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# Race Differences in Sexuality and Their Correlates: Another Look and Physiological Models

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Lynn (1989) rejects the *r/K* theory of race differences, claiming that (a) *r/K* analyses do not apply to large mammals like man; (b) the races do not really differ on attributes such as altruism, brain size and intelligence, and sexuality; (c) if they do, the differences do not relate to reproduction; and (d) if they do, environmental variables will explain them. In response I note that (a) *r/K* analyses do apply to large mammals, including primates; (b) the races differ quite markedly in altruism, brain size and intelligence, and sexuality; (c) reproductive variables are affected as in sexually transmitted diseases being a major cause of infertility among Negroid people and in the relative incidence of AIDS and HIV-1 seroprevalence in these populations; and (d) no alternative explanation has been provided for the covariant pattern of multifarious differences. These include morphological characteristics (depth of voice, salient muscularity, buttock size), personality traits (activity level, aggressiveness, cautiousness), social organization (marital stability, mental health, law abidingness), speed of maturation (age to walk alone, age of first breeding, age of death), and sexual variables (gamete production, intercourse frequencies, size of genitalia). On all the differences, Caucasoids fall *between* Mongoloids and Negroids. Possible physiological mediators of the covariant relationships are discussed.

## INTRODUCTION

M. Lynn (1989) faults my arguments in several ways. This reply is differentiated by the sections outlined in the abstract. As a general point, it is worth noting that no alternative theory is proposed to account for the numerous data sets discussed, only piecemeal criticisms too often falling back on theories of "stereotyping," "self-fulfilling prophecies," and "bias," none of which can account for critical variables such as gamete production nor explain why Mongoloids are typically more *K*

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than Caucasoids. Later in this paper I attempt to show the direction of development of such a formulation. First, let us deal with particulars.

## LIFE HISTORY VARIABLES IN PRIMATES

Lynn (1989) argues that $r/K$ analyses "have little effect on larger mammals like man" (Abstract), that their application to human race differences is "implausible," and that I misrepresented particular theories. His reading is incorrect on all counts. Life history measures vary enormously even among nonhuman primates. Following Harvey and Clutton-Brock (1985, Table 1) the following figures are provided for nonhuman primate species (with those for *Homo sapiens* in brackets). Gestation lengths range from 60 to 250 days (267); birth weight from less than 10 to over 2000 g (3300 g); litter size typically is 1 but twinning is very common in some species (1); weaning age from less than 50 to over 1500 days (720); female age at first breeding from less than 1 to over 9 years (>10); adult brain weight from less than 10 to over 500 g (1250); and longevity from less than 10 to over 40 years (70). Across the primate species most of the life history measures are positively correlated, although the relationships are not perfect. Note, for example, that humans do not have the longest weaning time. It was to account for this type of patterning in data that $r/K$ theory was formulated (Wilson, 1975), and several authors have applied it to the larger mammals (Wilson, 1975; Eisenberg, 1981). Differences between human subspecies (races) in these variables would thus seem to belong in a broader evolutionary context than has been considered to date.

Lynn perhaps misread my earlier papers, for in them are references to Lovejoy (1981) and Johanson and Edey (1981) that provide an $r/K$ perspective on how the *Homo* line became differentiated from nonhuman primates (see, for example, Figs. 1 and 2 in Rushton & Bogaert, 1987). Some of this was reiterated in my first reply to Lynn (Rushton, 1989), where it was also suggested that a move away from direct aggressive sexual competition to indirect cooperative competition through pair bonding underpinned human origins. Thus, far from $r/K$ strategies being irrelevant to the larger mammals, they have been postulated to underlie the very existence of our species. The new evolutionary approaches to the total "life history" of different species, as well as to their ecological and genetic determinants, constitute a revolution in the study of animal behavior (Wilson, 1975). In studies of dandelions, fish, flies, milkweed bugs, and field mice, many of the covariant $r/K$ traits are also found *within* species, and to be genetic in origin (see Rushton, 1985a, for review). There is no reason why such analyses should not be applied to human differences.

Lynn's claims that I misused a theory by Brooks and Wiley (1986) concerning the role of entropy in evolution is quite misleading. This theory was mentioned, without evaluation, as one of many possible approaches to the evolution of *r*/*K* covariant life history variables. It merits full consideration as a possible explainer of within-species differences, as do alternatives (Eisenberg, 1981; Partridge & Harvey, 1988; Stearns, 1977). Similarly, whether in any sense of the word evolution is "progressive" in nature and whether some people are "more advanced" than others may be worthy of attention.

