ARTICLES

Apportionment of Racial Diversity: A Review

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It has become increasingly popular to theorize and assert significant genetic differences between arbitrary regional, ethnic, and racial groupings of humans. Beginning with Livingstone, Brace, and Newman is the early 1960s, biological anthropologists have shown that variation in human traits is *non-concordant* along racial lines, as they are products of overlapping, dynamic selective pressures.

In 1972, Lewontin analyzed blood groups, serum protein, and red blood cell enzyme variants and found that only about 6% of total genetic variance was accounted for by race, while the majority of variance is accounted for by differences *between individuals.* Using similar assays, Latter obtained similar results in 1980. In 1982, Nei and Roychoudhury analyzed 62 protein variants and 23 blood groups, finding that roughly 10% of genetic variance was accounted for by race. Analyzing protein, blood group, and HLA variants, Ryman and coworkers obtained similar figures in 1983. More recently, Dean and coworkers (1994) and Barbujani and coworkers (1997) have used PCR techniques to analyze RFLP and microsattelite loci, again yielding estimates of around 10% for the amount of genetic variance accounted for by race. Furthermore, recent research on regional and racial variance in mtDNA (Excoffier and coworkers, 1992), a traditional marker for human racial groupings, shows a higher proportion of variance *within* than *across* racial categories.

These studies used a variety of assays and analytical techniques, some of which are designed to *maximize* the amount of variance accounted for by race. In light of this, the low proportion of genetic variance across racial groupings strongly suggests a re-examination of the race concept. It no longer makes sense to adhere to arbitrary racial categories, or to expect that the next genetic study will provide the key to racial classification.

In the last 30 years there has been an assault on the race concept.[1–35] Many anthropologists question the usefulness of the race concept and have discarded it as a research and teaching tool.[36–39] Even use of the race concept in textbooks is declining.[39] Only 50% of physical anthropologists and 31% of cultural anthropologists accept the validity of biological races in *Homo sapiens.*[40] There are many reasons for the decline in acceptance of race as a means of understanding human variation. Some claim that anthropologists are under pressure to maintain a politically correct position.[41–43] However, one of the underlying reasons for the decline is the arbitrary nature of racial classifications: The boundaries between races depend on the specific traits used and the classifier's own cultural norms.[11,25] Other reasons include the lack of correlation of traits used in classification[11,25] and the existence of alternative methods for explaining human variation.

The success of racial classification depends on the number of traits used in ordering the races.[25] A single trait such as skin color will result in a classification system that is easily determined. Add another trait and classification becomes a more difficult task, and there usually are groups that cannot be classified. As you increase the number of traits, the problems in racial classification become insurmountable.

Newman[44] used genetic traits as a means of systematically testing the validity of Garn and colleagues'[45,46] classification of geographic and local races. He selected a number of traits and systematically determined whether or not they clustered. Newman found that three of the geographic races (Asian, Amerindian, and African) appeared to "stand up well," three (Melanesian, Polynesian and Micronesian) fell within the categories of "suspense account" and "may be valid but the critical data is lacking," and three (European, Indian and Australian) were labeled "unwarranted abstraction." He commented that Garn and coworkers' list of local races "har-

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bor[s] many conceptual left-overs from the days of typological thought in racial anthropology" (p. 192). Newman was pleased with his results and said that with more study and the discovery of new genetic traits racial classification would be refined. Unfortunately, he appears to have missed the implication of his analysis and the issues it raises for racial studies.

Newman unwittingly had discovered that racial traits are nonconcordant; that is, there is no agreement between traits used in racial classification.[12] If there is concordance, every trait will result in the same classification. For concordance to occur, each trait must be selected for at the same rate and in the same direction. In reality, genetic traits are evolving at different rates and in different directions, and consequently become nonconcordant. In practice, racial classifiers have to select the genetic traits and morphological features that support their preconceived notions of race. Racial lines have been drawn along the axes of aggression,[47] sexual behavior,[48] intelligence,[49–53] athletic ability,[54–58] and just about every other behavioral and psychological characteristic one can think of. Often, these supposed racial differences are presumed to be genetic. However, there is a major line of evidence that makes claims about racial differences in aggression, sexual behavior, intelligence, and almost any other characteristic highly unlikely, if not completely untenable.

