

THE ROUTLEDGE COMPANION TO PHILOSOPHY OF RACE

For many decades, race and racism have been common areas of study in departments of sociology, history, political science, English, and anthropology. Much more recently, as the historical concept of race and racial categories have faced significant scientific and political challenges, philosophers have become more interested in these areas. This changing understanding of the ontology of race has invited inquiry from researchers in moral philosophy, metaphysics, epistemology, philosophy of science, philosophy of language, and aesthetics.

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By clearly explaining and carefully organizing the leading current philosophical thinking on race, this timely collection will help define the subject and bring renewed understanding of race to students and researchers in the humanities, social science, and sciences.

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Paul C. Taylor, Linda Martín Alcoff,
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RACE AND BIOLOGY

Rasmus Grønfeldt Winther

Introduction

Imagine landing in the largest city, the capital, of an alien planet. You are stunned to see that every humanoid is within a few centimeters of the same height, everyone has a nearly identical muscular body, and everyone's facial features are quite similar—high cheekbones, small noses, and black eyes. Perhaps most surprising to you, everyone has purple skin. The ambassador accompanying you tells you the purple skin is a consequence of interacting skin pigment proteins, the double sun of that planetary system, and generation upon generation of voluntary random breeding. As you walk to your important meeting, the ambassador also informs you that every adult humanoid on the planet looks the same. She is no exception. Call this planet “Unity.”

Now recall Darwin's natural experimental laboratory of evolution you learned about in high school biology, the Galápagos Islands, and the variety of finch and tortoise species found there. But let us now populate them, in our minds, with identical small populations of early humans. Add a few more dozen islands that are larger, have distinct environments, and are distant and mutually unreachable. Throw in a few million years of evolution. Humans on the islands of this thought experiment will come to be quite different indeed, in body, behavior, and culture. Call this scenario “Galápagos-Writ-Large.”

Is *Homo sapiens* anno 2020 more like the inhabitants of Unity or Galápagos-writ-large? Modern genetics and genomics teach that our species is much closer to Unity.

Although less reductionist approaches to the biological sciences exist (Maturana and Varela 1980; Levins and Lewontin 1985; Maynard-Smith and Szathmáry 1995; Oyama 2000; Noble 2006; Winther 2008; Pigliucci and Müller 2010), most scientific practice on race and biology today is performed at the genetic and genomic level. The thrust here is to explore this level. In particular, seven theses on human genetic variation establish a crisp evolutionary picture of our species as relatively young, quickly expanded, and fairly continuous in genetic variation across our entire geography. It seems crucial to cover such facts so that the reader may form her or his own judgments about statements such as the following ones made by philosophers in prestigious venues:

The lack of fixed traits for each so-called race means that race cannot be inherited as is popularly thought. Rather, the specific physical characteristics variably associated with races in cultural contexts are inherited through family

descent as is the rest of human biology. Race, therefore, supervenes on human genealogy or family inheritance.

(Zack 1999: 84)

There are no racial genes responsible for the complex morphologies and cultural patterns we associate with different races.

(Haslanger 2000: 43)

The concept of race is the concept of a group of human beings . . . (1) distinguished from other groups of human beings by visible physical features of the relevant kind, . . . (2) whose members are linked by a common ancestry, [and] (3) who originate from a distinctive geographic location.

(Hardimon 2003: 451–452)

After laying out the empirical facts, I consider their biological and philosophical implications. I survey proposals about the metaphysics of racial “kinds of people” (Hacking 2007a, 2007b), organized around *biogenomic*, *biological*, and *social* levels (Kaplan and Winther 2014). Theories of race require distinguishing at least four questions:

1. The “biogenomic race” question: is there genetic structure in human populations and what is it?
2. The “semantic” question: does the genetic structure correspond to extant designations of populations or kinds, in different languages?
3. The “biological race” question: does the genetic structure correspond to significant genetically based differences for socially valued phenotypes?
4. The “social race” question: are there racialized social kinds?

The Galápagos-writ-large scenario is an extreme version of the existence of biogenomic race. Biogenomic race exists when a species is subdivided into populations corresponding to standard uses of, for example, racial, national, or ethnic designations (see Winther and Kaplan 2013; Kaplan and Winther 2014). Most practitioners do not take their genomic work on human populations to be about race (Coop et al. 2014; Philosophy in a Multicultural Context Research Cluster: <http://ihr.ucsc.edu/portfolio/philosophy-in-a-multicultural-context/>).