## THE REALITY OF RACIAL DIFFERENCES

If racial differences are truly random they will be distributed around a mean of zero (no difference) with an equal number of negative results for *r*/*K* theory as positive. Instead most predictions are confirmed, with only a few being null and hardly any going in the opposite direction (see Table 1, Rushton, 1989, following Rushton 1988a, 1988b). Lynn claims this conclusion "is ridiculous" (p. 8). Here then is the place for refutation. If the opposite to predicted patterns could be observed in crime, rough and tumble play, marital stability, longevity, or other relevant variables, Lynn's critique would gain credence. Since there is an explicit 1, 2, 3 ordering on each variable, there is ample opportunity for disconfirmation. Such a refutation is not provided. Instead of contradictory evidence Lynn suggests that I "unconsciously selected and examined, only those hypotheses . . . supported by data," and that I ignored body size and altruism from consideration because these were anomalies for my argument. Lynn further suggests that because of inadequacies in some of the studies of brain size (e.g., nutrition was not controlled for) and sexuality (e.g., white males did the interviewing), the null hypothesis warrants support. He suggests that when "adequate" studies are carried out, they show "no difference."

Let us, then, consider more of the evidence. With respect to body size, I agree that large size is *K* and that this is an apparent anomaly. I said so quite explicitly in a work (Rushton, 1985a, p. 449) that Lynn has cited on two separate occasions; I also drew attention to body size being a problem for *r*/*K* analyses in other species. With respect to altruism, in which Lynn notes I have a long-standing interest, I have consistently argued that in humans, this depends on rule-following behavior in addition to temperament variables like empathy (Rushton, 1980, 1988c). As such, population differences opposite altruism, unlawful victimizing behavior, can be examined. (For evidence that altruism and aggression are usefully conceptualized as opposite ends of a continuum, see Ellis, 1988; Eron, 1987; Olweus, Block, & Radke-Yarrow, 1986; Rushton, 1988c.)

### *Altruism (Law-Abidingness) and Aggression*

Wilson and Herrnstein (1985) review much of the relevant literature on race differences in law abidingness. The Chinese and Japanese in North America have a lower incidence of crime than do Europeans, even when controlling for social class, on which they are higher (see also, Vernon, 1982). Afro-Americans currently account for about half of all arrests for assault and murder and two-thirds of all arrests for robbery in the United States, while constituting about one-eighth of the population. Since about the same proportion of victims say their assailant was black, the arrest statistics cannot be attributed to police prejudice. Blacks are also overrepresented among persons arrested for white collar offenses. In 1980, for example, blacks made up about one-third of those arrested for fraud, forgery, counterfeiting, and receiving stolen property, and about one-fourth of those arrested for embezzlement. Blacks are underrepresented only among those white-collar offenses that ordinarily require, for their commission, access to high status occupations (tax fraud, securities violations). Similar figures are found in Britain: while consisting of 13% of the population of London, African-descended people account for 50% of the crime. These findings also hold regardless of whether they are based on surveys of victims or on official police records. Mongoloid immigrants to Britain are underrepresented in crime. Cross-cultural studies of developing countries suggest that the findings may be generalizable (Wilson & Herrnstein, 1985).

A study carried out in Quebec suggests that the racial pattern in prosocial behavior is generalizable. A sample of 825 4- to 6-year-old children, from 66 different countries, speaking 30 different languages, were assessed by 50 teachers. All the children were in preschool French language immersion classes for immigrant children in Montreal to enable better integration into the school system. Only 20% of the children were born in Canada, with the black children often coming from French language countries like Haiti, the white children from Spanish-speaking countries like Chile, and the Oriental children from what used to be French Indo-China (Vietnam, Kampuchea). Teachers reported better social adjustment and less hostility–aggression from Mongoloid than from Caucasoid than from Negroid children (Tremblay & Baillargeon, 1984). Because this study was carried out in a unique cultural setting, examining children from parts of the world rarely studied, many of the circumstantial objections raised about research in English language countries are obviated.

### *Brain Size and Intelligence*

Lynn (1989) reiterates his belief that Gould (1981) had discredited published findings in this area and deals with my presentation of Table 2 (Rushton, 1989) by arguing that only "inappropriate aggregation of subsamples" could support my rank ordering, that "Ancient Caucasians" should be eliminated because they were smaller than Modern Caucasians, and that my arguments show the same sort of bias that Gould (1981) points out in Morton's original analyses. I urge the reader to look back to Table 2 (taken directly from Gould, 1978, 1981) and assess this argument for themselves. First, if one accepts Lynn's position and eliminates the Ancient Caucasians, this leaves a 4 in.$^3$ difference in internally measured cranial capacity between Mongoloids and Caucasoids on the one hand and Negroids on the other. Even if this is somewhat overestimated, the residual cannot be ignored. Second, if body size is controlled, as in the studies by Tobias (1970), and Ho, Roessmann, Straumfjord, and Monroe (1980a, 1980b), which Lynn dismisses on other grounds, the rank ordering is indeed Mongoloids > Caucasoids > Negroids when the brain–body allometric regression is taken into account, because Mongoloids are typically smaller in body size than Caucasoids, except for their heads.