This evidence comes from research on how human variation is partitioned and distributed among individuals and groups such as nations and races. The apportionment of human genetic diversity shows that only about 5% to 10% of genetic variation can be accounted for by traditional racial classification. Research that supposedly classifies individuals by their racial origins using genetic data is methodologically flawed and ultimately is not very useful for understanding biological variability.

A host of studies has concluded that racial classification schemes can account for only a negligible proportion of human genetic diversity. In 1972, Richard Lewontin[24] published "The Apportionment of Human Diversity," the first serious attempt to determine the extent to which racial groups account for human genetic variation. Drawing on existing studies of the distribution of various biochemical markers in populations around the world, Lewontin compiled data for nine blood groups (represented by differences in immunologic response to a specific challenge), as well as eight serum protein and red blood cell enzyme variants. In the absence of polymerase chain reaction technology, these markers acted as proxies for actual genotypes; each variant was presumed to represent one allelic variant.

Lewontin used two sublevels of population structure in his analyses: races (Caucasians, Black Africans, Mongoloids, South Asian Aborigines, Amerinds, and Oceanians) and populations (Chinese, Navaho, Portuguese, and so on). Employing the Shannon information measure, a measure of the frequencies of alleles at particular loci, Lewontin analyzed the proportion of genetic variation accounted for by differences between races, between populations within races, and between individuals within populations. He found that racial classifications accounted for about 6.3% of genetic diversity, population differences accounted for 8.3%, and individual differences 85.4%. Thus, genetic differences between individuals have little to do with racial or ethnic boundaries.

Restriction enzyme and polymerase chain reaction techniques make obsolete use of "genetic systems" as a proxy for genotype because they allow researchers to gain a more direct measure of the genetic code. To many who supported the racial model, this was the scientific breakthrough that would prove their case.

Lewontin's analysis was met with disbelief. It was thought that if the "correct" genetic traits were used, race would be shown to be a major source of human diversity. Critics argued that if Lewontin had used the right genetic markers, his results would have been quite different.

Lewontin's results have been replicated by many studies, which in turn have used a variety of different types of data and different analytical methods. In 1980 Latter[59] used major geographical contiguity to classify six "major human subgroups," subdivided these major divisions into "regions" (northern, southern, and eastern), and subdivided these regions into populations (Greek, Zulu, and so on). Thus, Latter added an additional layer of population substructure to Lewontin's analysis. Just as Lewontin had done, Latter used published data on 18 "genetic systems" (ten blood groups, three serum proteins, and five enzymes). However, in addition to the Shannon information measure, he used two new analytical methods for quantifying genetic diversity: the proportion of shared genes between two randomly selected individuals and the probability that two randomly selected individuals will have different (nonidentical) genotypes. Using these three measures, 7.5% to 10.4% of genetic diversity was accounted for by "major geographical groups" (races), while 83.8% to 87% of genetic diversity was due to individual variation within populations. Interregional and interpopulation differences accounted for the remaining 5.5% to 6.6% of genetic diversity. Variation in percentages depended on the type of biochemical marker used (blood groups) versus enzymes.

In 1982, Nei and Roychoudhury[60] analyzed data from 62 protein loci and 23 blood group loci for "Caucasoid," "Negroid," and "Mongoloid" populations. Assessing the proportion of total genetic heterozygosity attributable to racial classifications, they found that this was roughly 9% to 11% (8.8% for protein loci, 10.9% for

blood group loci). Interestingly, despite these very low figures, the authors went on to discuss "the pattern of evolution of the three major races" (p. 11). This speaks to the logical disconnect shown by many researchers who simultaneously prove the irrelevance of genetic race and then proceed to discuss the genetic evolution of races. The findings of Lewontin, Latter, and Nei and Roychoudhury were so counter to conventional wisdom that they stimulated more researchers to seek the "right set of genes" and "the right analytical techniques."

