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Anthropology, Race, and the Genome

The Profound Relevance and Irrelevance of Biology*

By *Jonathan Marks*
UNC-Charlotte

With racialized medicine and behavioral genetics everywhere—in the news, in popular science forums, and in funding opportunities—it is incumbent once again upon biological anthropology to attempt to bridge the gap between biology and anthropology. Although many biological anthropologists don't foster a connection between their interests and the rest of anthropology, with some actually believing the genetics hype and some even believing the racialized hype, there is a faithful remnant.

Anthropology, and especially biological anthropology, has some positive knowledge to offer on three things that have been in the news the last year: racial genetics, behavioral genetics, and the quest for the genes that make us human.

One of the most interesting paradoxes of the 1990s Human Genome Diversity Project (HGDP) was that the project tried to maintain a politically correct line that races don't exist, while simultaneously producing labeled figures that showed Australians are Red, Mongoloids are blue, Africans are Yellow, and Caucasoids are green. If races don't exist then what could that possibly mean?

The answer is obviously that the authors committed the fallacy of Linnaeus, namely, taking the most extreme populations as color-coded avatars, and calibrating the differences accordingly. But this goes Linnaeus one better, for Linnaeus was work-

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ing within a Platonic framework, which the geneticists weren't. Rather, they were simply translating quantitative variation into qualitative differences. If you make Africans a single color, whether yellow as in this high tech genetic study, or more popularly black, then it certainly follows that people assigned to a different color might well have different properties.

Anthropology certainly has something to offer here, such as the relativizing observation that the decision to lump Africans together as a single category is highly arbitrary, subsuming most of the genetic variation in the human species, and grouping together people who are physically and genetically quite different. A further observation is that populations straddling the color boundaries are more similar to one another than they are to other, more distant populations of the same color assignment.

But most importantly, saying races don't exist as discrete biological units is not the same as saying races don't exist. Jews and Muslims, Catholics and Protestants, exist and act regardless of whether their gods do; and very little of American social history and politics can be understood outside the context of race, regardless of whether it is a biological unit or not. The diversity project went so far as to tell a naive constituency that they were going to diminish racism, as if racism were predicated on the biological reality of race, and group-level hatreds could be ameliorated by a simple cold shower in the truths of genetics.

Even though the HGDP project collapsed, the haplotype mapping project was organized with its brain trust remaining intact in Palo Alto and New Haven, and using cell lines in Paris as a basis for their newer studies. In its most widely publicized discovery from nearly a year ago, a new mathematical analysis of the genetics in their genetic repository permitted the division of the populations of the world to be divided into whatever number of groups they asked for. When they asked their computer to divide the world's genetic variation into two groups, the result broadly separated Europe, Africa, and Western Asia from the Far East, Oceania, and the New World. When they asked the computer to divide our species'

gene pool three ways, they got Europe and West Asia; East Asia, Oceania and America; and Africa. A four-way partition distinguished the native Americans from the Asians and Oceanics. When asked to divide the world into five, the computer gave results corresponding to the peoples of Africa, Eurasia, East Asia, Oceania, and the Americas, that is, the continents; and when asked to divide the world into six, it separated out the Kalash people of Pakistan from those five.

On the face of it, this finding would seem to lend no support to popular ideas about race: the Kalash—whoever they are—are hardly biologically equivalent to the Africans; the number of groups is set by the programmer, not found by the computer; and there is certainly nothing racially commonsensical about juxtaposing Europe and Africa against East Asia, Oceania, and the Americas, artificially bisecting the human gene pool.

Two observations are in order here. One: paradoxically the authors of the HGDP are the same people who used to say they would demonstrate that there was no basis for race, and are now saying there is. And two, the sources of funding have changed: where earlier the pot was filled with government gold, the rainbow now seems to end at the doorsteps of pharmaceutical companies, whose goal is to develop drugs targeted for specific divisions of the human gene pool. And what more obvious way to divide the human gene pool than racially?

Consequently, when another group of population geneticists (led by Neil Risch of Stanford) goes public with the simple pseudo-discovery that there really are races, we shouldn't be terribly surprised. They do so partly by perversely using "population," "race," and "ethnic group" synonymously, although the first term is supposed to be local, the second global, and the third cultural in designation. If you fail to acknowledge that human groups are fluid, hierarchically organized, and symbolically bounded, it's hard to imagine that the science you do will be of much value, regardless of the extent or pattern of biological variation in the human species.

The point is that the nature and quality of the data hasn't changed appreciably, nor

have the kinds of analyses.

Money is nice, but there are a few isolated instances in the history of Western civilization where it has turned out to be something of a corrupting influence. The structure of human variation, I would argue, has become a site not so much for empirical studies by biological anthropologists, as for general anthropologists interested in the production of knowledge.

As "Deep Throat" told Woodward and Bernstein many years ago, "follow the money." In these postgenomic times, it is useful to remember that 95% of everything a geneticist says is code for "give me more money." My personal favorite is: "These results are preliminary but more research is needed." If the results are preliminary, they should not be quoted or published until they are firmed up. And further, the plea for validation is shamelessly self serving.