The existing data may be far from perfect, but on the basis of all that I have been able to find, data from around the world show the rank ordering as reported, charges of bias notwithstanding. Indeed, it can be debated whether bias is more manifest in the writing of Stephen Jay Gould, the Harvard paleontologist and anti-sociobiological ideologue, than of researchers like Samuel George Morton (1799–1851), one of the giants of the American scientific community of his time, who Gould attempted to discredit. Evidence has already been presented of self-confessed bias on the part of Gould (Rushton, 1989). Additional information comes from a recent history of the mental testing movement, seven or so decades ago (Sokal, 1987). While Gould's (1981) book denigrated the goals of these early efforts and cast aspersions on the scientific integrity of the pioneers, the newer work gives a more balanced view of events and personalities.

Gould's (1981) unreliability as a scholarly guide has been revealed for other parts of *The Mismeasure of Man.* For example Jensen (1982) documented Gould's tendency to represent in "false, misleading, or grossly caricatured" ways (p. 125) the view of those with whom he disagrees. More recently a spate of correctives in the September 1988 issue of the *American Psychologist* have shown that Gould's charge of "conscious skullduggery" in Goddard's (1912) study of the heritability of feeblemindedness in the Kallikak family was unwarranted. Gould had claimed that Goddard's photographs were "phonied" by inserting heavy lines to give eyes and mouth a "diabolical appearance." However, not only was such retouching quite common during the period but an empircal study has shown that the retouched photographs actually strike judges as appearing kindly (Glenn & Ellis, 1988). Finally, Michael (1988) remeasured a random sample of the Morton collection of human crania to check Gould's charge that Morton "unconsciously" doctored his results to show Caucasian racial superiority. Michael found that, in fact, very few errors occurred and these were not in the direction Gould had asserted. Instead, errors were found in Gould's assessment, and Michael concluded that Morton's research "was conducted with integrity" (while) "Gould is mistaken" (p. 353). Morton was trying to understand racial variation and not, as Gould claimed, trying to prove Caucasian racial or intellectual superiority. What is required to refute the findings on race differences in brain size is not character assassination, but better data, as could be gained from the new computer-assisted brain imaging techniques increasingly available.

A climate of fear, however, has descended upon researchers in connection with race, a climate fueled in part by anti-sociobiologically motivated scientists like Gould, Kamin, Lewontin, Rose, and their like. Brain size and genetic differences are typically not even listed as alternative hypotheses to explain the very clear differences between the races in educational achievement. In a review in *Science*, Steen (1987) examined data on mathematically precocious youth in the United States, finding that the proportion of Asian American students who achieve high mathematics scores (above 650) on the Scholastic Aptitude Test is twice the national average, while the proportion of black students is less than one-fourth the national average, and internationally, 5th and 12th grade level students in both China and Japan score higher than equivalent white Americans, whereas those from African countries do not. While Steen (1987) discussed the potential strategic problems this may create for the United States, she does not raise the question of whether the differences might be genetic.

A recent news feature in *Science* documents how black Americans fall behind their white and especially their Asian counterparts in mathematics (Norman, 1988). The study, sponsored by the National Science Foundation (NSF), involved 28,000 students in the public schools of Montgomery County, Maryland, a relatively affluent area bordering on Washington, D.C. which puts substantial resources into education. The NSF official who oversaw the project describes Montgomery County as having one of the best school systems in the country, with students performing above the national average in mathematics. Racial differences begin to show up as early as the first or second grade, and become more pronounced throughout the school years. By the eighth grade, almost half the black students in Montgomery County are performing below their grade level. The study discussed the attitudes of parents, teachers, counselors, and students; economic factors and disrupted families; and programs for training and retraining teachers, but did not consider the possibility that the differences may be genetic or involve the speed or efficiency with which the brain can process information (Vernon, 1987).

