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Genetic and Environmental Influences on Pre-schizophrenic Personality: MAXCOV–HITMAX and LISREL Analyses

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Abstract

Postulating that the predisposition to illness in Claridge's disease model of schizophrenia can be equated with the personality dimensions S or Insensitivity, (low) E or Extraversion, and N or Neuroticism, as measured by Van Kampen's 3DPT, and assuming that the mode of transmission of schizophrenia is basically polygenic, the genetic and environmental etiology of S, E, and N was assessed in a sample of 52 MZ and 76 DZ twin pairs and their parents by means of LISREL. Besides, in a sample of 2118 subjects MAXCOV–HITMAX analyses were conducted for these factors as well as for the personality dimension G or Orderliness, but now assessed by the 4DPT, in order to find out whether a discrete or quasi-discrete variable might also underlie these dimensions, giving support to the possibility of dominance or epistasis. The results obtained in these investigations favoured a model for all three dimensions, allowing for both additive and non-additive genetic effects in combination with non-shared environmental influences. It was not possible to choose between a model involving dominance and a model involving epistatic genetic effects. With the use of scores corrected for sex and age, which were converted to normal scores, the proportion of variance explained by additive genetic factors was 20% for S, 40–41% for E, and 26–29% for N. Dominance or multiple-gene epistasis accounted for 37–38% (S), 19–20% (E), and 30–31% (N), and unshared environmental influences for 42–43% (S), 41% (E), and 42–43% (N) respectively. Copyright © 1999 John Wiley & Sons, Ltd.

INTRODUCTION

According to Claridge's disease model (see e.g. Claridge, 1985; 1990), schizophrenia may best be compared with systemic diseases, such as essential hypertension

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(including its sequelae), and not with infectious or neurological illnesses or major gene disorders (see also Eysenck, 1960). The most obvious difference, therefore, between the model advocated by Claridge and the usual illness model in psychiatry concerns the assumption that continuity in illness not only refers to a possible spectrum of dysfunctional states (see for the schizophrenic spectrum e.g. Baron, Gruen, Rainer, Kane, Asnis and Lord, 1985; Kendler, Gruenberg and Kinney, 1994), but also to a predisposition to disease that can be described as 'fully dimensional' (Claridge, 1994), because it emphasizes continuity at the personality level (see also Foulds, 1965). This distinction is important with respect to etiology, for it suggests that the same mechanisms that are responsible for normal personality functioning are also operating in the development of the dysfunctional state. Just as illnesses, such as heart failure or a cerebrovascular accident, may be secondary to high blood pressure—measured on a scale running from low to high—, so schizophrenia may arise from extreme personality variations which, in moderate degree, are perfectly healthy. Moreover, in both conditions the full dimensional aspects may give rise only to dysfunction in interaction with environmental influences or triggering factors, such as diet, biological hazards, and life stress. According to Claridge, then, it seems very likely that it is the dispositional aspects of schizophrenia that are inherited, rather than schizophrenia as a full-blown disorder.

One problem, however, with Claridge's (1990; 1994) conceptualization consists of his choice of schizotypal personality factors as the most likely candidates to represent this predisposition. As Claridge (1994, p. 155) himself asserts, at least the 'positive' (or psychotic-like) and the 'negative' (or introvertive-anhedonic) components that have been identified in 'normal schizotypy' (see e.g. Mason, 1995; Vollema and Van den Bosch, 1995) map well into the symptomatic domain of both schizophrenia and schizotypal personality disorder. The same may be said with respect to the two remaining schizotypy factors, Impulsive Nonconformity and Cognitive Disorganization (see e.g. Heston, 1966; Kendler, 1985). As, however, schizotypal personality disorder clearly belongs to the dysfunctional spectrum, it seems rather risky to regard the four schizotypy factors as normal dimensions of personality instead of interpreting them as factors referring to attenuated symptoms of schizophrenia. This conclusion seems especially relevant with respect to the positive factor of schizotypy, but the other factors may also be seen as primarily symptomatic, especially if schizophrenia is defined in a Bleulerian sense, emphasizing associative loosening (Cognitive Disorganization) and negative symptoms, such as affective blunting, autism, and avolition (Introvertive Anhedonia) (see Andreasen, 1987).

