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SHORT COMMUNICATION

Genotype × Age Interaction for Neuroticism

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Twin data suggest that genetic variability in neuroticism increases with age.

KEY WORDS: neuroticism; genetic variability; age; twins.

INTRODUCTION

Eaves and Eysenck (1976) have reported a genetic analysis of adult neuroticism scores and showed that variation in the trait itself and in the inconsistency of measurement accords with a simple model assuming random mating, additive genetic variation, and within-family environmental effects. Variation between subjects' responses on two occasions separated by an interval of 2 years, however, showed no genetic component and reflected only environmental experiences unique to individuals.

Twin data of this kind do not readily allow us to separate the effects of genes which contribute to the mean expression of the trait in an individual from those which control an individual's responsiveness to the environment and thus contribute to genotype–environment interaction (*GE*).

A simple model for the components of variance of twins reared together demonstrates that analysis of variance alone is inadequate for the detection of *GE* from such data. The expectations of variance components between families (σ_B^2) and within families (σ_W^2) for monozygotic (MZ) and

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dizygotic (DZ) twins reared together are

$$\sigma^2_{BMZ} = G_1 + G_2 + E_2 + G_1E_2 + G_2E_2$$
$$\sigma^2_{WMZ} = E_1 + G_1E_1 + G_2E_1$$
$$\sigma^2_{BDZ} = G_2 + E_2 + G_2E_2$$
$$\sigma^2_{WDZ} = G_1 + E_1 + G_1E_1 + G_2E_1 + G_1E_2$$

The notation is that of Jinks and Fulker (1970). The GE terms do not denote multiples but serve to represent the additive statistical components of interaction between the four main effects whose variances are G_1 (the within-family genetic component), G_2 (the between-family genetic component), and E_1 and E_2 (environmental components within and between families). We have assumed no interaction between environmental components and no covariation of genotypic and environmental effects.

Estimable combinations of the parameters are $(E_1 + G_1E_1 + G_2E_1)$, $(G_1 + G_1E_2)$, and $(G_2 + E_2 + G_2E_2)$. Thus we find that every genetic or environmental component is confounded with some corresponding source of genotype–environment interaction if we have only data on twins reared together. This means that such data would lead to no detectable departure from expectations on the basis of a simple genetic model even if all the trait variation were due to GE. However unrealistic this possibility may seem in the light of our knowledge of GE, we must recognize the formal intractability of the problem provided that we restrict ourselves to the analysis of variance or intraclass correlations.

There are three possible solutions to the problem.

1. We may obtain data on individuals reared apart and on unrelated individuals reared together. Now we might analyze the components of interaction between G and E_2, although G_1E_1 and G_2E_1 cannot be separated from E_1 (Jinks and Fulker, 1970).
2. We may capitalize on the empirical finding of quantitative genetics (e.g., Mather and Jinks, 1971) that the effects of loci contributing to GE often covary with those contributing to G. This is the basis of Jinks and Fulker's test of GE, which examines the linear or nonlinear covariation between the mean scores of MZ twin pairs and the corresponding within-pair standard deviations. They indicate that their test is most safely employed with separated twins unless the between-family environmental deviations do not interact with environmental effects within families.
3. In the absence of genotype–environment covariation, we may attempt to detect heterogeneity between the variances of arrays of genotypes in different environmental circumstances. Such

heterogeneity is indicative either of GE or of genotype–environment covariation (Scarr-Salapatek, 1971).

The analysis reported here involves exploitation of both the second and third possibilities.

DATA

Age is a variable which, formally at least, may be regarded as environmental and which contributes to the E_2 component for twins reared together. Provided that we can assume no differential mortality for the trait in question, we can preclude genotype–environment covariation and can thus regard any heterogeneity of genetic variances at different ages as indicating an interaction of genetic differences with age. Since we may expect any heterogeneity to show some systematic relationship to age, we examine the covariation with age of genetic variability in our trait.

Our data are the neuroticism scores of adult volunteer twins derived from responses to an 80-item Personality Inventory. Some characteristics of the sample are given elsewhere (Eaves and Eysenck, 1975). We tabulate (Table I) correlations between pair means, absolute intrapair differences, and age for the N scores of MZ and DZ twins. The twins have mostly lived together prior to adulthood.

The negative correlations between pair sums and age confirm the decline in N scores with age (Eysenck and Eysenck, 1968) and justify regarding age as a significant common environmental component for the twins. The absence of any correlation between age and MZ intrapair differences suggests that there is no interaction of environmental components within families with age. The absence of any (linear) covariation between genetic effects and effects contributing to $(G_1E_1 + G_2E_1)$ is confirmed by the nonsignificant sum–difference correlation for MZ twins. The only significant correlation involving intrapair differences is that between age and

Table I. Correlations Between Age, Pair Means, and Absolute Intrapair Differences for Neuroticism Scores of MZ and DZ Twins[a]

	MZ (df = 402)		DZ (df = 212)	
	Means	Differences	Means	Differences
Age	−0.25[b]	−0.02	−0.19[b]	0.19[b]
Means	1.00	0.04	1.00	−0.04

[a] Data for males and females have been pooled.
[b] Significant at the 0.01 level.

differences for DZ twins, which implies that genetic differences in *N* become more pronounced with advancing age. Thus, although our earlier study revealed no genetic component of short-term variability for these twins, the present analysis suggests that long-term changes in neurotic behavior are under genetic control.

DISCUSSION

There are two possible interpretations of our finding. One suggests that additional genes become operative later in life and contribute to greater variability. Alternatively, the same genes may operate all the time but developmentally significant environmental experiences are not randomly distributed over genotypes so that neurotics tend to seek or create less therapeutic environments than normals, resulting in a slower decline in N scores for neurotics than normals. Only a long-term developmental study of the same subjects can discriminate between these alternatives.

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