### *Sexuality*

Lynn (1989) argues that methodological problems in the literature require a conclusion of "no differences" between the races in sexual behavior. This strains credulity. In our most recent work together, Anthony Bogaert and I have examined some of the consequences, including the dysfunctions accruing, to the racial group differences in sexuality. We concluded that relative to Caucasians, populations of Mongoloid ancestry are inclined to a greater frequency of inhibitory disorders such as low sexual excitement and premature ejaculation, with a concomitantly lower frequency of sexually transmitted diseases, including AIDS. Populations of African ancestry, on the other hand, are inclined to a greater frequency of uninhibited disorders such as rape and unintended pregnancy, with a concomitantly higher frequency of sexually transmitted diseases, including AIDS (Rushton & Bogaert, in press).

For example, Oriental populations are completely underrepresented in sex crimes, both as perpetrators and as victims (Wilson & Herrnstein, 1985). Arrest statistics show that blacks, however, are substantially overrepresented in such crimes. In the United States they account for about one-half of all those arrested for rape (Wilson & Herrnstein, 1985). Among adolescents the overrepresentation of blacks in crime is most marked for sexual offenses (Davis & Leitenberg, 1987). Across all criminal offenses in 1980 and 1981 committed by adolescents, 24% of the offenders were black; for sexual offenses, however, 35% were black; for forcible rape, 58% were black. Black sex offenders, compared to white sex offenders, are also more likely to use force in the commission of the offense and to engage in vaginal intercourse with their victims (Kirk, 1975). Often the victims of sexual assault are also black: A large study carried out in Philadelphia on the sexual abuse of children aged 2 to 12 showed that 82% were black and 18% were white (Peters, 1976).

The prevalence of sexually transmitted diseases (STDs) in different populations provides another way of estimating whether these populations differ in their sexual behaviors. The data are consistent in showing that for a range of STDs, Mongoloids < Caucasoids < Negroids. The World Health Organization Technical Reports and other studies examining the world wide prevalence of syphilis, gonorreah, herpes, chlamydia, etc., find very low levels in China and Japan, and very high levels in Africa, with European countries and populations intermediate (see Rushton & Bogaert, in press, for review). The racial pattern found worldwide in STDs is also found *within* the United States (USDHHS, 1986).

With respect to AIDS and HIV-1 seroprevalence, both intranational and international epidemiological studies reveal Mongoloids < Caucasoids < Negroids (Barnes, 1987; Curran, Jaffe, Hardy, Morgan, Selik, & Dondero, 1988; Piot, Plummer, Mhalu, Lamboray, Chin, & Mann, 1988). While the reporting of AIDS cases from Africa and the Caribbean has been delayed, partly due to the reluctance of many governments until 1987 to officially acknowledge their existence (Dickson, 1987; Norman, 1985), it has now become clear that relative to other populations, many of these predominantly Negroid countries have an enormous AIDS problem with up to 25% of the 20- to 40-year age group in some urban areas infected, including women of child-bearing age, and with up to 90% of the female prostitutes seropositive (Piot et al., 1988; Quinn, Mann, Curran, & Piot, 1986).

In Africa and the Caribbean countries the AIDS virus is transmitted predominantly through heterosexual intercourse. The age and sex distributions of HIV infection rates is similar to that of other STDs with higher prevalence among younger sexually active women (Quinn et al., 1986). At the other extreme, it is a characteristic feature of AIDS in Japan that most sufferers are hemophiliacs, accounting for 34 of Japan's 59 officially confirmed AIDS patients (Swainbanks, 1988). The prime infection route is strongly suspected to have been blood plasma imported from the United States for manufacture of blood coagulants. An intermediate amount of HIV infection is apparent in Europe and the Americas where to date it has occurred predominantly among homosexual men and intravenous drug abusers (Piot et al., 1988), although it may be breaking out into the heterosexual population (Masters, Johnson, & Kolodny, 1988).

A similar pattern of whites being intermediate to black and Oriental populations occurs *within* the United States. As of July 4, 1988, while blacks amount to less than 12% of the U.S. population they disproportionately account for 26% of adult and 53% of pediatric AIDS cases (Heyward & Curran, 1988). Whites amount to over 80% of the population and account for 59% of adult and 23% of child AIDS cases, with Hispanic populations being intermediate (Heyward & Curran, 1988). Oriental populations do not exist in the AIDS figures, including those from California and Hawaii where they constitute large percentages of the 16- to 40-year-old population. Confirmation of the U.S. racial pattern is found from the results of testing more than 1.2 million military applicants and blood donors. These studies show that black applicants have HIV-1 seroprevalence rates 3 to 10 times higher than white applicants (Curran et al., 1988).

## RACE AND REPRODUCTION

Lynn claims that I failed to adequately show racial group differences in rate of reproduction, arguing that the differences in egg production and fecundability are not sufficient and that other data are contradictory.