A probably more defensible description of the pre-schizophrenic personality has been given by Von Zerssen (1993; Pössl and Von Zerssen, 1990). According to his investigations, two personality types can be discerned that may be present prior both to schizophrenia and to neurotic illness. These personality types have been designated as the 'anxious–insecure type' and the 'nervous–tense type'. It is explicitly emphasized that the traits that these types comprise are different from schizotypal personality traits, because the latter ''traits'' are probably symptoms of a disorder within the schizophrenic spectrum ..., originally designated as ''schizophrenia simplex'' or ''latent schizophrenia'' ..., or they may represent symptoms of an initial stage of a later on typical schizophrenic psychosis' (Von Zerssen, 1993, p. 127). Although many individual traits are listed to characterize both personality types (see Pössl and Von Zerssen, 1990), a more global characterization has also been offered. For,

according to Von Zerssen (1993, p. 129), his two types are conceptually related to two Big-Five dimensions, as described, for instance, by Goldberg (1990): 'the ''anxious–insecure type'' corresponds to a high degree of Neuroticism; and the ''nervous–tense type'' holds a low position on the Agreeableness factor and a relatively high position on Neuroticism'. However, it might be added that the anxious–insecure type also seems to represent a low degree of Extraversion. With regard to these two (or three) personality dimensions (or to the personality types themselves), the term 'premorbid personality' is used in its literal sense. That is, the term does not refer to 'insidious changes of personality that are probably an expression of the disorder itself', but 'to the whole period from birth to the time *before* such changes have become obvious. The term thus covers the formation of character during childhood and adolescence, and its further modifications during adulthood when the subject was not yet afflicted by a functional mental disorder' (Von Zerssen, 1993, p. 117).

Independent of Von Zerssen's contribution, we have also offered a characterization of the pre-schizophrenic personality. Based on criticisms on Eysenck's Psychoticism model (e.g. Eysenck and Eysenck, 1976), an alternative model was postulated, comprising the dimensions Insensitivity (S), Extraversion (E), Neuroticism (N), and Orderliness (G) (Van Kampen, 1993; 1996b; 1997). Of these dimensions, the factors S and G took the place of Eysenck's P dimension, with S positively and G negatively related to P. In this conceptualization, the pre-schizophrenic personality is typified by high positions on S and N, and a low position on E. For the G factor, no relationship with the pre-schizophrenic personality was expected. As we could demonstrate (Van Kampen, 1997) that the factors S, E, N, and G, as measured by our 4DPT or Four-Dimensional Personality Test, are clearly correlated with the Big-Five dimensions Agreeableness (negative), Extraversion, Neuroticism, and Conscientiousness respectively, our characterization of the pre-schizophrenic personality is obviously similar to that of Von Zerssen. Moreover, we could demonstrate that in an inventory (the Schizotypic Syndrome Questionnaire; in Dutch: SSV), which we specifically constructed to measure a wide variety of schizotypic symptoms (12 scales), virtually all scales correlated positively with S and N, negatively with E, and did not correlate with G (see for some preliminary results Van Kampen, 1996a). In this context, it might be of interest that the Eysenck dimensions P, E (reversed), and N have been found by Mason (1995) to load, respectively, on his schizotypy factors Impulsive Nonconformity, Introvertive Anhedonia, and Cognitive Disorganization. So, even underlying the putatively dispositional factors of Claridge (see above), the same three-factor structure seems present as observed in Von Zerssen's typology and put forward in our own conceptualization of the pre-schizophrenic personality. Thus, summarizing the above, and taking Claridge's disease model for granted, we may assume that the hereditary basis of schizophrenia—and of other schizophrenic spectrum disorders—is most likely to be found in the contribution of genes to individual differences in S, E, and N. Because genetic modelling studies tend to favour the hypothesis that some sort of polygenic system is involved in the genetic transmission of schizophrenia (see for a review Faraone and Tsuang, 1985), we will at least here presuppose the influence of additive genetic factors.

