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Uniqueness, Diversity, Similarity, Repeatability, and Heritability

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This paper reexamines classic attempts at estimating the number of possible genotypes for a species. In the original computations (Hirsch, 1963), the probability that any two human parents will produce two offspring with the same genotype was calculated to be equal to $(1/2^{23})^2$, or over one chance in 70 trillion. The error lies in that this number reflects the number of cells in the matrix of zygotes, not the number of zygotic genotypes. When this is taken into account, the probability of two human parents producing two offspring with the same zygotic genotype is 1 in 160,000—almost a billion times more likely than previously suspected. The complexity of the genetic system is also discussed in the context of the concept of "heritability," often confused with that of "heredity." This confusion has led to the wrong view that heritability represents a nature/nurture ratio.

Since the emergence of genetics following the triple rediscovery in 1900 of Mendel's (1866) classic study, there have been attempts to calculate and estimate the number of possible genotypes for a species (Sutton, 1903). An example of such attempts is Borel (1961), who, according to Piéron (1962), had made it as early as 1941 (see also Corcos & Monaghan, 1993; Morgan, 1934; Snyder, 1949; Wright, 1932). I too once contributed to this literature (Hirsch, 1963). Reexamination of this work recently has revealed a previously unappreciated complexity.

I made and published the individuality calculation in an invited lead article in *Science* magazine 40 years ago (Hirsch, 1963) and have always been proud that my arithmetic has withstood the test of time. It was and still is correct. Nobody has succeeded in challenging it. But now, I find that I myself must challenge the interpretation that I gave to the calculation at that time, because the calculation does not answer the question I was then asking, however mistakenly at that time we all believed that it did. It answers a different question; however, as you will see, it is a related question.

My purpose now is to explain what formerly has been assumed and what must henceforth be analyzed more carefully. Formerly the situation had been analyzed in the following way: We estimated "the probability that the second offspring born to parents will have exactly the same genotype as their firstborn" to be "less than 1 chance in over 70 trillion," that is $(1/2^{23})^2$ because man, with 23 chromosome pairs, produces gametes with any of 2^{23} = 8,388,608 alternative genomes. For the second born to be identical to the firstborn, it requires both parents to produce a gamete that replicates the one that each had previously contributed to their firstborn and that these replicates combine once again. Because each individual can

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produce gametes with any of 2^{23} alternative genomes, the probability of a replicate occurring is $1/2^{23}$ in the case of each parent, independently and separately; and the probability of their joint and simultaneous occurrence in combination was logically inferred to be the product of their separate probabilities, that is $(1/2^{23})^2$, or one chance in over 70 trillion.

The logic is impeccable; we learn it as students in elementary mathematics: The probability of the simultaneous occurrence of two independent events is the product of their separate probabilities. But the actual relationship is different, and we did not appreciate that difference until we started to work with diagrams, which was impossible to do for such large numbers. Certainly, to date, no one else seems to have done so. My 1963 and earlier discussions used words, some genetic symbols, and a few elementary calculations, as others who have considered this problem have done. Under those conditions the complexity remained undetected.

