Science or prejudice?

By: Douglas Wahlsten

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Article:

Why BBS? Rushton invites commentary on his speculations by a wide spectrum of academics, yet he seems to pay no attention whatsoever to previous objections to sociobiology in the pages of this journal or elsewhere. *Vaulting Ambition,* by Kitcher (1985), the subject of peer commentary in *BBS* (1987), provided an incisive critique of Wilsonian sociobiology, and most of his points apply directly to Rushton's article. Yet Rushton proceeds as though those words had never been written. His article is presented as a development *within* a narrowly conceived sociobiology, and he cites the works of like-minded colleagues whom he considers authorities on everything from marriage and child abuse to patriotism and war.

Where's the model? Much is vague and informal in this article Rushton talks about gene frequencies, differential heritabilities, and relative strengths of conflicting tendencies but never does he commit himself to a quantitative model that would allow a decisive evaluation of his ideas. Population genetics is supposed to be a precise discipline, yet we find no equations, no path diagrams or anything of the sort in this article. Is Rushton hoping the *BBS* commentators will do the mathematical thinking for him? Much more is required before this can be regarded as a scientific hypothesis.

When does the rigor begin? Sloppiness abounds. Phenotvpes are said to be "the product of multiple alleles" (section 7.1), though the context implies Rushton really means multiple genetic *loci.* The meaning and consequences of multiple allelism at *a* locus and multiple loci heredity are quite different. He argues that mechanisms for both detecting and producing features of the organism are "genetically coupled" (section 7. which implies genetic linkage, when he apparently means pleiotropic gene action or genetic correlation. Again, the disparate effects of linkage and pleiotropy in a model hardly make them synonyms. Rushton claims that "it is advantageous for a single gene to work for copies of itself" (section 7.1), having earlier claimed (section 6.1) that too much genetic similarity is bad and leads to inbreeding. He imbues the little gene with a narcissistic affinity for its own kind, but instructs it not to do what comes naturally if this would contradict the theory. If such a contradiction were somehow integrated into a comprehensive and nonlinear quantitative model, one might think of it as part of science and call it dialectic; but lacking this, the conflicting claims appear to be equivocation.

Are the genes communist? Rushton maintains that "genes maximize their replication by benefiting any organism in which their copies are to be found" (section 10) and claims that this property is broadly representative of "the overall genuine" (section 7.1). It just so happens that techniques of molecular biology indicate there are from 60,000 to 200,000 structural genes in the human cell nucleus coding for distinct proteins, but only 2,208 loci have been validated, many of which are known from very few individual cases and cannot be considered polymorphic (McKusick 1988). Considerably less than 10% of the loci in the human genome are polymorphic at the level of polypeptide gene products. What humans have in common genetically vastly exceeds their differences; hence genetic similarity theory would seem to require that those genes at fixed loci should do their utmost to guarantee the propagation of all people, regardless of ancestry, and that, being most

numerous, they should prevail over their vacillating and sparse neighbors. Rushton's views about patriotism, xenophobia, and war simply do not follow from his premises.

Polygenic favoritism in the family? In section 6.2 Rushton uses a fictitious example as the basis to estimate that a child could have 60% genetic similarity to its mother and 70% to its father. This is absurd. First, over 90% of the loci will be fixed in the population. Second, at loci with many alleles in the population, the parents will usually not have any alleles in common. If the two parents have four different alleles, the child will always have the same 50% genetic similarity with both parents. Third genetic similarity to the mother and to the father at a locus can differ only when the parents have two or perhaps three alleles among them. If there are two alleles in the population, then three genotypes (AA, Aa, and aa) and nine mating combinations of male and female are possible. In five of these combination' including Aa × Aa, the genetic similarity of a child to it mom and dad must be identical. If the frequencies of the alleles are *p* and *q*, and assuming the population is in Hardy-Weinberg equilibrium, a little algebra reveals that the expected genetic similarity of a child to its mom is 100 (1 — *pq)* and that the variance is $100^2\ pq\ (\frac{1}{2} - pq)$ for the one locus. If there are *N* such independent loci in linkage equilibrium, the expected mean genetic similarity across the *N* loci is also 100 (1 - *pq*) and the 95% confidence interval for the genetic similarity to mom is

$$100\ (1 - pq) \pm 1.96\ (100) \sqrt{pq\ \frac{\left(\frac{1}{2} - pq\right)}{N}}$$

Likewise, the 95% confidence interval for the *difference* between genetic similarities to mom and dad is

$$0 \pm 1.96\ (100) \sqrt{pq\ \frac{(p^2 + q^2)}{N}}$$

If the alleles are equally prevalent (p*q*, the maximum variance case) and there are 5,000 such loci, the interval for genetic similarity to mom is from 74.931% to 75.069%. If there are about 100,000 structural genes in the human genome, of which 90% are fixed for one allele, 5% are highly polymorphic, such that the parents usually have four alleles, and 5% have two equally frequent alleles, the 95% confidence interval for overall genetic similarity of a child to its mom is 96.247 to 96.253, and the interval is 0 ± 0.049% for the *difference* in genetic similarities to mom and to dad. Rushton's figures of 60% for mom and 70% for dad would be plausible only if the mating were between a man and something much more remotely related than a chimpanzee (Jones 1986). He simply conjures up these numbers, rather than deriving them from established facts.

Conclusion or sentiment? The premises in Rushton's article are incompletely specified and major components of the nascent model are grossly at variance with facts. His article provides yet another example of how "Neo-Darwinian sociobiology can be used to give pseudo-scientific support to what are actually mere prejudices" (Saunders 1988). Rushton's argument for a genetic cause of ethnic conflict and xenophobia (section 9) must be regarded as an asseveration of personal belief rather than a tentative scientific conclusion. [See Wahlsten: "Insensitivity of the analysis of variance to heredity-environment interaction." *BBS* 13 (1) 1990.]