Ryman and coworkers[61] replicated the previous findings[24,59,60] using two human lymphocyte antigen (HLA) types, nine blood groups, and 14 electrophoretically detectable protein types. As in previous studies, these data represented proxies for actual genetic loci. Ryman and coworkers searched the literature for data from 18 populations, which they subsequently grouped into six to seven more inclusive groups (races). An important point is that they created these broader groupings using two distinct methods. First they used traditional "anthropological races," based on classical racial typology; next they used a computer program designed to create groups using dendrograms of genetic relatedness.

Using the Shannon information measure, Ryman and colleagues[61] found that individual variation within populations accounted for 86% of all genetic diversity. The numbers ranged from 82.7% (blood group loci) to 90.3% (electrophoretic protein subtypes). Interestingly, use of the computational dendrogram slightly increased the proportion of genetic diversity attributable to the broader groups (an average of 1.3%) as compared to the use of classical racial typology. This is expected, given that the program was designed to maximize genetic differences between groups. It does not counter the claim that racial groupings account for a relatively minor proportion of total genetic diversity, given the small increase yielded by reorganizing the racial categorization scheme. Using a dendrogram to define races only increases the genetic diversity from blood groups attributable to race to 15.4%. This still represents only a minor proportion of total human genetic variation.

The salvation of those who believe in the importance of race in understanding human variability was a more basic measure of genetic structure. Restriction enzyme and polymerase chain reaction techniques make obsolete use of "genetic systems" as a proxy for genotype because they allow researchers to gain a more direct measure of the genetic code. To many who supported the racial model, this was the scientific breakthrough that would prove their case. Not surprisingly, the use of restriction enzyme and polymerase chain reaction to determine the portion of human genetic variability that is attributable to race yields results that are similar to those of past studies that used genetic traits. For example, Dean and coworkers[62] conducted a study of 257 restriction fragment length polymorphism loci in American Caucasian, African American, Asian, and American Indian individuals. Using Lewontin's[24] statistical techniques for approximating heterozygosity, as well as a technique that describes gene diversity more generally, they found that between 9.5 and 10.1% of the diversity in restriction fragment length polymorphism loci was attributable to racial categories.[62] Interestingly, comparisons of racial groups that allowed the use of more restriction fragment length polymorphism loci yielded much lower estimates of interracial variation, 4.4% to 4.75%. In a similar study, Barbujani and colleagues[63] collected and analyzed polymerase chain reaction data for 30 microsatellite loci and 79 restriction fragment length polymorphisms in 16 human "populations." When analyzed by major geographic group, these combined DNA data yielded 10.8% of the total genetic diversity. Only a single locus was found in which individual differences within populations accounted for less than 50% of genetic variation.

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A recent study by Hartmann and colleagues[64] claims to have found evidence of greater genetic diversity between than within races. This study sampled Hispanic, African American, and East Asian populations in Southern California, using polymerase chain reaction techniques to examine variation due to genetic differences in restriction fragment length polymorphism sites. The authors claim to have found evidence counter to the conclusions of Lewontin.[24] However, they base their claim on an analysis of restriction fragment length polymorphism size diversity that compares the genetic diversity between racial groups to that between regional groups, not between individuals. In doing so, Hartmann and coworkers[64] blatantly ignore the findings of Lewontin and all of the subsequent studies on population substructuring and the proportion of genetic diversity attributable to race. These studies showed that the majority of variance in genetic diversity is attributable to individual variation within racial categories, not differences between ethnic or regional groups. Hartmann and colleagues[64] do not perform the necessary analysis to determine what percentage of size diversity in restriction fragment length polymorphism is attributable to individual variation.

