A Response to Rushton's "Race Differences in Behaviour"

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In this paper I refute J. P. Rushton's claim to having demonstrated heritable race differences in intelligence, cranial capacity, and brain weight. I also refute his claim to having established that heritable behavior traits, when ordered by gene-culture coevolutionarily based r/K reproductive strategies, determine the level of human evolutionary development to be, in descending order, Mongoloids, Caucasoids, and Negroids. Refutation is accomplished through analysis of the causal chain governing explicit theory and unstated assumptions; documentation of inadequate, spurious and misapplied data sources; and demonstration of scientifically unacceptable methodology.

Dans cet article je réfute l'énoncé de J.P. Rushton qui croit avoir démontré les différences raciales héréditaires de l'intelligence, de la capacité cranienne et du poids du cerveau. Je réfute également sa prétention d'avoir établi que des traits comportementaux héréditaires, commandés par des statégies de reproduction génétiques et culturelles déterminent que le niveau de développement humain est, par ordre décroissant, mongolien, caucasien et nègre. Ce rejet se fait d'abord par l'analyse de la relation causale qui soutend la théorie et les énoncés. De plus, la position de Rushton est basée sur une documentation inappropriée et sur des données dont l'application est mise en doute. Finalement, sa méthodologie est scientifiquement inacceptable.

In his paper "Race Differences in Behaviour: A Review and Evolutionary Analysis" (1988) J. P. Rushton argues that heritable behavior traits place Mongoloids at the pinnacle of human evolution, with Caucasoids on the slopes beneath them, and Negroids at the bottom of the mountain (his human categories). He contends that the validity of his theory and, by virtue of his theory being validated, the validity of his choices of categories for classification are demonstrated through his analysis of the following traits: intelligence, maturation rate, personality and temperament, sexuality, and social organization. According to Rushton's theory, development of these traits is entirely controlled by the racially based inheritance of the genotype; that is, the traits are measurably different among races and genetically fixated within the individual. Further, he states that this ordering of races can be explained in part by a second theory: "gene-culture coevolutionarily based r/K reproduction strategies" (Rushton, 1988, p. 1009).

Rushton's theories and the analysis upon which they are based demand a reasoned response. Although, as Rushton argues, refutation requires the citing of major studies demonstrating results opposite to those he reports, such a response, by itself, would not be sufficient. The array of flaws in Rushton's work is so vast and occurs on so many levels that a deeper analysis is required: The very epistemological basis of Rushton's thought must be called into question. There are three levels at which this can be accomplished: underlying theories and assumptions, methodology, and information (data) sources.

Analysis of Rushton's methodology and data sources is relatively straightforward. However, analysis of the underlying theories and assumptions is complicated by Rushton's attempt to unify a wide range of theoretical issues and by his consistent failure to present and account for theories which are in competition with those supporting his thesis.

This misrepresentation, by omission, of extreme theoretical positions as mainstream positions, accepted without significant challenge by the relevant scientific communities, stands as Rushton's single most important epistemological failure. This is true not only because these theories are contentious when applied to the attempted establishment of a racial hierarchy, and not only because they often represent major areas of intense paradigm dispute within the fields from which they are drawn, but also, and especially, because Rushton is presenting an argument which relies for its power upon the unbroken necessity of its causal chain.

In science, the links in a causal chain owe their strength not to the theory per se, but to the rigorous testing and verification procedures which are the hallmark of empirical method. A causal link originates when a theory is proposed, usually as the result of an observed effect receiving a hypothesized cause; however, it is not until subsequent testing leads to hypothesis verification that researchers can proceed on the assumption that the theory is "correct," that a link in the causal chain has been established.

An essential flaw in Rushton's work stems from his treatment of contentious, unverified theories as assumptions which do not require testing and verification. The result is that he often assumes the "cause" in a cause-and-effect relationship, when the relationship requires further investigation before the theory in question can be accepted as correct. For instance, although Rushton explicitly recognizes that intelligence is not strictly heritable, he nevertheless proceeds on the "unstated assumption" that heritable intelligence is "the cause" of variation in I.Q. test results among groups. (That his argument must be based upon this assumption is clearly demonstrated by a complete lack of analysis of alternative explanations.) However, if Rushton had treated this assumption as theory, as he should have, then he would have had to establish (identify, test, and verify) each link in his causal chain. In doing so he would have had to establish, among other

things, that no other factor or factors could account for the effect which he attributes to heritability.

This pattern of mistakenly treating unverified theories as assumptions is evident throughout Rushton's paper. It appears to be based upon the specious logic that his assumptions, taken together, verify his argument; therefore, his assumptions are correct. As a result, whatever the reasons may be, Rushton's causal chain is not always explicit. To evaluate the validity of Rushton's argument, it is necessary to identify and test the links in his causal chain.

A response to Rushton's theories, therefore, requires not only an analysis of methodological errors and flawed data sources but also the presentation of alternative theories to those proposed as assumptions by Rushton. In this way inadequate or contentious theories and unstated assumptions can be clearly illuminated. It is through this process that the links, or lack thereof, in Rushton's causal chain can be clearly established and that confounds capable of providing alternative explanations can be examined. Such detailed analysis is constrained by limitations of space and time. As a result, close analysis of Rushton's paper will be restricted to the first section, "Brain Size and Intelligence," and the last section, "r/K Reproductive Strategy," with subsidiary comments provided only to reinforce the argument that a similar analysis of the whole would produce corresponding results.

In the opening section of his 1988 paper, subtitled "Brain Size and Intelligence," Rushton claims a causal relationship between intelligence and cranial capacity, brain weight, and I.Q. scores. An analysis of this section shows that Rushton's argument is governed by a number of important but unstated assumptions which are central to his causal chain. In addition, it relies for support upon inadequate, spurious, and misapplied data sources and it exhibits faulty methodology.

To begin by restating a central thesis of this section, Rushton claims that intelligence is heritable and scores on intelligence tests demonstrate that Mongoloids are more intelligent than Caucasoids who are more intelligent than Negroids. To establish this causal chain, Rushton must demonstrate, at a minimum, that a) intelligence is sufficiently heritable that other factors could not be the cause of measurable differences among populations; b) intelligence tests measure innate (heritable) rather than functional (applied) intelligence; c) intelligence tests' cultural bias is insufficient to account for measurable differences in test scores among populations; and d) given that intelligence tests are norm referenced, test scores are comparable across tests and demonstrate the order claimed.

However, Rushton does not demonstrate causality, he assumes it. One of his assumptions is that intelligence is fixed at birth and cannot be significantly influenced by environment: The size of your head says it all. This theory lacks

empirical support; rather, the evidence points toward an interaction of biological and environmental factors as contributing to the development of intelligence (Piaget, 1977; Bouchard & McGue, 1981; Scarr & Kidd, 1983; Bouchard, Lykken, McGue, Segal, & Tellegen, 1990). As a result, it is necessary to determine the part which environment plays in the development of intelligence to establish whether purely environmental factors are capable of accounting for any real, measured differences in tested intelligence among populations.

According to Piaget, intelligence, by its very nature, has evolved as a strategy for people to interact successfully with their environment. Just as the mind is able to affect the environment, so does the environment affect the development of the mind. What we have, then, is not a fixed point for intelligence at birth, but rather a fixed potential. And the degree to which that potential is maximized or minimized over its inborn reaction range is directly controlled by environmental factors.

