Race and Biology

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Chapter for Linda Alcoff, Luvell Anderson, and Paul Taylor (eds.). *The Routledge Companion to the Philosophy of Race*. London: Routledge.
August 13, 2015

Abstract
The ontology of race is replete with moral, political, and scientific implications. This book chapter surveys proposals about the reality of race, distinguishing among three levels of analysis: biogenomic, biological, and social. The relatively homogeneous structure of human genetic variation casts doubt upon the practice of postulating distinct biogenomic races that might be mapped onto socially recognized race categories.

Comments Welcome.

1. Introduction

Imagine landing in the largest city, the capital, of an alien planet. As you disembark, 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 more surprising to you, everyone has purple skin, a consequence of interacting skin pigment proteins, the double-sun of that planetary system, and generation upon generation of voluntary random breeding, the ambassador tells you. This ambassador accompanying you as you head for your important meeting also informs you that every adult humanoid on the planet looks like that, as indeed she does. Call this planet "Unity."

Now recall the variety of finch and tortoise species on the 19 islands of the Galápagos, Darwin's natural experimental laboratory of evolution that you learned about in high school biology. But let us 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 incommunicable, and throw in a few million years of evolution. Humans on these distinct islands will come to look and 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 therefore to explore this level. In particular, seven theses on human genetic variation are explored below in order to 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 "logical core" of the "ordinary concept of race" involves identifying a "group of human beings" "(1) distinguished from other groups… 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, 451-2).

After laying out the facts, I consider their biological and philosophical implications. I survey proposals about the metaphysics of racial "kinds of people" (Hacking 2007a), organized around *biogenomic*, *biological*, and *social* levels (Kaplan and Winther 2014). Theories of racial metaphysics 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 important variables?
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, e.g., racial, national, or ethnic designations and kinds (Winther and Kaplan 2013; Kaplan and Winther 2014; on kinds see Hacking 2007b, Kendig 2015). Most practitioners do not take their genomic work on human populations to be about race (Coop et al. 2014; comments during "Philosophy in a Multicultural Context" events: 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's chapter in 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, Nielsen 2015).

Biological races exist when a stable correlational or, better yet, causal mapping can be drawn between group genetic differences and socially significant 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).

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 pernicious effects of employing racial categories, and of the existence (or not)—or at least the perception of 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 normativity, 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 a pragmatic, conventionalist, or more sociological approach would argue, is a difficult matter also bracketed in this entry (Spencer 2012; Ludwig 2015; Winther, Millstein, Nielsen 2015)

2. 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 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, often used coextensively with *gene*, and often but not necessarily functional; an *allele* is one of various versions of a locus (or gene), differing with another allele at one nucleotide or more, 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 are the sex chromosomes, our mitochondria also have 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 a function of allele-frequency change within and across populations and species. Population geneticists subject 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 population genetics can be summarized in the following seven theses:

1. Low nucleotide diversity

2. Relatively small genomic differences between *Homo sapiens* and our nearest allies, e.g., chimpanzees and bonobos

3. Widely distributed alleles

4. Non-African variation is basically a subset of African variation

5. Most of what little genomic variation there is in our species, is among individuals within populations, and not between populations

6. Even so, clustering and classifying individuals in groups 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 significant 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 1000 (Li and Sadler 1991; Yu et al. 2002). Given a total genome size of 3 billion nucleotides, and an average difference of about .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 .077%, chimpanzees by .134%, and gorillas by .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. **Relatively small genomic differences between *Homo sapiens* and our nearest allies, e.g., chimpanzees and bonobos.** Average across-genome nucleotide identity between humans and chimpanzees is 98.77% (i.e., 1.23% nucleotide divergence), between humans and bonobos is 98.69%, and between humans and gorillas is 98.36% (Yu et al. 2004). Even human and mice genomes are roughly 85% identical (Batzoglou et al. 2000). The 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 which nucleotide sequences can be traced back to common ancestors and which converged independently need to be made (Dicks and Savva 2007; Gerstein et al 2007; Hahn et al. 2007).

3. **Widely distributed alleles**. Most populations contain most of the common alleles present in the human population. Over 90% of common alleles (i.e., alleles not private to 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 (Rosenberg 2011). Over

82% of common alleles are found in three or more regions, and approximately 47% of common alleles are found in *all* regions.

