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# Has the “Equal Environments” Assumption Been Tested in Twin Studies?

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A recurring criticism of the twin method for quantifying genetic and environmental components of human differences is the necessity of the so-called “equal environments assumption” (EEA) (i.e., that monozygotic and dizygotic twins experience equally correlated environments). It has been proposed to test the EEA by stratifying twin correlations by indices of the amount of shared environment. However, relevant environments may also be influenced by genetic differences. We present a model for the role of genetic factors in niche selection by twins that may account for variation in indices of the shared twin environment (e.g., contact between members of twin pairs). Simulations reveal that stratification of twin correlations by amount of contact can yield spurious evidence of large shared environmental effects in some strata and even give false indications of genotype  $\times$  environment interaction. The stratification approach to testing the equal environments assumption may be misleading and the results of such tests may actually be consistent with a simpler theory of the role of genetic factors in niche selection.

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## Background

For several decades, twin studies have provided a critical facet of psychiatric and behavior-genetic research. In the simplest paradigm, estimates of concordance or correlation of monozygotic (MZ) and dizygotic (DZ) twins are manipulated to provide estimates of the contributions of additive genetic effects, shared or common family environmental effects, and unique, individual-specific within-family environmental effects (Eaves et al., 1989; Jinks & Fulker, 1970; Neale & Cardon, 1992). Further nuances of twin resemblance are taken as indicative of other, more complex sources of variation such as non-additive genetic effects, twin interaction, or genotype  $\times$  environment interaction (see e.g., Eaves, 1982). Interpretation of quantitative estimates of these variance components depends critically on the so-called “equal environments assumption” (EEA) (i.e., that monozygotic [MZ] and dizygotic [DZ] twins experience equally correlated environments). The EEA is viewed as a major weakness of the twin method for quantifying genetic and environmental components of human differences (e.g., Martin et al., 1997). It is well known that MZ twins are more similar than DZ twins for a wide range of indices of environmental treatment (e.g., Loehlin & Nichols, 1976) although such environments often fail to correlate with major behavioral outcomes (Kendler, 1993). It has been proposed to test the EEA by stratifying twin correlations by indices of the amount of shared environ-

ment (Kendler & Gardner, 1998). However, it is also often claimed that exposure to relevant environments may be influenced by genetic differences (Lykken et al., 1990; Lytton, 1977). We present a model for the role of genetic factors in niche selection in twins that may account for variation in indices of the shared twin environment (e.g., contact between members of twin pairs). Simulations reveal that stratification of twin correlations by amount of contact yields patterns of twin resemblance that might be mistaken for large shared environmental effects in strata with greater contact and even give misleading indications of genotype  $\times$  environment interaction. Claims to have falsified the equal environments assumption, therefore, need to be regarded with caution. Indeed, the results of such tests may actually be consistent with a simpler theory of the role of genetic factors in niche selection.

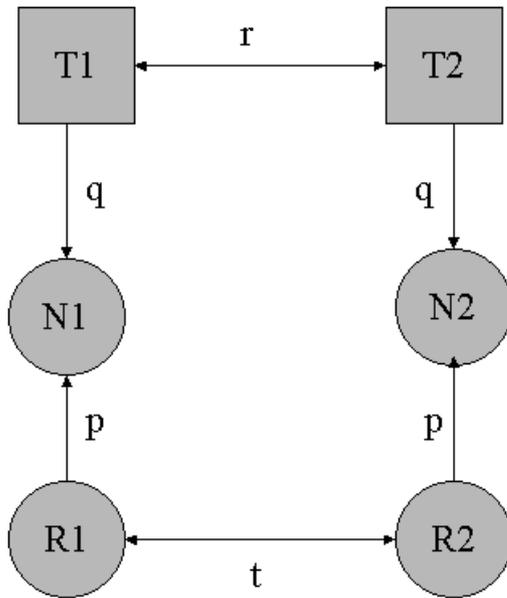
## Methods

Figure 1 summarizes our basic model. Trait,  $T$ , is measured in pairs of twins.  $T_1$  and  $T_2$  are the measures of the first and second members of a pair and the correlation between twins is  $r$ .  $T$  might be a measure of disruptive behavior or cognitive ability. The twin correlation will depend on the contributions of genetic effects and the shared environment to twin resemblance. In the absence of non-additive genetic effects and effects of the shared environment, the MZ correlation is expected to be twice that for DZ twins (Eaves, 1982).