Worrying about the semantics of race involves reflecting on appropriate conditions of application of racial terms, kinds, concepts, and names; on the nature of the reference relations of such terms (etc.) to the world; and on the processes of baptism and justification, whether in ordinary discourse, behaviors, and norms, or in biology, of these terms (etc.) in the first place (see Sarah-Jane Leslie, this volume; Mallon 2006; Spencer 2014; Ludwig 2015). As one example of addressing semantic concerns, the very baptism of “biogenomic race” is justified because slippage between the terms “populations” and “races” is common but can be avoided when we recognize complex and subtle differences between the two terms (Reardon 2005; Morning 2011; Kaplan and Winther 2014; Winther, Giordano, Edge and Nielsen 2015).

Biological races exist when a stable correlational or, better yet, causal mapping can be drawn between group genetic differences and socially significant or valued phenotypic characters such as cognitive abilities and perhaps also disease proclivities. It is especially

in the domain of the biological race question (i.e., in the metaphysics of biological race) that fraught political and moral questions and challenges emerge. Indeed, exploring human genetic variation and the existence (or not) of biogenomic race would be a wholly abstract and intellectual endeavor except for its politically and morally relevant consequences (Lewontin 1970; Hacking 2005; Kitcher 2007; Winther and Kaplan 2013).

Finally, social races exist when there are psychologically and communally perceived stable kinds of racialized people, often leading to systematic discrimination and oppression (Mills 1998; Haslanger 2000; Hacking 2005).

Importantly, a normative question lurks: what are the beneficial and what are the pernicious effects of employing racial categories, and of perceiving the existence (or not) of various sorts of race, and who is affected and how? Whether this should be made a distinct question, thereby making ontological and semantic questions logically distinct and perhaps even prior to the normative question, as a standard analytical metaphysical approach would prefer, or whether such a normative question should not be separated out since it suffuses all the others, as pragmatic, conventionalist, or more sociological approaches would argue, is a difficult matter also bracketed in this entry (Spencer 2012; Ludwig 2015; Winther, Millstein and Nielsen 2015).

Fact Sheet: Seven Theses About Human Genetic Variation

A few basic definitions are necessary. The *genome* is the entire DNA sequence in an individual of a species; the genome is made up of long strings of DNA *nucleotides*, which take four forms represented by the letters A, C, G, T. A *locus* is a specific part of a genome, a “chunk” of the nucleotide sequence (though it can be composed of non-continuous nucleotide sequences), often used in our language coextensively with *gene*, and often but not necessarily functional, in the sense that the locus (or gene) is causally or mechanistically associated with some (part of a) phenotype. An *allele* is one of various versions of a locus (or gene), differing with another allele at one nucleotide or more, and existing in a population of a species. Our genome is divided into 23 pairs of *chromosomes*, 22 of which are standard autosomes and one of which is the sex chromosomes, our mitochondria also have unique genes. The facts below focus on autosomes, unless otherwise specified. Given this conceptual background, population genetics is the attempt to make evolutionary theory mathematically explicit by viewing evolution as the change of allele frequencies across generations, within and across populations and species. Population geneticists develop mathematical models. Furthermore, they subject the explanatory and predictive population-genetic theory to empirical tests (Lewontin 1974; Hartl and Clark 1989; Nielsen and Slatkin 2013). Complex facts about human genetic variation revealed by our best contemporary population genetics can be summarized in the following seven theses:

1. Low average nucleotide diversity
2. Small inter-species differences
3. Widely distributed alleles
4. Non-African variation is basically a subset of African variation
5. Most genetic variation is among individuals within populations
6. Even so, clustering populations and classifying individuals is reliable
7. The further apart on human migration routes that two populations are, the less genetically similar they are.

These basic quantitative features of human genetic variation can be understood without reference to population genetic theory. Or they can be the targets of population genetic models deploying important theoretical parameters such as mutation rate (μ), selection coefficient (s), and effective population size (N_e) (Hartl and Clark 1989; Nielsen and Slatkin 2013; Winther, Giordano, Edge, Nielsen 2015). Let us explore the seven theses.

