COMMENTARIES

Genetic similarity theory and human assortative mating: a reply to Russell & Wells

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The role of genetic similarity theory (Rushton 1989a) in understanding human relationships has been questioned by Russell & Wells (1994) who contend that (1) alternative theories are equally able to explain assortative mating, (2) spousal similarity on blood groups is not yet established and (3) people select spouses on environmentally based traits. These three points, they argue, weaken the theory sufficiently 'that an open verdict should be recorded' (page 464). In reply, I hold that Russell & Wells have (1) ignored both the role of adaptation in evolutionary theorizing and the distinction between proximal and distal causation, (2) erroneously assumed that weak support for a null hypothesis cancels strong support for the alternate hypothesis, and (3) mistakenly implied that environmentality is a dichotomous alternative to heritability.

In an earlier paper (Rushton et al. 1984), Russell, Wells and I incorporated the kin selection theory of altruism under a more general hypothesis so as to apply it to humans. In people, altruism is often directed at non-kin such as spouses, friends, neighbours and fellow-ethnics. We hypothesized that a gene could ensure its own survival by acting to bring about the reproduction of any organism in which copies of itself were found. To pursue this strategy a gene must, in effect, detect copies of itself in other organisms. Our 1984 formulation built on my earlier work (1980, page 31), and by others (Dawkins 1976; Thiessen & Gregg 1980).

Much of the evidence for genetic similarity theory comes from studies showing that both best friends and spouses assort most positively on the more heritable components of various attributes (reviewed by Rushton 1989a). Examples of differing heritabilities used to establish the theory include: for physical characters, 80% for mid-finger length versus 50% for upper-arm circumference; for intelligence, 80% for the general factor versus less than 50% for specific abilities; for personality, 41% for having a preference for reading versus 20% for having many different hobbies; and for attitudes, 51% for agreement with the death penalty versus 25% for agreement with bible truth. The heritability/similarity relationship is only expected to apply within sets of homogeneous traits; for example, among physical attributes, cognitive abilities or social attitudes. There is no claim that people assort more on height than, say, on social attitudes just because height is more heritable. This qualification is necessary because some traits are more important for mate choice than are others, so trait importance must be held constant.

This brings me to Russell & Wells' first point. They do not dispute the predictive utility of differential heritabilities and indeed, they cite a study of their own (1991), corroborating it in the context of marital satisfaction. Their claim now is that there are many alternative explanations for the data other than genetic similarity theory. Russell & Wells provide a proximal example of such an alternative following Gangestad (1989). They reason as follows. If person A mates with person B because of a gene-based preference for tall people, then offspring will tend to inherit genes for (1) tallness and (2) a preference for tallness in mates. Thus, Russell & Wells conclude, people will inherit the tendency to find attractive in others the attributes they themselves possess. But their analysis then stops, as did Gangestad's (1989) before them, and is thereby incomplete for it misses the next vital step. Unless the chance
configuration is adaptive, it will break up in later generations. Furthermore, although Russell & Wells cite Chiarelli & Rabino Massa (1985) to show that people inherit a tendency to be attracted to particular physical features in others, they are silent about why such a tendency evolved in the first place. The virtue of genetic similarity theory is its specification of altruism as the distal, evolutionary process enabling adaptive behaviour to be maintained across generations. Increased altruism to genetically similar others has the effect of replicating genes more effectively and thus can explain Chiarelli & Rabino Massa’s finding.

Assortative mating is found in taxa ranging from insects to birds to primates, and it can be observed in the laboratory as well as in nature. To have evolved independently in such a wide variety of species, assortative mating must confer substantial advantage. Advantages thought to accrue in human mates include (1) increased marital stability, (2) increased relatedness to offspring, (3) increased within-family altruism and (4) greater fecundity. Evidence for these putative advantages is summarized in Rushton (1989a). For example, fetal loss has been shown to increase with the distance between the birthplace of parents and with each additional country of birth among great-grandparents (Bresler 1970).

The second point made by Russell & Wells (1994) is that in five large samples they failed to find evidence for any departure from Hardy-Weinberg equilibrium such as would be expected if people tended to marry others of similar genotype. Their analysis, however, was limited to the ABO blood group, a relatively insensitive metric. In contrast, I carried out two studies using seven polymorphic marker systems (ABO, Rh esus (R h), MNSs, K el, Duffy (F y), Kidd (J k) and HLA) at 10 loci across six chromosomes to confirm predictions about social assortment. In the first, I found that fecundity was predicted by genetic similarity in 1000 cases of disputed paternity among sexually interacting couples of North European appearance (judged by photographs; Rushton 1988). Couples who produced a child together were 52% similar on this metric, whereas those who did not were only 44% similar (P <0.05). In the second study, I used the same genetic markers to examine similarity among male friendship pairs (Rushton 1989b). The best friends were 54% similar to each other on this metric. An equal number of randomly chosen pairs from the same overall sample was significantly less similar (48%). Stratification effects were unlikely because within-pair differences in age, education and occupation did not correlate with the blood similarity scores.

In both of these studies (Rushton 1988, 1989b), blood analyses were done blind with regard to knowledge of the relationship. Moreover, I had carefully chosen blood tests sensitive enough to identify paternity in 95% of cases and to distinguish reliably between fraternal twins reared in the same family. Russell & Wells weight their results as equal to mine, ignoring the fact that results from an insensitive hypothesis test cannot cancel those from a more powerful one.

The third point made by Russell & Wells concerns the role of environmental factors. Clearly, environmental factors such as propinquity, to name but one, play a role in determining social assortment. This issue has never been in question. Thus, Russell & Wells’ observation that spouses also assort on the basis of ordinal position in the family is irrelevant to whether assortment also results from genetically based preferences. Social scientists often err in thinking that if an environmental factor has influence, then it follows that genetic factors have no influence. Although Russell & Wells do not quite fall for this fallacy of ‘nothing but-ism’, they do make the curious claim that if assortative mating occurs on a putatively non-heritable characteristic, then this ‘must be regarded as weakening the [genetic] theory’ (1994, page 464).

Much data have now accumulated showing that genes bias the development of complex social behaviour in every examined human phenotype from personality and psychopathology to political attitudes and choice of social partners (Plomin et al. 1994). As Turkheimer & Gottesman (1991) proposed, it is time to enshrine $H^2\neq 0$ as the ‘first law of behaviour genetics’ and to argue that $H^2=0$ is no longer an interesting null hypothesis. But no behavioural geneticist believes that genes are 100% responsible for complex social behaviour. The battle is between those who believe in a 100% environmental determinism and those who think that both genes and environments affect behaviour. From an evolutionary point of view, individual differences are the alternative genetic combinations and adaptations that compete through the mechanism of natural selection. It seems unlikely that human mating will turn out to disconfirm Turkheimer & Gottesman’s ‘first law’.
It is also surprising that Russell & Wells limited their analysis of the value of genetic similarity theory to assortative mating. They ignored all the within-family data showing, for example, the fine distinctions that relatives make even while grieving for dead children who, because of assortative mating, resembled one side of the family more than the other (Littlefield & Rushton 1986). They also ignored many data on ethnic differences and ethnic nepotism that require theoretical analysis (Rushton 1995). 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. New, imaginative research has also been forthcoming. For example, Tesser (1993) has confirmed a genetic basis for the attitude similarity/attraction relationship between individuals who had only just become acquainted. Now that DNA analysis allows the mapping of human populations (Cavalli-Sforza et al. 1994), there should be accelerated research to examine preferences, not only in families and small groups, but also in large ones, both national and international.

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REFERENCES