We assume further that  $T$  contributes to a latent trait (“niche score”) that affects the niche that an individual twin selects in the human ecosystem. Examples of relevant niches may be peer groups, educational activities, etc. Let  $N_1$  and  $N_2$  denote the values of the trait in first and second twins. The standardized path coefficient,  $q$ , reflects the impact of the measured trait. Additional residual effects,  $R_1$  and  $R_2$ , also influence  $N$ . The path from residual to niche score is  $p$ . These residual effects may be correlated if other genetic and/or shared environmental factors influence the choice of niche. The correlation between the residual components adds an amount  $tp^2$  to the correlation between  $N_1$  and  $N_2$ .

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**Figure 1**  
 Path model for outcome trait T and niche, N, in twin pairs. R represents residual effects due to shared environment or genes that correlate between twins. Unique environmental influences on N and T are not shown.

In practice,  $N_1$  and  $N_2$  may not be measured directly on individual twins. Instead, we may gather data on a variable that reflects the similarities or differences between the niches of twins. Examples could be measures of the amount of contact between twins, or ratings of the extent to which they share similar peers or social activities. Let the difference in niches be  $D = |N_1 - N_2|$ . We scale the relative amount of contact between a pair so that:

$$0 < C = e^{-D} < 1 \tag{1}$$

The model in Figure 1 yields the following correlation matrix between variables  $N_1, N_2, T_1$  and  $T_2$ :

$$S = \begin{matrix} & N_1 & N_2 & T_1 & T_2 \\ N_1 & 1 & s & q & rq \\ N_2 & s & 1 & rq & q \\ T_1 & q & rq & 1 & r \\ T_2 & rq & q & r & 1 \end{matrix}$$

Where  $s = rq^2 + tp^2$ .

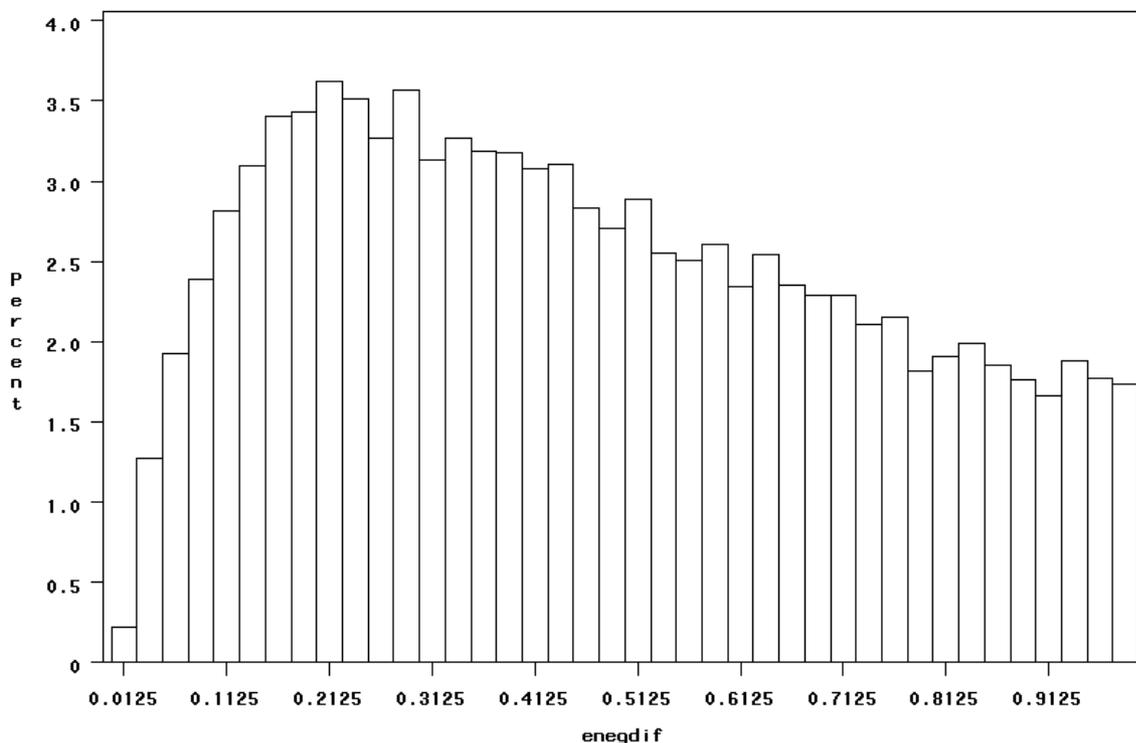
Numerical values may be substituted for the parameters in **S** to reflect different hypotheses about the magnitude and kind of genetic and environmental influences on T and N. For a given **S** we simulated 20,000 multivariate normal vectors and generated values for the scaled amount of contact, C, between each pair by substitution in (1). The values of C were sorted in ascending order into 10 bins of equal width, each corresponding to 10% of the total range in contact. Thus the first bin contained pairs with the least contact (0–0.1) and the last contained pairs with the greatest contact (0.9–1.0). Correlations were computed for pairs within each bin. Simulations and statistical analysis were conducted in PC SAS (SAS Institute, 1999).