Fortunately, for E and N several behavioural–genetic investigations have already been conducted (see e.g. Eaves, Eysenck and Martin, 1989; Floderus-Myrhed, Pedersen and Rasmuson, 1980; Loehlin and Nichols, 1976; Pedersen, Plomin, McClearn and Friberg, 1988; Rose, Koskenvuo, Kaprio, Sarna and Langinvainio,

1988). The most popular design is the classical twin study, in which intrapair similarity in MZ or identical twin pairs is compared with that in DZ or fraternal twin pairs. Particularly in earlier twin studies, a simple model was believed to be valid, assuming only the influences of additive genetic factors and an unshared environment. More recently, however, the finding that MZ correlations for E and N are often more than twice the magnitude of DZ correlations (see Henderson, 1982; Plomin, Chipuer and Loehlin, 1990), together with the use of much larger twin samples, resulted in the testing of more elaborate models, in which, for instance, the presence of contrast effects (see e.g. Carey, 1986; Rose, 1995) or the presence of both additive and non-additive genetic effects was assumed. Evidence of a significant effect of dominance on E, but not on N, for instance, was obtained in the re-analysis by Eaves *et al*. (1989) of data of Floderus-Myrhed *et al*. (1980). A similar effect, but this time for N and not for E, was reported in a study by Price, Vandenberg, Iyer and Williams (1982). A further complication of this picture is that both in Loehlin's (1992) meta-analysis and in a study by Finkel and McGue (1997) non-additive factors were found to influence the two dimensions, while in a study by Riemann, Angleitner and Strelau (1997) significant dominance effects were not demonstrated, at least in self-report data. However, the consistent finding that the parent–child correlations for E and N are very low (see e.g. Eaves *et al*., 1989, p. 127; Carmichael and McGue, 1994; Bratko and Marušić, 1997) may in itself enhance the possibility that non-additive genetic effects play an important role in the development of these dimensions.

In this article we will also examine the genetic and environmental background of the dimensions E and N, measuring these factors by the E and N scales of the 3DPT or Three-Dimensional Personality Test (Van Kampen, 1993), a forerunner of the 4DPT. However, as the sample of MZ and DZ twins and their parents in our study is relatively small, it seemed rather risky to compare several models merely on the basis of this sample. Therefore, to direct our comparison, we will first present the results of a taxometric investigation in which we tested whether the separate 4DPT factors, that were still interpreted as 'continuously distributed behavioral tendencies' (see Gangestad and Snyder, 1991, p. 145), might be influenced by a latent class variable. With respect to the possibility of non-additive effects (dominance or epistasis), the demonstration that such a class variable may exist is, of course, most welcome. Moreover, the 'raw data', including parent–child correlations, obtained in our twin study will be checked for their correspondence with the above-mentioned findings of 'non-additivity'.

Besides these two personality factors, our twin and taxometric studies will also address factor S. In the case of this dimension, however, we could locate only three twin studies in which a scale was used with the explicit intention of measuring the Big-Five factor Agreeableness (Bergeman, Chipuer, Plomin, Pedersen, McClearn, Nesselroade, Costa and McCrae, 1993; Jang, Livesley and Vernon, 1996; Riemann *et al*., 1997). Unfortunately, the results of these studies are very different. Bergeman *et al*. (1993) observed only a small, but nonsignificant, dominance effect (in combination with both unshared and shared environmental influences), while Jang *et al*. (1996) found evidence of an additive genetic component (in combination with specific environmental effects). In the study by Riemann *et al*. (1997), a model encompassing additive effects, dominance effects, and specific environmental effects yielded the best fit for a self-report measure of Agreeableness, although the inclusion of the dominance parameter did not significantly improve the fit as compared with a reduced

model which allows for merely additive and specific environmental effects. With respect to the S dimension, therefore, the results of our taxometric study might be especially revealing.

