypes: "social control," wherein the environment inhibits the influence of G_i on P_i ; "social expression," wherein social variables at some high levels are required to trigger genetic expression; and "social distinction," wherein genetic expression occurs only at low levels of some social variables. Following Freese, we characterize this second form of moderation as the contingency of the self.

These two forms of social and genetic moderation arguably rely on different interpretations of the same interaction coefficient. The first interprets the interaction as a deviation from the social-only coefficients, whereas the second interprets the interaction as a deviation from the genetic-only coefficients. Qualitatively,

the stickiness of the self would appear in the variable responses of different persons with similar social characteristics to the same environment, whereas the contingency of the self would appear in the distinctive responses of persons with a particular genome to different environments despite their social similarity to other persons without that genome. Our review of the four cases finds that three cases—the studies of racial stratification (Conley 1999), ethnic assimilation (Alba and Nee 2003), and racial/ethnic interactions (J. Lee 2002)—are vulnerable to the possibility of genetic contributions, namely, the placeholders for ethnocentrism and strategic action.

In the cases of ethnic assimilation and racial/ethnic interactions, we suggest that individual tendencies for ethnocentrism are likely to moderate social effects. To illustrate, a central question in the study of ethnic assimilation has been whether the “new” immigration since the Immigration Act of 1965 will follow in the footsteps of the “old” immigration that concluded with the Immigration Act of 1924. Richard Alba and Victor Nee’s (2003) *Remaking the American Mainstream* uses survey and census data to reexamine historical and contemporary indicators of adaptation for both Europeans and non-Europeans. In Stinchcombe’s terms, their conclusions are that in both eras, organizations in the form of state institutions regulate the reception of different national origin groups, to which individual families respond through the symbolic and material resources of their coethnic networks by making decisions that tend to alter their linguistic, occupational, educational, residential, and marital characteristics—in the direction of acculturation. In this explanation, partially heritable tendencies for ethnocentrism may moderate how families respond to their institutional reception and network resources, wherein individual parents with higher propensities for ethnocentrism may make decisions that attempt to limit, or otherwise influence, their families’ interactions with the mainstream. Furthermore, this moderation effect may involve clinal class differences in the frequency of ethnocentrism.⁴

The studies of racial stratification and ethnic assimilation advance explanations vulnerable to the possible effects of a partially heritable capacity for adaptation to new and/or stratified environments, that is, strategic action. A significant issue in the study of racial stratification has been the race-class debate over the relative contributions of racism and economic disadvantage in causing racial disparities in individual life chances. Dalton Conley’s (1999) *Being Black, Living in the Red* examines the causes of black-white disparities using an existing longitudinal survey with measures for not only the traditional socioeconomic indicators of income, occupation, and education but also parental assets. In Stinchcombe’s terms, his conclusions are that historical events have produced racially segregated networks in the form of neighborhoods with unequal property values and families with unequal assets. Although these events have passed, they indirectly affect the young adult children from these networks through the unequal resources available to them for pursuing their socioeconomic goals. We suggest that individual capacities for strategic action might moderate the effect of network resources on their pursuit of socioeconomic achievement. However, after controlling for his broader operationalization of class, Conley does not find significant group differences on most filial economic outcomes, that is, assets, college graduation, unemployment, and welfare receipt, which suggests the presence of individual variation without clinal class differences. Similarly, in the study of ethnic assimilation, capacities for strategic action potentially moderate the success with which immigrant parents perceive and pursue opportunities. Unlike Conley’s study, however, Alba and Nee’s (2003) analysis does not reduce the effect of group membership to “zero”; thus, both individual and clinal class variations in strategic action may moderate how families respond to their institutional reception and network resources.

Social-Genetic Exaggeration as Illustrated by Racial/Ethnic Interactions

Social and genetic variation may be correlated even without interaction at the person level through the distal causation of genetic effects by social variation or vice versa (Freese 2008). In particular, small genetic or environmental differences may become exaggerated in E_i or G_i , respectively. The environmental exaggeration of genetic differences can occur through large differences in social response to small differences in phenotype or through the selection of different phenotypes into different environments, both leading to gene-correlated outcomes unrelated to differences in genetic potential. By contrast, the genetic

exaggeration of environmental differences, that is, geography, is arguably the mechanism of evolution, whether toward speciation or the emergence of subspecies. That said, where the geography in question is partly social, that is, involving mating restrictions, genetic exaggeration is also likely the result of sexual selection, not simply selection for fitness (Diamond 1994). Our review finds that all four cases are vulnerable to the social exaggeration of genetic differences, namely, the placeholder for visible ancestry.

