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Sex differences and non-additivity in the effects of genes on personality

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New large-sample data show that non-additive genetic effects, probably epistatic interactions between loci, and sex-limited gene expression are significant features of the genetic architecture of human personality as measured by questionnaire scales of extraversion and neuroticism. Three large data sets – new data on large samples (n = 20 554) of US twins, their spouses, parents, siblings and children, correlations for Australian twins (n = 7 532), and previously published twin data from Finland (n = 14 288) – are subjected to an integrated analysis to test alternative hypotheses about the genetic causes of family resemblance in personality. When allowance is made for differences in reliability of the scales, the combined data are consistent with the same model for variation. There are significant amounts of genetic non-additivity for both dimensions of personality. The evidence favours additive × additive epistatic interactions rather than dominance. In the case of neuroticism, there is especially strong evidence of sex differences in genetic architecture favouring a greater relative contribution of non-additive genetic effects in males. The data confirm previous claims to find no major contribution of the shared environment of twins and siblings to these dimensions of personality. Correlations between spouses are zero, and the correlations for very large samples of siblings and non-identical twins do not differ significantly.

Keywords: personality, twins, genetics, sex differences, non-additivity, neuroticism

Three sources of data have been exploited in separate studies of the inheritance of personality: twins reared together[1–5] or apart;[6–9] nuclear families;[10,11] adoption studies.[12–15] These studies generally concur in showing a moderate contribution of genetic factors to variation in the major dimensions of personality and in showing that the contribution of the environment shared by family members to the correlations between relatives is usually small. Indeed, even when attempts have been made to measure (rather than simply to estimate) the environmental factors salient to personality variation, their contribution to family resemblance has been pitifully small.[2,4,16]

Beyond this, however, there are large areas of uncertainty, even apparent inconsistency, which our new study tries to resolve. It has been claimed that estimates of heritability derived from studies of separated twins, and studies of monozygotic and dizygotic twins reared together, exceed those obtained from nuclear family and adoption data.[12,13] There are at least three possible reasons for this: twins are not typical of the population with respect to the degree of family resemblance; twins are typical but the resemblance of MZ twins is inflated by non-additive genetic effects;[15–18,1] the effects of genes on personality are partly age-specific 'genotype × age interaction',[19,20]) so the similarity between twins exceeds that for non-twins because twins are always measured at the same age but other relatives are not.

Most attempts to analyse such non-additive effects have been tentative because sample sizes have been far too small to allow reliable discrimination between alternative hypotheses. It has seldom been possible to detect genetic non-additivity reliably in humans,[21,22] let alone decide on the kinds of gene action which may contribute to it. Thus, although Price et al[23] suggested the genetic effects on personality may be non-additive, Hewitt's reanalysis of their data[24] showed that there was no statistical reason to reject a purely additive genetic model. Several recent large studies of twins living together indicate that a purely additive model for genetic effects on personality cannot explain the fact that the correlation between DZ twins is significantly less than half that for MZ twins. The magnitude of the estimated non-additive component is very large compared with the additive component which suggests that the non-additivity may involve epistatic

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interactions between loci rather than simple dominance interaction between alleles.[25]

Data on twins alone, however, whether they be reared together or apart, cannot resolve the contribution of dominance and epistasis to non-additive genetic effects. A study is needed which can resolve the inconsistency between data on twins and non-twins and elucidate further uncertainties about the causes of family resemblance in personality at the same time, namely, the effects of sex on the expression of genes and environment and the effects of assortative mating. We summarise results of a new study of extremely large samples of adult US twins, their spouses, siblings, parents and children which allow us to test with the same instrument, at the same time and in the same population, alternative hypotheses to account for the inconsistency between twin and family data. The correlations from this study are integrated in a joint analysis of two other large twin studies using similar instruments in Australia and Finland. Correlations between MZ twins, siblings and parents and offspring provide enough information to detect non-additive genetic effects and, in large samples, to go some way to resolving dominance and epistasis. Pairs which differ with respect to sex permit analysis of sex differences in gene expression. Spouses allow detection of assortative mating. Some effects of genotype $\times$ age interaction and special twin environmental effects may be detected by comparing the correlations of dizygotic twins and non-twin siblings.

