Psychological Medicine, 2003, **33**, 817–825. © 2003 Cambridge University Press
DOI: 10.1017/S0033291703007840 Printed in the United Kingdom

Does the level of family dysfunction moderate the impact of genetic factors on the personality trait of neuroticism?

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ABSTRACT

Background. While the family environment can directly influence later risk for psychopathology, dysfunction in the family of origin may also moderate the impact of genetic factors on liability for psychiatric disorders. Can a similar pattern be seen for the personality trait of Neuroticism (N) – which is a risk factor for many psychiatric conditions?

Method. Our sample of 957 complete female–female twin pairs from a population-based register had measures of self-reported N and multiple reporters (twin, co-twin, mother, father) for family dysfunction (FD). Statistical analysis was conducted by traditional regression analysis and a moderator structural equation twin model operationalized in the computer program Mx.

Results. Dividing the sample into quartiles based on increasing levels of FD, the mean of N increased substantially while correlations of N in monozygotic (MZ) and dizygotic (DZ) twins were relatively constant. Regression analyses did not suggest greater twin resemblance for N with increasing levels of FD. The best-fit structural equation model was the standard un-moderated model in which the proportion of variance in N due to genetic (39%) and unique environmental effects (61%) remained constant across values of FD.

Conclusions. Although a false-negative result due to limited power cannot be excluded, these analyses do not support the hypothesis that FD moderates the impact of genetic factors on levels of N.

INTRODUCTION

The quality of the home environment in which a child is raised has long been thought to be a crucial determinant of that child's later psychological functioning (Bowlby, 1980; Maccoby, 1992; Perris *et al.* 1994; Parker & Gladstone, 1996). A larger number of studies have documented a direct effect of various measures of family dysfunction on risk for later psychopathology (Burbach & Borduin, 1986; Holmes & Robins, 1988; Gerlsma *et al.* 1990; Parker, 1990; Perris *et al.* 1994; Moffitt *et al.* 2002). In this report, we examine a different mechanism by which the home environment may impact on later indices of mental health – by moderating the effects of genetic influences.

Such a mechanism – technically termed a 'shared environment × gene interaction' – has been proposed for a range of psychiatric disorders. In particular, adoption studies of conduct disorder (Cadoret *et al.* 1995), antisocial personality (Cadoret *et al.* 1983), schizophrenia (Tienari, 1991) and some subtypes of alcoholism (Cloninger *et al.* 1981; Sigvardsson *et al.* 1996) suggest that genetic effects on risk are increased in those exposed to a pathogenic rearing environment. Recent studies have shown family environment × gene interactions for disinhibition (Boomsma *et al.* 1999) and verbal intelligence

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(Rowe *et al*. 1999), with higher heritabilities observed in less restrictive and more educated families, respectively.

In this report, we examine whether the level of family dysfunction (FD) influences the heritability of the personality trait of neuroticism (N). Originally proposed by Eysenck & Eysenck (1964), N was designed to measure an individual's level of 'emotionality' or emotional instability and vulnerability to stress and has been identified as a major personality dimension by nearly all subsequent investigators (John, 1990).

For those seeking to understand how genetic and environmental risk factors inter-relate in the aetiology of psychiatric illness, N is of interest for at least five reasons. First, elevated levels of N are associated with an increased risk for a range of psychiatric syndromes, including major depression (McGuire *et al*. 1963; Kendell & DiScipio, 1968; Hirschfeld & Klerman, 1979; Wetzel *et al*. 1980), anxiety disorders (Marks, 1987; Clark *et al*. 1994), alcoholism (Prescott *et al*. 1997) and drug abuse (Kendler *et al*. 1999; Degenhardt *et al*. 2001). Furthermore, prospective studies show that high levels of N predict first-onset of major depression (Nystrom & Lindegard, 1975; Angst & Clayton, 1986; Hirschfeld *et al*. 1989; Boyce *et al*. 1991; Kendler *et al*. 1993). Secondly, N is approximately normally distributed in the general population (Eysenck & Eysenck, 1964) and so represents a quantitative index of risk. Thirdly, in adulthood, N is relatively stable over time (McCrae & Costa, Jr., 1990; Kendler *et al*. 1993), thereby reflecting trait vulnerability. Fourthly, in a wide range of twin and twin-family studies, genetic factors have consistently been shown to influence N, with most estimates of heritability ranging from 35 to 50% (Szmukler *et al*. 1986; Eaves *et al*. 1989; Loehlin, 1992; Loehlin *et al*. 1998; Lake *et al*. 2000). Fifthly, genetic risk factors for N and for major depression are closely related (Kendler *et al*. 1993).