In human behavioral genetics, everything is preliminary, more research is always needed, but somehow it rarely if ever seems to support the original contention. But by the time the rest of us discover that, the original contention has made it into the realm of folk knowledge. Human behavioral genetics is something of an odd field in that it has rather few practitioners, and most of them aren't even geneticists, but rather it has a lot of interested parties. And there is a convergence of interests in human behavioral genetics that makes it a particularly powerful site of knowledge production. First, people who believe that group-level differences in performance, or deed, or thoughts are ultimately innate will be attracted to human behavioral genetics. We may call those people racists, as they begin with a folk belief about the reality of bounded human groups, and add to that belief value judgments about the qualities of the members of those groups.

Behavioral geneticists focus on the genetic differences between groups, which we already know to comprise between one-seventh and one-twentieth of the detectable genetic variation in our species. To argue that any particular behavioral difference between groups is genetically based requires some considerable empirical evidence. It would have to be demonstrated that this particular genetic difference is patterned unlike the great majority of genetic diversity

in our species.

Avoiding this latter pitfall is the second group, the old fashioned hereditarians, who commonly now gather under the large umbrella of evolutionary psychology and tend to focus not so much on group-level differences in behavior, but on personality traits that vary within groups. The major axis here is between those who emphasize the genetic differences as causal agents of diversity in human thought and deed, and those who smooth over the diversity and extract an ideal form, and then proceed to speculate wildly about how we haven't really evolved, but are stuck genetically in a Pleistocene of their own invention. In general, the evolutionary psychologists are zealots for adaptation, yet rarely acknowledge that only a subset of adaptations are in the strict sense evolutionary. Consequently it is never quite clear what evolutionary psychologists believe about the structure of the human gene pool, which is where the effects of Darwinian adaptation accrue.

Noting how the hereditarians complement their agendas, are the third group, consisting of right-wing ideologues like Charles Murray, who discovered genetics late in life. And like the political allies of the eugenics movement decades ago, they appreciate the convergence of interests between the work of this category of ostensible science and a justification of their own social and political goals.

Some of the most widely cited work in this area comes from the studies of identical twins separated at birth. But taking inferences about behavioral genetics from identical twins separated at birth at face value is comparable to taking 16th century ethnographies by missionaries at face value. There may be a kernel of truth there, but it is buried deeply under layers of assumptions, belief systems, and conflicts of interests. Nearly all of what is cited these days comes from the University of Minnesota's Twin Study, run by psychologist Thomas Bouchard. The most famous of these is the Jim twins, who were reunited at age 39, and discovered that they had been given the same name,

married women with the same name twice, gave their sons and their dogs the same name, etc. It is hard to know whether these are intended even to be genetic narratives or ESP narratives, and probably they are both, which should suffice to invalidate them.

If we follow Deep Throat's advice again and follow the money, we see that Bouchard, a former protege of Arthur Jensen, began the study thanks to the largesse of the Pioneer Fund. The Pioneer Fund is now administered by Philippe Rushton, who needs no introduction for his beliefs that Africans evolved to be promiscuous and stupid, Asians evolved to be chaste and brilliant, and Europeans evolved to a happy medium.

The more cynical among us might speculate that those interested in twin studies expect to help establish the inheritance of personality traits within groups, and extrapolate from that to the innateness of racial behaviors. Consequently it is always useful to examine the basic patterns of human cognitive and behavioral variation and compare them to the basic patterns of human genetic variation. The result is that most genetic variation is clinal and differentiates people within groups; but most cognitive or behavioral variation is discrete and differentiates the groups themselves from one another, and by consequence, their members. Rates of change and immigrant acculturation studies teach us that the differences in thought and act that differentiate groups from one another are cultural and cultural differences are not rooted in the genetic differences between the groups, but rather, are external or extrasomatic.

This has two important consequences: First, the different patterns evident in the variation in human genetics and human behavior makes it very unlikely that the latter could be a significant cause of the former. And second, if culture is the major component of variation in human behavior and reflects nongenetic differences, then behavioral genetics has carved for itself a very small piece of the pie that is human cognitive or behavioral variation.

The behavioral genetics puzzle involves some real geneticists working on the problem. I once saw a scientist working on the genetics of alcoholism in the Navajo challenged as to how large a part of the alcoholism problem on the reservation he was actually tackling. Without batting an eyelash, he volunteered "less than 5%." And when the challenger demanded to know why he was investing his time and the government's resources to the genetic study of less than 5% of the problem, he lamely replied, "because I'm a geneticist—that's what I do."

Two genetical fallacies permit hereditarian propaganda to persist: first, the invalid extrapolation of patterns of within-group variation to between-group variation; and second, the invalid extrapolation of patterns of pathological variation to patterns of normal variation.