Piaget argues that children learn by the strategies of accommodation and assimilation and through the process of equilibration; they progress through a series of unique, sequential stages of intellectual development, from sensorimotor to formal operational. Children must meet and assimilate into their schema or accommodate their schema to problem solutions at one level before they are able to operate on the next higher level. To a significant extent it is the environment, not the mind, which presents or withholds the necessary interactions (Piaget, 1977). The important aspect of this understanding as it applies to Rushton's argument is that the development of what we commonly consider to be intelligence, what we attempt to measure through intelligence tests, is formed through an interaction between genetic endowment, personal environment, and cultural environment.

The importance of cultural factors in the development of intelligence has been confirmed by subsequent investigators who found that, while some individuals in highly industrialized cultures never reach the formal operational stage (usually reached in our society at 11-15 years of age and characterized by the ability to solve problems of a highly abstract and hypothetical nature for which the terms are merely symbolic), attainment of formal thought and even its precursor, concrete operations, are completely absent in other cultures (Dasen, 1972). They are absent because the cultural environment has never demanded, or for that matter permitted, that the mind develop the ability to operate at the concrete level or to think in symbols. (Those who doubt this interpretation are referred to Piaget's discourse on the Origins of Algebra in *Psychogenesis and the History of Science* (1989, pp. 143-146) where he demonstrates that symbolic thought originated in 16th century European culture.)

The conclusion that intelligence is not fixed at birth, but rather that various environmental factors play an important role in its development or retardation,

receives further support from studies seeking to determine the part which genetic inheritance plays in the development of intelligence. Of these, the study of monozygotic (MZ) and dizygotic (DZ) twins reared apart provides perhaps the most powerful tool, because MZ twins have identical genetic inheritance while DZ twins show the normal intrafamily distribution of genes but share with MZs other twin characteristics. Comparing data for reared-apart MZ and DZ twins with other family data allows researchers to compute an estimate of heritability for middle class, white Americans. Such computations are based on the assumption that the similarities demonstrated by monozygotic twins reared-apart (MZA) represent the inherited fraction of intelligence as manifest by identical genes reared in different environments.

In their review of "Familial Studies of Intelligence," Bouchard and McGue (1981) state that "the data support the inference of partial genetic determination for IQ is indisputable" (p. 1058). And Scarr and Kidd (1983), reviewing twin studies of intelligence, report, "From the comparison of MZ and DZ correlations from the 30 studies, one can calculate an estimate of the broad heritability of IQ in the white U.S. population as somewhere between .3 and .7, with a most likely value in the .5 range" (p. 395). Plomin and DeFries (1980) concur. Bouchard et al., in their review of the Minnesota Study of Twins Reared Apart (1990), state, "Under the assumption of no environmental similarities, genetic factors account for approximately 70% of the variance in IQ" (p. 224).

It is important to note that Bouchard et al. (1990) feel it necessary to make the assumption of no environmental similarities when accounting for the heritable factor in intelligence, and moreover, to point out that "in individual cases environmental factors have been shown to be highly significant" (p. 225) as demonstrated by a 29 IQ point difference between one set of monozygotic twins in their study. Further, "since only a few of these MZA twins were reared in real poverty or by illiterate parents and none were retarded, this heritability estimate should not be extrapolated to the extremes of environmental disadvantage still encountered in society" (p. 277). They conclude that "in the current environment of the broad middle-class, in industrialized societies, two-thirds of the observed variance in IQ can be traced to genetic variation" (p. 227). However, they also point out that "the MZT [monozygotic twins reared together] correlation apparently declines with age (for example, as a result of the accumulation of nonshared environmental effects)" (p. 226); and they conclude that "the proximal cause of most psychological variance probably involves *learning through experience*, just as radical environmentalists have always believed" (p. 227).

The 29-point difference in I.Q.s between one set of monozygotic twins in Bouchard et al.'s study appears to indicate that, in at least one instance, a radically different rearing environment caused a two standard deviation difference in intelligence as measured by I.Q. tests. This difference is nearly

twice the 15-point difference between Black and white American populations pointed to by Rushton as evidence of racial differences in intelligence.

Before expanding upon this argument, it is worth quoting Scarr and Kidd (1983) on the limitations of the concept of heritability.

> In its precise form, the heritability concept has several limitations. First, the heritability of a trait depends on the range of relevant environmental variation in the population being studied. If the population is subject to a wide range of relevant environments, heritability will decrease; conversely, if the relevant aspects of the environment are held constant, heritability will increase. Furthermore, it frequently happens that the range of environments in the sample studied is not representative of the general population, so the heritability estimate cannot be safely extended to the population as a whole. (p. 379)

These caveats should act as cautions in two ways. It may be inappropriate to assume that heritability estimates for a relatively homogeneous, white middle-class population are the same as heritability estimates for a Black underclass. Further, if the degree of heritability appears to vary between environmentally different populations, then we may suspect that a portion of what the studies are measuring as heritability is, in fact, environmental influence. Therefore, it seems reasonable to conclude that intelligence is less strictly heritable than Bouchard's review of the Minnesota twins might lead us to believe and that heritability may have less effect in disadvantaged Black families.

Another study by Fischbein (1980) appears to lend support to this thesis. From an analysis of Swedish data on 94 MZ and 133 DZ pairs of twins reared together, Fischbein determined that socioeconomic status was important to the degree of heritability as determined by I.Q. tests.

> Genetic factors seem to account for a greater proportion of the variance in social group I compared to social group II or III. This is evident when studying the within-pair correlations for MZ and DZ in different social groups. . . . The intra-class correlations for MZ and DZ will thus tend to be of the same magnitude in a less stimulating environment, where other factors than genetic endowment will account for a larger part of the variance in test scores. In a stimulating environment, on the other hand, children will develop in accordance with their genetic potentials, and the MZ twins with identical inheritance will tend to be more similar than the DZ twins. (pp. 60-61)

The conclusion that both heredity and environment play an important part in the development of testable intelligence would not have surprised Piaget, nor would the conclusion that environmental deficits mitigate against the full development of heritable potential; he believed that intelligence is created through an interaction between the mind and the environment. On the other hand, I believe that Piaget's work argues for the interpretation that attempting to distinguish heritable factors from environmental factors is meaningless, as the development of functional intelligence derives from the interaction of inherited mental aptitude and learning environment.

However, leaving Piaget aside, it could be argued that, contrary to Rushton's assumption, the evidence already assembled provides ample scope for other than heritable explanations of any observed differences in standardized test scores between populations. Nonetheless, three more assumptions which Rushton utilizes in this section have direct bearing upon his argument and deserve attention. These theoretical assumptions are (a) I.Q. tests measure innate intelligence, (b) such tests are culturally and environmentally bias free, and (c) scores on such norm referenced tests are equivalent across populations and across tests.

I.Q. tests certainly seem to offer accurate predictions about future scholastic success and they seem to correlate with socioeconomic status; beyond this the contention begins. Although many psychologists believe that I.Q. tests measure intelligence, no study has demonstrated that such tests do so, let alone that they measure innate intelligence. There are two reasons for this. The first is that, as Wechsler (1958) puts it, "few psychologists are willing to spell out what they mean by intelligence and, when they do, seldom agree" (p. 3). In other words, while a general, broad consensus of what constitutes intelligence exists, sufficient conceptual disagreement persists that no precise, widely accepted definition has been constructed. In the absence of a precise definition, there is nothing to test for. The second is that, as Weinberg (1989) points out, I.Q. tests were not designed to measure innate intelligence. Binet, the test's originator, was not interested in measuring innate intelligence; rather, he was interested in measuring "manifestations of intelligence in everyday life" (Weinberg, 1989, p. 100), a more functional conception of intelligence which "is heavily influenced by educational, cultural, and socioeconomic factors as well as by a range of individual factors that distinguish one person's life from another's" (p. 100).