In a sample of 957 complete female–female twin pairs ascertained from a population-based registry, we examine whether dysfunction in the family of origin, as reported by both twins and their parents, moderates the impact of genetic risk factors of N. Our *a priori* hypothesis was that FD would moderate the impact of genes on N, so that the heritability of N (that is, the proportion of variance in N that was due to genetic factors) would increase with increasing levels of FD.

METHOD

Sample

The twins in this study were sampled from the population-based Virginia Twin Registry (Kendler & Prescott, 1999), which now constitutes part of the Mid-Atlantic Twin Registry. These female–female twin pairs, from birth years 1934–1974, became eligible if both members previously responded to a mailed questionnaire, the response rate to which was ~64%. They have been approached for four subsequent waves of personal interviews from 1988 to 1997, with cooperation rates ranging from 85 to 92%. In 1990–1991, all cooperative parents (90% of those available) were personally interviewed. Zygosity was determined by a combination of standard questions (Eaves *et al*. 1989), photographs and DNA analysis (Spence *et al*. 1988; Kendler & Prescott, 1999). For these analyses, relevant data were available on 561 complete monozygotic (MZ), 396 complete dizygotic (DZ) pairs and 69 unpaired twins (35 from MZ and 34 from DZ pairs).

Measures

N was measured using the 12-item scale from the short EPQ (Eysenck *et al*. 1985) in the original questionnaire completed in 1987–1988. A total summed score was computed only if at least six of the items were completed, although 99% of the sample completed $\geqslant$10 items. The N scores were then standardized.

Family dysfunction (FD) was measured using 14 items chosen from the Family Environment Scale (Moos & Moos, 1986), which reflected the general emotional tone of the home when 'the twins were growing up'. Data were collected from the twins and their parents in 1990–1991 and rated on a four point-scale (often to never). Two sample items are: family members really helped and supported on another; family members would get so angry sometimes that they would throw things or hit each other.

A categorical variable factor analyses on these 14 items, carried out in the program Mplus (Muthen & Muthen, 1998), produced eigen values for the first four factors of 5·56, 1·73, 0·91