1. *Low average nucleotide diversity.* Of species whose genomes have been extensively mapped, *Homo sapiens* has unusually low average nucleotide diversity. All members of *Homo sapiens* are basically identical at, on average and approximately, 999 base pairs out of 1,000 (Li and Sadler 1991; Yu et al. 2002). Given a total genome size of 3 billion nucleotides, and an average difference of about 0.1% between any two humans, two individuals will typically differ at approximately 3 million nucleotides. For comparison: *Drosophila* fruit flies, the standard workhorse for genetic studies, differ from each other on average by 1%, which is 10 times our diversity (Li and Sadler 1991); bonobos differ by 0.077%, chimpanzees by 0.134%, and gorillas by 0.158% (Yu et al. 2004). Maize has even more nucleotide diversity than *Drosophila*, and soybeans have slightly more than humans (Brown et al. 2004). Admittedly, *Homo sapiens* has more diversity than most big cats—roughly twice that of lions and leopards (unfortunately for their future prospects, cheetahs have near 0% diversity) (O'Brien et al. 1985). Wherever you may be from, you and I are genetically quite similar. Unity indeed.
2. *Small inter-species differences.* Average across-genome nucleotide identity between humans and chimpanzees is 98.77% (i.e., 1.23% nucleotide divergence), 98.69% between humans and bonobos, and 98.36% between humans and gorillas (Yu et al. 2004). Even human and mice genomes are roughly 85% identical (Batzoglou et al. 2000). Actual similarity is less than immediately apparent since genes (or parts of genes) are rarely linearly and continuously arranged on the genome, and genes change order and structure across species. Meaningful species-to-species comparisons of gene structure and gene number can therefore not be straightforwardly made only by comparing average nucleotide sequence similarities. Further evolutionary inferences about, for example, which nucleotide sequences can be traced back to common ancestors and which converged independently need to be made, and gene functionality must be assessed (Dicks and Savva 2007; Gerstein et al. 2007; Hahn et al. 2007).
3. *Widely distributed alleles.* Most human populations contain most of the common alleles present in our species. Approximately 92% of common alleles (i.e., alleles not present in just one person or a few people) are found in two or more of the following regions: Africa, America, Central/South Asia, East Asia, Europe, Middle East, and Oceania). That is, only about 8% of all common alleles are geographically private (i.e., the allele is unique to a single region). Over 82% of alleles are found in three or more regions, and approximately 47% of alleles are found in *all* regions (Rosenberg 2011).
4. *Non-African variation is basically a subset of African variation.* Africa is much more genetically variable than the rest of the world, and much of the rest of the world's variation is a subset of African genetic variation. African populations have approximately twice the nucleotide diversity of non-African populations. That is, two people whose recent ancestors are of African origin differ on average by about 1:900

nucleotides (0.11%), whereas two people whose recent ancestors are of European origin differ on average only by approximately 1:1600 (0.063%) (Yu et al. 2002; Campbell and Tishkoff 2008; Wall et al. 2008). Second, Africa also has approximately half of the total number of private alleles (Rosenberg 2011). As a third measure of variation, consider the distribution of all the approximately 8,000 alleles surveyed in Rosenberg (2011). Of these, roughly 82% were found in Africa, much more than any other single continent. Furthermore, most alleles (87%–90%) found in one non-African continent were also found in Africa, but not the converse. For example, only 74% of alleles observed in Africa are also found in Europe, and only 63% of alleles identified in Africa are also located in the Americas (Rosenberg 2011; Figure 22.1). Indeed, the number of total alleles per region diminishes, unsurprisingly, as we move farther away from Africa (along human migration routes), in the following ranked order: Middle East, Europe, Central/South Asia, East Asia, Oceania, and America (Figure 22.1). A final measure of genetic variation relevant here is heterozygosity, which is a measure of how evenly distributed alleles at