I urge the reader to reexamine Rushton (1989) and the data presented on menstrual cycle length, speed of pregnancy, and international fertility rates (Brazil, 1958, notwithstanding; see Demeny, 1984) to see whether they are insufficient. With respect to Lynn's other "contradictory" data, that in the U.S. blacks suffer higher infertility rates than whites (13 vs 8 per 1000), Lynn fails to note the reason: sexually transmitted diseases. As we have just reviewed in sobering detail, STDs including AIDS constitute an increasing problem for Negroid populations worldwide. Africa, for example, is known to be differentiated from other areas of the world in having STDs as the major cause of infertility (Cates, Farley, & Rowe, 1985).

It would be foolish to commit what some critics of sociobiology have called the "adaptationist fallacy" and naively assume that each and every component of an organism's life history directly contributes in some simple way to reproductive functioning. Racial differences in numerosity of sweat glands, for example (in which the ordering is also Mongoloid < Caucasoid < Negroid) are past adaptations to heat. Some *r*/*K* attributes may be consequences of the adaptive significance of other *r*/*K* attributes. As suggested in my 1985a paper, the functional questions of the selection pressures which brought about the constellation of associated attributes can be left, temporarily at least, in abeyance.

Nonetheless, as Bogaert and I discussed in relation to the AIDS pandemic which is directly affecting the reproductive success of millions of people, many of the *r*/*K* attributes conjoin to differently affect susceptibility (Rushton & Bogaert, in press). Most obvious will be that less *K* individuals will be drawn to have sexual intercourse with more numerous partners, even when they are married. Lowered levels of intelligence must also be considered a risk factor since observation of contingent danger will be less, in terms both of acquiring the disease and in transmitting it to others. Social organizational capacity also differentiates at-risk groups both within and without the United States. In addition, the tendency to follow societal rules is directly implicated, as are temperament factors such as cautiousness. In short, we have argued that *r*-strategy individuals are genetically inclined to those cofactors placing them at risk.

Many may find disagreeable our approach to race differences in the sensitive area of sexual behavior and AIDS, and particularly to our linking them to genetically based evolutionary processes. It may be useful to remember that even if genetic predispositions are operating, this does not deny the importance of environmental influences. Distal genetic effects are *necessarily* mediated by proximate neuroendocrine and psychosocial systems which have independent effects on phenotypic behavior and provide potential mechanisms for intervention. Knowledge of which groups are most at risk may facilitate the alleviation of suffering. Ignorance in this area will not be of benefit.

## ARE THE RACIAL DIFFERENCES HERITABLE?

### *Sexuality*

Lynn (1989) incorrectly claims that a genetic basis has not been shown for the race differences in sexuality. Both direct and indirect evidence indicate a genetic contribution. Direct evidence is available for the heritability of gamete production (egg production is a defining feature of the *r*/*K* continuum, see Fig. 1 in Rushton & Bogaert, 1987). The more eggs produced per unit of time, the more *r* the organism. The rate of double ovulations resulting in dizygotic twins per 1000 births is <4 in Mongoloids; about 8 in Caucasoids, and >16 in Negroids, with some African populations having rates as high as 57 per 1000. The incidence of nonmonozygotic triplets and quadruplets shows comparable rank orders (Bulmer, 1970; Nylander, 1981). Moreover, and perhaps as a result of matching evolutionary processes, measures of the size of the testes show the difference is twofold lower in Mongoloids than in Caucasoids (9 g vs 21 g), too large a difference to be accounted for in terms of body size (Diamond, 1986; Short, 1984). Although the data are less conclusive, larger scrotal circumferences have sometimes been found in Negroids than Caucasoids (Ajmani, Jain, & Saxena, 1985).

I have drawn attention to the heritability of the racial differences in gamete production three times now in this series of interchanges. For example, "This is because the tendency to double ovulate is inherited largely through the race of the mother, independently of the race of the father, as observed in Mongoloid–Caucasoid crosses in Hawaii and Caucasoid–Negroid crosses in Brazil (Bulmer, 1970)" (Rushton, 1989; see also Rushton & Bogaert, 1987, p. 534; 1988, p. 269). I have also shown that the production of dizygotic twins is related to many other *r*-characteristics, including lowered age of menarche, shorter menstrual cycles, higher numbers of marriages, higher rates of coitus, more illegitimate children, a closer spacing of births, a greater fecundity, more wasted pregnancies, a larger family, an earlier menopause, and an earlier mortality (Rushton, 1987a). In a review by Allen (1981), the greater fecundability of mothers of twins is shown not only by their larger families (e.g., 4.3 vs 2.6 in one study, discounting one member of the twin pair), but also by the speed with which they become pregnant (e.g., following the return of husbands from war). Additional, more indirect evidence for the heritability of racial differences in sexuality will be discussed in a later section.