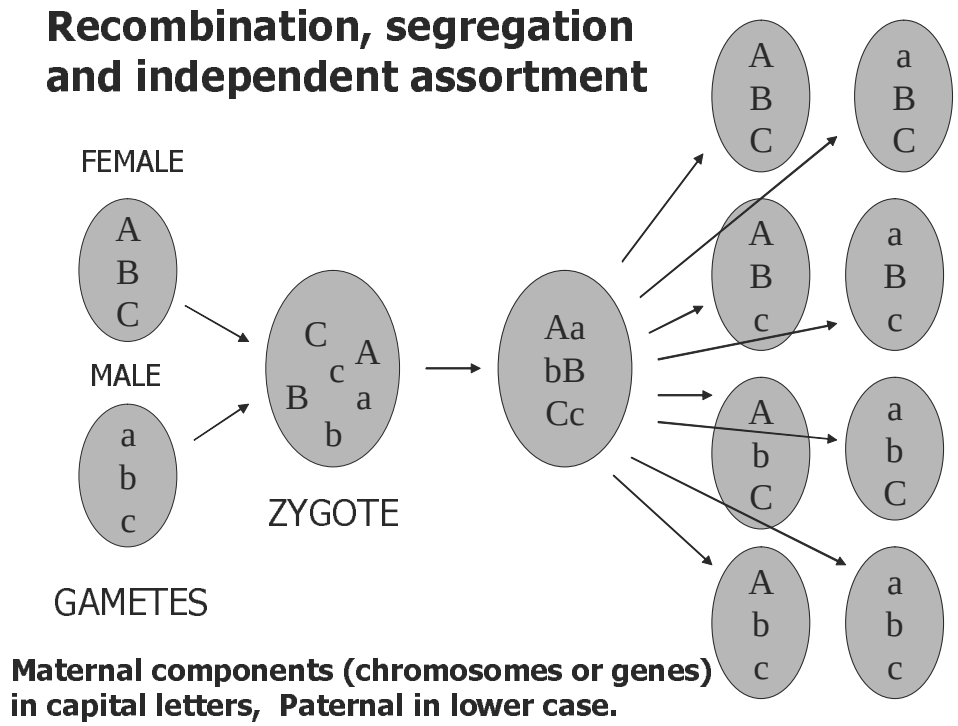


Figure 1. Recombination, segregation, and independent assortment. Genetic components (chromosomes or genes) are undifferentiated in this exercise, except the maternal components are in capital letters and paternal components are in lower case to designate which parent contributed what components. Adapted with permission, courtesy of W. S. Sutton (1903) and E. B. Wilson (1924).

The different picture emerges, however, when we employ the diagrams with which we explain some of elementary genetics to our classes—using numbers small enough so such diagrams are possible. For a species like *Drosophila willistoni*, with only three pairs of chromosomes, the diagram shown in Figure 1 displays the reproductive cycle from fertilization through gametogenesis for an individual. Two parental gametes (sperm and egg) combine into a zygote on the left and on the right side the resulting individual at gametogenesis produces gametes with $2^3 = 8$ alternative genomes. But, the probability that a given breeding pair will

have two identical offspring is not $(1/2^3)^2$. Yes, $1/2^3$ is the probability of the occurrence of a particular gamete from one parent, and the same is true for the other parent. So, why does not its square, that is $(1/2^3)^2 = 1/64$, give the probability of a second zygote being identical to the first zygote, as we all had previously assumed it does?

The answer can be found by examining the matrix of zygotes (called "Punnet squares" in genetics) produced by arraying along the margins the eight possible male and female gametic genomes where each cell entry is the zygote resulting from the intersection of row and column gametes. This examination is illustrated in Figures 2 and 3. Remember, our examination is being done for the case of a species with three or fewer pairs of chromosomes.

The probability that the second offspring born to parents will be genotypically the same as their firstborn depends upon (1) the number of genotypes that are possible and (2) the relative frequencies of their expected occurrences. Consider the matrices of genotypes produced by gametic genomes arrayed on their margins for some easily illustrated cases, such as 1, 2 and 3 pairs of chromosomes (assuming for simplicity no crossing-over, no mutation, and only two forms [homologues] of each chromosome in a population). Under those assumptions the number of types of gametic genomes on the margins is 2^N, where *N* is the number of chromosome pairs.

	A	a
A	AA	Aa
a	Aa	aa

$$\frac{1^2+2^2+1^2}{4^2}=\frac{3}{8}$$

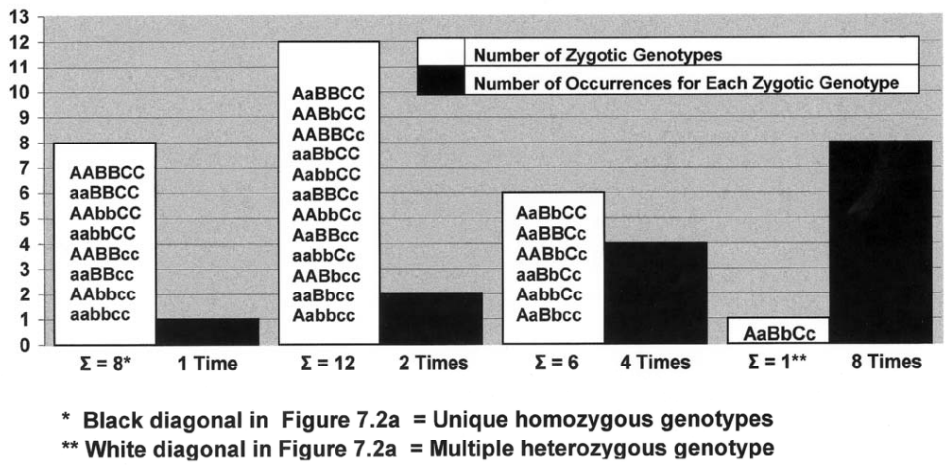
	AB	aB	Ab	ab
AB	AABB	AaBB	AABb	AaBb
aB	AaBB	aaBB	AaBb	aaBb
Ab	AABb	AaBb	AAbb	Aabb
ab	AaBb	aaBb	Aabb	aabb