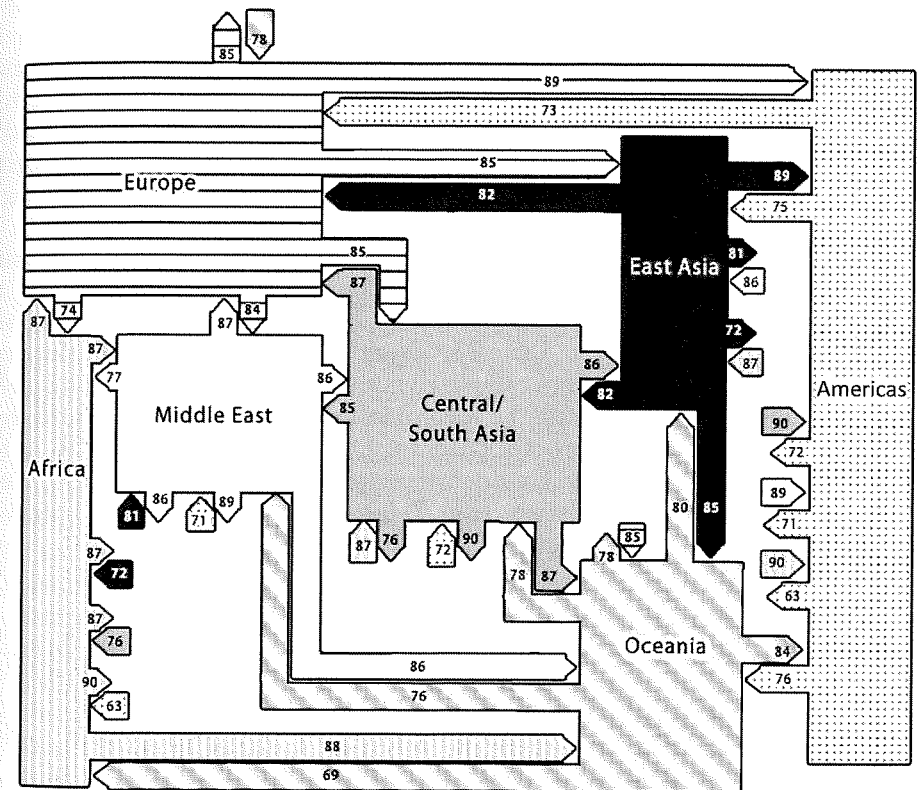


Figure 22.1 “Schematic world map of the ‘flow’ of microsatellite alleles. . . . boxes represent regions of the world, positioned geographically. Links entering into a geographic region indicate the percentages of distinct alleles from the geographic region found in other regions. . . . For example, averaging across loci, 87% of alleles observed in Europe are also observed in Africa, whereas 74% of alleles observed in Africa are also observed in Europe.” Source: Figure 9, Rosenberg 2011, 680; redrawn by Michelle Dick, UC Santa Cruz. Reprinted with kind permission.

each locus are in a given population, averaged across loci. Low heterozygosity for a locus means that most genotypes in that population are homozygous (e.g., AA or aa) rather than heterozygous (Aa) (Hartl and Clark 1989). Interestingly, within-population heterozygosity diminishes as a tight linear function of geographic distance from Addis Ababa, Ethiopia (Figure 22.2).

The loss of (i) nucleotide diversity, (ii) private alleles, (iii) total alleles, and (iv) genetic heterozygosity as we move away from Africa can be explained in terms of a serial founder effect model (see #7). As *Homo sapiens* migrated out of Africa, we went through a series of genetic bottlenecks in which relatively smaller groups colonized new areas (Ramachandran et al. 2005; Lawson Handley et al. 2007). These groups represented only some of the genetic variation of the parental population, as measured by (i)–(iv) (these are all theoretically correlated measures; Kaplan and Winther 2013). People reaching America via the Bering Strait went through this bottleneck process the highest number of times (though indigenous Oceanian populations also experienced almost as many, some of them non-overlapping with the Americans). Is Africa like the capital of the Unity world?

5. Most genetic variation is among individuals within populations, and not across populations. How much more genetically similar (on average) are two randomly chosen individuals from the same population as compared (on average) to a randomly chosen individual from that population and an individual from another population, either from anywhere within the same continental region or from anywhere within another continental region? Lewontin (1972) was the first to address this question explicitly, proposing an information-theoretic measure of genetic variation and applying it to genetic data on 17 blood proteins from worldwide blood samples of people. To the surprise of many at the time, his measure of genetic variation was, on average, 85% as large when calculated for a single population as it was when calculated for the world as a whole. (The earliest published data explicitly giving just this statistical result can be found in Table 12, “World Variation of Gene Frequencies,” Cavalli-Sforza 1966: 367.) More completely, Lewontin found that the total global heterozygosity could, on average, be (approximately) divided or partitioned thus: 5% at the continental region level (African, European, and Asian); 10% at the across-population level, within continental regions (e.g., Ghanaian, Kikuyu, Tutsi, Zulu), and 85% within populations (e.g., Zulu). For instance, Table 22.1 shows a