However, Rushton argues that I.Q. tests do measure innate intelligence, because they measure a general factor: "g". Rushton takes his argument from Spearman, by way of Jensen. It was Spearman (1927) who first proposed that intelligence was comprised of a single factor which he denoted "g" and of which he said, "the effects of heredity upon g are very large indeed" (p. 391). Jensen (1972, pp. 76-77; 1981, p. 162) makes use of Spearman's theory. And he does so knowing that Thurstone (1937), responding to Spearman, had long since demonstrated that Spearman's theorem was only a special case of a more general factor theorem and, further, that Thurstone had proposed that g was merely an artifact of the factor analytic technique Spearman used. Although the debate has not been entirely resolved, as Wechsler (1958) says of Spearman's theory, "his concept of one central or unifactor has largely been abandoned by psychologists" (p. 9). In its place, Thorndike, Bregman, Cobb, & Woodyard (1927), Thurstone (1937; 1947), Wechsler (1958), and Torrance and White (1975), among others, have proposed a number of variously strong factors which are necessary to make up general intelligence.

Therefore, in contrast to Rushton, I propose that I.Q. tests measure applied or functional intelligence. I define applied intelligence as the phenotype, the genotype modified through experience and learned problem solving techniques: the mind's interaction with its environment.

Support for the proposition that I.Q. tests measure something other than innate intelligence is provided by numerous, authoritative studies. Emanuelsson and Svensson (1986) report that results from a study of nearly 32,000 Swedish 13-year-olds indicate that I.Q. scores increased between 1961 and 1966 and again between 1966 and 1980. How is this possible, unless some environmental factor or factors changed? Surely we aren't evolving that fast. Similarly, Nagoshi, Phillips, and Johnson (1987) found that Americans of Japanese ancestry scored better than their parents on I.Q. tests and conclude that "this AJA generational change undoubtedly reflects historical developments in Hawaii that produced increased social mobility and status for the AJA parental generation" (p. 314).

While some environmental factors have been shown to raise I.Q.s, others have been shown to depress them. A Dutch study of Raven's Progressive Matrices (a nonverbal test of g) for all 386,114 Dutch males who reached the age of 19 between 1963 and 1966 documents a drop in I.Q. scores across birth order and through increased family size. Zajonc and Markus (1975) report that

> intellectual performance as measured by Raven's Progressive Matrices declines with family size. . . . The magnitude of these effects is revealed in the fact that the highest (firstborn of two) and the lowest (last born of nine) scores in the Bellmont-Marolla data are separated by about two-thirds of one standard deviation. (pp. 74-75)

Zajonc and Bargh (1980 a & b) confirmed these findings and extended them to cover other studies.

Blake (1989), using an American sample, confirmed Zajonc's finding that increased sibsize has a negative effect upon cognitive ability. She also established that "verbal ability is found to be related negatively to sibsize and nonverbal ability is found to bear little or no relation" (p. 131). In addition, she found that in the group of 4511 white six- to eleven-year-olds tested, there was "close to a standard deviation difference between those from small and large families" (p. 131). She contends that since the results were controlled for parental education and since sibsize correlates with verbal ability but not with nonverbal ability, these results cast doubt upon "the claim that the association is due primarily to inherited I.Q." (p. 132). Nearly identical conclusions had been reached by Steelman and Doby (1983).

The research of Zajonc, Blake, and Steelman casts doubt upon any thesis requiring I.Q. tests to measure only innate intelligence. But it does more: When combined with family data on Black and white Americans, it helps explain part of the 15-point mean difference in I.Q. between populations. In 1988 the

percentage of Black families with four or more children was 5.6%, while the percentage of white families with four or more children was 2.5% (U.S. Department of Commerce, 1989, Table 65).

Another study by Zigler, Abelson, and Seitz (1973) lends further support to the theory that environment affects cognitive performance as manifest through I.Q. scores. They found that "disadvantaged children approach testing situations with a general situational wariness which results in their obtaining I.Q. scores beneath the level dictated by their cognitive competence" (p. 301). In contrast to the scores of nondisadvantaged children, who showed no such gain, there was a "mean 10-point I.Q. increase made by those disadvantaged children who were retested within a 1-2 week period" (p. 300). Kalechstein, Kalechstein, and Docter (1981), conducting similar studies, found that explicitly teaching disadvantaged Black children to follow oral instructions, mark responses, and use time and guessing strategies increased SAT scores by almost 40%. The importance of test-taking strategies to test scores was confirmed by Scruggs and Mastropieri (1986), Dolly and Williams (1986), and Frierson (1986). Clawson, Firment, and Trower (1981) found that test anxiety was higher among Blacks than whites and that this "seems to be a probable influence on poorer test performance" (p. 214).

Thus, numerous studies support the contention that I.Q. test scores are affected by environmental factors (i.e., I.Q. tests do not only measure innate intelligence). Some of these studies go so far as to claim the potential for almost a standard deviation's difference for one environmental effect.

The proposition that I.Q. tests are bias-free is no less contentious than the proposition that they measure innate intelligence. Canadian psychologists have expressed concern about cultural bias in the Wechsler tests for the past 30 years (Cyr & Atkinson, 1987, p. 101). Offering further support for the role of environmental and cultural bias in I.Q. tests, Scarr and Weinberg (1981) report a study of 130 interracial adoptions in which they found that "the range of reaction of socially classified Black children's I.Q. scores from average (Black) to advantaged (white) is at least one standard deviation" (p. 130). Scarr and Kidd (1983) also report that adopted Black children reared in advantaged white homes did as well as adopted white children reared in similar homes. Further, they report that the amount of Black ancestry is not a factor in cognitive achievement. And they state that "it is highly unlikely that genetic differences between the races could account for the major portion of the usually observed differences in the performance levels of the two groups" (p. 408).

A study by Fredrickson (1977) adds support to the thesis that I.Q. tests are culturally biased. On average, I.Q. tests demonstrate an approximately 15-point difference between Black and white American populations. However, Fredrickson found no such effect in his three year study of lower socioeconomic

preschool children in Cedar Rapids, Iowa. He reports that: "The mean I.Q. for the Black children was 101.40 and the mean I.Q. for the white children was 104.03" (p. 98). Fredrickson's study was conducted on, as he put it, "a very homogeneous group of children in socioeconomic status" (p. 97). The study controlled for occupation of breadwinner, family income, and education of mother and father. Fredrickson concludes that

> the most reasonable interpretation of the statistically nonsignificant differences in mean I.Q.s between black and white children in this study is the result of the equivalent cultural context for both populations. . . . The fact that black families in this study have roots extending back over 100 years in Cedar Rapids, that few blacks are unemployed, or that there has been little immigration during the last generation may account for the environmental equivalence with lower socioeconomic white families. (pp. 101-102)

This question of cultural bias has been addressed from a slightly different perspective by Blau (1981). She looked at the cultural background factors which predict the test scores of Blacks and whites. Blau found that different predictors operate for these two groups. For instance, demographic origin and sex of the child were important white predictors but these had no significance for Blacks; on the other hand, parents' social-class origins and mother's religious affiliation were significant predictors for Blacks, but not for whites (p.168). Increased family size, as in other studies, was found to be of major significance in depressing I.Q. scores of both groups. Speaking of Black children, she concludes:

> This book has presented in considerable detail evidence that their cognitive defects are not inherent but result from identifiable environmental deficits of a cumulative and complex nature that are associated with poverty, but which constitute independent impediments to the intellectual development of minority children.
>
> Poor Black ghetto children with several closely spaced siblings reared by poorly educated mothers without work experience, organizational ties, or exposure to white friends and co-workers are most at risk of entering school with severe cognitive deficits. In both races, sons of nonworking mothers in father-absent homes average the lowest I.Q. scores, but the incidence of such families is much higher among Blacks than among whites. (p. 222)

The question of cultural bias on I.Q. tests is addressed directly by Wechsler (1958).