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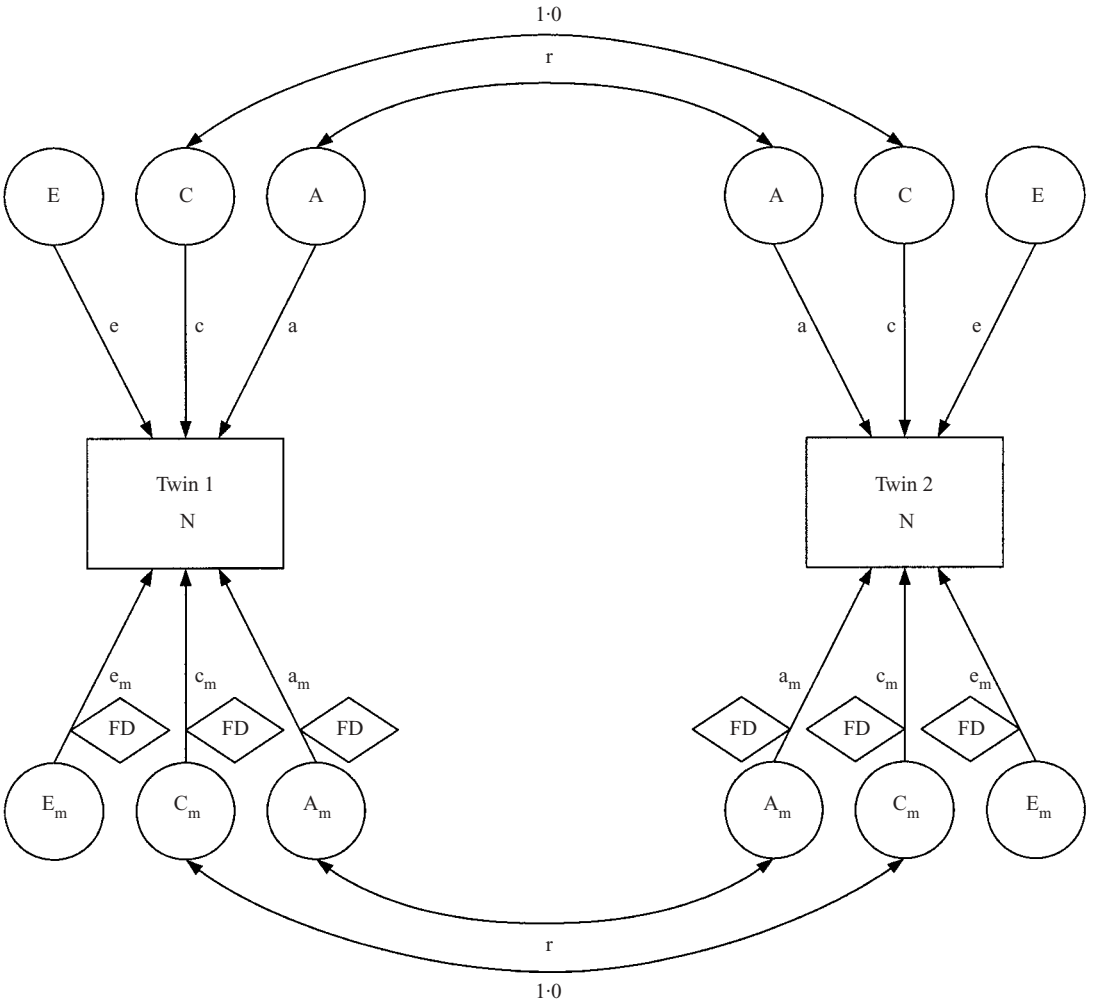

FIG. 1. A structural equation twin model for Neuroticism (N) with Family Dysfunction (FD) as a moderator variable. This model contains standard effects of additive genes (A), shared family environment (C) and unique-environment (E) on N as well as moderated genes (A_m), moderated shared family environment (C_m) and moderated unique-environmental effects (E_m). The observed dependence variable is depicted in rectangles, latent variables in circles and the moderator variable in a diamond. When a path is moderated, it means that the strength of that path depends on the value of the moderator variable; r equals 1·0 for MZ and 0·5 for DZ twins. Because FD scores are the same for each member of a twin pair, this model has no ability to determine the relationship between A and A_m, C and C_m and E and E_m. The model also contains direct and moderated effects on the mean of N but these are not pictured for the sake of clarity.

and 0·83, respectively. The first unrotated principle factor had loadings in excess of +0·40 for 12 of the 14 items. In the interest of parsimony, therefore, our analyses treated these 14 items as a single dimension, reverse coding certain items so that increasing scores reflected higher levels of FD. Scores obtained from the mother, father, and twins were separately standardized. The number of reporters for the measures of FD were as follows: four (twin, co-twin, mother, father), 50%; three (most typically, twin, co-twin and mother), 32%; two (usually twin and co-twin), 13%; and one (self), 5%. The inter-informant correlations for FD scores ranged from +0·35 to +0·58 with a mean (S.D.) of +0·41 (0·08). The final FD score was the within family average of *Z* scores from all available reporters re-scaled to range between 0 and 1. This variable – which was always the same for both members of a twin pair – served as the

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definition (moderator) variable for the interaction model.

Model-fitting

Our major goal was to determine whether the heritability of N was influenced by the level of reported FD. Since both N and FD were continuously distributed, we fitted a moderation model as presented in Fig. 1. As in typical twin modelling, we divided the sources of individual differences into those due to additive genetic effects (A or a^2), shared or 'common' environment (C or c^2) and individual-specific or 'unique' environment (E or e^2). This model allows for the direct regression of FD onto N, the 'basal' or unmoderated effect of A, C and E on N and an effect of A, C and E on N that is moderated by the level of FD. (When, as in these analyses, the environmental effects are perfectly correlated within twin pairs, the model in unable to determine the degree of correlation between the genetic or shared environmental factors that impact on the basal *versus* moderated levels of N.)