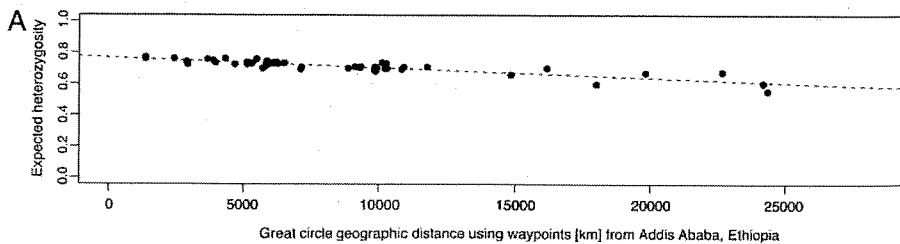


Figure 22.2 The amount of heterozygosity of each of approximately 40 worldwide populations diminishes as a function of their respective distance, along (approximate) migration routes, from Addis Ababa

Source: Figure 4A, Ramachandran et al. 2005, 15946. Reprinted with kind permission.

Table 22.1 Allele frequencies of three distinct genes across continental regions, as presented in Cavalli-Sforza and Bodmer (1971), and used in Lewontin (1972) and (1974). Frequencies are rounded from four to two significant figures. Empty cells indicate lack of data. The data gathering methodology involved “selecting whenever possible populations from Northern Europe . . . Central or South Africa . . . and China” (Cavalli-Sforza and Bodmer 1971: 724). See especially data tables in Cavalli-Sforza and Bodmer (1971), 724–733; Lewontin (1974), 152–157.

Gene	Alleles	Africans	Europeans	Asians
Duffy	Fy	0.94	0.03	0.1
	Fya	0.06	0.42	0.9
	Fyb	0	0.55	0
Auberger	Aua	0.64	0.62	
	Au	0.36	0.38	
Xg	Xg ^a	.55	.67	.54
	Xg	.45	.33	.46

range of kinds of loci, using data from Cavalli-Sforza and Bodmer (1971). Lewontin’s results imply that at most variable loci, different human groups tend to have relatively similar allele frequencies. Thus, the *Duffy* gene is an atypical example, as it is more extremely diverged than average (e.g., 0.94%: 0.03%: 0.1% for one of three alleles), and *Auberger* indicates less variation across populations than the average human locus. The Xg gene is typical of the human genome, showing some small variation across continental regions (see #6 for a simple statistical toy case). More technically, Lewontin’s measure shares important theoretical properties with the standard inter-population heterozygosity F_{ST} measure developed by Wright (1931), which is a measure comparing expected and actual heterozygosities of populations, or (equivalently) a measure assessing the amount of inter-population genetic difference (Hartl and Clark 1989; Holsinger and Weir 2009; Nielsen and Slatkin 2013; Winther 2014). Interestingly, Lewontin’s partitioning numbers have withstood the test of time, although the percentages vary slightly, often with a higher within-population percentage partition (Barbujani et al. 1997; Rosenberg et al. 2002; Rosenberg 2011; Li et al. 2008). Remind you of Unity?

6. Even so, clustering populations and classifying individuals is reliable. Even if most variation is within populations, if we accumulate information across loci, then reliable inferences can be made both about existent populations (clustering) and the population membership(s) of any particular individual (classifying) (Rosenberg et al. 2002; Edwards 2003; Tal 2012; Edge and Rosenberg 2015; Rosenberg 2017; Winther 2017). A computer program, Structure, was designed to do this (Pritchard et al. 2000). Consider a simple case of 100 loci spread across a haploid genome (i.e., single, not paired, chromosomes). Each locus can have a G or an H allele. The frequency of G alleles is 2/5 in population A and 3/5 in population B—very much in line with the expected allele-frequency differences in #5, and analogous to tossing a biased coin (in statistical jargon: binomial sampling). Now imagine that we genotype a person of unknown origin and find that she has a total of 40 G alleles across these 100 sites. Around 8% of people from population A will have this many G alleles, but only about one in 40,000 people from population B will