> Clearly, the statistical definition of intelligence implies that norms obtained on any particular sample are valid only for such groups as the sampled population represents. . . . Thus, test norms obtained on Englishmen cannot be used for classifying Fiji Islanders. This is obvious to everybody. The principle involved, however, becomes less obvious when applied to less divergent groups, for example, the use of identical test norms for Negroes which are identical to tests originally standardized on white populations; it becomes still less so when the differentiae which might distinguish the groups, such as nationality, economic condition, and social status, are themselves hypothetical. Nevertheless, the limitations still hold. (p. 45)

Thus, a norm referenced test is only valid for the population which forms the normative group. As the foregoing analysis has attempted in part to demonstrate, different cultural norms adhere for whites and Blacks in America.

The same problem of norm referencing holds for comparisons between populations. As Wechsler said, if a test is norm referenced to a specific population as it must be to be valid for that population, then it is invalid for testing other populations with different norms. Similarly, normative tests, even though they have the same name (i.e., I.Q. tests) cannot be compared directly across normative groups because no standard of comparison exists. Therefore, to compare the scores of Black and white Americans on a white referenced I.Q. test is meaningless. And it is equally meaningless to compare American scores with Japanese scores, since the normative groups are distinct and different; no standard of comparison exists between tests.

To reiterate: Rushton's assumptions require that (a) intelligence be sufficiently heritable that other factors cannot account for any measured difference in intelligence between populations; (b) I.Q. tests measure innate (heritable) intelligence; (c) they measure I.Q. in a manner which is not culturally biased; (d) scores on independent, norm references tests are equivalent; and (e) I.Q. scores are greatest for Mongoloids and lowest for Negroids.

A review of the literature shows that current estimates for the fraction of intelligence due to heritability for a white, middle-class, American population ranges from 1/2 to 2/3. If these estimates are reasonably accurate, other factors than heritability may account for measurable I.Q. differences between Blacks and whites. Further, no data demonstrate that only heritable intelligence is measured by I.Q. tests; rather, functional or applied intelligence may be what is measured and that, at least in North America, this may be done in a culturally biased manner, since these tests have traditionally been norm referenced to a white, middle-class population. In addition, from a review of the literature and on the basis of Piagetian theory, we may suspect that the functional intelligence of the Black population is depressed from its innate level through the process of environmental and cultural deprivation. Finally, comparisons between populations defy the meaning of norm referenced, while comparing norms across populations requires a method of standardization which is absent by definition.

In the opening section of his paper Rushton makes another set of assumptions which can be treated in like manner. He hypothesizes that "the bigger human brain evolved to increase intelligence. . . . It seems reasonable, therefore, to assume a positive relation between brain size and intelligence" (p. 1010). If this assumption were sound, then elephants and whales would be very much smarter than humans; yet they are, by all accounts, less intelligent. However, further analysis of Rushton's causal chain would be wasteful. Rather, the time has come to analyze his use of data sources and his methodology.

Rushton treats cranial capacity and brain weight as independent, correlated variables; as a result, he considers them as independent subtraits of intelligence. However, they should only be treated as independent if brain density (brain weight divided by brain capacity) varies between populations (racial groups), which it has not been shown to do. Although Rushton points out that brain weight begins to decrease at some point after physical maturity is achieved, he does not put this observation to use. Therefore, it seems reasonable that, after detailing cranial capacity and brain weight findings separately, he should have reported these results as a single category. But this lapse is minor. Rushton's use of data to support his thesis that cranial capacity and brain weight are largest for Mongoloids and smallest for Negroids exhibits other, more important flaws than those of confusing dependent and independent variables and the resulting taxonomic problems.

Rushton's data for cranial capacity and most of his data for brain weight are unscientific and therefore inappropriate. Insufficient data are given by his sources, or even his sources' sources, for meaningful statistical tests to be performed on the data set. And Rushton's own manipulation of the data is methodologically unsound. In order to understand the failure of data and method, it is necessary to review first the data sources which he reports as original, then the intermediate accounts he utilizes, and finally his use of them.

Rushton cites two original data collections for cranial capacity: Howells'(1973) and Montagu's (1960). He does not, however, refer directly to either author for his data; instead he uses Coon (1982) and Molnar (1883) — second-hand sources. And it is no small wonder, for Howells did not produce any direct measures of cranial capacity, while Montagu's data are not original; rather they are invalid and unable to undergo meaningful statistical processing.

In his study Howells (1973) was not creating data for future use by Rushton; he was concerned with discriminating the relative importance among aspects of skull shape according to place of origin. For his measurements he used skulls from institutional collections. Many were old, while those from two of the three European samples were Medieval, and the sample from Gizeh, Egypt, dated from about 600-200 B.C. Howells's intent was to "represent a real population and time span" (p. 6). What he meant by this was not a cosmopolitan population, (e.g., Europeans) but a specific population "of narrow genetic origin" (p. 6). He selected thus so that the cranial variations within samples would be minimized; thus, he could determine the regularities within each population. Howells used three samples from Europe (e.g., Medieval Norse from Oslo, 55 males, and 55 females); he used only one sample from Asia, Buriats, "pastoralists occupying the region on either side of the southern end of Lake Baikal" (p. 28); and he used five samples from Africa, but two of them, Egyptians and Bushmen, were very dissimilar from the other three. In all Howells measured 17 populations around the world.

Howells's data, his measurements, were essentially of the outside of skulls; as Passingham (1982), an expert on primate brains, states: "The relation between cranial capacity as measured from outside the skull and from inside the skull is not at all close" (p. 122). In addition, Howells had few complete skeletons and did not report any data on body height. According to Passingham, adjusting brain size for body height is a necessary minimum but lacks accuracy, while measuring neocortex against medulla would provide accurate data on relative brain size (p. 83). This adjustment is necessary because bigger bodies require bigger brains to control them.

In addition, Howells's multivariate analysis did not indicate that all skulls were strictly classifiable into the racial categories which Rushton proposes. For both analysis by successive splits and by successive mergers, female and male skulls grouped differently (Howells, 1973, pp. 62-64). For instance, when clustered by successive splits, it was found for males that the three European populations grouped with the Amerindians and Hawaiians, while the Buriats grouped with the Eskimos in what Howells had previously termed "a reluctant combination" (p. 62). For the females, by contrast, even the Eskimos had moved closest to the Europeans and the Buriats were left on their own. And Howells's three-dimensional representation of the data (p. 65) shows clearly why this is so; the Buriats are isolated from all other populations. Therefore, of Howells's 17 populations, only seven represent samples from the three populations of interest to Rushton: Berg, Zalavar, and Norse from Europe; Teita, Zulu, and Dogon from Africa; and Buriat from Asia.

Coon (1982) reproduces Howells's diagram of clustering by successive splits, one of three different models he could have selected. To Howells's diagram he adds a column of cranial capacities and the statement that "Howells did not record cranial capacities. Those listed here were calculated by a standard formula" (p. 18). Unfortunately, the standard formula is not given. Coon goes on to state clearly and explicitly which populations group together on the basis of cranial features and that the North Asiatic Mongoloids are exemplified only by the Buriats. For added emphasis he quotes Howells to the effect that Europeans, American Indians, Greenland Eskimos, and Hawaiians are broadly related (p. 23).