Four simulations were conducted, corresponding to two sets of MZ and DZ twins. In both sets we set  $r$ , the MZ and DZ correlations in T, to 0.6 and 0.3 respectively. In each case we set  $q = 0.7071$ . The two sets differed in the residual correlations between  $N_1$  and  $N_2$ . In the first set we fixed MZ and DZ values of  $s$  to 0.3 and 0.15 respectively. These values imply that there is no residual genetic or environmental correlation in niche scores. In the second set we set the values of  $s$  to 0.5 and 0.25 in MZ and DZ pairs respectively, corresponding to modest additional residual genetic correlations in N.

**Table 1**  
 MZ and DZ Correlations for Simulated “Conduct Problems” as a Function of Amount of Contact Between Twins (1 = Least Contact, 10 = Most Contact) Assuming Conduct Problems Influence Niche Selection

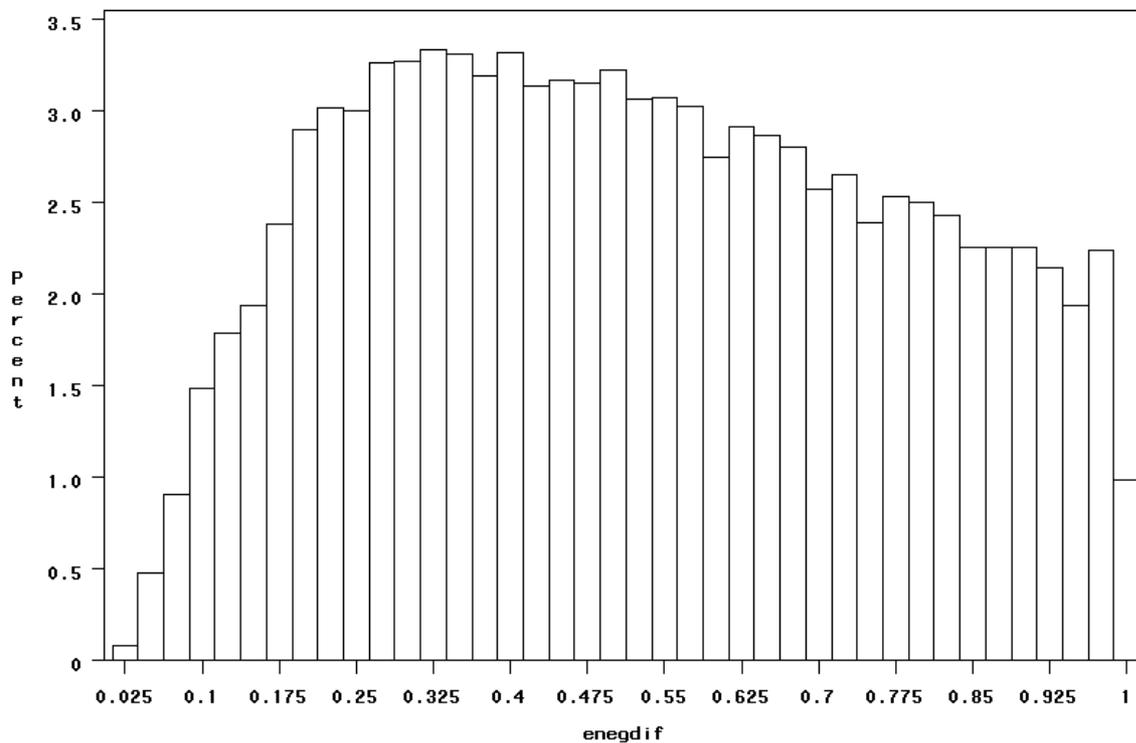
Niche correlation (s)	0.3 $\rho = 0, t = 1$		0.15 $\rho = 0, t = 0.5$		0.5 $\rho^2 = 0.2, t = 1$		0.25 $\rho^2 = 0.2, t = 0.5$	
	MZ		DZ		MZ		DZ	
Contact 1	0.308	-0.127	0.135	-0.200				
2	0.469	0.117	0.320	0.056				
3	0.588	0.307	0.503	0.264				
4	0.629	0.372	0.592	0.364				
5	0.648	0.414	0.635	0.431				
6	0.673	0.482	0.706	0.496				
7	0.695	0.495	0.718	0.519				
8	0.695	0.507	0.730	0.544				
9	0.706	0.516	0.734	0.548				
10	0.685	0.553	0.730	0.566				

Note: The correlations are based on a total of 20,000 simulated MZ and DZ Twins Pairs. It is assumed that the twin correlations for “Conduct Problems” are 0.6 and 0.3 for MZ and DZ twins respectively. The first pair of twin correlations assume that twin correlations between niches are based only on conduct. The second set allows for residual genetic effects on contact.



**Figure 2a**

Distribution of "contact" in 20000 simulated twin pairs for  $s = 0.25$  twin correlation in continuously distributed "niche scores". Amount of contact ( $C = \text{enegdif}$ ) is scaled as  $\exp(-D)$ , where  $D$  is absolute difference in niche score. Increasing amounts of contact are associated with increasing values of the abscissa in the figure.