METHOD

In order to test the latent class model, MAXCOV–HITMAX analyses were conducted (Meehl, 1973; Gangestad and Snyder, 1985). Essentially, this method capitalizes on the fact that the covariance between two taxon or class indicators will be greatest when evaluated in a 'mixed' group consisting of as many taxon members as members in the taxon complement, and will be smallest in a group of individuals who share only membership in the same single class. The procedure begins with the selection of k indicators that are believed to represent the underlying class variable. In the present investigation, for instance, we avoided excessive interitem correlations within taxon or complement by selecting from each 4DPT scale seven items that seemed to sample different aspects of the scale (see the Appendix), and that indeed later on were found to intercorrelate almost zero for subjects scoring in the highest and in the lowest quartile on a scale of the sum of these items (cf. Trull, Widiger and Guthrie, 1990, p. 45). Moreover, we selected our items on the basis of an item–total correlation of at least 0.40, which agrees with the assumed capacity of each item to discriminate between taxon and complement. Following the selection of k indicators, the procedure continues by examining the covariances for each pair of indicators (keyed in the same direction) among the subsamples of subjects scoring at each level of the scale formed by the remaining $k - 2$ indicators. In this investigation, the number of covariances for each level is 21. Averaging these covariances, a single curve can be obtained relating the interitem covariances to these scale levels. In order to reduce sampling variation and the effects of model assumption departures, this curve will usually be presented after smoothing the means. In the case of a latent class variable, the relationship mentioned is quadratic, which normally corresponds to a curve peaking towards the middle of the scale and drooping towards the extremes. However, if the taxon base rate is relatively low (0.25 or less), the curve may be not concave down, but may be characterized rather by an upward-sweeping right-end peak (Lenzenweger and Korfine, 1992, p. 568). The base rate of a taxon can be estimated by solving the quadratic equation for the proportion of taxon members in each scale interval, multiplying these values by the frequencies observed for each interval, and dividing the sum of these latter values by the total number of subjects. Obviously, if the latent structure is nontaxonic, there will be no variation in covariation, and the resulting curve will be flat.

The MAXCOV–HITMAX analyses were carried out in a sample of 2118 subjects of 20–59 years of age (1225 females, 820 males, and 73 subjects of unknown sex) that was composed of several subsamples previously used in other studies examining the 4DPT (e.g. Van Kampen, 1997). All subjects had responded to a written request by their family doctor to fill in this inventory. The subjects are inhabitants of the Dutch cities of Amersfoort, Haarlem, Gouda, Leiden, and Tilburg. The mean age of the sample is 38.94 with a standard deviation of 11.29 years. The sample divided by sex and age can be said to be fairly representative of four subgroups within the Dutch population: females aged 20–39, females aged 40–59, males aged 20–39, and males aged 40–59 (see Van Kampen, 1996a, p. 98).

In our study of MZ and DZ twins and their parents, three models were fitted by means of LISREL (Jöreskog and Sörbom, 1989), allowing for additive gene action (parameter *h*), additive gene action plus dominance (parameter *d*), and additive gene action plus epistasis (parameter *i*), in all three cases combined with nonshared environmental influences (parameter *e*) (see Heath, Neale, Hewitt, Eaves, and Fulker, 1989). These models will be denoted here, respectively, as the AE, ADE, and AIE models. Each model has been specified in terms of KSI (assumed causative factors), ETA (phenotype), and Y (observed variables), with the correlations (in PHI) between the two additive genetic, dominant or epistatic causative factors all set to unity for MZ twins, to 0.5, 0.25, and 0 for DZ twins, and to 0.5, 0, and 0, respectively, for parents and offspring. The correlations between the non-shared environmental factors for the two types of twin and for the parent–offspring pairings are, of course, zero. The variables observed—the 3DPT scores for S, E, or N—were believed to be completely determined by ETA, and, so, the diagonal values in LAMBDA-Y, representing the regressions of Y on ETA, were set to unity. The S, E, and N scores were corrected for the main effects of sex and age as well as for their interaction, by computing the standardized differences (or residuals) between the scores that were predicted merely on the basis of the subjects' sex and age and their actual scores. This was done for all subjects collectively (see McGue and Bouchard, 1984). Equality constraints were introduced for each parameter to be estimated, meaning that the coefficients for the paths leading from KSI to ETA were thought to be the same in twins from MZ and DZ pairs and in parents and offspring, as well as in first and second twins from each zygosity group. The parameters in GAMMA not belonging to the model to be tested (for instance, *d* in the AIE model) were fixed at zero. The models were fitted to the MZ, DZ, and parent–offspring covariance matrices for S, E, or N under maximum-likelihood estimation (see e.g. Neale, Heath, Hewitt, Eaves, and Fulker, 1989). This was done both prior to and after converting the scores to normal scores, i.e. the *z* scores the values would have if the observed distributions were perfectly normal. The χ^2 statistic that is provided by the LISREL program was used to assess the goodness of fit.