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 sub-set 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 (.11%), whereas two people whose recent ancestors are of European origin differ on average only by approximately 1:1600 (.063%) (Yu et al 2002; Campbell and Tishkoff 2008; Wall et al. 2008). Africa also has approximately half of the total number of geographic "private alleles"—i.e., types of genes at a locus that are unique to a particular region—with the rest distributed across the other six regions; note that only about 8% of all common alleles are private (Rosenberg 2011). As a third measure of variation, consider the distribution of all the approximately 8000 common alleles surveyed in Rosenberg (2011). Of these, roughly 82% were found in Africa, much more than any other single continent. Furthermore, to a first approximation, most common alleles (87%-90%) found in one non-African continent were also found in Africa, but not the converse. For example, only 74% of common alleles observed in Africa are also observed in Europe, and only 63% of common alleles identified in Africa are also located in the Americas (*Figure 1*; Rosenberg 2011). Indeed, the number of common alleles diminishes as we move farther from Africa, in the following ranked order: Middle East, Europe, Central/South Asia, East Asia, Oceania, and America (*Figure 1*). A final measure of genetic variation relevant here is heterozygosity, which is a measure of how evenly distributed alleles at each locus are in a given population. 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 2*; Ramachandran et al. 2005).

The loss of (i) nucleotide diversity, (ii) private alleles, (iii) common alleles, and (iv) genetic heterozygosity as we move away from Africa can be explained in terms of a "serial founder effect" model. As *Homo sapiens* migrated out of Africa we went through a series of genetic bottlenecks in which small 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 related 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?

