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Human assortative mating: more questions concerning genetic similarity theory

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In a previous short communication on the subject of human assortative mating, we concluded that 'an open verdict should be recorded concerning the claims and counterclaims surrounding genetic similarity theory, pending more precise theorizing and more analytic investigations' (Russell & Wells 1994, page 464). Rushton (1995) has disputed the arguments that led to this conclusion. We question his rebuttal of the three points that we originally made, and present some further questions that genetic similarity theory needs to address satisfactorily.

Our first point was that if genes produce variation in, for example, height, and in preference for a mate with a particular height, then an individual of a particular height will be chosen by a mate with the relevant preference, and their offspring will tend to inherit both the attribute and the preference for it. Thus, we stated, 'people will inherit the tendency to find attractive in others the attributes they themselves possess' (page 463). Rushton does not accept this point, saying that our 'analysis then stops . . . and is thereby incomplete for it misses the next vital step. Unless the chance configuration is adaptive, it will break up in later generations' (page 547).

As a step towards resolving this issue, we ran a computer simulation. For the sake of simplicity, we assumed that mate choice was confined to preference for a single attribute. The program presumed a population of 200 'people', 100 of each sex. Each person had 20 pairs of chromosomes. The first 10 pairs each carried a 'gene for height', which received the value 0 or 1 with a probability of 0·5 at the outset. The second 10 pairs each carried a 'gene for preferred height', again taking the value 0 or 1 with a probability of 0·5. We determined the height of each individual simply by summing across the first 10 chromosome pairs. Thus, height varied according to the binomial distribution, taking a value between 0 and 20. Preferred height was determined in the same way, except that it was computed from the genetic material in the last 10 chromosome pairs.

The rule for mate choice worked as follows. Each female sequentially chose the male whose height most closely resembled her preferred height, from among the pool of those eligible. Once a male had been chosen, he was swapped to the location of the female, to remove him from consideration by females who had yet to make their choice. When a female found more than one male tying for first place in her affections, she chose the last of those encountered. Thus, the first female had 100 males to choose from, and the last had no choice at all. At this point in the program, the assortative mating coefficient for height was calculated. Then, each couple produced one male and one female offspring. Each offspring's first chromosome (in each pair) was selected from the father. Whether the gene on a chromosome came from the first or second of each pair of the father's chromosomes was separately determined at random in each instance. The gene on the second chromosome of each pair was selected in the same way from the mother. Thus, each offspring received a genetic makeup that came equally from the father and the mother, and equiprobably from each of the four grandparents. Although males were able to inherit height preferences, these preferences were not expressed, but could be partly passed on to sons or daughters. After 'reproduction', the offspring became the parental generation, and the cycle started again.

We ran this simulation for 100 generations, and replicated the whole process 50 times. Assortative mating coefficients were averaged across the 50 replications (Fig. 1). On generation zero, the assortative mating coefficient is just under zero. In 10 generations it climbed to around 0·5 and gradually reached a plateau at around 0·7.

There is little evidence for Rushton's assertion that, in the absence of adaptive mechanisms, 'the chance configuration . . . will break up in later

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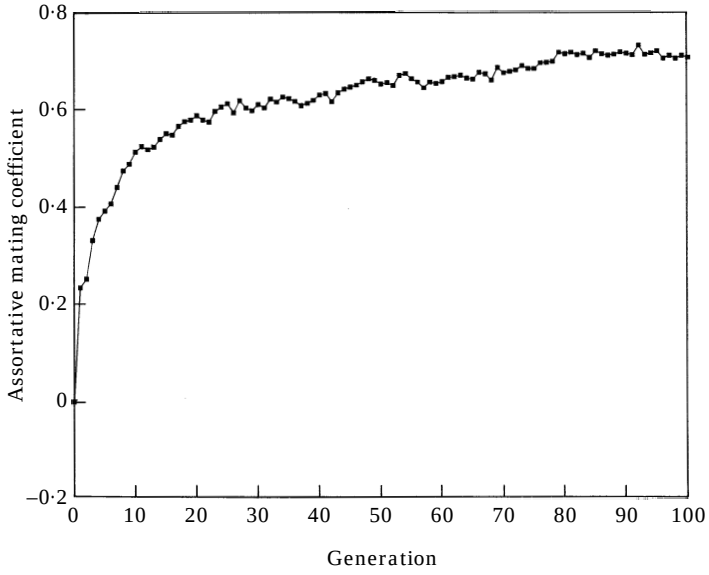

Figure 1. Averaged results of 50 replications of mate-choice simulation.

generations' (page 547). Although this simulation is an oversimplified model of human behaviour, it nevertheless suggests the viability of an alternative biological theory for assortative mating on a range of partly inherited attributes for which there are partly inherited varying preferences.