Recent analyses of the genetic apportionment of mitochondrial (mtDNA) and Y chromosome diversity complement and support the results obtained for autosomal genetic diversity. For example, Excoffier and coworkers[65] found that roughly 75% to 80% of haplotypic diversity in mtDNA resided between individuals within populations, with 15% to 22% attributable to regional (racial) origin. Seielstad and colleagues[66] found that roughly 81% of mtDNA diversity was attributable to individual differences within popu-

lations and that close to 16% was attributable to regional differences. The slightly higher proportion of mtDNA diversity attributable to continent of origin is likely the result of a lack of significant selective pressure on mtDNA (thus making convergent evolution on different continents unlikely), as well as its haplotypic pattern of inheritance. Nevertheless, intrapopulational differences still make up the bulk of mtDNA genetic diversity. This is remarkable, given that mtDNA is said to be the best marker of "racial origin."

A recent investigations of Y chromosome genetic diversity claims that individual differences within populations may account for as little as 35% of diversity in the Y chromosome,[66] with between-continent variation accounting for almost 53%. However, these figures hold only for single nucleotide polymorphisms on a nonrecombining segment of the Y chromosome. When microsatellite diversity is considered on parts of the Y chromosome that do recombine, individual variation accounts for more than 83% of Y chromosome genetic diversity. It is important to note that these apportionment figures were obtained from the analysis of a noncoding segment of the Y chromosome. Thus, relaxed selection pressures could lead to increased variation by continent, for the reasons described earlier. Likewise, the haplotypic nature of Y chromosome inheritance might also lead to increased intercontinental variation. Most importantly, neither mtDNA nor the Y chromosome DNA discussed previously code for anything functionally significant. Thus, these studies do not provide evidence of racial variation in behavior, intelligence, or morphology.

Not surprisingly, these results have not been well received by racial typologists and others who dislike the political implications of the lack of appreciable genetic divergence between races. Some have argued that Lewontin and others simply have not used "the right genes" in their analyses. For example, Miller[67] has argued that local frequency-dependent selection should increase the local diversity of HLA and other antigenic blood group types due to evolutionary arms races with infectious diseases, and that this dynamic biases results in favor of individual variation within populations. However, localized frequency-dependent selection does not preclude broader geographical (racial) differentiation. In fact, the differential geographical distribution of infectious disease types would likely favor racially based genetic diversity in these systems under Miller's[67] scenario of disease-based selection.

Lewontin[24] and Latter[59] present evidence that is even more damaging to Miller's[67] argument. They used a variety of nonantigenic systems, which generally yield even more conservative estimates of the percentage of genetic variation attributable to race. For example, Ryman, Chakraborty, and Nei's[61] results show that nonantigenic systems, in this case electrophoretically detectable proteins, exhibit the least racially contingent diversity. Furthermore, Ryman, Chakraborty, and Nei[61] suggest that these data are most likely to be representative of total genomic diversity due to closer mapping between codon differences and electrophoretically detectable differences. Given that these authors' figures for electrophoretic "loci" estimate racially contingent genetic diversity to be 5.4% to 7.3%, depending on the classification scheme, it appears that Lewontin's original figure of 6.3% is close to the mark. Notably, microsatellite and restriction fragment length polymorphism studies[62,63] also produced similar estimates.

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Some evidence presented in genetic apportionment studies seems, on the surface, to support Miller's[67] "right genes" argument. Notably, the percentage of genetic variation accounted for by race differs for different loci. Both Lewontin[24] and Barbujani and coworkers[63] list summary statistics for individual loci. Lewontin[24] found two loci in which racial differences accounted for roughly 25% of total genetic diversity (Rh and Duffy blood groups, 25.9% and 25.3%, respectively). Meanwhile, Barbujani and coworkers[63] found four restriction fragment length polymorphism sites in which racial ("geographic") grouping accounted for 22%, 23.9%, 35.5%, and 42.7% of total genetic variance. However, these loci are extremely rare (two of 17 and four of 109). The high proportion of genetic diversity explained by race in these examples could easily be the result of coincidences between genetic drift and culturally transmitted racial typologies.