Data

The correlations between relatives for the three populations are given in Table 1. The US sample was drawn from the Virginia Population-based Twin Registry[26] and a volunteer sample of older twins drawn from the American Association of Retired Persons. Ages ranged from 18 to 88 years. Extraversion (E) and Neuroticism (N) scores were obtained from responses to a mailed questionnaire which included the short form of the Eysenck Personality Questionnaire (EPQ-R).[27] Heteroscedasticity was removed by the angular transformation and analysis conducted for the residual scores of the entire sample (n = 20 544 individuals) after eliminating the effects of sex, sample, twin vs non-twin, the linear and quadratic regressions on age and the two-way and three-way interactions between these terms. The correlations between relatives for the residuals did not change significantly from the correlations for the uncorrected transformed E and N scores.

The Australian study yields correlations for the arcsine transformed E and N scores of the full Eysenck Personality Questionnaire (EPQ)[28] gathered from a large volunteer sample of adult Australian twins.[3] The linear component of the regression on age was partialled out of the correlations separately for each of the five groups of twins. Correlations and sample sizes for the Finnish twins were taken from the publication of Rose et al.[2] The E and N scales used in this study are shortened forms of the E and N scales of the earlier Eysenck Personality Inventory (EPI).[29]

A model

The correlations between spouses for both personality dimensions in the US sample are unequivocally zero and justify our subsequent assumption that mating is random with respect to differences in Extraversion and Neuroticism. The assumption of random mating is further justified by correlations of 0.065 and 0.052 for E and N respectively in an English sample of 889 spouse pairs.[1] In view of the repeated strong claim that the shared environment of

Table 1 Correlations between relatives for Extraversion (E) and Neuroticism (N)

	USA			Australia			Finland		
		Correlation			Correlation			Correlation	
Relationship	No. of pairs	E	N	No. of pairs	E	N	No. of pairs	E	N
Male MZ	646	0.431	0.347	566	0.472	0.457	1027	0.460	0.326
Female MZ	1418	0.507	0.393	1233	0.517	0.497	1293	0.489	0.427
Male DZ	370	0.137	0.105	351	0.083	0.154	2304	0.153	0.124
Female DZ	702	0.098	0.170	751	0.159	0.241	2520	0.144	0.184
M–F DZ	1052	0.117	0.084	905	0.159	0.098	–	–	–
Male siblings	844	0.168	0.112	–	–	–	–	–	–
Female siblings	1787	0.190	0.187	–	–	–	–	–	–
M–F siblings	2299	0.123	0.128	–	–	–	–	–	–
Father–son	1082	0.120	0.128	–	–	–	–	–	–
Mother–daughter	2274	0.166	0.159	–	–	–	–	–	–
Father–daughter	1422	0.122	0.162	–	–	–	–	–	–
Mother–son	1562	0.195	0.197	–	–	–	–	–	–
Spouses	2212	0.002	0.008	–	–	–	–	–	–

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siblings contributes little to variation we shall assume, unless the data provide compelling reason to the contrary, that all family resemblance is genetic, but allow for the contribution of the unique environments within families since their contribution has been established beyond doubt.[1,4,30]

As far as the genetic component is concerned, we assume that genetic effects on personality are autosomal. The model always allows for additive genetic effects, and non-additive effects may also be included. We let h denote the (standardised) path coefficient from the additive genetic effect to phenotype, and d be the path coefficient from the dominance effect to phenotype. Subscripts m and f are used to denote the paths from genetic effects to phenotype in males and females respectively. We let r be the correlation between the effects of genes on the phenotypes of male and female members of unlike sex pairs. A demonstration that r = 1 amounts to showing that the same genes affect both sexes even though the h may be different in males and females. If r < l it is implied that at least some genes having an effect on one sex do not have a consistent effect on the other sex and possibly that different genes affect the two sexes. Although, in principle, r may differ for additive and non-additive effects, our capacity to resolve such subtleties even with the present sample sizes is expected to be small, so we shall assume that r is the same for both additive and non-additive effects. When non-additive genetic effects are entirely due to dominance (ie there are no epistatic interactions) the assumption of random mating yields the following pattern of expectations for the correlations between relatives:

	Male	Female	Male–female
MZ twins	$h^2_m + d^2_m$	$h^2_f + d^2_f$	–
DZ twins (siblings)	$\frac{1}{2}h^2_m + \frac{1}{4}d^2_m$	$\frac{1}{2}h^2_f + \frac{1}{4}d^2_f$	$\frac{1}{2}r(h_m h_f + \frac{1}{2}d_m d_f)$
Parent–Offspring	$\frac{1}{2}h^2_m$	$\frac{1}{2}h^2_f$	$\frac{1}{2}h_m h_f$
Spouses	–	–	0

The model assumes that the environments of twins and siblings are uncorrelated (and, by implication, that DZ twins are no more correlated for the environments that affect personality than non-twin siblings). The effects of epistatic interaction between additive genetic effects ('additive × additive epistasis') contribute to these correlations in exactly the same way as dominance, except that the parent–offspring correlations also have a non-additive component identical to that for siblings and DZ twins in addition to the additive component.[31] In theory other types of epistasis may also be incorporated in a full genetic model,[32] but such subtle effects are highly confounded with additive and dominance effects in human data and are unlikely to be resolved at this stage.

Model-fitting

The twofold task of model-fitting is to provide estimates of the parameters of the model and to provide a goodness-of-fit test of the assumptions of the model. Failure of these assumptions would make the observed pattern of correlations differ from that predicted under the model. Examples of such assumptions which could lead to failure of the model are:

1) if environmental resemblance of siblings is less than that for DZ twins;
2) if there is a substantial effect of the shared home environment on twins and siblings;
3) if there is significant assortative mating;
4) if there is cooperative or competitive social interaction.[33,34]

The full model described above and various submodels were fitted to all three data sets separately by the method of weighted least squares applied to the z-transforms of the observed correlations. The Statistical Analysis System's NLIN procedure[35] was used for model-fitting. Observed correlations were treated as independent even though the same individual may contribute to more than one correlation and more than once to the same correlation (in sibships having more than two members, for example). The application of weighted least squares to family data summarised in this way gives estimates which are close to maximum likelihood and tends, if anything, to underestimate chi-squares used to test and compare models.[36] In our data, with relatively small correlations between relatives and small family sizes, the approximation is unlikely to lead to serious errors of inference.

Where theoretically possible, three models for the relationship between gene expression and sex were fitted in all possible combinations, with three models for the contribution of additive and non-additive effects. With respect to sex-dependent gene expression, models were fitted in which

1) the same genes had identical effects in both sexes (eg r = 1, $h_m = h_f$, $d_m = d_f$);
2) the same genes had different effects in males and females (eg r = 1 but $h_m = h_f$);
3) different genes (r < 1) had different effects on the phenotypes of the two sexes.

Models considered for the additive and non-additive components were:

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1) additive effects with no non-additivity;
2) additive plus dominance effects;
3) additive plus additive × additive epistatic effects.

In practice, the full range of models can be fitted only to the US data set where the twin and sibling data are supplemented by parent–offspring data. In the Australian and Finnish data sets, which lack parents and offspring, the effects of dominance cannot be resolved. The lack of published data on unlike-sex pairs in the Finnish sample forces us to assume the same genes affect variation in males and females in our separate analysis of these data.[37]

Initially, models were fitted to the separate samples to allow comparison of estimates over populations. Then we conducted a combined analysis of all three populations by fitting the same model and parameter values jointly to all 22 correlations for each variable. Since the three studies used scales of different length and, in the case of the EPI, slightly different sets of items to measure E and N, we expect the scales to have different reliability coefficients. In the joint analysis we multiplied the expected correlations for the US and Finnish samples (EPQ-R and EPI) by the parameters r_2 and r_3 respectively to allow for the reduction in reliability of these two shorter scales compared with that of the full EPQ employed with the Australian sample. These additional parameters were estimated along with the parameters of the genetic model in the combined analysis.