We are particularly interested in three models. We present the models here from simplest to most complex (although this is not the order in which they will be tested). The simplest is the standard model, in which the moderated pathways to N are set to zero. The model estimates a single value for the proportion of variance in N that is due to a^2, c^2 and e^2 and this value is independent of the level of FD. This model also predicts that the variance of N is independent of the level of FD. The scalar model predicts that the variance of N changes as a function of FD. However, across the range of FD, the scalar model predicts that the proportion of variance in FD that is due to a^2, c^2 and e^2 is invariant. Since genetic variance changes proportionally with total variance in this model, heritability remains constant. The key assumption of the moderator model is that the proportion of variance that is due to a^2, c^2 and e^2 changes as a function of the level of FD. The moderator model used in these analyses also predicts that the total variance in N changes as a function of FD.

The full model, in these analyses, is the moderator model and the scalar and standard models are nested submodels. Mx fits these models to the raw data by maximum likelihood. We compare the fits of these models by both the χ^2 difference test (where $\Delta\chi^2 = -2(\ln L_i - \ln L_j)$ where L_i and L_j are the likelihoods of models i and j) and Akaike's Information Criterion (AIC) (Akaike, 1987) where lower values indicate a more favourable balance of parsimony and explanatory power (Williams & Holahan, 1994).

RESULTS

Preliminary analyses

Using general estimating equations to correct for the correlational structure of the twin pairs, and controlling for zygosity and age at interview, the level of FD strongly predicted the level of N ($b = +0{\cdot}17$, $z = 6{\cdot}55$, $P < 0{\cdot}0001$). Because of this, we allowed FD to directly influence mean levels of N in all of our twin models.

The central prediction of our *a priori* hypothesis, that genetic risk factors and FD positively interact in the aetiology of N, is that the similarity of neuroticism scores in twin pairs should increase as the level of FD increases. Furthermore, we would predict that this increase should be more pronounced in MZ than in DZ twin pairs. Prior to formal model-fitting, we explored the support for this prediction in our data in three different ways. First, we divided our sample into quartiles based on FD scores and examined, in each quartile, the mean and variance of N and the product-moment correlation for N in MZ and DZ pairs (Fig. 2). Both the mean and variance of N increase monotonically with increasing levels of FD, although the mean increases much more markedly than does the variance. The pattern of twin correlations does not resemble that predicted by our hypothesis. The correlation in MZ twins is basically invariant across levels of FD. The pattern in DZ twins is less stable but no general monotonic trend is seen.

Secondly, in individual twins, we predicted, in a standard regression, N in twin1 from N in twin2, FD in twin2 and the interaction between them. The interaction between FD and twin2 N was not significant in MZ ($t = 1{\cdot}52$, df = 557, $P = 0{\cdot}13$) or DZ pairs ($t = 0{\cdot}86$, df = 392, $P = 0{\cdot}39$). Thirdly, using twin pairs, we predicted, from FD scores, the absolute value of the difference in N scores between the twins. These effects were also non-significant in MZ ($t = -0{\cdot}11$, df = 559, $P = 0{\cdot}91$) and DZ pairs ($t = -0{\cdot}42$, df = 394, $P = 0{\cdot}67$).