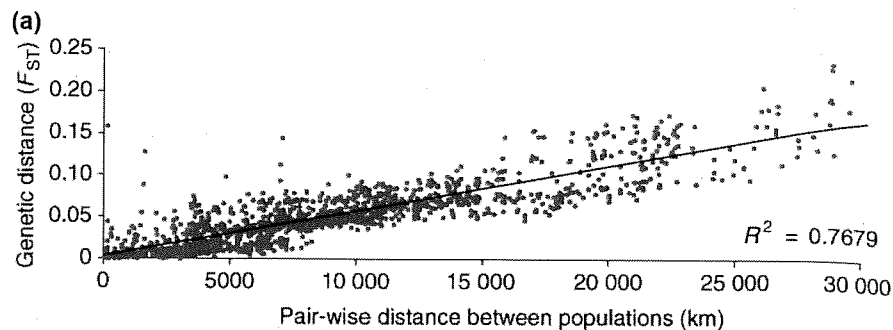


Figure 22.3 High correlation between pairwise F_{ST} and pairwise geographic distance of worldwide populations

Source: Figure 1a, Lawson Handley et al. 2007, 435. Reprinted with kind permission.

have this many G alleles. We can thus infer with high confidence that the person is from population A, and our confidence will only increase as we sample more loci. After all, each new locus is analogous to another coin toss—the more times we toss a biased coin, the closer we get to the actual bias frequency (e.g., 0.4 in population A). Thus, with even small differences in allele frequencies at each locus for distinct populations, by examining enough loci, we can use this procedure to become as confident about population membership as we like (Edwards 2003, Fig. 1, 799). Might subtle genetic differences between humanoids from distinct regions exist even on Unity?

7. *The further apart on human migration routes that two populations are, the less genetically similar they are.* If we plot F_{ST} between pairs of worldwide populations against the geographic distance between population pairs, a clear linear inverse correlation is found (Figure 22.3; $R^2 = 0.77$; Serre and Pääbo 2004; Ramachandran et al. 2005; Lawson Handley et al. 2007). To simplify, the further apart two populations are from one another, the higher their F_{ST} —namely, the more dissimilar their allele frequencies are. Indeed, changes in allele frequency differences across increasingly different populations will tend to occur smoothly. That is, the differences are clinal, under the *isolation by distance* model (Wright 1943; Malécot 1955). In addition to clinal variation, there are small discontinuous jumps in genetic distance associated with geographic barriers including the oceans, the Himalayas, and the Sahara. A serial founder effect model, based on, but slightly more nuanced than, the isolation by distance model, thus seems the appropriate theoretical model of human migrations (Ramachandran et al. 2005; Rosenberg et al. 2005; Rosenberg 2011).

In combination, these seven theses suggest that we are a relatively young species that has expanded in geography and numbers fairly quickly. Our genetic variation still very much overlaps across even continental regions.

Biological Implications

These basic patterns of human genetic diversity can be used for further biological inferences. Trees can be drawn representing our evolutionary history. Since a variety of phy-

logenetic tree-building methods are available (Felsenstein 2004; Winther 2017) and because our species has on occasion mated across large distances, such trees are useful primarily at low levels of resolution. Human trees tend to be consonant with a map of migrations (e.g., Cavalli-Sforza and Feldman 2003: 270; Sommer 2015; for the first population genetic map of human genealogy, see Edwards and Cavalli-Sforza 1964: 75, presented and discussed in Winther 2017). At higher levels of detail, a *trellis*, *network*, or *reticulate* rather than a *tree* model of human evolution seems more plausible (Templeton 1999, 2002; Winther and Kaplan 2013: 63–66; consult Andreasen 2007 for a *cladistic* race concept, and Kitcher 1999 for a *reproductive isolation* race concept; but see Kitcher 2007; Millstein 2015).

Using significant population genetic theory, we can also build trees that represent the history of individual genome segments (Nielsen and Slatkin 2013). Using coalescent methods, the *mitochondrial Eve*—copies of whose mitochondrial DNA, which is transmitted maternally, exist (with variations) in every person today—has been estimated to have lived 99,000 to 148,000 years ago. Similarly, the *Y-chromosomal Adam*, copies of whose Y-chromosome exist in every man today, lived anywhere from 120,000 to 338,000 years ago (Mendez et al. 2013; Poznick et al. 2013). The Most Recent Common Ancestor for European populations can also be estimated (Ralph and Coop 2013).