$$\frac{1^2+2^2+1^2+2^2+4^2+2^2+1^2+2^2+1^2}{16^2}=\left(\frac{3}{8}\right)^2$$

	ABC	aBC	AbC	abC	ABc	aBc	Abc	abc
ABC	AABBCC	AaBBCC	AABbCC	AaBbCC	AABBCc	AaBBCc	AABbCc	AaBbCc
aBC	AaBBCC	aaBBCC	AaBbCC	aaBbCC	AaBBCc	aaBBCc	AaBbCc	aaBbCc
AbC	AABbCC	AaBbCC	AAbbCC	AabbCC	AABbCc	AaBbCc	AAbbCc	AabbCc
abC	AaBbCC	aaBbCC	AabbCC	aabbCC	AaBbCc	aaBbCc	AabbCc	aabbCc
ABc	AABBCc	AaBBCc	AABbCc	AaBbCc	AABBcc	AaBBcc	AABbcc	AaBbcc
aBc	AaBBCc	aaBBCc	AaBbCc	aaBbCc	AaBBcc	aaBBcc	AaBbcc	aaBbcc
Abc	AABbCc	AaBbCc	AAbbCc	AabbCc	AABbcc	AaBbcc	AAbbcc	Aabbcc
abc	AaBbCc	aaBbCc	AabbCc	aabbCc	AaBbcc	aaBbcc	Aabbcc	aabbcc

$$\frac{1^2+2^2+1^2+2^2+4^2+2^2+1^2+2^2+1^2+2^2+4^2+2^2+4^2+8^2+4^2+2^2+4^2+2^2+1^2+2^2+1^2+2^2+4^2+2^2+1^2+2^2+1^2}{64^2}=\left(\frac{3}{8}\right)^3$$

Figure 2. Probability of two offspring having identical genotypes illustrated with Punnett squares in which the margins contain genomes and the matrix cells contain zygotic genotypes as the products of matings involving 1, 2, or 3 pairs of chromosomes (represented as upper or lower case letters or both).



* Black diagonal in Figure 7.2a = Unique homozygous genotypes
** White diagonal in Figure 7.2a = Multiple heterozygous genotype

Figure 3. Zygotic genotype frequency of occurrence as shown in the Punnett square of Figure 2 involving 3 pairs of chromosomes (represented as upper or lower case letters, or both).

Our collective error has been not to appreciate that when the number of gametic genomes, 2^N is squared, $(2^N)^2$, the result describes the number of cells in the matrix of zygotes, not the number of zygotic genotypes. Figure 4 illustrates this issue.

Number of Gametic Genomes:	$2^1=2$	$2^2=4$	$2^3=8$
Number of Cells in Matrices:	$(2^1)^2=4$	$(2^2)^2=16$	$(2^3)^3=64$
Number of different Zygotic Genotypes:	$3^1=3$	$3^2=9$	$3^3=27$

Figure 4. Number of possible different genotypes in matrices using 1, 2, and 3 pairs of chromosomes. Our collective error has been not to appreciate that when the number of gametic genomes (2^N) is squares $(2^N)^2$, the result describes the number of *cells* in the matrix of zygotes, *not* the number of zygotic genotypes (3^N).

For the two-allele case, the number of different genotypes in each matrix is a smaller number, 3^N. In the examples shown, there are $2^1 = 2$, $2^2 = 4$, and $2^3 = 8$ gametic genomes, matrices with $(2^1)^2 = 4$, $(2^2)^2 = 16$, and $(2^3)^2 = 64$ cells but only $3^1 = 3$, $3^2 = 9$, and $3^3 = 27$ different genotypes in the cells for the one, two, and three chromosome-pair cases, respectively. Examination of the matrices reveals, and dramatically so in the off diagonal, that many genotypes are not uniquely determined by a single combination of maternal and paternal gametes, as our calculation had assumed. In fact, what can be seen in the off diagonal is that any gamete can enter the multiple heterozygote, if it combines with the appropriate gamete from the opposite sex. Figure 5 illustrates the expected occurrences of relative genotype frequencies.