The second point made by Russell & Wells (1994) was that assortative mating for blood groups would lead one to expect a departure from Hardy–Weinberg equilibrium. We analysed data on ABO blood group frequencies in five samples, and found no evidence of departure from Hardy–Weinberg equilibrium. Rushton (1995) described two of his previous studies, which he considered superior to ours because they were not entirely limited to the ABO blood groups. He reported above-chance similarity between mates (Rushton 1988) and between male friends (Rushton 1989a) on a number of blood group measures. He concluded that 'results from an insensitive hypothesis test cannot cancel those from a more powerful one' (page 548).

A number of aspects to this criticism warrant a reply. First, failure to find departure from Hardy–Weinberg equilibrium is not confined to the ABO blood groups. For example, one can use data from Burns (1983, page 275) on the MN blood group, which were based on a sample of 6129 white Americans from three cities. The chi-squared value for departure from Hardy–Weinberg equilibrium was not significant ($\chi^2 = 0{\cdot}0227$, $df = 1$).

Second, it is frequently held to be unsound to support a null hypothesis, because samples are typically small and, therefore, unlikely to disprove a false null hypothesis. One of the samples used in Russell & Wells (1994), however, exceeded half a million people. Failure to reject the null hypothesis in this situation strongly suggests that the null hypothesis is true.

We made no claim for any special merit of this or our previous analyses. However, if there is assortative mating on genotype as genetic similarity theory suggests, one would expect to find demonstrable departures from Hardy–Weinberg equilibrium. Failure to find such departures requires explanation.

It is of interest to note a feature of the results that Rushton (1988) reported, namely, where he 'found that fecundity was predicted by genetic similarity in 1000 cases of disputed paternity among sexually interacting couples' (Rushton 1995, page 548). The couples (986 in number) came from the files of a company providing legal testimony in cases of disputed relationships, using blood analyses. In the 799 instances where the male could not be excluded from paternity, a mean similarity of 52·02 was found, using the metric outlined by Rushton (1989b). In the

remaining 187 instances, where the male was excluded from paternity, a mean similarity of 44·42 was found.

The point we wish to make is that it is easier to disprove paternity if the putative father is genetically different from the mother. If the adults are alike, the child will tend to have the same genes as the male even if he is not the father. Thus, genetic similarity may not predict fecundity; rather, dissimilarity assists disproof of paternity. This point was made by Daly (1989) and Ridley (1989), but appears not to have been noted by Rushton.

We cannot tell what proportion of the 799 cases involved genuine fathers. All we can presume is that all 986 couples interacted sexually. Genetic similarity theory predicts that they should be of above average similarity on genetic markers. Combining both groups (those in which the male could and could not be excluded from paternity), the weighted mean similarity score is 50·58, which falls within the 95% confidence interval for the randomly paired control group in Rushton's (1989a) study of male friends.

Rushton's third criticism of Russell & Wells (1994) is that we 'mistakenly implied that environmentality is a dichotomous alternative to heritability' (page 547). We disagree. Nevertheless, we think it of interest that there is assortative mating on an attribute, position in the family of origin, that does not signal genotype. At least it shows, as we said, that people 'cannot discount environmental influences to detect the underlying genotype' (1994, page 464). One is led to wonder what novel finding, consonant with but additional to existing knowledge, would lead Rushton to regard genetic similarity theory as disproved.

In his concluding remarks, Rushton (1995) purports to find it surprising that we limited our analysis to assortative mating and 'ignored all the within-family data' (page 548) together with 'many data on ethnic differences and ethnic nepotism' (page 549). It was not our intention to present a review of the entire range of genetic similarity theory, but to raise some questions concerning its application to assortative mating. While considering the first of these two points, however, we are tempted to question the accuracy of Rushton's statement about 'children who, because of assortative mating, resembled one side of the family more than the other' (pp. 548–549).