Nevertheless, it is likely that those such as Miller[67] will seize upon these examples as giving hope to the theory that "those important genes" actually do differ significantly along racial lines. For example, commenting in racial apportionment studies, Miller[67] declared, "None of these genes affected skin color, nose shape, body build, size, etc. to mention [sic] characteristics that differ between races" (p. 97). Counter to Miller's argument, it seems that the morphological characteristics that racialist scientists and others do find useful are determined by simple variations in very specific regions, and are quite direct results of climactic selective pressures.[68] Owens and King[68] argue that variation in "stereotypic features of 'race'" such as skin color, hair form, and facial features are "literally superficial" and does not support the utility of the race concept.

A number of studies have emerged that use genetic data to classify individuals based on race or "geographical origin," perhaps the most notable recent attempt being Cavalli-Sforza and coauthors' *The History and Geography of the Human Genes*.[69] Such

TABLE 1. STUDIES OF RACIAL APPORTIONMENT

Study	Data	Statistic	% race	% region	% population	% individual
Lewontin[24] (1972)	17 blood groups	Shannon information measure	6.3		8.3	85.4
Latter[59] (1980)	10 blood groups, 3 proteins, 5 enzymes	Shannon information measure, Hedrick genic similarity, Latter paired comparison	2.8–14.0[a]	0.4–6.1	1.7–5.4	80.2–90.7
Nei and Roychoudhury[60] (1982)	62 protein, 23 blood group	Nei's standard genetic difference,[b] but parsing of variance unclear	15.0–19.0[c]			81.0–85.0
Ryman, Chakraborty, and Nei[61] (1983)	14 protein, 9 blood group, 2 HLA[d]	Chakraborty hierarchical gene diversity	6.4–15.4[e]		2.0–6.6	82.7–90.3
Excoffier, Smouse, and Quattro[65] (1992)	34 mtDNA loci	Bootstrapping, null distribution assumption[f]	15.7–22.0[g]		3.3–3.6	74.7–80.7
Dean and coworkers[62] (1994)	115 RFLP loci	Unweighted averaging of allele frequencies	9.5–10.1[h]			89.9–90.5
Barbujani and coworkers[63] (1997)	79 RFLP, 30 microsattelite loci	Bootstrap multiple allelic frequency comparison	10.0–11.7[i]		3.9–5.5	84.5
Seielstad, Minch, and Cavalli-Sforza[66] (1998): single nucleotide	34 mtDNA loci,[j] 22 biallelic polymorphisms (Y)[k]	Bootstrapping (Y and mtDNA), Unclear (autosomal)	12.5 (mtDNA), 52.7 (Y)[l]		6.1 (mtDNA), 11.8 (Y)	81.4 (mtDNA), 35.5 (Y)
Seielstad, Minch and Cavalli-Sforza[66] (1998): microsattelite	29 autosomal loci, 10 Y chromosome loci	Bootstrapping[m]		2.0 (autosm) 2.5 (Y)	0.2(autosm) 14.0 (Y)	97.8 (autosm), 83.5 (Y)

[a] Depending on data type (blood group, protein, or enzyme) and statistic used.
[b] Approximates number of codon differences.
[c] Parsing of variance unclear, range of percentages unexplained.
[d] From Nei and Roychoudhury.[60]
[e] Depending on data type and classification (a priori or post hoc to maximize group differences).
[f] For analysis at each level of population substructuring. Assumes that other levels are artificial and random.
[g] Depending on analytic technique (Euclidean distances between haplotypes, assumption of equal distance between haplotypes, network-based with racial assumptions, network-based with allowance for nonlinearity).
[h] Depending upon method of estimating heterozygosity (Lewontin,[24] Nei).[77]
[i] Depending on assay type (microsattelite or RFLP).
[j] From Excoffier et al.[65]
[k] Autosomal statistics taken from Barbujani et al.[63].
[l] On nonrecombining part of Y chromosome (from Underhill et al.).[76]
[m] Similar methods to Excoffier et al.[65]

studies select the small proportion of genetic variability that is roughly apportionable by race to plot out dendrograms of essentially false categorizations of human variability. To accomplish this, these studies use a priori categorizations of human variability that are based on the inaccurate belief that classical racial categorization schemes delineate a series of isolated breeding populations. This pursuit is not only futile; it leads to faulty and misleading conclusions about the nature and origin of human differences.