Rushton makes use of Coon's calculations, but ignores his groupings. He reports: "I averaged the figures provided in Coon and found: Mongoloids 1401 cm^3, Caucasoids 1381 cm^3, and Negroids 1321 cm^3" (p. 1010). Rushton does not state which of Coon's figures he used to arrive at his "averages"; however, a simple recalculation demonstrates that he used Norse, Zalavar, and Berg for his Caucasoid calculations; Zulu, Dogon, and Teita for his Negroid calculations; and Arikara (Amerindian), Peru, Eskimo, Mokapu (Hawaiian), and Buriat for his Mongoloid calculations. Moreover, he added male and female data together in his process of "averaging."

The second set of data Rushton cites for cranial capacities is even weaker than the first. Rushton uses data from Molnar (1983) which he says are "based on independent data by Montagu (1960)" (p. 1010). However, Montagu's data are not original: he uses cranial capacities other authors had listed. None of these give sample size, but in some instances — for example, Gibraltor boy — a single individual obviously comprises the sample. And in others, like Piltdown man, we know that the sample was both singular and a fraud. In addition, cranial capacity was in some instances estimated visually, in others calculated from external measurements, and in yet others measured with mustard seed or water. However, Montagu was reliable in one way: His figures were meant to represent males only. When a female skull formed the sample he added 10 per cent to the size and listed it in italics (Montagu, 1960, p. 459).

Molnar's (1983) list is nothing more than a selection of 11 of the figures cited by Montagu. From Montagu it is clear that Molnar's list includes a visual estimate, measured samples, and calculated samples. And, unlike Montagu's listing, Molnar's does not provide names of the original author so that the data can be checked back to their source; as well, information on the method of determining cranial capacity is not provided. And yet Rushton relies upon this at least third-hand source for his data. In doing so he ignores Molnar's exhortation, which surrounds the table itself, that

> more nonsense has, perhaps, been written about the size of the human brain and its relationship to intelligence than about any other aspect of our anatomy. . . . It is improbable that the differences in modern populations' brain size have any relevance to variations in mental ability. . . . As Von Bonin (1963) a foremost neuroanatomist, once stated, the correlation between brain size and mental capacity is insignificant in modern Homo sapiens. (pp. 64-65)

Rushton "averages" this second- and third-hand data without regard for sample size. He does not adjust them for height or body size, a minimum necessary adjustment according to Passingham (1982). He combines data from Coon's sample without regard for Howells's findings regarding population fit. He combines data on males and females from Coon's calculations with data from Montagu which are for males only. And he does so without any adjustment whatsoever. He combines visual estimates with externally measured calculations and internal measures. He utilizes incomplete data sets, so that no meaningful statistical tests can be conducted on the data to assess them. Data which cannot be assessed are not valid: What Rushton is practicing is not science; it is not even quasi-science; it is pseudo-science.

Rushton continues by asserting that "concomitant differences emerge when calculations are made of brain weight" (p. 1011). Again he cites two data sources: Tobias (1970) and Ho, Roessmann, Straumfjord, and Monroe (1980). None of Tobias's data is original and little of the information necessary for statistical calculations is presented; Ho, on the other hand, except for the "brief

updating review" Rushton utilizes, provides a significant source of original and meaningful data.

Tobias's (1970) article is an attempt to demonstrate that valid comparisons between the brain weight of human populations require that account by taken of "sex, body size, age of death, lapse of time after death, temperature after death, anatomical level of severance, presence of absence of cerebrospinal fluid, of meninges, and of blood vessels" (p. 3). In particular, he notes that the studies he cites have not complied with these requirements.

Tobias provides a review of studies on brain weight on page 6, which contains table 2, utilized by Rushton for his calculations. Tobias, in his text and tables, variously lists the mean for each study and the range of means across studies for each population. To utilize the data, Rushton has to make a choice. Either he can average the specific means given by various researchers for each population, which provide nine pieces of data, or he can select the two extreme means given for each population and average the resulting six data points. Although as science neither data set has merit, the superiority of the first choice is that it utilizes all of the data points available without sacrifice. Nonetheless, Rushton chooses the second possibility. Thus, he calculates brain weight for Mongoloids 1368 gms, Caucasoids 1378 gms, and Negroids 1316 gms, which, when averaged together with subsequent data, will still leave Mongoloids with the heaviest brains. If, however, Rushton had chosen to maximize his data set, he would have calculated Mongoloids 1368 gms, Caucasoids 1411 gms, and Negroids 1308 gms. The result would have been a negligible difference between Mongoloid and Caucasoid brain weights when his final "averages" were computed.

Before moving on, it is worth listing Tobias's references for the studies of brain weight used by Rushton in his calculations, as we shall soon meet all of them again: Pearl (1934), Vint (1934), Kusumoto (1934), Shibata (1936), Appel and Appel (1942), Bailey and Von Bonin (1951), and Pakkenberg and Voigt (1964).

Rushton's second source for brain weights is Ho, Roessmann, Straumfjord, and Monroe (1980). This is the only source of useful scientific data that Rushton has included in his cranial capacity and brain weight analysis. It provided him with all the information he needed to draw a logical conclusion about the relationship between the brain weight of Blacks and whites: A conclusion he failed to reach.

Instead, following his figures from Tobias, Rushton says, "A briefer updating review was provided by Ho, Roessmann, Straumfjord, and Monroe (1980) from which I calculated: Mongoloids, 1334 g; Caucasoids 1307 g; and Negroids 1289 g" (p. 1011). To do this, Rushton ignored the figures reported by Ho's study and added up the figures provided by Ho for previous studies. The authors of these

studies, as listed by Ho were: Pearl (1934), Vint (1934), Kusumoto (1934) in Bailey and Von Bonin (1951), Shibata (1936), Appel and Appel (1942), Bailey and Von Bonin (1951), and Pakkenberg and Voigt (1964). This is an identical listing of authors, dates, and figures given by Tobias; yet Rushton uses it a second time as though the figures were all new. Not only that, he selects from the data, using only part of the data available, ignoring some of the higher white means. By combining data he has already used once with previously available but unused data, Rushton arrives at and presents a second set of "averages."

For a third set of data he turns to Ho's original data. He utilizes these data regardless of Ho's assertions that his population is older than others studied and that brain weight must be adjusted for age, regardless of Ho's assertion that brain weight must be adjusted for body dimensions, and regardless of the fact that only two of the three populations are represented in Ho's sample. In short, he adds his unrepresentative data to the duplicated data sets provided by Tobias and Ho's reviews and once again "averages" them to achieve his results. This is science?

The necessary caution is given explicitly by Ho. Nonetheless, explicit as Ho is, Rushton claims he

> also provided original brain weight data for 1261 subjects aged 25-80 collated from autopsy records after excluding those brains obviously damaged and found significant means differences between American whites and American Blacks: 1323 g vs 1252 g, a difference which held when controlled for body weight, height, sex or surface area. (p. 1011)

Ho did present original brain weight data as Rushton reports. He did not, however, find a significant mean difference between populations which held for all the controls Rushton claims. Instead, Ho reports:

> All the differences in brain weight between the white and Black populations are statistically significant when adjusted for any of the body dimensions, except for that between white and Black men, when adjusted for body weight. (p. 645)

In other words, Ho found that when adjusted for body weight, there was no statistical difference between the brain weights of white and Black men in his sample. Remember Passingham's minimal requirement!