Notably, some loci are almost certainly experiencing natural selection. Some are targets of local selection, in which different alleles are favored in distinct parts of the globe. Consider the gene *SLC24A5*, which influences skin color variation. The ancestral allele, strongly associated with dark skin in African populations, is practically fixed—that is, present in every individual genome—in most African populations (as well as in most global populations outside of Europe or the Middle East). The derived allele, highly correlated with light skin in European populations, is effectively fixed in most European and Middle East populations (Sabeti et al. 2007). The explanation for selection on skin color is not yet completely clear, but there are several plausible explanations (Wilde et al. 2014).

Clear signatures of local selection are rare. Indeed, *SLC24A5* was one of only “twenty-two strongest candidates for natural selection” out of more than 300 candidate regions (Sabeti et al. 2007: 913–914). It was studied for its unusually divergent geographic distribution (Clark et al. 2003) and its link to known molecular mechanisms of melanin production (Lamason et al. 2005). Though there almost certainly are other loci like this (e.g., altitude adaptation in Tibetans, itself perhaps caused by gene mixing with archaic hominids; Huerta-Sánchez et al. 2014; Sabeti et al. 2006), their frequency and phenotypic consequences are unknown. Despite journalistic yarn-spinning by some (Wade 2014; effectively reviewed by Orr 2014), and despite a few other genes such as *FOXP2*, the so-called language gene (Enard et al. 2002), our knowledge about causal links between genes and behavior remains scant. And this is precisely the fraught epicenter of interest. Might some of the genes for cognitive abilities or certain diseases lie in the relatively small fraction of geographically private alleles (8%) or across-continental heterozygosity (5%–10%)?

Philosophical Implications

Philosophers interested in biological aspects of race tend to focus on two issues: (1) concepts of race and (2) the metaphysics of race. The metaphysics of race involves debates

among three kinds of attitudes towards the reality of race, at three possible operational levels: realism, anti-realism, and conventionalism, about biogenomic, biological, and social race, respectively.

The biogenomic racial realist (e.g., Dobzhansky and Edwards; consult Winther and Kaplan 2013; Kaplan and Winther 2014) concedes that human sub-populations should be admitted as legitimate biological entities (i.e., the biogenomic question above). This realist believes that biogenomic races correspond, at least sometimes, to socially entrenched categories of, for example, racial, national, or ethnic designation (i.e., the semantic question above).

In contemporary literature, Sesardić (2013) accepts the existence of biogenomic races, which he takes to be foundational for biological racial realism. Spencer's tempered defense of biological race in *Homo sapiens* explicitly appeals to biogenomic race, and he distances himself from both standard conceptions of race and from social concerns (Spencer 2012, 2014). Spencer should probably be read as a biogenomic racial realist, arguing that we are more like Galápagos-writ-large than most everyone else admits. When Hochman (2013) denies the reality of human races by noting that human F_{ST} 's would hardly force the identification of similar populations in non-human populations, he critiques biogenomic race. A Unity scenario is accepted. Long and Kittles (2003) attempt to destroy biogenomic race in a manner analogous to Hochman.

In addition to realist and antirealist positions, there are at least two other live options on the metaphysics of biogenomic race. Conventionalism about biogenomic race is defended by Winther (2011, 2014), Kaplan and Winther (2013, 2014), Winther and Kaplan (2013), and Ludwig (2015). According to a conventionalist perspective, interpreting the reality (or not) of biogenomic races depends on the variety of "explanatory interests" deployed (Ludwig 2015: 245–247), and the measures and models used, in particular analyses. Strictly speaking, a fourth option is the ontological *reification* of race (Gannett 2004; Kaplan and Winther 2013; Winther 2014; critique in Spencer 2013), in which "what is cultural or social is represented as natural or biological, and what is dynamic, relative, and continuous is represented as static, absolute, and discrete" (Gannett 2004: 340), or, alternatively, mathematical models are "conflated and confused with the world" (Winther 2014: 204; Winther 2018). Both conventionalism and reification can be interpreted as lying either between realism and antirealism, or perhaps outside of any spectrum defined by realism and antirealism as the two extremes. The reader may wish to draw her or his own conclusions on the metaphysics of biogenomic race based, among other considerations, on section two of this chapter.