**Figure 2b**

Distribution of "contact" in 20000 twin pairs for  $s = 0.5$  twin correlation in continuously distributed "niche scores".

## Results

Figures 2a and 2b summarize the distributions of contact values for two illustrative values (0.5 and 0.25) of the correlation between niches,  $s$ . It is no surprise that the median amount of contact increases with the twin correlation in niche values. This finding corresponds to the observation that MZ twins typically have greater contact with one another than DZ twins.

Table 1 gives the correlations between twins grouped according to amount of contact. The results show two main trends in both simulated examples. Firstly, when the amount of contact is low the DZ correlations are typically less than half the MZ twin correlations. It might be inferred, mistakenly, that non-additive genetic factors were contributing to the measured trait,  $T$ , in twins exposed to different environments. In contrast, the correlations for DZ twins having larger amounts of contact are typically more than half those for MZ twins with comparable environmental similarity. It is tempting to conclude that increased contact between twins creates the opportunity for greater environmental sharing. The fact that MZ twins have greater contact than DZ twins might then be assumed to vitiate the “equal environments” assumption. In fact, this is not the case. The data were simulated under a model that assumed no shared environmental effects in any stratum. Heterogeneity of differences between MZ and DZ correlations stratified by an environmental covariate is sometimes regarded as evidence for genotype  $\times$  environment interaction (e.g., Eaves, 1982; Silberg et al., 2001). It might be tempting to conclude that the lower correlations and increased apparent genetic non-additivity in twins with less contact indicates that genetic differences are interacting with environmental differences within or between twin pairs. Again, this is not the case in the simulated data so care must be taken not to misinterpret the pattern of twin correlations stratified by contact.