But adjusting brain weight for body weight is not the only requirement. Ho states that

> the average brain weight for adults in our study, 1288 g, is lower than that reported from Europe, where means varied from 1300 to 1508 g. One of the reasons for our lower average is that the mean age of the population in our study is 60 years, whereas, in the reports cited (with the exception of one), it ranged between twenty and fifty years. (p. 637)

In fact, Ho reports that brain weight must be adjusted for age (p. 639) and for increase in body dimensions (p. 640). Moreover, he adds that "the relationship between brain weight and body weight must be established during the

developmental stages" (p. 644) because at six years of age "the brain reaches 92% of adult size" (p. 639) and "the brain does not respond to later fluctuations of body weight . . . in contrast to the developing brain for which malnutrition is said to result in a decrease in weight" (p. 644). The foregoing clearly demonstrates that Rushton misstates Ho's findings. This example is only one of many that could be given, in which Rushton cites an authority in support of his argument, when in fact the authority's statements directly contradict Rushton's thesis.

In his opening section, Rushton consistently misconstrues information paraphrased from his sources. While developing his hypothesis about intelligence and brain size into an assumption, he purports to paraphrase from Passingham when he says, "Mammals with larger brains learn faster than those with smaller brains" (p. 1010). The relevant passage from Passingham's *The Human Primate* (1982) actually states, "Primates can be ranked according to the amount of neocortex in relation to the medulla" (p. 124).

The reality of what Passingham said, as opposed to Rushton's paraphrase, strikes at the core of Rushton's argument that cranial capacity or brain weight can be used as a direct indication of intelligence. This is especially true since Rushton cannot, and hence does not, even adjust his figures for body weight, Passingham's minimal requirement for valid comparisons. As Passingham states, "Within the primates comparisons of the different groups produce anomalous results when they are based on encephalation indices using brain and body weight. . . . The picture becomes more orderly when the brain is related not to the size of the body but to a more direct measure of the inputs and outputs of the brain, such as the medulla" (p. 79). Although Passingham made this statement with regard to comparing primates of different body size, the possibility exists that, should Rushton be correct insofar as some real difference in brain size occurs between human populations, any observed variations in brain size could be accounted for through an equal difference in medulla dimensions.

Rushton continues by asserting that "Passingham (1982) updated the evidence for a positive correlation between brain size and intelligence within human populations" and that "He carried out additional analyses to discover that intelligence, indexed by occupational status was also related to brain weight at autopsy even allowing for differences in height" (p. 1010). It is worth noting what Passingham actually said.

> It is possible to demonstrate a very loose relation between cranial capacity, as measured from outside the skull, and intelligence as assessed in a modern population. But this effect disappears when account is taken of differences in height. We should not be surprised at this negative result, since the relation between cranial capacity as measured from outside the skull and from inside the skull is not at all close. The lack of a close correspondence between external measurements and internal cranial capacity, and therefore brain size,

> could well mask a small, but genuine, relation between brain size and intelligence. (pp. 121-122)

Note that, although Passingham undoubtedly wishes he could confirm a direct correlation between brain size and intelligence within the human population since that would dovetail neatly with the thesis that the human brain has evolved in size to evolve in intelligence, he does not do so. In fact, in the section to which Rushton refers, Passingham says:

> Unfortunately there are no records of intelligence as assessed on proper tests, and we must make do with the occupation in which the people engaged in their lifetime, on the assumption that these give some indication of abilities. When differences in height are allowed for we find that there is indeed a relation, though slight, between occupation and the weight of the brain. In this sample relative brain size is marginally bigger in professional people than in semi-skilled and unskilled workers. But the effect is very small, and there is considerable overlap between the groups. We should be very cautious in interpreting this finding. (p. 122)

Rushton, of course, is not cautious at all. His thesis is that larger brains are more intelligent and therefore achieve higher status. However, the data are amenable to alternative explanations, such as occupational status tends to reproduce itself across generations, within families; nutrition and health care tend to be better in higher status homes; and quality of nutrition and health care correlate closely with brain size; therefore, on average, the well nourished brains of high status children are likely to grow larger than those of other status groups.

In fact, in his opening section, "Brain Size and Intelligence," Rushton offers no valid data, open to statistical analysis, which support his contention that intelligence is heritably fixated. Nor does he produce a causal relationship between intelligence and cranial capacity, brain weight, and test scores. Instead, in an effort to convince without evidence he utilizes unstated and unwarranted assumptions, he infers causality where none exists, he uses unacceptable statistics in an unscientific manner, he uses invalid and unreliable sources, and he claims support from sources which contradict his thesis.

The same pattern of assuming instead of demonstrating a causal chain; of errant methodology; and of spurious, inappropriate, and inaccurately paraphrased data sources continues as Rushton's analysis progresses. In section two, *Maturation Rate*, Rushton states, "For some measures of ossification development [author's note: skeletal hardening], Mongoloids appear to be as advanced as Negroids" (Eveleth & Tanner 1976, p. 1013). Turning to Eveleth and Tanner we find that

> African children under good nutritional and environmental circumstances are more advanced that Europeans in skeletal development from birth to adolescence. There are no data showing whether the advancement continues into adolescence, though it seems probable. Quite a different pattern emerges for Asiatics, represented by Chinese and Japanese. Although similar during

> childhood to Europeans they become advanced at adolescence. (p. 206)

In fact, although Mongoloids are skeletally slow to develop during early childhood, they consistently catch up to and surpass both Caucasoids and Negroids on the various measures of skeletal development. In some areas they have caught up by age eight, and they have caught up and moved ahead of both groups, in all areas, by age 13-14 (Eveleth & Tanner, pp. 198-206). In addition, in refutation of Rushton's assumption that maturation is wholly heritable, it is worth noting the findings of other studies reported in the same book.

> Skeletal maturation is slower in lower socio-economic groups compared with upper and middle groups. . . . The characteristic Asiatic increase in rate at adolescence began a year earlier in the better-off boys and still earlier in the better-off girls. (p. 203)

It becomes clear that, as opposed to Rushton's contention that skeletal development in Negroids occurs sooner than skeletal development in Caucasoids, which occurs sooner than comparable development in Mongoloids, Rushton's own authority contends that the true order changes with age and that, as maturity is approached, Mongoloids are more mature and maturing faster than Negroids who are more mature and maturing faster than Caucasoids.

Another example of spurious and inaccurate data sources is contained in Rushton's claim that sexually, as determined by first menarche, first intercourse, and first pregnancy, Mongoloids mature later than Caucasoids, who mature later than Negroids. In support of this he refers to an unnamed French Army Surgeon, a specialist in genito-urinary diseases, and studies by Malina (1979) and Rushton et al. (1987). There are several severe problems with Rushton's data sources, not the least of which is that, at least with regards to first menarche, Rushton has his order wrong again.

However, before moving on to the data which refute Rushton's claim, it is worth taking a closer look at his data sources. Rushton begins by referring to the ethnographic record, specifically an unnamed French Army Surgeon. That the unnamed surgeon is Dr. Jacobus is made clear from Rushton's citation of *Untrodden Fields of Anthropology*. No serious scholar would accept this work as part of the legitimate ethnographic record. Rather, it dates from an era when pornography was strongly suppressed and represents an effort to present sexually titillating material under the guise of scholarship.