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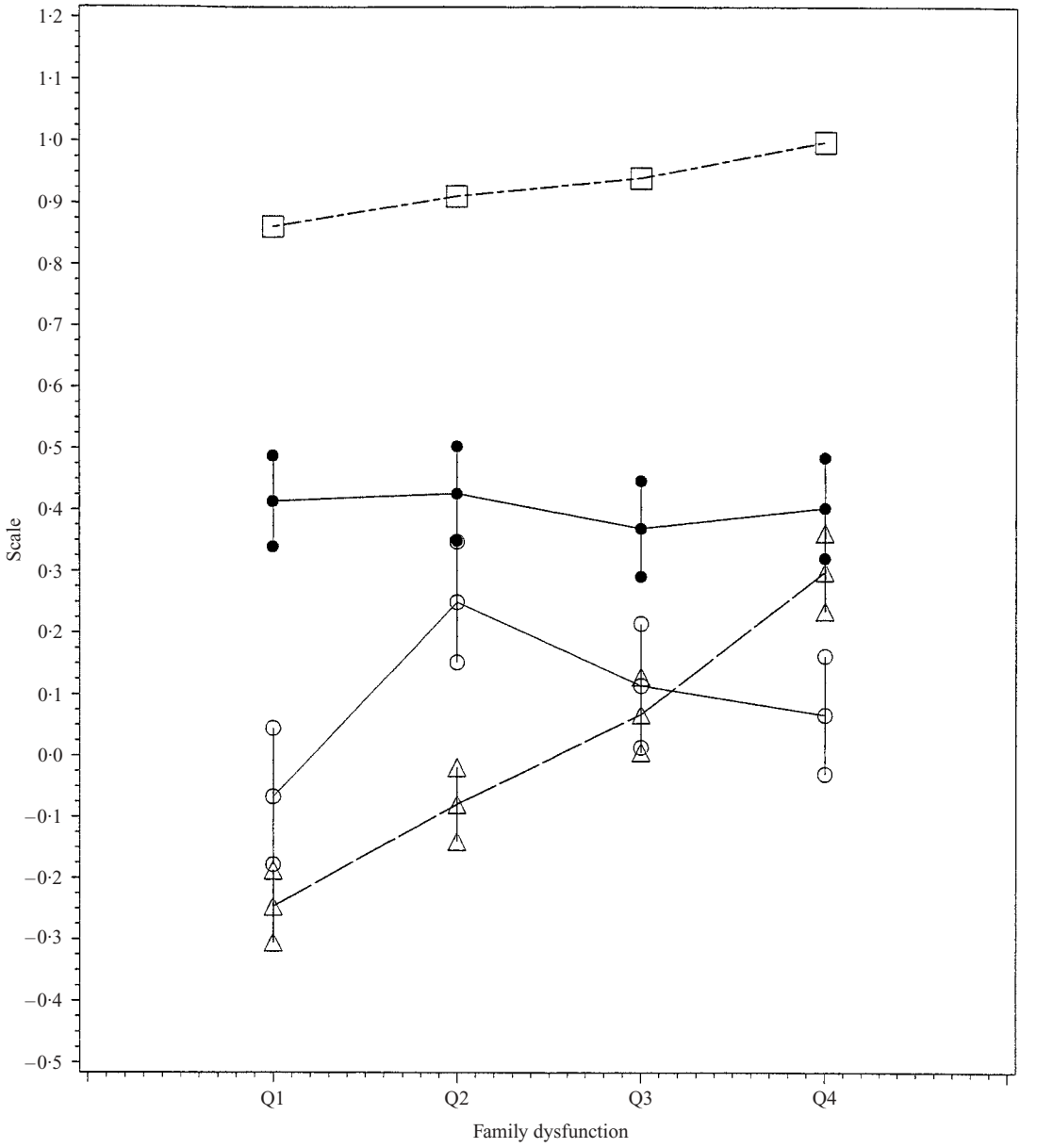

FIG. 2. Changes (±S.E.) in the mean (△), variance (□) and MZ (●) and DZ (○) twin correlations for standardized Neuroticism scores across quartiles of the level of family dysfunction.

Model-fitting

The results of model-fitting are presented in Table 1 with parameters estimated for two conditions: (*i*) unmoderated (i.e. when the moderator variable equals its minimal value of zero and FD is absent); and (*ii*) with the moderator variable is at its maximum value of unity (and levels of FD are very high).

Our full or moderator model produced a fit of $-2\ln L = 5377{\cdot}4$ with 1,945 degrees of freedom. We set the AIC for this model at zero as a comparison for the subsequent submodels. As can be seen in the table, the moderator model

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Table 1. *Parameter estimates for the sources of variation in neuroticism as a function of the level of family dysfunction*