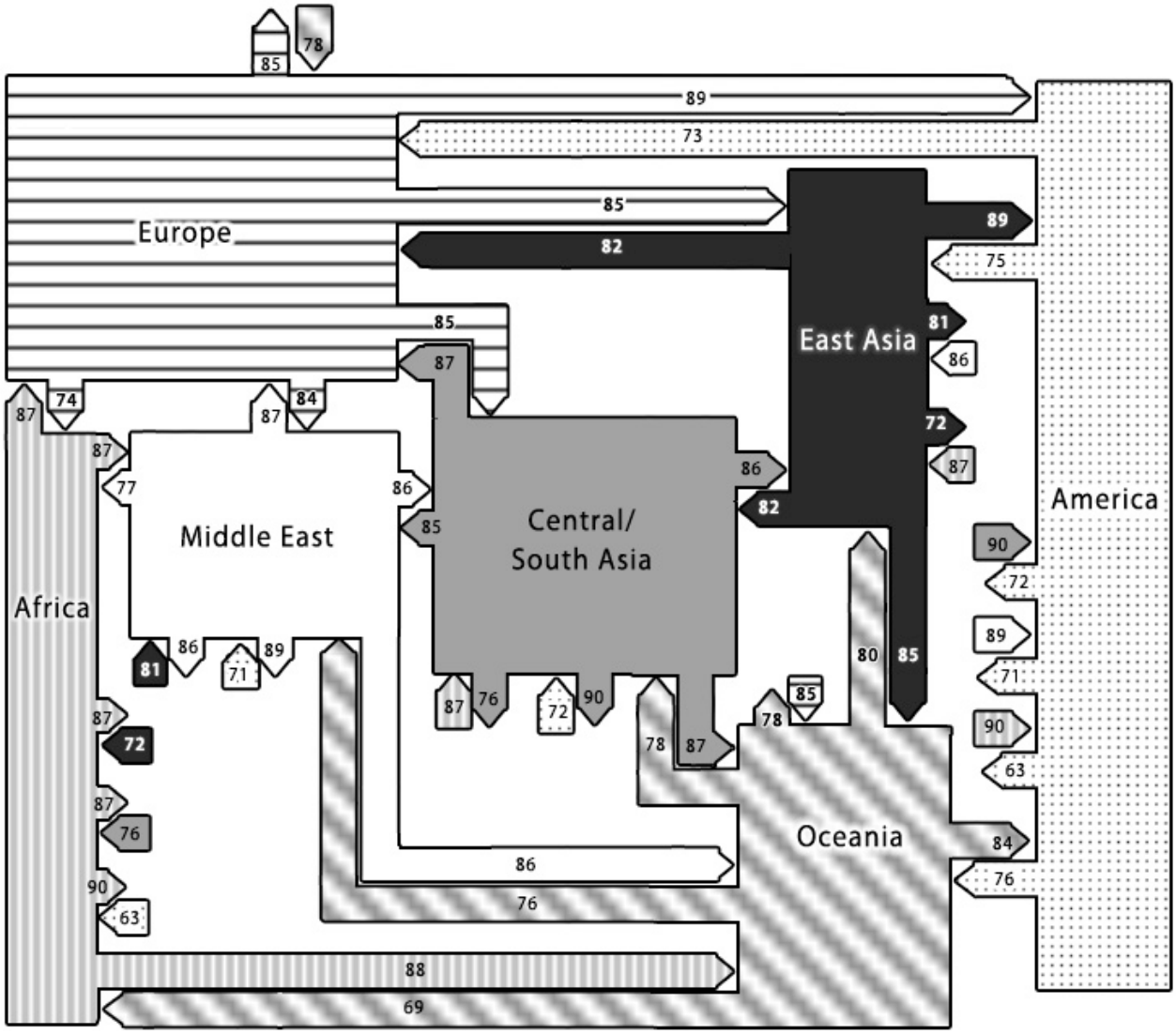

Figure 1. "Schematic world map of the 'flow' of microsatellite alleles. …boxes represent regions of the world, positioned geographically. Links entering into a geographic region indicate the percentages of distinct alleles from the geographic region found in other regions… For example, averaging across 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.)

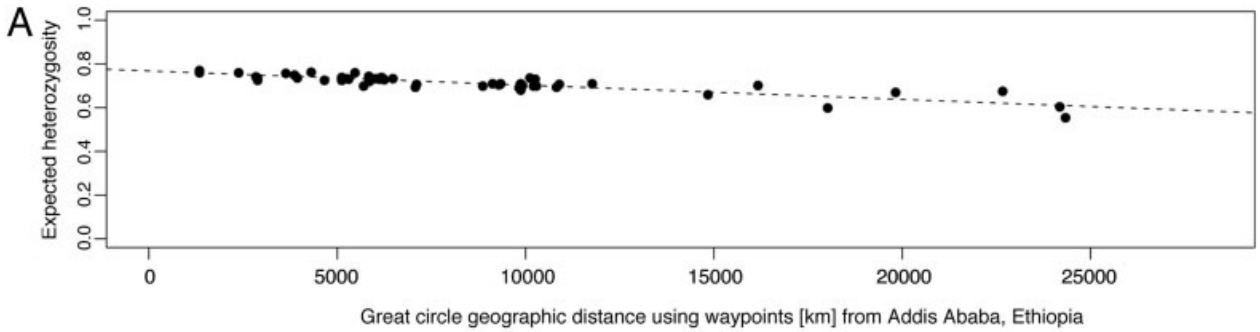

Figure 2. The amount of heterozygosity of each of approximately 40 worldwide populations diminishes as a function of their respective distance, along (approximate) migration routes, from Addis Ababa (Source: Figure 4, Ramachandran et al. 2005, 15946).

5. **Most of what little genomic variation there is in our species, is among individuals within populations, and not between 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 within the same continental region or from another continental region? Contrary to the expectations or prejudices of some, the simple answer is that you are almost as genetically different from any randomly chosen member of your group of ancestral origin as you are from any random person chosen from basically anywhere on Earth. Lewontin (1972) was the first to address this question, 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. These results imply that at most variable loci, different human groups tend to have similar allele frequencies. Thus, not only are alleles widely distributed (#3), they are also fairly uniformly distributed across populations. More technically, Lewontin's measure shares important theoretical properties with the standard comparative heterozygosity F_{ST} measure developed by Wright (1931), which is a measure comparing expected and actual heterozygosities of populations or (equivalently) within and across populations (Hartl and Clark 1989; Holsinger and Weir 2009; Nielsen and Slatkin 2013; Winther 2014). More completely, Lewontin found that the total global heterozygosity could, on average, be (approximately) divided or partitioned thus: **5%** at the continental region level ("Negroid," "Caucasoid," and "Mongoloid" in Lewontin 1972 terms); **10%** at the across-population level, within continental regions (e.g., Ghanaian, Kikuyu, Tutsi, Zulu), and **85%** within populations (e.g., Kikuyu). Table 1 shows an example using data from Lewontin (1972) and Cavalli-Sforza and Bodmer (1971). The *Duffy* gene is an atypical example, as it is more extremely diverged than average (e.g., .03%:94%:.1% for one of three alleles). The *Auberger* and *Xg* genes are much more typical of the human genome, since allele frequencies are fairly similar across "races," and there are few private alleles (#4).