### *Intelligence*

Lynn (1989) spends considerable effort trying to explain away the numerous data sets I presented for the heritability of race differences in IQ. When I point out that black children raised by white middle class families regress to their population mean in IQ scores, educational achievement, and social deviance, Lynn dismisses it as a "self-fulfilling prophecy," although there is scant (if any) evidence for such effects (Jensen, 1980). When I point out that Mongoloids and Negroids often show racially recognizable forms of behavior across diverse cultural and historical settings, Lynn rebuts by pointing to Mongoloid subpopulations such as North American Indians, although no doubt appropriate Caucasoid subpopulations could be selected to maintain the rank order. When I review the evidence from adoption studies showing small effects to "shared environment" on individual sibling scores, Lynn counters by presenting a study showing substantial effects to shared environment on *grouped* scores, a procedure adequately refuted by Plomin and Daniels (1987, p. 51) in the article from which Lynn drew his information. When I discuss how combining within-family and between-family analyses can rule out between-family sources of variance such as social class and thereby leave in genetic and within-family sources of environmental variance, Lynn dismisses it as "faulty logic." Lynn similarly dismisses regression to the mean effects and arbitrarily attributes them to the effects of the environment. When I draw attention to the fact that it is the more heritable items on IQ tests which are the ones which most differentiate the races, Lynn ignores it, and dismisses my finding that Japanese inbreeding depression scores predict the magnitude of the black–white difference on the same tests as a "coincidence." One begins to wonder what kind of evidence *could* make Lynn accept the plausibility of a genetic hypothesis.

Consider more fully the data on *g*, the general factor of intelligence that emerges when factor analysis is carried out on numerous measures of complex mental ability. When the scores of blacks and whites are compared on specific items and subtests, those items judged to be least culturally biased but loading most highly on *g* show the greatest differences (Eysenck, 1984; Jensen, 1980, 1985, 1987a). Thus Jensen (1985) examined 11 large-scale studies, each comprising anywhere from 6 to 13 diverse tests, and showed that a significant and substantial correlation was found in each between the test's *g* loading and the mean black–white difference on the same tests. Subsequently, Naglieri and Jensen (1987; see also Jensen, 1987a) matched 86 black and 86 white children in the 4th and 5th grades for age, school, sex, and socioeconomic status and tested them with the Wechsler Intelligence Scale for Children-Revised (WISC-R) and the Kaufman Assessment Battery for Children (K-ABC) for a total of 24 tests. They found that the standardized black–white differences on the various tests were positively correlated ($r = +0.78$, $p = +0.75$) with the test's *g* loadings.

Of importance to the argument for the heritability of the racial group differences is that *g* loadings correlate with estimates of genetic influence. Thus Jensen (1983) reported a correlation of +0.81 between the degree of inbreeding depression (defined as a lowered mean of the trait relative to the mean in a non-inbred population) from cousin marriages in Japan and the *g* loadings of the 11 subtests of the WISC. This is especially interesting because inbreeding depression indicates genetic dominance which arises when the trait confers evolutionary fitness (defined as increased survival of genes through successive generations). Subsequently, Jensen (1987b) reported rank order correlations of +0.55 and +0.62 between estimates of genetic influence from two twin studies and the *g* loadings of the Wechsler Adult Intelligence Scale (WAIS) subtests. Most recently Vernon (in press) has found a correlation of +0.60 between the heritabilities of a variety of speed of reaction time tasks and their relationship with the *g* loadings from a psychometric test of general intelligence.

### *Other Behavior Genetic Designs*

Lynn fails to consider that the distinction between "extrinsic" social class explanations and "intrinsic" genetic and within-family explanations are also obtainable from the study of twins. These dovetail with the adoption studies showing that the crucial environmental variables influencing postadolescent behavior are those that occur *within* families, not *between* them (Bouchard, 1984; Plomin & Daniels, 1987). This important discovery implies that since the environmental variables usually proposed to explain racial differences, such as social class, religious beliefs, cultural practices, father absence, and parenting styles account for so little variance *within* race, they are unlikely to *between* races.

The decrease in variance due to the common family environment that occurs during adolescence is found even for traits such as altruism and aggression which parents are expected to socialize heavily (Rushton, Fulker, Neale, Nias, & Eysenck, 1986). In this study 573 pairs of adult monozygotic (MZ) and dizygotic (DZ) twins were mailed questionnaires measuring their altruism, empathy, nurturance, aggressiveness, and assertiveness. Approximately 50% of the variance on each scale was found to be associated with genetic effects, virtually 0% with the twin's common environment, and the remaining 50% with each twin's specific environment and/or error associated with the test. Correcting for the unreliability in the tests raised the heritabilities to 60% and reduced the specific environment variance to 40%. A summary of the results is presented in Table 1.