The sticking point here is the reality (or not) of *biological* race. The entire issue of biogenomic groups, populations, or races would not be so politically, socially or morally challenging if nothing rode on it (Helen Longino, pers. comm.). If putative group membership only determined socially insignificant characters such as toenail width, normative concerns would be much less salient. The open possibility of finding genes correlated with, for instance, cognitive abilities, makes the study of human genetic variation consequential. For instance, Lewontin (1972) concludes thus: "since . . . racial classification is now seen to be of virtually no genetic or taxonomic significance . . . no justification can be offered for its continuance" (397). Elsewhere he makes his position more explicit:

The taxonomic division of the human species into races places a completely disproportionate emphasis on a very small fraction of the total of human diversity.

That scientists as well as nonscientists nevertheless continue to emphasize these genetically minor differences and find new "scientific" justifications for doing so is an indication of the power of socioeconomically based ideology over the supposed objectivity of knowledge.

(Lewontin 1974: 156)

Lewontin's statement does not imply that he denies #6 (Feldman and Lewontin 2008). His intelligent ire has been aimed less at biologists studying human genetic variation (Lewontin 1978 is a brief response to Mitton 1977) and more at hereditarians including Jensen (1969), Herrnstein and Murray (1995), Lynn and Vanhanen (2002), Wade (2014) and others. Hereditarians argue that many contemporary social, political, and economic inequalities are partly due to hereditary differences in the (average) innate capacities of different continental region "races." They endorse a Galápagos-writ-large picture of biological race, and are less concerned with the details of biogenomic race. In addition to Lewontin, many commentators, such as Coop et al. (2014) and Kaplan and Winther (2014) also deny the existence of biological race. Another option is to withhold judgment until individual genes for socially and morally significant traits such as cognitive or behavioral abilities are identified and clear and explicit selective scenarios and mechanistic penetrance established (consult Longino 2013). The discourse around biological race highlights the *pragmatics* of race, namely, the ways scientific practices intertwine with social concerns and context, and with normativity (Winther, Millstein, and Nielsen 2015).

Characterizations of social race are explored in detail elsewhere in this volume. Kendig (2011) baptizes a hybrid "physiosocial" form of realism: "race can be best understood in terms of one's experience of his or her body, one's interactions with other individuals, and one's experiences within particular cultures and societies" (191). The only philosophical point I shall make here is that, broadly speaking, most realists about social race are making a descriptive point: social race is real in most societies—it is measurable and is experienced within a system teeming with discrimination and oppression. However, some of these same social racial realists imply a prescriptive point of wishing to resist current power structures in order to attain a *post-racial* society, which remains an elusive social vision to characterize (e.g., Mills 1998; Taylor 2015).

Conclusion

As a species, we are closer to Unity than to Galápagos-writ-large. Moreover, there are multiple views on offer regarding the metaphysics and pragmatics of race. Should we stop racializing ourselves and abandon our conceptualizations and uses of biogenomic or biological race?

Perhaps not. First, a number of ongoing areas of biomedicine and forensics lean heavily on population genetics deploying biogenomic and biological race categories. For instance, health outcome disparities between racial groups in the United States are dramatic (Murray et al. 2006). Some researchers explain such disparities by hypothesizing factors in the average genome, so to speak, of distinct groups (Collins et al. 2003; Risch et al. 2002). Others argue that such systematic differences result from shared social circumstances, especially various consequences of racism. That is, "race becomes biology" via mechanisms of "embodiment of social inequality," such as "allostatic load"

(see Gravlee 2009; Kaplan 2010; see also Lorusso and Bacchini 2015). Less controversially, some kinds of racialized categories are critical for matching potential donors in stem cell/bone marrow transplants (Hacking 2005; Bergstrom et al. 2009). Second, DNA forensics via genetic profile matching on related individuals is used increasingly throughout the world. Lest misidentifications occur, DNA forensics requires knowledge of the background population from which individuals are sampled, or statistical methods for correcting the lack of such knowledge (Rohlf et al. 2012).

Many biological researchers of good will are astutely aware of the potential discriminatory and oppressive social effects of biomedicine and forensics. Even so, there are clearly many legitimate and historical reasons to worry that at least some forms of research on biogenomic and biological race could perniciously exacerbate social problems, as we saw in the critiques of the hereditarians by Lewontin and others. Vigilance, dialogue, and mutual co-teaching are crucial in the complex area of race and biology.

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