Since the 1992 Los Angeles riots, studies of racial/ethnic interactions have paid increasing attention to interminority relations within the context of minority groups' distinct relations with the white majority. Jennifer Lee's (2002) *Civility in the City* examines the merchant-customer relations of Jewish-, Korean-, and African American-owned businesses in black neighborhoods using in-depth interviews with 75 African American, Jewish, and Korean merchants and 75 black customers collected through field research in five low-income and middle-income neighborhoods in two cities. In Stinchcombe's terms, her conclusions are that historical events have produced (1) racially segregated networks in the form of predominantly black and white neighborhoods associated with a black and nonblack inequality in assets; (2) cultural stereotypes and status hierarchies of white over nonwhite and nonblack over black, which persist in segregated neighborhoods in the absence of contact; and (3) ethnically segmented networks in the form of business niches stratified by the movement of older ethnics into services and newer ethnics into retail. In turn, these factors produce the situations of largely nonblack merchant-black customer relations in black neighborhoods and white sales associate-black customer relations in white neighborhoods. The nonblack merchants and sales associates share a residential isolation from their black customers that shapes their internal states, but the merchants also experience regular contacts with blacks that reshape their attitudes over time. Furthermore, an important aspect of this circumstance is the internal response of black customers to their experiences with, and observations of, the largely white sales associates outside of black neighborhoods. The result is a latent resentment of the nonblack domination of businesses in black neighborhoods.

In this study, the social exaggeration of genetic differences, that is, visible ancestry, is pervasive in (1) the large difference in the historic response of whites to black versus nonblack phenotypes among prospective neighbors, (2) the resulting selection of racial phenotypes into segregated neighborhoods with unequal resources for business development, (3) the large difference in the response of business owners to coethnic versus other-ethnic ancestry among prospective business contacts, (4) the resulting selection of ethnic phenotypes into segmented business niches, and (5) the exaggeration, by persons, of average group characteristics into confirmations of a wide range of cultural stereotypes.

Biosocial Causal Chains

Individual phenotypes, actions, and internal states may be the result of "[biosocial] causal chains of indeterminate lengths combining" two or more of the first three modes (Perrin and Lee 2007:308). In fact, our review finds that all four cases employ explanations vulnerable to at least two modes of biosocial causation. Bobo and Tuan's (2006) study of racial attitudes is a primarily socially determined explanation that also involves the use of visible ancestry to distinguish different out-groups (social-genetic exaggeration), whereas the remaining studies are more equally vulnerable to both social-genetic exaggeration and social and genetic contributions: J. Lee's (2002) study of racial/ethnic interaction emphasizes a wide range of social-genetic exaggerations but remains vulnerable to the unmeasured effects of individual and group variations in ethnocentrism, Conley's (1999) study of racial stratification emphasizes residential network segregation but remains vulnerable to the unmeasured effects of individual variation in strategic action, and Alba and Nee's (2003) study of ethnic assimilation emphasizes institutional reception and network resources but remains vulnerable to the unmeasured effects of individual and group variations in both ethnocentrism and strategic action.

Our integration of biosocial causal modes into select social explanations has several implications. First, biological and social determinations are likely rare if they require explanations that rely exclusively on person-level causal units and racial/ethnic categories without basis in clinal classes, respectively. Second, given the prevalence of the nondeterminist modes, researchers might assume that social and biological inputs are likely entangled until they have evidence for specific forms of interdependency or determinism.

Third, the familiar phenomena of discrimination, inequities in neighborhood resources, ethnic solidarity, and group stereotyping qualify as the most pervasive form of biosocial causation: the social exaggeration of genetic differences. Fourth, certain partially heritable characteristics potentially moderate the effects of situations, networks, organizations, and cultural processes on persons or, more precisely, the ways persons respond to those higher-level units. A tendency for ethnocentrism may moderate how immigrant families respond to their institutional reception and network resources and also how nonblack merchants respond to regular contact with black customers. Also, a capacity for strategic action may moderate how those same families respond to their environment and also moderate how black and white young adults respond to their residential and educational network resources. However, certain empirical findings limit these moderations: The out-group-specific nature of racial attitudes suggests a role for higher-level units in directing ethnocentric tendencies, and the nonsignificant race coefficients for many racial stratification outcomes suggest that social class mediates group differences in capacities for strategic action. In sum, relatively little of the empirical explanations made by sociologists of race/ethnicity require the claim of biological nonreality traditionally associated with racial constructionism.

CONCLUSION

In this article, we have reformulated the theory of race as a social construction by replacing obsolete claims about human genetic variation with the findings of recent genetic research, clarifying the nature of the group differences consistent with recent research and embedding the potential effects of genetic ancestry in biosocial causal processes. Specifically, we advance the concept of *clinal classes* as a measurement of genetic ancestry that is complementary to the existing measurement of ancestry as solely clinal, coin the term *genomic individualism* to include ancestry in a theoretical model of genetic effects, reconceptualize racial/ethnic categories from purely social creations to social perceptions of biological ancestry, and reinterpret major research in racial/ethnic sociology to illustrate how individual genomes might mediate the effects of genetic ancestry on phenotypes and social environments.