## Conclusions

Our simulations imply that complex patterns of twin resemblance stratified by niche similarity may only be the consequence of family resemblance for simple additive genetic differences in the causes of niche selection without any real violation of the equal environments assumption, as it is commonly understood. Thus, although the *absence* of any association between twin similarity and contact may still justify the claim that contact is not a factor in twin resemblance, the counter-claim that differences in similarity with contact falsify the EEA is clearly unfounded. A significant association in cross-sectional or retrospective studies between measures of contact and pattern of twin similarity may indicate simply that genetic factors influence how or with whom twins choose to spend their time. Our simulations challenge the uniqueness of any purely environmental interpretation of such data. It remains to be seen

how easy it will be to derive numerical estimates of the parameters of the model for niche selection with real data. Recent success with Markov Chain Monte-Carlo methods give some hope that the problem of parameter estimation may be tractable (Eaves & Erkanli, 2003; Eaves et al., 2003). At the very least, our theoretical findings qualify claims that the current approach through stratification by indices of putative environmental similarity sheds any unambiguous light on the impact of inequalities in the environments of MZ and DZ twins.

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## References

- Eaves, L. J. (1982). The utility of twins. In V. E. Anderson, W. A. Hauser, J. K. Penry, & C. F. Sing (Eds.), *Genetic basis of the epilepsies* (pp. 249–276). New York: Raven Press.
- Eaves, L. J., & Erkanli, A. (2003). Markov–Chain Monte Carlo approaches to the analysis of human development. *Behavior Genetics*, 33(3), 279–299.
- Eaves, L. J., Silberg, J. L., & Erkanli, A. (2003). Resolving multiple epigenetic pathways to depression. *Journal of Child Psychology and Psychiatry*, 44, 1–9.
- Jinks, L. J., & Fulker, D. W. (1970). A comparison of the classical, MAVA and biometrical-genetical approaches to the analysis of human behavior. *Psychological Bulletin*, 73(5), 311–314.
- Kendler, K. S. (1993). Twin studies of psychiatric illness: Current status and future directions. *Archives of General Psychiatry*, 50, 905–915.
- Kendler, K. S., & Gardner, C. O. (1998). Twin studies of adult psychiatric and substance dependence disorders: Are they biased by differences in the environmental experiences of monozygotic and dizygotic twins in childhood and adolescence. *Psychological Medicine*, 28, 625–633.
- Loehlin, J. C., & Nichols, R. C. (1976). *Heredity, environment and personality: A study of 850 sets of twin pairs*. Austin, TX: University of Texas Press.
- Lykken, D. T., McGue, M., Bouchard, T. J., & Tellegen, A. (1990). Does contact lead to similarity or similarity to contact? *Behavior Genetics*, 20, 547–561.
- Lytton, H. (1977). Do parents create, or respond to, differences in twins? *Developmental Psychology*, 13, 456–459.
- Martin, N., Boomsma, D., & Machin, G. (1997). A twin-pronged attack on complex traits. *Nature Genetics*, 17, 387–392.
- SAS Institute (1999). *The statistical analysis system. PC SAS: Version 8*. Cary, NC: SAS Institute Inc.
- Silberg, J. L., Neale, M. C., Rutter, M., & Eaves, L. J. (2001). Genetic moderation of environmental risk for depression and anxiety in adolescent girls. *British Journal of Psychiatry*, 179, 116–121.