Results

Chi-square statistics which measure the goodness of fit of the models fitted to the separate populations are given in Table 2. Large chi-squares indicate a relatively poor fit. Differences in chi-square between different models are a guide to the reduction in fit which occurs when specific combinations of parameters are eliminated from the model.[1]

Estimates of the proportions of variation in males and females due to various genetic sources in the populations severally and jointly are given for selected models in Table 3. The separate analyses show the qualitative findings to be remarkably consistent in the different populations. In all three populations and for both variables, the assumption of additive gene action and no sex differences in gene expression fails badly. In every case, including non-additive genetic effects in the model significantly improves the fit for both variables, especially for extraversion. The US data, which include the critical sample of parents and offspring, suggest that models including epistatic effects perform somewhat better than those which allow for dominance, either by the strict criterion of goodness of fit, or because models involving dominance give opposite signs to the non-additive genetic effects in the two sexes. Some improvement is generally found when sex differences in gene expression are included in the model but this effect is more marked for neuroticism than extraversion and more striking for extraversion in the US data than in the other samples. There is little gain for either variable in allowing for different genes to be expressed in the two sexes.

The differential pattern of results for extraversion and neuroticism becomes consistent and clear in the combined analysis. For both E and N, the effects of non-additivity cannot be ignored and the balance of evidence points to epistasis rather than dominance as the primary non-additive component. For extraversion, however, sex differences in gene expression are either absent or very small and the reliabilities of the three scales are all quite similar. For neuroticism,

Table 2 Goodness of fit chi-squares for genetic models of personality in three populations

		USA χ^2			Australia[a] χ^2			Finland[a,b] χ^2		
Sex limitation	Non-additivity	E	N	df	E	N	df	E	N	df
None	None	75.96	26.08	12	23.58	21.76	4	31.69	17.23	3
None	Dominance	34.21	21.97	11	3.08	9.96	3	0.90	12.48	2
None	Epistasis	18.44	16.64	11						
Same genes	None	64.00	18.87	11	20.34	18.03	3	31.08	4.20	2
Same genes	Dominance	15.00[c]	9.07[c]	9	1.10	0.01	1	[d]	[d]	0
	Epistasis	12.64	10.91	9						
Different genes	None	46.26	16.77	10	15.18	1.92	2	–	–	–
Different genes	Dominance	13.61[c]	5.78[c]	8	–	–	–	–	–	–
	Epistasis	12.21	10.70	8				–	–	–

Notes: [a]Effects of dominance and epistasis cannot be distinguished formally with twin data alone
[b]No unlike-sex pairs reported for Finnish data so 'different genes' model for sex limitation cannot be tested
[c]Nonsense values for parameters (opposite signs for dominance effects)
[d]Perfect fit solution

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sex differences in the magnitude of genetic effects are highly significant and imply a larger non-additive effect in men than in women although the same genes are being expressed in both sexes (r = 1). The reliability of the short form EPQ-R and EPI neutoricism scales are estimated to be significantly less than the reliability of the longer EPQ scale. Population differences in the effects of the stable within-family environment would lead to the same pattern of correlations.

The fact that we obtain a good fit to the correlations from a model which omits all environmental effects apart from those which give rise to differences between individual members of the same family suggests that there are no marked long-term effects of the shared family environment on the adult personality dimensions of extraversion and neuroticism. Our large samples of siblings and parents and offspring give no reason to believe that the results for DZ twins are not typical of first degree relatives in general. The low correlation for DZ twins compared with that for MZs has led to the suggestion in the past that the phenotype for extraversion was affected by competition between twins based on genetic differences. The fact that the sibling correlation is not markedly greater than the DZ correlation confirms that, if there is competition, it is not made much more intensive by the twin condition. The large sample of spouse pairs shows beyond reasonable doubt that mating is truly random for both extraversion and neuroticism. These data add overwhelming support to abandoning the assumption of purely additive genetic variation in favour of a model which recognises that genetic effects on personality have a substantial non-additive component. These results are strengthened still further by correlations for scales related to extraversion and neuroticism in samples of separated monozygotic twins[6,7] since it is difficult to claim that these had been subjected to a 'special MZ twin environment' which is uncorrelated with genotype. The finding that the non-additive genetic effects are, if anything, epistatic is consistent with earlier speculation based on twin data alone[25] that the relative amount of non-additivity at loci responsible for extraversion was too great to be caused by dominance alone.