Turning now to refutation of Rushton's claim for order of first menarche: According to numerous (approximately 100) and wide-ranging studies assessed by Eveleth and Tanner, median age at menarche for Hong Kong Chinese is 12.5 (well-off), 12.8 (middle-class) and 13.3 (poor). For Americans of European extraction median age at menarche is 12.8 and for Europeans in Europe it ranges from 12.5 to 13.2. "Though Africans in Africa, even those apparently well-off are relatively late (13.4 Uganda, 14.1 Nigeria) Afro-Americans are early,

averaging 12.5 years" (p. 213). Similarly, Vernon (1982) found that "Japanese in California reach menarche 20 months earlier than Japanese in Japan" (p. 75). Therefore, as the data stand, on average, the median age of first menarche is lowest for Mongoloids and Caucasoids and highest for Africans, exactly the opposite of what Rushton claims. However, the range across countries but within populations is considerable and the variation accounted for by socioeconomic status is equally substantial. Therefore, not only can it be said that Rushton erred in his findings, but also that effects confounding heritable factors must be significant. In addition, it is worth pointing out that between 1840 and 1970 mean age at menarche in Europe (the only population Eveleth and Tanner give records for) has dropped from 17 years to 13 years (p. 218). They note that "improvement in environmental conditions, chiefly nutritional, is a principal cause of earlier maturation" (p. 217).

Equally invalid are Rushton's claims that age of first intercourse and age of first pregnancy are indicative of rate of maturation. Age of first intercourse in Caucasoid populations has been dropping steadily for 30 years. Given that puberty is usually reached before first intercourse, cultural and environmental factors, not heritability, determine the timing. The same can be said for first pregnancy. Any attempt to develop a causal link between first intercourse or first pregnancy and maturation rate is spurious.

Rushton makes the same claims for longevity that he does for skeletal development and age at menarche. According to him, Mongoloids outlive Caucasoids, who outlive Negroids. However, Rushton only cites statistics for the United States. If longevity is a fixated heritable trait across populations, as it would need to be to fit his theory, then global figures should confirm his American data. They do not. Life expectancy depends more on where one lives than on race. For instance, using only statistics for males, 500 million Mongoloids in China can expect to live 67 years, while across the water in Japan, males can expect to live 75 years. In Hong Kong males can expect to live to be 74. On the other end of the scale, Mongoloid males in Vietnam have a life expectancy of 57 years, in East Timor of 39 years, and in Kampuchea of 42 years. By comparison, male Caucasoids in India can expect to live to be 56 but if they live in North America or Western Europe they can expect to live into their early 70s. Africans are not so lucky. If they are born in North America, males can expect to live into their mid 60s, but in most parts of Africa life expectancy is much shorter. It varies from a low of around 33 to 34 in Sierra Leone and Gambia, rises into the 40s and 50s for most of continental Africa, and jumps to from 70 to 74 on islands off the coast (United Nations, pp. 154-164).

Clearly, there is vast disparity between life expectancies within races, but across countries. The same disparities exist within races but through history. In the past 300 years the life expectancy of Europeans has risen from the mid-30s to the low 70s: It has doubled. Comparable changes have occurred within the

United States for Blacks between 1920 and 1985. In 1920 a Black man could expect to live 45 years, today he can expect to live 65 years. In fact, in relative terms, increases in his life expectancy have been outpacing those of his white counterparts.

It is impossible, on the basis of the available figures, to give any meaningful rank ordering to life expectancy between races. What can be said is that the confounding factors of nutrition, climate, medical care, cultural attributes, socio-economic status, and doubtless others which this author has not recognized, affect life expectancy to such a great degree that no meaningful comparison across populations is possible.

At the other end of life, Rushton uses gestational period in his ranking of races. He claims that "In the USA, Negroids have a shorter gestation period than Caucasoids" (p. 1012), but does not rank Mongoloids. Rushton claims that "By week 39, 51% of Black children have been born, while the figure for whites is 33%; by week 40, the figures are 70 and 55% respectively" (p. 1012). Although cross cultural gestation studies have been impossible to obtain, Neligan, Kolvin, Scott, and Garside (1976) completed a seven-year controlled study, *Born Too Soon or Born Too Small*, of premature and low birth weight children in Newcastle, England. They found that short gestational period (defined by them as 36.5 weeks and earlier) was strongly correlated with low social class (pp. 11-14). It should not be surprising then, that Black Americans, who have historically been discriminated against and as a result are primarily of low social class, have a shorter gestational period than do whites.

With regard to multiple birthing, Rushton claims that the rate of dizygotic twinning is highest in Negroids and lowest in Mongoloids (p. 1014), thus confirming his r/k reproductive strategy claims for egg production and Negroids's relative r strategy approach. However, Gedda, Parisi, and Nance (1981) report data in *Twin Research 3* which indicate that the incidence of dizigotic twinning is not heritably fixated. They indicate that rates of dizigotic twinning have fluctuated wildly over the period during which records have been kept and that the rate for industrialized countries has been dropping sharply since the 1950s and in some cases earlier. Specifically, data from Scotland indicate that the rate was 12.6 in 1860, 8.9 in 1939, and 10.9 in 1958; however it has fallen consistently since then to present levels around 5.0 (pp. 16-17). Similarly, in Japan the rate had fallen from 2.5 in 1958 to 1.9 by 1974 (p. 23). In Italy, where the rate had been relatively constant throughout the period 1879 to 1940 at 9.0 and then climbed to 10.0 for the period 1940 to 1950, it had fallen to 5.0 in 1970 (p. 45). Studies conducted in Virginia indicate that the rate of dizygotic twinning was 13 for whites and 16 for Blacks in 1918 and has since declined consistently to reach its present level of 9 for whites and 11 for Blacks (p. 63). Clearly, the rate of dizygotic twinning is not heritably fixated, since it has declined in some populations by up to 50% in the last 20 years, and since it

shows a consistent decline through the latter half of this century for all industrialized populations studied.

The time has come to declare that Rushton has prejudiced his study beyond redemption through his unstated assumptions, his unwillingness or inability to address and account for theories in competition with those supporting his thesis, his use of spurious and inappropriate data sources, his inaccurate paraphrasing of statements made by other authors so that they seem to corroborate his thesis when in fact they oppose it, his omission of contradicting data from within the sources he quotes, and his unscientific methodology. At least for the specific traits reviewed, Rushton has failed to demonstrate the validity of his thesis that "Racial differences exist on numerous behaviour traits such that Caucasoids fall between Mongoloids and Negroids" (p. 1009). He has failed for all the reasons just mentioned; but most importantly, he has failed because the available empirical evidence does not support his thesis.

However, before putting this matter to rest, it may be worth reviewing the underlying theory through which Rushton seeks to unify his argument: the theory that "these observations may be explained in part in terms of gene-culture coevolutionary based r/K strategies" (p. 1009).

According to Rushton, the basis of r/k reproductive theory is that there are two reproduction strategies: r strategy involves "maximum egg production and no parental care," while K strategy involves "elaborate parental care in which the birthrate is reduced to a minimum" (p. 1018). He further contends that all members of the animal kingdom operate at some point along the continuum from r to K; humans are the most K of all, but some humans are more K than others (pp. 1018-1019). He adds:

> The more K the family the greater the spacing between births, the fewer the total number of offspring, the lower the rate of infant mortality, the better developed the parental care. The more K a person, the longer the period of gestation, the higher the birth weight, the more delayed the onset of sexual activity, the older the age at first reproduction, the longer the life, the lower the sex drive, the higher the intelligence, the more efficient the use of energy, the lower the dispersal tendency, the more social rule following the behaviour, and the greater the altruism. (p. 1019)

That is, Rushton seeks, through an analysis of supposedly racial-genetic traits, to demonstrate that evolution has made Mongoloids more K-selected than Caucasoids and Caucasoids more K-selected than Negroids in an evolutionary system where, he contends, more K represents more advanced genetic development.

Further, Rushton seeks to link r/K reproductive strategies with a theory from evolutionary- and socio-biology denoted as "gene-culture coevolution." This theory proposes that genetic development and cultural development evolve in parallel and that each factor influences the direction of development taken by the

other. Thus Rushton proposes the theory of "gene-culture coevolutionarily based r/K reproductive strategies" to explain the ordering of traits and races.