	Model: parameter estimates (95% CI)			
	Parameter	Moderator	Scalar	Standard
Unmoderated (when moderator at minimum)	a^2	0·30 (0·13–0·49)	0·39 (0·29–0·46)	0·39 (0·30–0·46)
	c^2	0·00 (0·00–0·06)	0·00 (0·00–0·05)	0·00 (0·00–0·05)
	e^2	0·70 (0·51–0·87)	0·61 (0·55–0·68)	0·61 (0·54–0·68)
	Variance	0·84 (0·68–1·00)	0·86 (0·71–1·02)	0·97 (0·91–1·04)
When moderator at maximum	a^2	0·51 (0·24–0·63)	0·39 (0·29–0·46)	0·39 (0·30–0·46)
	c^2	0·00 (0·00–0·16)	0·00 (0·00–0·07)	0·00 (0·00–0·06)
	e^2	0·49 (0·37–0·74)	0·61 (0·55–0·68)	0·61 (0·54–0·68)
	Variance	1·22 (0·94–1·56)	1·18 (0·93–1·50)	0·97 (0·91–1·04)
$\Delta\chi^2$		—	0·8	2·7
Δdf		—	2	3
AIC		—	−3·2	−3·3

a^2, Additive genetic effects; c^2, shared environmental effects; e^2, unique environmental effects; df, degrees of freedom; and AIC, Akaike's information criteria.

estimates the heritability of N to be lower when FD levels are at a minimum ($a^2 = 0{\cdot}30$) than when they are at a maximum ($a^2 = 0{\cdot}51$). Also, as expected, the variance of N is predicted to increase with increasing levels of FD.

By constraining the proportion of variance due to a^2, c^2 and e^2 to be constant across values of FD, we produced the scalar model. The change in χ^2 for this model compared to the moderator model is very slight ($\Delta\chi^2 = 0{\cdot}8$, $\Delta df = 2$) and produced an improvement in the AIC because of its greater simplicity ($-3{\cdot}2$). As predicted, in this model as the level of FD increases, the heritability of N is constant but the variance of N increases.

By constraining the variance of N to be constant across values of FD, we then produce the standard model which fits slightly better than the scalar model (AIC = $-3{\cdot}3$). As expected, the heritability estimates for N were identical for the scalar and standard models: 0·39.

DISCUSSION

We sought to determine whether FD moderates the impact of genetic risk factors on N. In accord with several previous studies of psychopathology, we predicted that genetic effects on N would become more important as levels of dysfunction in the family of origin increased. More specifically, we predicted that the moderator model would provide a better fit to the data than the alternative models. However, our predictions were not supported. The best explanations of the data were provided by the scalar and standard models which in turn produced very similar AIC values. These two models agreed in most important features. Both predicted an absence of shared environmental effects and the same heritability for N across varying levels of FD. They only disagreed in that the moderator model predicted a modest increase in variance of N with increasing levels of FD.

Our inability to detect evidence for a moderation of genetic effects on N by levels of FD can be interpreted in two different ways. One possibility is that it represents a type II statistical error – an acceptance of the null hypothesis when it is false. Two points can be made in favour of this position. First, parameter estimates from the moderator model did suggest a positive interaction with the heritability of N increasing in the predicted direction. Secondly, the detection of interactions is statistically difficult and so many attempts to do so have low power (Wahlsten, 1990). The two most comparable recent twin studies that report significant interactions between genes and indices of family environment had larger sample sizes: (Boomsma *et al*. 1999), 1967 twin pairs; (Rowe *et al*. 1999), 1909 sibling pairs.

The second plausible interpretation of our results is that there is no meaningful interaction between FD and genetic factors in the aetiology of N. Five points can be made in favour of this position. First, unlike previous studies, we used multiple measures for our modifier variable (FD) thereby potentially reducing measurement

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error and increasing power. Secondly, unlike one of the two most comparable previous twin studies, which used a dichotomous (Boomsma *et al*. 1999) modifier variable, our modifier variable (FD) was continuous thereby increasing statistical power. Thirdly, in contrast to the two most similar prior studies (Boomsma *et al*. 1999; Rowe *et al*. 1999) an examination of the raw twin correlations as a function of the modifier variable revealed no systematic trend for changes in heritability. Fourthly, in accord with our modelling results, two different regression analyses also provided no evidence that an FD × gene interaction influenced levels of N. Fifthly, the statistical evidence in favour of an interaction was extremely modest, the model deteriorating only $0{\cdot}8\chi^2$ with df = 2 when the interaction effects were constrained to zero.

Without detailed power analyses of moderation models, which are not currently available, it is not possible to chose definitively between these two interpretations although a review of the available evidence leads us to favour the latter.