On the subject of ethnic nepotism, the applicability of genetic similarity theory has been questioned elsewhere (Russell 1987). Although Rushton predicts a tendency to befriend and marry within rather than across ethnic groups, we suspect that there may be less inter-racial friendship and marriage than would be predicted if people were trying to find another person on the basis of genetic similarity. Figures for all marriages in Britain in 1981 are provided by Coleman (1985, his Table V). Of 396 West Indian men and 51 African women who married in that year, not one of those men married one of those women. Of the 53 481 white men and 197 Pakistani women who married, only four of those men married four of those women. Rushton would presumably regard these figures as consonant with his views. How, then, would he explain that, according to this same data set, significantly more marriages occurred between African men and white women, or between Pakistani men and African women, than are predicted by the assumption of random mating? We argue that the subjects of inter-racial marriage and, more generally, ethnic nepotism, are complex and poorly understood (e.g. see discussion in Hamilton 1987).

Finally, Rushton concludes that 'although alternatives to genetic similarity theory may be proposed for subsets of the data, none has been proposed to explain the whole array. This explanatory power may suggest a simplicity indicative of truth' (page 549). To us it suggests instead an unwillingness to acknowledge the complexity of the phenomena under consideration. Let us ignore side issues and turn back to the subject of assortative mating to provide additional examples of questions that genetic similarity theory should address.

When theories are derived in evolutionary biology, the account may start with 'Imagine a gene with a particular effect'. That this represents an oversimplification is well understood. One aspect of the oversimplification is that pairs of genes should be considered. Thus, let us imagine a passive allele, a, and a mutation, A, which acts to spread through the population by favouring individuals that contain it, even if in the body of another individual. Before it becomes frequent, it is likely to be paired with a. Most individuals will be aa, but some Aa, leading eventually to a few AAs. For an Aa to maximize the chance of A spreading, it should exhibit most favouritism to any AA individual that it encounters, rather than acting most favourably to an Aa individual. Thus,

contrary to what genetic similarity theory would predict, its optimal behaviour is not to favour individuals exhibiting most genetic similarity, but to favour individuals more the more A genes they possess.

An interesting example of something of this sort happening in reverse was described by Lenington (1983). Wild house mice may have genotype + +, or carry a lethal t-allele, genotype +t. The tt individuals die. Females tend to avoid mating with +t males, especially females who are +t themselves. Furthermore, +t males are more likely to attack +t oestrous females than + + oestrous females. Thus, the +t mice seem to prefer mates who are genetically dissimilar to themselves, whereas + + mice prefer other + + mice, but without the fervour of +t mice.

When one considers human assortative mating, too, there are findings that cause embarrassment to genetic similarity theory. Take, for example, a recent paper by Lykken & Tellegen (1993) reporting a series of four studies. They used the Minnesota Twin Registry, which contains information on a large number of psychological variables, and includes not just MZ and DZ twins, but, in a large number of cases, the spouses of one or both twins. Of the results of their second study, Lykken & Tellegen say that 'Pairs of individuals who were selected as spouses of MZ twin pairs show no more similar scores than do DZ spouse–spouse pairs and hardly more than do random pairs of same-sex adults' (page 62). Needless to say, genetic similarity theory predicts that spouses of MZ twins should be roughly twice as similar to each other than spouses of DZ twins.

Of the results of their third study, they said that 'twins consider their cotwin's choices of wardrobe and of household furnishings to be similar to their own, and this is more true for MZ than for DZ twins. They also feel positively disposed toward their cotwin's choice of vacations and of jobs and, once again, this similarity in choice behavior is greater for MZ than for DZ twins. On the crucial question of mate selection, however, a very different picture emerges. About as many twins of both sexes and both twin types disliked as liked their cotwin's choice of fiancée or fiancé . . . The MZ twins did not approve of their cotwin's choice significantly more than did the DZ twins' (page 63). Genetic similarity theory predicts that twins should tend to like their cotwin's choice of partner, especially when the twins are MZ rather than DZ.

Although we do not necessarily accept all of the conclusions reached by Lykken & Tellegen (1993), we believe that they have presented interesting new data and arguments. It is almost inevitable that scientists will encounter new observations that require modification of their views. Rushton's critique of Russell & Wells (1994) presents no new observations or signs of any refinement of his views.

In conclusion, we stand by the position represented in our first sentence, and are content for readers to decide for themselves whether it is fair comment.

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