Dominance Alone Is Not Enough

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It has often been noted that the correlation between dizygotic (DZ) twins is smaller than predicted from the monozygotic (MZ) correlation under a simple additive genetic model. Possible genetic explanations of this finding are considered. It is shown that duplicate gene interactions between pairs of moderately frequent alleles at polygenic loci are sufficient to produce surprisingly small (approximately 0.12) genetic correlations between siblings.

KEY WORDS: dominance; epistasis; emergenesis; extroversion; twins; genetic correlation.

INTRODUCTION

Given random mating, the classical model of additive gene action is expected to yield a genetic correlation between dizygotic (DZ) twins or siblings which is exactly half that of monozygotic (MZ) twins. It has been a cause of some puzzlement among investigators that the correlation between DZ twins often appears to be much less than expected. How is the discrepancy to be explained? One school (e.g., Eaves and Eysenck, 1975) has adopted a conservative position which has claimed that, although the DZ correlation looks too small, the discrepancy is not larger than might be expected by chance alone and so should not be used as a foundation for theories of unwarranted complexity. Although it is true that many studies are still too small to justify any other position, there is now replication of the small DZ correlation for extraversion (perhaps sociability rather than impulsivity), in samples which are so large as to render the
discrepancy from classical additivity far greater than might be expected by chance alone (see Eaves et al., 1987).

Thus, in this case at least, the failure of the usual additive genetic model has to be taken seriously. Two kinds of explanation can be offered. The first is genetic, in terms of nonadditive genetic effects. The second seeks an explanation in social terms, for example, competitive social interactions between twins based on their genetic differences. The purpose of this paper is to examine the value of purely genetic explanations of the phenomenon.

Among genetic explanations, there have been two major contenders. The relatively conservative position, from a scientific point of view, exemplified by Eaves et al., has parameterized the extra difference between MZ and DZ correlations in terms of genetic dominance. Such a model fits the data for extroversion by purely statistical criteria but usually leads to a (nonsignificant) negative estimate of the component due to additive gene action. Such estimates can arise quite often by chance because of the high correlation between estimates of additive and estimates of dominance effects derived from twin data alone. However, the large numerical values of the dominance parameters obtained in such analyses are consistent only with a heterozygous advantage or very extreme gene frequencies and dominance effects. Even when the allele frequencies are so extreme as to all but eliminate the contribution of homozygous effects to variation, the limiting value of the sibling genetic correlation is one-fourth of the MZ correlation. This value is greater than that sometimes observed in practice.

An alternative genetic explanation for the low DZ correlation is advanced by Lykken (e.g., 1982), who invokes very high-order interactions ("emergenesis") between large numbers of relatively infrequent alleles at different loci.

The problem with the "dominance" and "emergenesis" models is that they are not consistent with what is known about the mechanisms of gene action inferred from experimental studies in other organisms (see, e.g., Mather and Jinks, 1982). Careful experimental analysis in species such as *Nicotiana rustica* suggests that virtually all cases of apparent "overdominance" at polygenic loci can be resolved in terms of epistatic effects. There is little evidence in experimental organisms that heterozygous effects exceed the additive genetic deviations. On the other hand, although higher-order interactions (e.g., trigenic interactions) can be detected in careful breeding studies, their effects are far outweighed by interactions between pairs of loci ("digenic interactions"). Thus, we would tend to reject any model which requires extreme amounts of dominance, on the one hand, or large contributions of high-order interactions,