To elucidate further the possible significance of these findings, it is necessary to discriminate between environmental factors which influence mean levels of a trait and those which influence the sources of individual differences. Our data contains a strong positive association between FD and N for which there are at least three possible causal pathways. First, the twin herself might directly influence level of FD so that a highly neurotic child might cause family conflict. Secondly, since levels of N are correlated in family members, the highly neurotic parents or siblings of a twin with high N might cause high FD. Thirdly, high levels of family conflict might directly cause high levels of N in the twin.

While we have no direct evidence for the importance of these three possible pathways, it is plausible that all are acting to some extent. Assuming that there is some direct causal effect of family FD on twin N, our results suggest that while family functioning can influence mean levels of N and its variance, they do not influence the proportion of individual differences that are due to genetic and environmental factors.

It might be useful to contrast our findings with recent results for an adoption study of adolescent aggression and conduct disorder (CD) (Cadoret *et al*. 1995). In this study, an adverse home environment not only increased mean levels of CD symptoms but also increased the differences in the levels of CD between those at low *versus* high genetic risk. That is, not only did a dysfunctional environment on average cause more conduct problems, but it will actually provide more opportunities for the expression of a genetic liability to behaviours such as fighting, lying to parents, truancy and running away from home. Furthermore, families with high levels of FD may monitor their children less effectively, thereby maximizing genetic influences on peer selection, with peer environment in turn feeding-back on the phenotype of the child. These results are consistent with other adoption study evidence that the genetic predisposition to externalizing behaviours may be maximally expressed under adverse rearing environments (Crowe, 1974; Cloninger *et al*. 1982).

The pattern with N is fundamentally different. While mean levels of N tend to increase in highly conflictual families, the increase is approximately the same in those with low *versus* high genetic liability to N. That is, the phenotypic expression of the genetic liability to N appears to be insensitive to the variation in family dysfunction. This difference might arise because conduct disorder is a behavioural phenotype while N is a personality trait. Shared environment × gene interactions will be more robust for phenotypes that are behavioural than for those that reflect trait characteristics.

Limitations

These results should be interpreted in the context of five potential methodological limitations. First, the sample was restricted to Caucasian female twins born in Virginia. It is unknown whether these results would extrapolate to males or to other ethnic or geographical populations. Secondly, the analytical models applied in this report are relatively new and their power is not yet well understood. Thirdly, it might seem paradoxical that we detected a main effect of FD on N and yet our twin modelling found no evidence for shared environmental effects. This is easily explicable, however, given the power of the twin method (Neale *et al*. 1994). In our analyses, FD accounted for $<3\%$ of the total

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variation in N and far larger sample sizes would be needed to detect, by latent variable modelling, a contribution of shared environmental variance of this magnitude in the presence of substantial genetic variation. Fourthly, we have only tested one global measure of family functioning. It is possible that the genetic influences on N may be moderated by other aspects of the family environment. Fifthly, by combining parental and twin reports of FD, we may have confounded rather than clarified the effects of FD on N. Therefore, we re-ran our models twice now defining FD as, respectively, the mean of twin reports only and the mean of parental reports only. Using twin reports, the pattern of results was nearly identical to that seen in the table with the scalar model again producing the best fit. Using parental reports, in the moderator model, estimates of the heritability of N were indistinguishable at minimum and maximal values of FD, the variance in N increased only slightly with increasing levels of FD and the standard ACE model fit best. Our combining of twin and parental reports for FD did not obscure evidence for a genotype–environment interaction in the aetiology of N.

The power of a number of gene–environment interaction models using the general approach employed in this study has recently been examined (Purcell, 2002). The results suggest that the sample size employed in the present study should have adequate power to detect moderate gene–environment interactions.

This work was supported by NIH grants MH-40828, MH/AA/DA-49492, DA-11287 and AA-09095. We acknowledge the contribution of the Virginia Twin Registry, now part of the Mid-Atlantic Twin Registry (MATR), to ascertainment of subjects for this study. The MATR, directed by Dr L. Corey, has received support from the National Institutes of Health, the Carman Trust and the W. M. Keck, John Templeton and Robert Wood Johnson Foundations.

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