Gene	Alleles	Caucasoid	Negroid	Mongoloid
Duffy	*Fy*	0.03	0.94	0.1
	Fya	0.42	0.06	0.9
	Fyb	0.56	0	0
Auberger	*Aua*	0.62	0.64	
	Au	0.38	0.36	
Xg	Xg^a	.67	.55	.54
	Xg	.33	.45	.46

Table 1. Allele frequencies of three distinct genes across continental regions, as used in Lewontin (1972) and (1974) and Cavalli-Sforza and Bodmer (1971), cited in Lewontin (1974). Frequencies are rounded from four to two significant figures. Empty cells indicate lack of data. See especially Lewontin (1974), 152-157.

Lewontin's initial numbers have held up across subsequent studies, although the percentages vary a bit, often with a higher within-population percentage partition (Barbujani et al. 1997; Rosenberg et al. 2002; Rosenberg 2011; Li et al 2008). In short, because human F_{ST} is relatively low, between .05 and .15 (i.e., most heterozygosity is within populations), two individuals from the same population are almost as likely to differ genetically at typical loci (e.g., *Auberger* and *Xg* rather than *Duffy*) as two individuals chosen from any two random global populations. Remind you of Unity?

6. **Even so, clustering and classifying individuals in groups is reliable.** Even if most variation is within populations, if we accumulate information across loci, rather than averaging across loci, then clustering and classifying individuals is reliable. Despite the relatively small allele-frequency differences between human groups (#5), it is possible to pool information from many loci to make reliable inferences about both the ancestral populations that exist (clustering) and the population membership(s) of any particular individual (classifying) (Rosenberg et al. 2002; Edwards 2003; Tal 2012; Edge and Rosenberg 2015; Rosenberg 2016). A computer program, *Structure*, was designed to do this (Pritchard et al. 2000). For example, consider 100 loci spread out across the genome where 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 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 increase the number of loci sequenced. After all, each new locus is analogous to another coin toss, and, roughly speaking, the more times we toss a biased coin, the closer we get to the actual bias frequency (e.g., .4 in population *A*). Thus, given even small differences in allele frequencies at each locus for different populations, by examining enough loci, we can use this procedure to become as confident about population membership as we like. Even though populations are quite similar, accumulation of small differences makes it possible to classify individuals as originating from discrete population clusters. 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.** Although genetic differences between any two populations are relatively small, populations that are farther apart are more genetically dissimilar. If we plot F_{ST} pairs of world-wide populations against the geographic distance between population pairs, a clear and smooth linear inverse correlation is found (*Figure 3*; R^2 = .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 more different their allele frequencies are for typical loci. Moreover, this change tends to be clinal, viz., varying smoothly, 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 (Ramachandran et al. 2005; Rosenberg et al. 2005; Rosenberg 2011).

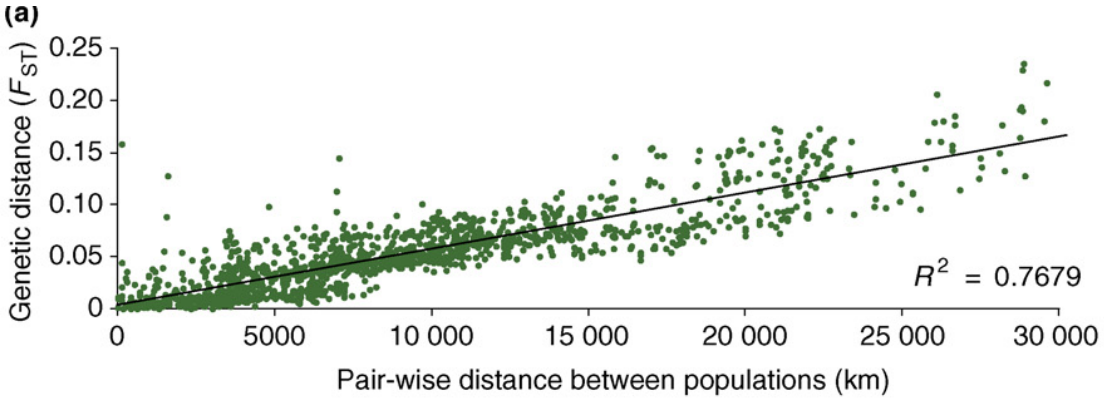

Figure 3. High correlation between pairwise F_{ST} and pairwise geographic distance of world-wide populations. (Source: Figure 1a, Lawson Handley et al. 2007, 435)

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.