What follows are some examples that illustrate the relationship between pathology and normalcy. It is generally accepted that the range of normal height, while obviously affected by diet and other environmental factors, is affected by a handful of genes. Nothing is known about them, but they can be invoked and modeled as if they had additive effects. Achondroplasia is a genetic disease that affects the connective tissue, resulting in dwarfism. The gene is located on chromosome 4, it obviously affects height, but it is not one of the handful of genes. Only one in 25,000 people has achondroplasia, but many people are short. To understand the genetics of height, you don't need to know anything about the achondroplasia gene; it merely fouls up the complex developmental processes and results ultimately in body size outside the normal range of variation.

Likewise, many people mutilate their bodies in diverse ways, from biting nails to tattoos, scarification and footbinding to circumcision, castration and clitoridectomy. Lesch-Nyhan Syndrome is caused by a gene located on the X chromosome, which when mutated in 1 of 100,000 boys causes a buildup of uric acid that is not

properly excreted and somehow causes them to bang their heads and bite their lips and fingers uncontrollably, i.e., a self-mutilation syndrome. What does Lesch-Nyhan have to do with self-mutilation in the human species? Absolutely nothing. The great majority of instances of what we would identify as self-mutilation occur in the total absence of the Lesch-Nyhan mutation.

So, like shortness, self-mutilation is a phenotype that is widespread among completely normal people. The fact that genes have been identified that cause a pathological imitation of a feature or behavior has no bearing at all on understanding their etiology as they are expressed in the great majority of normal human beings. Likewise, the allele for monoamine oxidase A deficiency may well be a violence allele, but most instances of human violence have nothing to do with it.

Another example of pathology and normalcy involves FOXP2, a so-called language gene discovered several years ago. The original report was called, "A forkhead domain gene is mutated in a severe speech and language disorder." If you have this mutation, you have problems speaking and understanding properly. When the gene was first announced, reporters claimed "First Gene Linked to Speech Identified." In some sense, this is uncontroversial: if we had the genes of a cow, we would go "moo" and if we had the genes of a dog we would go "woof woof," but we have the genes of people, and so we are going blah blah blah anthropology blah.

But what does FOXP2 have to do with speech? It is identical in people who speak Russian, Kikuyu, and Post-modern; it is identical in orators and stutterers; it is identical in people who got 800 on their SAT verbal and people who got 400. It has nothing to do with the normal range of variation in human vocalauditory communication.

In another research project concerning apes and speech, the researchers compared the gene across species. Knowing that language is something that separates

us from the apes, the researchers sequenced it in different primates. They found that of 715 amino acids, the sequence of rhesus monkey, chimp, and gorilla were identical and were one amino acid away from the mouse, but there was a mutation in the orangutan protein and two in the human.

One of the investigators conjectures that "Maybe this gene provided the last perfection of language, making it totally modern." The gene became, in its least circumspect form, "the best candidate yet for a gene that enabled us to become human," as one geneticist gushed; and in its most circumspect form, just one of the genes that evolved to permit the emergence of speech and language.

In a wider context, if the question is what genetic changes enabled us to become human, the answer is all of them. Every genetic change that diagnoses a human genome from an ape genome contributed to making us human, whether it is something unexpressed, like the fusion to form human chromosome 2, or expressed, like whatever gives us longer legs than arms. They all make us human.

Do two amino acid changes in the human lineage of FOXP2 make it a good candidate gene for phonemes, morphemes, and syntax in language and speech? Answer, well, as good as anything. But important genetic differences are unlikely to be translated directly into important normal phenotypic differences. The big buzzword these days is "epigenetics," the differential expression of genes that is not a consequence of different DNA sequences. It raises the question of that mutation in the orangutan, a species that appears to be as mute as chimpanzees in spite of having an amino acid difference in this gene. It also raises the question of the rhesus macaque, gorilla, and chimpanzee, all having different cognitive functioning and different vocalizations in spite of the same FOXP2 sequence.

We end up with a gene that fouls up either the cognitive or the productive aspects of human communication or both,

is highly conserved in mammals despite wide differences in their communication and cognition, and has accumulated two differences in the human lineage and one in the orangutan lineage. Since language is one of the things that separates us from the apes, although it doesn't separate orangutans from anybody, this is touted as a relic of the evolution of language, assuming there is a direct and simple translation of a genetic change into a normal phenotype.

Everywhere we look we will find small subtle differences between the human genome and the chimpanzee genome. Why? Because we are not chimpanzees. A gene that hypothetically causes deformed knees wasn't necessarily involved in the development of bipedalism, any more than the hundreds of genetic syndromes known that manifest mental retardation as a pleiotropic phenotype should be considered integral to the development of the large brain that also physically marks us in relation to the apes.

Conclusion

Understanding our biology is paramount to understanding our anthropology, but much of that understanding must come from circumscribing biology, or at least from circumscribing what is commonly represented as biological knowledge. One could reasonably argue that understanding our anthropology is even more paramount to understanding our biology. Perhaps to counter the rhetorical power of the genome and its study, we might relabel the human species as the anthropome and establish a field of anthropomics, the holistic and thoughtful study of human diversity as a more scientific-sounding counterweight to the reductive and conflicted, if well-hyped and well-funded, genomics.

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