These data not only signify a strong association of genetic factors with the characteristics in question but also indicate a negligible influence of the twin's shared environment. Rather the distinct experiences of the individual account for almost all of the environmental variance. Theories to explain this finding, which occurs after puberty, emphasize an increasingly active organism capable of shaping its own environment in a direction canalized by its underlying genotype (Lumsden & Wilson, 1981;

TABLE 1

ESTIMATES OF VARIANCE COMPONENTS AND ESTIMATES CORRECTED FOR UNRELIABILITY FROM A BIOMETRICAL ANALYSIS OF AGGRESSIVENESS, ASSERTIVENESS, ALTRUISM, EMPATHY, AND NURTURANCE QUESTIONNAIRES FROM 573 ADULT TWIN PAIRS[a]

| Trait | Additive genetic variance (%) | Common environmental variance | Specific environmental variance |
|---|---|---|---|
| Aggressiveness | 39 (54) | 0 (0) | 61 (46) |
| Assertiveness | 53 (69) | 0 (0) | 47 (31) |
| Altruism | 51 (60) | 2 (2) | 47 (38) |
| Empathy | 51 (65) | 0 (0) | 49 (35) |
| Nurturance | 43 (60) | 1 (1) | 56 (39) |

[a] Reproduced, by permission of the publisher, from Rushton et al. (1986a).

[b] Estimates in parentheses corrected for unreliability of questionnaire.

Rushton, Littlefield, & Lumsden, 1986; Scarr & McCartney, 1983). This, perhaps, is the reason why black children adopted by white middle-class families regress to their population mean in IQ, sexual behavior, and social deviance after they reach adolescence (Scarr, Weinberg, & Gargiulo, 1987).

Although the twin data and adoption data are compatible in showing very small effects due to shared environment, they have sometimes differed in the magnitudes estimated for heritabilities, with twin studies typically suggesting estimates of about 0.50 and adoption studies of about 0.20. Debate consequently ensued as to whether twin studies overestimated heritabilities (perhaps due to MZ twins being treated more similarly) or whether adoption studies underestimated them (perhaps due to subjects being tested at different ages). A unique recent study of MZ and DZ twins raised together *and* apart and measured on the same tests has circumvented both the problems mentioned above and confirmed the typical heritability of 0.50 across diverse traits, including those under consideration, such as aggression, behavioral restraint, and traditional morality (Tellegen, Lykken, Bouchard, Wilcox, Segal, & Rich, 1988).

These findings raise the possibility of another, more indirect approach to the heritability of race differences. This is to generalize from within-group heritabilities to between-group differences. While it is true that, as Lynn says, this is not generally thought to follow because heritability estimates are properties of particular populations (Falconer, 1981), recent evidence shows that estimates of genetic influence are, in fact, more generalizable across distinct ethnic and national groups than is often thought likely. For example, estimates of genetic influence on cognitive tasks calculated from Korean parent–offspring regressions (in Korea) significantly correlate with similar estimates calculated in European and Japanese Americans living in Hawaii, and, moreover, predict the degree of assortative mating thereon (Rushton, in press-b). Similarly, heritabilities calculated for personality and social attitudes in Australian twins predict heritabilities in British twins, and the degree of similarity in Canadian male friendships. And, as mentioned in my last response to Lynn, inbreeding depression scores calculated in Japan on the WISC subtests in the 1950s predict black–white difference scores on the WISC-R in the 1970s (Rushton, in press-a). These results cannot be attributed to the confounding effects of unreliability of measurement for the relationships remain even after controlling for reliability. Thus, the fact that libido and sexual attitudes have been found to be heritable within Caucasian groups (Eysenck, 1976; Snyder, 1987) implies that it is probably heritable within Negroid and Mongoloid groups, and helps to explain some of the differences between them.

More sophisticated designs are also possible. Thus a critical test of *r*/*K* theory is whether the covariance between traits is genetic or environmental in origin. Several lines of evidence suggest that when the studies are carried out the outcome will be favorable to *r*/*K* theory. In studies of dandelions, fish, flies, milkweed bugs, and field mice, many of the covariant *r*/*K* traits are genetic in origin (Rushton, 1985a) and in humans, studies have already reported genetic linkage for some variables (e.g., between anger, impulsivity, and delinquency, see Rowe, 1986), and implied them for others (e.g., between delinquency and sexual behavior, see Rowe, Rodgers, Meseck-Bushey, & St. John, in press). Unpublished analyses of the heritability of altruism study (see Table 1) also indicate latent genetic correlations between the components.