Discussion

The finding that there is a significant and substantial amount of non-additive genetic variation for these personality measures provides a strong indication that the scales reflect differences in biologically significant dimensions of behaviour. It has been argued that traits which have been subject to strong directional selection are characterised by marked directional dominance or duplicate gene interactions, whereas traits subject to stabilising selection display weak, ambidirectional dominance and/or complementary gene interactions.[38,39] Strong directional selection is expected to modify the action and interaction of alleles to optimise the phenotypic expression of the trait. It is premature to draw strong conclusions from second-degree statistics in which the direction and type of non-additive effects cannot be inferred reliably. However, Eaves[25] indicated that a very much reduced DZ and sibling correlation relative to that for MZ twins was only likely if there was epistasis of the duplicate-gene type. If this is true, then it is very tempting to conclude that the traits measured by these instruments have been

Table 3 Estimated contribution (% variance) of additive and epistatic effects to E and N under two models for genetic effects on males and females

Population	Sex limitation	Trait	Genetic variance (%) Additive Males	Additive Females	Epistatic Males	Epistatic Females	Relative reliability USA	Relative reliability Finland	Goodness of fit χ^2	Goodness of fit df
USA	Absent	E	11.1	11.1	37.2	37.2	–	–	18.4	11
		N	22.1	22.1	15.8	15.8	–	–	16.6	11
	Present	E	8.5	14.7	34.8	35.9	–	–	12.6	9
		N	16.1	29.2	18.2	10.3	–	–	10.9	9
Australia	Absent	E	8.0	8.0	42.3	42.3	–	–	3.1	3
		N	16.3	16.3	32.1	32.1	–	–	10.0	3
	Present	E	5.3	12.9	41.6	38.9	–	–	1.1	1
		N	14.0	45.9	31.8	0.0	–	–	0.0	1
Finland	Absent	E	11.7	11.7	35.9	35.9	–	–	0.9	2
		N	23.8	23.8	14.5	14.5	–	–	12.5	2
	Present	E	15.2	8.7	30.8	40.2	–	–	–	–
		N	17.0	30.9	15.6	11.8	–	–	–	–
Combined	Absent	E	11.3	11.3	38.9	38.9	0.97	0.95	22.6	18
		N	26.1	26.1	21.8	21.8	0.80	0.81	40.6	18
	Present	E	10.2	12.7	36.6	38.9	0.97	0.96	16.1	16
		N	15.7	38.2	21.9	13.1	0.80	0.83	18.8	16

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linearly related to reproductive fitness for a long time. This represents a change in what has been claimed in the past for personality measures[40,41] because additive models for gene action tended to fit the smaller samples then available.

Although the formal contributions of epistasis to family resemblance are well known, we have little beyond preliminary work[25] to help understand whether the patterns expected under stabilising and directional selection can be distinguished without breeding studies that can only be undertaken in animals and plants. Further, it might be thought that there would be marked assortative mating (and even non-genetic parent–offspring transmission) for traits that are adaptively significant. Yet extraversion and neuroticism show no evidence whatever of assortative mating. Does this indicate an inconsistency in our models for family resemblance in personality, or is the intuition fundamentally unsound that individuals mate for what really matters biologically? Finally, the sex-difference in the relative amounts of additive and non-additive variation for neuroticism might betoken sex differences in its adaptive significance. These are issues for which we have neither theory nor data at present, but it is to be hoped that our findings stimulate the production of both.

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