$3^1=3$
(1:2:1)

$3^2=9$
(1:2:1:2:4:2:1:2:1)

$3^3=27$
(1:2:1:2:4:2:1:2:1:2:4:2:4:8:4:2:4:2:1:2:1:2:4:2:1:2:1)

Figure 5. Expected occurrences of relative genotype frequencies in Punnett square matrices with 1, 2, and 3 pairs of chromosomes and 3, 9, and 27 genotypes.

The coefficients in the binomial expansion happen to give the relative frequencies with which the different possible genotypes may be expected to appear in each matrix. In the cases shown there are the frequencies 1:2:1 for (1) a single pair of chromosomes, (2) 1:2:1:2:4:2:1:2:1 for two pairs of chromosomes, and (3) 1:2:1:2:4:2:1:2:1:2:4:2:4:8:4:2:4:2:1:2:1:2:4:2:1:2:1 for three pairs of chromosomes, respectively.

2^{23} = 8,388,608 possible genomes
$(2^{23})^2$ = 70,368,744,177,666 cell matrix
3^{23} = 94,143,178,827 different possible human genotypes

Figure 6. Number of possible human genotypes with 23 chromosome pairs is 94+ billion distributed over a 70+ trillion-cell-matrix. In order to understand the genetic process of reproduction, we present a simplified situation using chromosomes with only two alternative forms in each pair and without cross-over or breakage occurring. Reality is much more complex.

As illustrated in Figure 6, humans, with 23 chromosome pairs, produce gametes with any of 2^{23} = 8,388,608 possible genomes, and would require a matrix with $(2^{23})^2$ = 70,368,744,177,666, or over 70 trillion cells, and 3^{23} = 94,143,178,827, or over 94 billion different possible genotypes distributed over those 70+ trillion cells.

Therefore, the answer to the question about the probability that the second born may have the same genotype as the firstborn requires a different approach. As displayed in Figure 7, starting with the Punnett square in the simplest case of one chromosome pair with two possible homologues, we have four equally likely possibilities (two of them the same) for each individual. Then taking the possible combinations that produce two individuals, we have entered a “yes” when they are identical and a “no” when they are not.

We find agreement occurring in 6 of the 16 cells of this matrix, with the probability of two identical genotypes being 6/16 = 3/8. More succinctly, instead of duplicating Aa, we could enter the three genotypes on the margins with their respective probabilities: AA - _, Aa - _, aa - _ and notice that a “yes” can occur only when the same entry value defines the row as defines the column. So the rea-

soning could be abbreviated by recognizing that our 3/8 value is obtained by summing the following terms: the probability of the first genotype multiplied by the probability of an identical second genotype—for AA, Aa, and aa, these are, as shown in Figure 8, respectively:

$$\frac{1^2 + 2^2 + 1^2}{4^2} = \frac{6}{16} = \frac{3}{8}$$

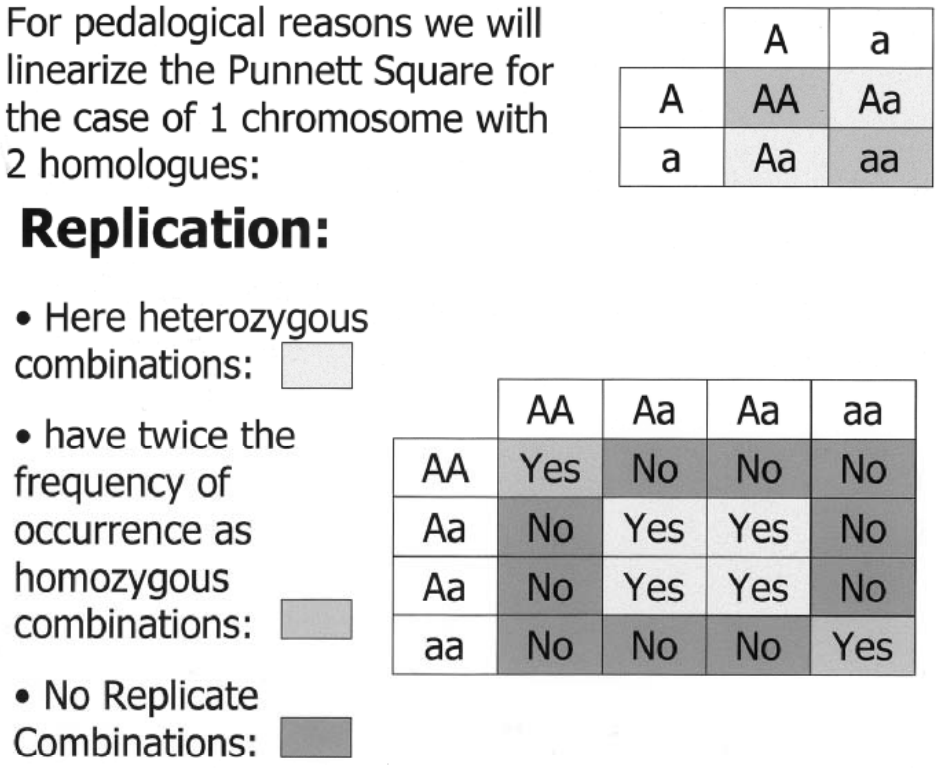


Figure 7. Probability of replication of heterozygous or homozygous combinations.

In general, by a similar line of reasoning we arrive at the values appearing in Figure 8. Note that, where previously we had estimated the probability of two identical children from one couple as 1 in 70 trillion, now we find that it is slightly less than 1 in 160,000—almost a billion times more likely! Our analysis also reveals that the previous estimate was an answer to a question somewhat different from the one we had stated, that is, instead of two children from the same parents, it was in fact addressing the question of two identical children from any (same or different) parents in the population. Nevertheless as shown in the table, the answer to the question involving any parents is also grossly different, that is, now seen to be one in 6.27 billion, also a much more likely event.

Number of Distinct Homologues	P(two children the same) with any parents	P(two children the same) with the same parents
2	3/8	19/32
3	5/27	25/54
4	7/64	103/256
m	**$2m-1)/m^3$**	**$\{(m^3 + 2m^2 + 2m - 1)\}/4m$**

Results: m = 2, n = 23

P{any parents} = $(3/8)^{23}$ = $(96/256)^{23}$ = 1.59×10^{-10} (1 in 6.27 billion)

P{same parents} = $(19/32)^{23}$ = $(152/256)^{23}$ = 6.21×10^{-6} (1 in 161,107)

Results: m = 4, n = 23

P{any parents} = $(7/64)^{23}$ = $(28/256)^{23}$ = 8.05×10^{-10} (1 in 1.24 billion)

P{same parents} = $(103/256)^{23}$ = $(103/256)^{23}$ = 3.37×10^{-9} (1 in 337 million)

Figure 8. Probability of identical genotypes of two children produced by (1) any parents, or (2) the same parents.

Furthermore, evidence and analysis also reveal the unrealistic nature of our assumption that there are in a population only two alternative forms of the chromosomes in each pair. In many cases, for example, maximum heterozygosity, both parents were required to have the same genotype—a ridiculous assumption. Whereas that assumption was certainly not concealed, effectively all of us seem to have ignored, or at least failed to appreciate, it.

One might object and raise the "so what" question: that my foregoing discussion has merely shown that, an event once believed to be infinitesimally unlikely, now appears merely very unlikely. At this point reference to the Punnett squares should remind us that our analysis has shown that the populations we study can no longer be considered to comprise almost exclusively unique genotypes. As was illustrated in Figures 2 and 3, there is diversity of genotypes and their frequencies: Figure 2's main diagonal (upper left to the reader's lower right) contains the unique genotypes, they are homozygotes. The off diagonal contains *N* replicates of the single most frequent genotype, the multiple heterozygote. In the cells on both sides of the main diagonal are distributed duplicate to multiple copies of the several homozygote-heterozygote combinations.