Ultimately more powerful techniques based on DNA sequencing will be available to test evolutionary theories of individual and group differences. With DNA sequencing now being an automated routine, the entire human genome will soon be charted. This will usher in the opportunity to study human genetics in its most complex polygenic forms, be they predispositions to behavioral traits, anatomical characteristics, or diseases. Genetic distance studies have already been discussed, suggesting a divergence time at least twice as long for the Negroid–non-Negroid split as for the Caucasoid–Mongoloid split (Stringer & Andrews, 1988).

## PHYSIOLOGICAL MODELS OF *r*/*K* COVARIANT TRAIT DEVELOPMENT

One advantage of a gene-based evolutionary perspective is the focus it brings to underlying physiology and one preliminary hypothesis would implicate testosterone and other sex hormones. As outlined by Rushton and Bogaert (1987) there is evidence to be found in support of such a system. In an early study, Freeman (1934) found group differences in the weight of the hypophysis (pituitary) with blacks having the heaviest (800 mg), whites being intermediate (700 mg), and Orientals the lightest (600 mg). The pituitary is directly involved with the release of gonadotropins which stimulate the testicles and ovaries in their functions (the release of testosterone, estradiol, and progesterone on the one hand, and sperm and eggs on the other). This would order the racial differences in rate of multiple birthing, for gonadotropin levels differentiate the populations in the predicted directions (Soma, Takayama, Kiyokawa, Akaeda, & Tokoro, 1975), as well as distinguishing mothers of dizygotic twins from mothers with no dizygotic twins (Martin, Olsen, Thiele, Beaini, Handelsman, & Bhatnager, 1984). Gonadal hormones are also involved in the development of sexuality, including secondary sexual characteristics such as muscularity and depth of voice (Haeberle, 1978; Hudson & Holbrook, 1982), the organization and structure of the brain (Gray, 1987; Kimura, 1987); temperament including aggression and altruism (Baucom, Besch, & Callahan, 1985; Olweus, 1986), and may also order the racial pattern that has been observed for premenstrual syndrome (Janiger, Riffenburgh, & Kersh, 1972), health (Rushton, 1987b), and male/female sex ratios where a geater frequency of male births occurs in Mongoloid than Caucasoid than Negroid populations (James, 1987).

A more central locus for research attention is brain functioning. It is of interest, for example, to wonder where the one cubic inch or more of grey matter that differentiates the races is located. Gray (1987) has described the cytoarchitecture and functioning of the behavioral inhibition system postulated to underlie such relevant components of temperament as cautiousness and sociability, which also differentiate the races (Rushton, 1985b). Brain imaging techniques may be used to ascertain such neurological differences. One agenda now is to go beyond the racial differences in genetically based covarying traits and to identify the underlying physiological mechanisms. Explaining racial differences may thus become a crucible for understanding individual differences, for the former constitute an aggregate of the latter.

## CONCLUSIONS

The juxtaposition of evolutionary biology with racial differences in behavior is today almost as taboo a subject as sexuality was for the Victorians. Moral and political sensitivities abound and many humanitarians argue that such treatments should be eschewed given the current state of knowledge. Focusing on population differences is considered by many to constitute an affront to the universal search for brotherhood, democracy, and equality. Intellectual curiosity, however, does not readily bow to moralistic guidelines no matter how laudable the motive or self-imposed the restraints. The fact is that we simply do not know what the evolutionary origins of the races are, nor the extent of their similarities and differences, and this ignorance is unlikely to be dispelled by the chilling pronouncements too often made about those who conduct such research.

From an evolutionary point of view it is to be expected that populations will differ, genetically, in the mechanisms underlying their behavior. Adopting such an outlook does not disconfirm the democratic ideal. As E. O. Wilson (1978) put it: "We are not compelled to believe in biological uniformity in order to affirm human freedom and dignity" (p. 52). He went on to quote Bressler (1968) that "An ideology that tacitly appeals to biological equality as a condition for human emancipation corrupts the idea of freedom. Moreover, it encourages decent men to tremble at the prospect of 'inconvenient' findings that may emerge in future scientific research." The dramatic discoveries of genetic and fossil evidence on human origins (Stringer & Andrews, 1988) and the remarkable popular interest therein (*Newsweek*, January, 1988) as well as the increasing willingness of front line journals to consider the social implications of group differences (Gottfredson, 1986; Steen, 1987) are, it is hoped, heralding a more enlightened research climate for the study of human variation. This would be of benefit, for ignorance is not to be preferred to knowledge.

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