Current formulations of racial constructionism deny any biological basis for race/ethnicity by employing an unrealistic criterion for biological differentiation that exceeds species-level differences. These formulations argue that human genetic variation is entirely composed of clines and regard any claims of a biological basis for racial/ethnic categories as embarking on a slippery slope to biological determinism. In fact, they counter that it is society that determines the consequences of human biology. Against these positions, we recommend that sociology set aside the claim of biological nonreality and adopt an approach more consistent with recent genetics research.

Accordingly, we have offered a reformulation of racial constructionism that accepts that recent genetic research has identified a biological basis for race/ethnicity that exceeds the more realistic threshold of statistically identifiable clusters. Our approach argues that human genetic variation is composed of both clines and clinal classes that are homologous to certain racial/ethnic classifications and that this biological ancestry has nondeterministic effects and may contribute to average group differences. We agree that recent research may permit a “backdoor” for biological racism, but we regard that possibility as resulting from a social perception of the findings rather than an essential characteristic of them. Instead, our bounded-nature alternative envisions the relationship of society and human biology as thoroughly entangled and invites researchers to explore its complex causal chains of social determinations, gene-environment interactions, and gene-environment exaggerations.

In sum, we reframe the social construction of race as the social processes that iteratively (1) condition the perception of human ancestry, (2) are moderated or “resisted” by partially heritable individual characteristics and their distributions across clines and clinal classes, (3) exaggerate individual variation along ancestral lines, and (4) determine the meaning and social consequences of often categorical perceptions of ancestry. These social processes are components of a racial social reality that is historical, processual, stratified, and analytically multilevel but that is also entangled with biological inputs inherited from the geographic distribution of humans in genetic watersheds over the past 50,000 years. In brief, our approach

seeks to acknowledge both the identifiability of a biological basis for race/ethnicity and the complexity of its social construction.

Our approach has implications for existing sociological accounts of race/ethnicity and future research on racial/ethnic classification and the role of psychology in social theory. First, if the claim that racial/ethnic categories have no biological reality is actually tangential to the empirical explanations in most racial/ethnic sociology, the implied minimum revisions are mainly contextual. Scholars could simply replace language associating racial constructionism with the “creation” of race/ethnicity with instead language associating it with the “perception” of ancestry. However, we believe that a theoretically deeper engagement would require some discussion of clinal classes, if not also genomic individualism.

Second, if clinal classes at different scales, e.g., $K = 5$ versus $K = 14$, are hierarchically related to each other, they cannot fully account for the variability of their social perception across time and place. This implication raises the question of how clinal classes influence the perception of ancestry, especially in historically and nationally different racial-classification systems. Applying Peterson’s (2010) distinctions between forms of social influence on innate ability, we sketch a range of influences for future research to examine: Clinal classes may be the natural boundaries for racial/ethnic classifications that vary simply according to the patterns of who has had contact with whom. Similarly, clinal classes may serve as the objective variation on which racial/ethnic classifications make variable divisions, similar to the color perception of the light spectrum, that is, whether blue and green are considered shades of the same color or distinct colors and by what terms “blue” and “green” are called. Alternatively, clinal classes may provide an objective but less pressing variation from which racial/ethnic classifications make selections and specifications, for example, giving some clinal classes more cultural “weight” than others (Danna 2011). Most distinctly, clinal classes may provide no constraint on racial/ethnic classifications where socially recognized ancestries have no basis in verifiable clinal classes.

Third, if genetic effects occur primarily through partially heritable psychological characteristics, then different frequencies of personality and cognitive characteristics—both actual and perceived—constitute an important missing construct in social theory. This possibility suggests (1) a logic of causal resistance to social construction wherein psychological characteristics moderate the effects of situations, networks, organizations, and cultural processes, and (2) an important role for negotiations of compatibility wherein perceptions of group psychology exaggerate the responses of persons to each other. Future research should explore the challenge of psychology for social theory in both the social and genetic contributions to “sticky” individualism and the cultural beliefs about how well different persons and groups “fit” together.

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NOTES

1. Kalleberg, Reskin, and Hudson (2000) find that indicators for “bad jobs” are not evenly distributed among occupations but instead form clusters that approximate social classes. Their existence is a separate issue from whether they originate from class relations.

2. For example, there are “black” and “white” clinal classes, not in the sense that contemporary blacks and whites have completely or even mostly distinct ancestors but, rather, in the sense that in the past 50,000 years, African and western Eurasian ancestral populations became more isolated from each other than among their respective subpopulations and that mating restrictions have contributed to maintaining their relative isolation into the present.
3. Keller and Miller (2006) use the metaphor of a single watershed to model the flow of genetic effects toward a given polygenic trait, whereas we use the metaphor of multiple watersheds to model the division of ancestral populations.
4. That said, Alba and Nee (2003) use families, not parents, as their proximate causal unit, and in relation to household decisions, families might approximate situations with interacting roles rather than simply persons.

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