Consider the following: Up to this point we have treated the uniformity versus diversity question as an absolute, either two genotypes are identical or they are different. A more realistic approach would be to recognize gradations of similarity from as much alike as monozygotes (identical twins, triplets, etc.) through gradations of partial similarity, that is, varying proportions of shared elements. If two or more individuals are the same with respect to 22 out of our 23 pairs of chromosomes, and differ on one pair of chromosomes, or 21 pairs and differ on two pairs of chromosomes, or 20 pairs and differ on three, and so forth, they will have much in common. Of course, exactly how much will depend on which chromosomes are shared and which are not. Those diagrams show a diversity of genotypes and their frequencies: The main diagonal contains the unduplicated unique genotypes—the homozygotes, the off diagonal contains *N* copies of the single and

most frequent genotype—the multiple heterozygote. In the cells of the N^2 matrix, exclusive of the two diagonals are distributed duplicate to multiple copies of the several homozygote-heterozygote combinations, which vary in frequency.

In sum, the import of these analyses is not merely to adjust a 41-year-old computation. Rather, it is to underscore the complexity of genetic combinations and to signal the dangers of simplistic approaches to representing genetic variability. For many decades (see Hirsch, 1970, 1997), such dangers have been ignored in several treatments of the concept of heritability.

Heritability is not Heredity

After our experience of the last decade with *The Bell Curve* (Herrnstein & Murray, 1994), which so badly misused the heritability statistic, and in a recent issue of *Science* devoted to mapping the human genome, an article by McGuffin, Riley, and Plomin (2001), which also badly misused the heritability statistic, we should consider the meaning of heritability (see Hirsch, 1997, for analyses and interpretation).

The heritability concept must be distinguished from, rather than confused or conflated with, the heredity concept. Usually, the heritability statistic measures the additive genetic variance of a trait in a population and may be quite different in one population from that in another population of one species. Heredity is our name for the biological system that makes possible the existence and reproduction of each species.

Unfortunately, a recent *Dictionary of Psychology* (Sutherland, 1996, p. 201) ignores completely the assumptions under which heritability may be used:

heritability. The extent to which a given characteristic is determined by heredity, usually measured as the proportion of the variance of the characteristic in a given population that can be accounted for by hereditary factors. The heritability will depend both on the variance of the genes and on the variance of the environment; both factors may differ from one population to another within the same species.

heritability ratio (h^2). A measure of heritability, namely $h^2 = V_g/V_t$ where V_g is the variance in a trait accounted for by hereditary factors and V_t is its total variance.

Heritability was developed in the mid 1930s to predict the outcome of plant and animal breeding studies and was borrowed by some psychologists in the belief that it could be applied to human data, for example, to determine what proportion of IQ is inherited (nature) and how much is acquired through experience (nurture). Analysis of the limitations of heritability reveals its inappropriateness as a measure in human psychology (Hirsch, 1997; Jacquard, 1983; Kempthorne, 1978) where breeding experiments are off limits. Social policy based on inappropriate heritability statistics risks misguided predictions about human psychology in present and future society.

Unfortunately, well-intentioned critics also confounded heredity and heritability: “There exist no data which should lead a prudent man to accept the hypothesis that I.Q. test scores are in any degree heritable” (Kamin, 1974, p. 1), thus like Watson (1914, 1936, 1959), implying the irrelevance of heredity to intelligence as measured by IQ tests. Several comments are relevant here:

(1) Merely because the heritability statistic might be inappropriate in a given situation does not permit one automatically to infer environmental causality;

(2) The presence of heredity-environment interaction precludes generalization about the effects of both heredity and environment;

(3) The presence of interaction is itself evidence that both heredity and environment may be exerting influence, for example, when it appears that the same environmental condition "causes" different phenotypic expressions in different genotypes, that in itself may be evidence of genetic effects; and

(4) The detection of interaction usually requires greater statistical power than does the measurement of the effects of the primary variables; attaining that power usually entails relatively large samples (Wahlsten, 1990), that is, if one wants the power to detect an interaction to be 90%, the power to detect the main effects will usually then have to be higher than 90%.