Rushton refers to various authorities on r/K reproductive strategies and Lumsden and Wilson (1981) on gene-culture theory. He also refers to specific authorities in support of particular statements he makes. Analysis of these authorities demonstrates that Rushton misrepresents both the meaning of r/K strategies and their applicability to his thesis. Further, analysis of Lumsden and Wilson's gene-culture theory shows it to be highly contentious and apparently without particular merit.

Rushton claims that

> the symbols r and K originate in the mathematics of population biology and refer to two ends of a continuum involving a trade-off between egg production and parental care. These range from r, involving maximum egg output and no parental care, to K, emphasizing elaborate parental care in which the birthrate is reduced to a minimum. (McArthur & Wilson, 1967; Wilson, 1975; Rushton, 1988, p. 1018)

Turning to McArthur and Wilson (1967), we find that they are interested in changes to island biogeography following colonization. They define K (a term originated by MacArthur in 1962) as the carrying capacity of the environment (p. 69) and r as the intrinsic rate of increase (p. 83). They theorize that, during initial or re-colonization, an uncrowded environment exists and success will be promoted by a strategy of r-selection, where fitness will be defined by the ability to harvest the most food and hence rear the largest families. "At the other extreme, in a crowded area, (K selection), genotypes which can at least replace themselves with a small family at the lowest food level will win" (p. 149).

Wilson (1975) defines r selection as "selection favouring rapid rates of population increase, especially prominent in species that specialize in colonizing short-lived environments or undergo large fluctuations in population size" (p. 593). He defines K selection as "selection favouring superiority in stable, predictable environments in which rapid population growth is unimportant" (p. 587).

It becomes apparent that, rather than K selection being postulated as a superior evolutionary survival strategy, the two opposing strategies are postulated as each being superior under certain conditions. Moreover, neither MacArthur and Wilson (1967) nor Wilson (1975) defines r selection and K selection in like manner to Rushton, contrary to his claim.

Rushton continues by asserting that "these reproductive strategies are correlated with other life history attributes. Following Pianka (1970), Wilson (1975), Eisenberg (1981), and Barash (1982), these are summarized in Table 3" (p. 1019). Indeed, Pianka (p. 593) and Wilson (p. 101) have produced nearly identical tables; however, these are not identical to Rushton's. Eisenberg and

Barash, on the other hand, do not speak directly to this issue. A comparison of Rushton's Table 3 with the original and identical table published in both Pianka and Wilson demonstrates how Rushton has extended this theory beyond the experts' intent and warped it to fit his purposes.

In fact, none of the authorities Rushton cites makes any direct connection between r/K theory and variations within or among human populations, nor does anyone propose such a theory. Only Lovejoy (1981), an anatomist and anthropologist, not an expert in population biology or sociobiology, connects humans directly with r/K strategies. In doing so he directly contradicts at least one of Rushton's central theoretical assertions.

Lovejoy is interested in bipedality and why it occurred. He applies r/K theory, as he understands it, to the evolution of hominoids (primates) in general and to the evolution of hominids (man) in particular. According to Lovejoy's theory, early hominoids had developed K strategy (few offspring and stable habitat) to such an extent that they were unable to take advantage of opportunities to disperse to new habitats. Lovejoy postulates that a shift to r-strategy population growth was required. However, of birth spacing, gestational period, infant dependency, and sexual maturity, "only birth spacing can be significantly shortened without alteration of primate ageing physiology" (Lovejoy, 1981, p. 343, Figure 2). Therefore, according to Lovejoy, bipedality arose as a behavioral pattern which allowed for shorter birth spacing and better survivorship. The result was an r selected shift which affected reproductive rates and enabled hominids to disperse more readily to fill a vacant ecological niche. It also provided the opportunities required for the development of greater intelligence and "it accounts for a functional, rudimentary material culture of long-standing, and it accounts for the greater proportion of r-selected characters in hominids relative to other hominoids" (Lovejoy, p. 348; see also Johanson & Edey, 1981, p. 321).

Therefore, not only does Lovejoy contradict Rushton's assertion that "Primates are all relatively K-strategists and humans are the most K of all" (Rushton, 1988, p. 1019), but he also theorizes that a shift of direction toward r-strategy was the precursor to the development of increased hominid intelligence and material culture, among other things.

Underpinned by their extension of r/K theory, Rushton and Lovejoy have proposed theories which are in direct conflict and result in opposite conclusions. This is not entirely surprising, since r/K theory is outside the scope of their expertise and their application of it is far removed from the realm in which it was created and the purpose for which it was intended to be applied.

The absence of an explicit, authoritative link between r/K strategies and gene-culture coevolution theory parallels the absence of an explicit link between r/K strategies and within-human-population variations; however, Rushton is not

deterred, which well he might be, considering that Edward Wilson was a prime mover in both fields and might have been expected to make clear any link that existed. Nonetheless, Rushton explicitly links variations in K-selection among races and gene-culture theory.

Gene-culture coevolution theory predicts that changes in gene frequencies cause changes in culture, which in turn causes further changes in gene frequencies (Lumsden & Wilson, 1981, pp. 11, 372); as well it predicts that, by extension, culture develops differentially for different gene pools and that this explains the variety of cultures among human populations.

Gene-culture theory is a development of sociobiology, itself a highly contentious discipline (Ruse, 1979). Although it ought not be dismissed lightly, the relationship between genetic change and cultural change is certainly no less contentious. Gene-culture coevolution has yet to be clearly and unambiguously demonstrated for any organism at an advanced level of evolution. Moreover, the history of human cultural development seems to deny gene-culture coevolution any importance in recent human history. During the last five thousand years various locales have been seen to attain and then lose cultural dominance. Gene-culture theory does not explain through what mechanisms dominant populations lose sway. Nor does gene-culture coevolution explain why the pattern of cultural dominance in the Western world has seen a counter-clockwise progression around the Mediterranean Basin and up the Atlantic coast of Europe which then jumped across to North America. The long-term motion of cultural dominance and the rapid techno-cultural changes of the last 300 years seem to testify to cultural transmission and cultural motion via adjacent learning environments rather than alteration through rapid gene-culture interaction.

Shifting back to Rushton's linkage of r/K strategy and gene-culture, if Mongoloids are most K and if most K is the highest level of gene-culture, then Mongoloids should be dominant. But any claim to Asian parity, let alone dominance, was lost at least 500 years ago. Rushton's thesis should require not only that K-strategy and gene-culture combine to explain reality, which they do not appear to do, but also that once Mongoloids or any other race achieved cultural dominance, dominance was maintained. Clearly, such has not been the case.

This analysis of Rushton's work is at an end; there is no question but that he failed in every respect. Not only has Rushton failed to demonstrate that the traits he assesses are ordered among races as he theorizes, he has also failed to demonstrate linkage between the traits themselves and K-strategy. In addition, he has failed to demonstrate that K-strategy is a superior evolutionary strategy to r-strategy, that more K is better. Finally, he has failed to demonstrate linkage between r/K theory and gene-culture coevolution theory. In short, there is no causal chain.

The final question is one of response. The correct response is not, I believe,

one of suppression. That is no solution. Instead, I propose that it is up to us, as teachers, to make available a wide range of data and theoretical perspectives so that our students and the public can fully inform themselves. This process involves presenting not one, but all theoretical perspectives and their supporting data. It is only through such a process that contending theses can be explored and an informed position arrived at by each individual. It is in this way that critical thinking skills will develop and the truth, relative as it may be, will out.

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