3. Biological Implications

These basic patterns or features can be used for further biological inferences. Trees can be drawn that represent our evolutionary history. Since a variety of phylogenetic tree-building methods are available (Felsenstein 2004) 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; see the first population-genetic-based map, Edwards and Cavalli-Sforza 1964, 75). At higher levels of granularity, a *trellis*, *network*, or *reticulate* rather than a *tree* model of human evolution seems more plausible (Templeton 1999, 2002; see Winther and Kaplan 2013, pp. 63-6 "Phylogenetics: Getting out what you put in?"; 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 such 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 to 148 thousand years ago. Similarly, the *Y-chromosomal Adam*, copies of whose Y-chromosome exist in every man today, lived anywhere from 120 to 338 thousand 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. Specifically, some are targets of local selection, in which different alleles are favored in distinct parts of the globe. An example is the gene *SLC*24*A*5, which influences skin color variation. The ancestral allele, strongly associated with dark skin in Africans, is practically fixed—that is, present in every chromosome—in most African (and most Asian and Southeast Asian) populations. The derived allele, highly correlated with light skin in Europeans, 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, *SLC*24*A*5 was one of only "twenty-two strongest candidates for natural selection" (Sabeti et al. 2007, 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 *FOXP*2, 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 epicenter of interest. Might some of the genes for cognitive abilities or certain diseases lie in the relatively small fraction of private common alleles (8%) or across-continental heterozygosity (ca. 5-10%)?

4. Philosophical Implications

Philosophers interested in biological aspects of race tend to focus on on two issues: (1) concepts of race and (2) the metaphysics of race. The latter efforts can be seen as endorsing three positions at three levels: realism, anti-realism, or 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) is concerned with whether human sub-populations should be admitted as legitimate biological entities (i.e., the biogenomic question above) and whether these at

least sometimes correspond to socially entrenched categories of, e.g., racial, national, or ethnic designation (i.e., the semantic question above). One concern here is whether we can use standards and practices employed by biologists in other domains (e.g., conservation biology; ecology, Winther and Kaplan 2013) to identify human sub-populations worthy of biological attention.

In contemporary literature, Sesardić (2013) seems to accept the existence of biogenomic races as the primary question for biological racial realism, although under my analysis these are two separate questions. 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 properly 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 2017). Both conventionalism and reification can be interpreted as lying either between realism and antirealism, or perhaps beyond or outside of any spectrum with 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 the fact sheet above.

The sticking point is about the reality (or not) of *biological* race. Again, the entire issue of biogenomic groups, populations, or races, as it were, would not be so politically, socially or morally challenging if nothing rode on it (Helen Longino pers. comm.). If putative group membership only determined relatively 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 sustained 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 due significantly 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) effectively deny the existence of biological race. Another option is to withhold judgment until individual genes for socially and morally significant traits such as cognitive abilities are identified and clear and explicit selective scenarios and mechanistic penetrance established. Especially the discourse around biological race highlights the *pragmatics* of race, viz., 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, measurable and experienced by extant systematic discrimination and oppression. However, some of these same social racial realists at least 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, Kaplan and Winther 2013; Taylor 2014).

5. Conclusion

As a species, we are closer to Unity than the Galápagos-writ-large. Human genetic variation exhibits strong overlap across even continental regions, and certainly within continents. Moreover, there are a broad variety of views on offer regarding the metaphysics and pragmatics of race. Should we stop racializing ourselves, as it were, 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 USA 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 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). 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 lack of such knowledge (Rohlfs 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 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.

Acknowledgements Paul Taylor kindly invited me to contribute to this volume. Quentin Coudray, Michael D. Edge, Joseph Hendry, David Ludwig, Lucas McGranahan, and Mette Smølz Skau critiqued earlier versions of this chapter. Exchanges on these topics with Anthony Edwards, Ian Hacking, Jonathan Kaplan, Richard Lewontin, Helen Longino, Carlos López Beltrán, Amir Najmi, Rasmus Nielsen, Noah Rosenberg, Bøllemis Skau, Omri Tal, and Michael J. Wade are very much appreciated.

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