The challenge Cavalli-Sforza made (Cavalli-Sforza & Cavalli-Sforza, 1995) to those he calls "IQ hereditarians, or enthusiasts" is that: In science we all have an obligation to be familiar with the current state-of-the-art and to incorporate previous developments into whatever we do. He showed that the current literature, including his own work, published in good journals yielded much lower heritabilities than those reported by the IQ enthusiasts, who simply go on ignoring the findings of others. I congratulate him on his forthrightness and agree completely with the argument he put forward. But, I go further and say (challenge) directly to him that, by the logic of his very same argument, neither party can ignore the analyses, I now quote below, by the two distinguished statistical geneticists, Oscar Kempthorne and Albert Jacquard, and both published in the well-known and highly respected journal *Biometrics*. They both analyzed the limitations of heritability analysis (it should also be noted that the later publication, by Jacquard (1997), acknowledges with full reference the earlier one in the same journal by Kempthorne that is featured as "A Biometrics Invited Paper;" furthermore, Jacquard (1997), now reports that his text has "the approval of the leading American biometrician Oscar Kempthorne"; author's translation):

> The idea that heritability is meaningful in the human mental and behavioral arena is attacked. The conclusion is that the heredity-IQ controversy has been a "tale full of sound and fury, signifying nothing." To suppose that one can establish effects of an intervention process when it does not occur in the data is plainly ludicrous. Mere observational studies can easily lead to stupidities, and it is suggested that this has happened in the heredity-IQ arena. The idea that there are racial-genetic differences in mental abilities and behavioral traits of humans is, at best, no more than idle speculation. (Kempthorne, 1978)

> The need for great rigor exists particularly in the case of research projects that have serious implications for us all; this is the case when psychologists study the "heritability of intellectual aptitudes." They should take the precaution of systematically defining in a precise way the sense in which they use the word "heritability"; they should also state whether the assumptions under which this word can be used hold true in their studies. It is highly probable that most of the time this exercise in rigor would lead them to the conclusion that none of the three parameters proposed by geneticists can be of any use in solving their problems. (Jacquard, 1983)

The complexity of the widely misunderstood, and so often misused, heritability statistic is further emphasized by the recent posthumous publication of Haldane's discovery of a case of negatively valued heritability. He reported the

example “as a warning against the assumption that where a character is mainly determined genetically it will be more frequent in the progeny of those who manifest it than in the progeny of those who do not. This assumption is taken for granted in popular expositions of Darwinism and of eugenics” (Haldane, 1996, p. 5). He considered the case to be “not trivial” because some “Other characters may have similar negative heritability” (Haldane, 1996, p. 3; also see accompanying commentary by Woodrow, 1996, who arranged for the publication).

With respect to the tragic and widespread confusion about heritability, consider the following: In both *IQ in the meritocracy* (Herrnstein, 1973) and *The bell curve* (Herrnstein & Murray, 1994), it was asserted the meritocracy-cognitive elite organization of our society. For the moment, let’s act as if he were correct; certainly, he believed that he was. That would mean that he was asserting a correlation between genotype and environment. Genotypes are not randomly distributed over the social environment, they are arranged in his meritocratic hierarchy. Not any randomly selected genotype can be trained at Harvard or other elite schools, because there is an interaction between genotype and environment. Furthermore, our species mates assortatively, not randomly; for example, my wife and I met and married in Paris as American students at the Sorbonne (Sanders, 1996; analogous stories are true of countless colleagues). Yet, heritability estimation assumes both random mating in an equilibrium population (including therein the equally likely occurrence of every culturally tabooed form of incest) and the absence of either correlation or interaction between heredity and environment. In fact, when one or more of those assumptions are violated, that is, random mating in an equilibrium population, correlation or interaction, heritability is undefined (see Kempthorne, 1997). What must be appreciated is that heritability is not a nature/nurture ratio measuring contributions to individual development and heritability is not heredity—two entirely different concepts that have been hopelessly conflated in *The bell curve* and many other texts where most of the hereditarian interpretations have been based on unjustifiable human heritability estimates (see Platt & Bach, 1997). Unfortunately, because of their assonance, when we hear one of the two words, automatically we think the other. Either these authors knew what they were perpetrating and are therefore responsible, or they did not know what they were doing and are therefore irresponsible. It is this level of “scholarship” that Bouchard (1995, p. 418) has recommended as “a superbly written and exceedingly well-documented book… It deserves the attention of every well-informed and thoughtful citizen.”

Herrnstein had neither dark skin nor kinky hair. He was a white, Jewish, Harvard professor. Otherwise, such scholarly incompetence, as has here been revealed, might, if one were to apply his professed high standards, have had to be interpreted as an unmistakable sign of his own genetic inferiority.

The problem being considered—the probability of the occurrence of replicate genotypes or replicate components thereof—whether or not it is of particular interest to you, has the advantage of requiring us to recognize the complexity of the genetic system—a reality that intervenes into the consideration of many problems involving genetics.

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