Thus, studies that attempt to define individuals on the basis of racial or "geographical" location are both flawed in design and easily misinterpreted. An example of this can be found in Bowcock and coworkers.[70] Despite a research design that should have maximized the degree to which the researchers were able to classify individuals by racial category, the results are something less than "high resolution" with respect to this goal. For example, 88% of individuals were classified as coming from the right continent, while only 46% were classified as coming from the right region within each continent. Notably, 0% success was achieved in classifying East Asian populations to their region or origin. These results occurred despite the fact that Bowcock and coworkers[70] entered their genetic information into a program that already used the a priori racial categories they were trying to replicate.

It is important to note that these researchers' results do not in any way locate diagnostic genetic markers for racial origin. Rather, they are based on pairwise comparisons of relative genetic distances. Thus, individuals were clustered by distance and then labeled based on a priori information about their origin. These labels were assigned in a way that characterized the largest proportion of individuals in each sorted group, a tactic that again gives Bowcock and colleagues[70] the advantage. This makes the fact

that only 88% of individuals were classified correctly by race and 46% by region even less impressive.

Other studies that classify individuals into racial groups produce results that argue against racial typology. For example, Excoffier, Smouse, and Quattro[65] used a computer to create racial groupings that maximized the amount of genetic diversity between groups. Thus, while defining regional groups a priori, they allowed genetic distance to define racial groups. As a result, these authors observed that the ideal racial typology using mtDNA grouped South American Pima Indians with Finns and Mayans with Sicilians!

The inadequacy of thinking about human diversity in terms of isolated breeding populations is well illustrated by the work of Keita and Kittles,[23] who have demonstrated how the pairwise genetic differences found by Bowcock and coworkers[70] and other researchers most likely are not the result of separate breeding populations evolving in different directions. Rather, this variation (which of course is very small, as illustrated by Lewontin and others) is more probably the result of selection pressures and genetic drift. Likewise, similarities between population samples are not necessarily a result of isolated cases of population admixture between breeding populations. Rather, they are likely a consequence of the expression of latent variability that was present before humans left Africa or parallel microevolution due to parallel ecological pressures. These evolutionary forces, well demonstrated in a variety of species, help deconstruct the notion underlying polygenic racialist thought that "the homes of the traits are essentially unique"[23] (p. 535).

Keita and Kittles[23] also effectively attack the predominant research methodology used by Cavalli-Sforza, Menozzi, and Piazza,[69] Bowcock and coworkers[70] and others, noting the inappropriateness of using a priori predefined racial categories and then sorting genetic diversity as much as possible into these categories. Interestingly, when these a priori classificatory schemes are not used, different assortments of genes and genetic systems in racial typology studies yield different classificatory schemes.[23] This is strong evidence of the clinical distribution of traits due to drift and differential selective pressure, a pattern that anthropologists have known about for decades.

The evidence against genetically mediated differences in behavior along racial lines is overwhelming (Table 1). First of all, a host of studies, beginning with those by Lewontin[24] in 1972 and most recently by Barbujani and colleagues[63] in 1997, have shown that the amount of human genetic diversity that is attributable to race is only about 5% to 10%. Following this, any particular "population" includes roughly 85% or more of the total human genetic diversity.[68] Also, racial genetics research that categorizes individuals by genetic profile, as does that of Bowcock and coworkers[70] or that defines genetic differences between hypothetical human "populations"[60,71–75] is plagued by a host of methodological flaws and faulty assumptions about human evolution. Thus, results that on the surface seem to demonstrate genetic differences between the human races are actually quite meaningless underneath. We support Owens and King's conclusion:[68] "The possibility that human history has been characterized by genetically relatively homogeneous groups ('races'), distinguished by major biological differences, is not consistent with genetic evidence . . .. The myth of major genetic differences across 'races' in nonetheless worth dismissing with genetic evidence."

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