The present sample of twins consisted of 129 twin pairs between 13 and 21 years of age. These subjects formed part of a somewhat larger sample of 160 adolescent twin pairs who had originally been used by Boomsma (1992) in her study of cardiovascular risk factors, and who for the present study were approached by mail once again. One hundred and twenty-eight fathers (mean age 47.70, s.d. = 6.47) and 130 mothers (mean age 45.19, s.d. = 5.79) were also willing to cooperate in the present study. Originally, most addresses of twin pairs were obtained from the population registry of the City Council of Amsterdam. However, a small number of families who heard of the study from other twins also volunteered. The zygosity of the twins was determined by blood typing, and in 36 twin pairs also by DNA fingerprinting. The sample of 129 twin pairs consisted of 53 MZ pairs (mean age 15.96, s.d. = 2.16) and 76 DZ pairs (mean age 16.50, s.d. = 1.84). Usable 3DPT data were obtained from 51 (for S and E) or 52 (for N) MZ pairs and from all 76 DZ pairs. Of the MZ pairs with usable data, 24 or, with respect to N, 25 pairs were male and 27 female. The group of DZ pairs consisted of 26 male, 25 female, and 25 opposite-sex pairs. For all 128 fathers, and for 129, 127, or 128 mothers, a score on the S, E, or N scale respectively could be calculated. There were 122 complete cases with S, E, and N scores for both parents and two co-twins.

As mentioned before, the instruments used in our taxometric and in our twin study are the 4DPT (Van Kampen, 1997) and the 3DPT (Van Kampen, 1993) respectively. The scales of these instruments consist of 16 items each, to be answered with '*yes*' or '*no*'. All scales have been found to be sufficiently reliable, with coefficients of internal consistency (Cronbach's α) ranging from 0.78 (3DPT-S) to 0.90 (4DPT-N), and test–retest reliabilities ranging from 0.75 (4DPT-G) to 0.92 (3DPT-E) (Van Kampen, 1993; 1997; Van Kampen, unpublished data). The above-mentioned difference in assessment is due to the fact that the 4DPT had not yet been developed at the time when the twin study was conducted. However, it is important to note that the 16-item E and N scales of the two instruments are virtually identical, which results in a correlation between the two versions of each scale both of 0.98 ($n = 550$; Van Kampen, unpublished). The two 16-item S scales are more dissimilar with only 11 items in common. Notwithstanding, the S scales, too, proved to correlate very highly ($r = 0.92$). Of even more importance is the fact that the two sets of five items that are specific to the S scale of the 3DPT and the S scale of the 4DPT were found to correlate 0.88 ($n = 538$), when corrected for attenuation. Both sets measure thus clearly the same construct. Finally, it might be emphasized that of the 4DPT items selected for the MAXCOV–HITMAX analyses (see the Appendix), only two S items (3 and 19), one E item (61), and one N item (54) are not included in the 3DPT scales.

RESULTS

MAXCOV–HITMAX analyses

Prior to the MAXCOV–HITMAX analyses, the internal consistency values (Cronbach's α) for the 4DPT scales were calculated for the total sample of 2118 subjects. Like the α values originally determined in some of the subsamples (see above), the α coefficients in the total sample proved to be sufficiently high. For the S, E, N, and G scales, these α values were 0.77, 0.88, 0.90, and 0.78, respectively. Besides, the correlations between the items and the scales to which they belong were computed to aid in the selection of the $k = 7$ indicators for each scale (see the Method section).

Figure 1 shows the covariance curves (both unsmoothed and smoothed) that are based on the average covariances across 21 indicator pairings for each interval on the corresponding six-point scales for S, E, N, and G. The smoothed curves were obtained by applying a three-point running means filter to the unsmoothed covariance values, copying the curves on the endpoints (see e.g. Tukey, 1977). Due to the high positive skewness of S and the high negative skewness of G (1.05 and -0.89 for all 16 items), the number of subjects associated with each interval varied considerably. The mean covariances, for instance, associated with the scores zero and five on the six-point S scale were computed in a subsample of, on average, 628.52, and in a subsample of, on average, 31.29 subjects respectively. Also, a relatively small number of subjects (mean = 33.81) was found to have a G score of zero. At least for the scale levels $S = 5$ and $G = 0$, therefore, the estimates of the interindicator covariances are relatively unstable. All other subsamples, however, were relatively large, varying in magnitude from a mean number of 91.95 to a mean number of 753.76 subjects. As is evident from Figure 1, the four covariance curves are clearly consistent with the existence of a latent taxon. In the case of the six-point scales for S, E, and N, the curves are concave down, and in the case of the G scale a left-end peak is observed. The taxon base rates,

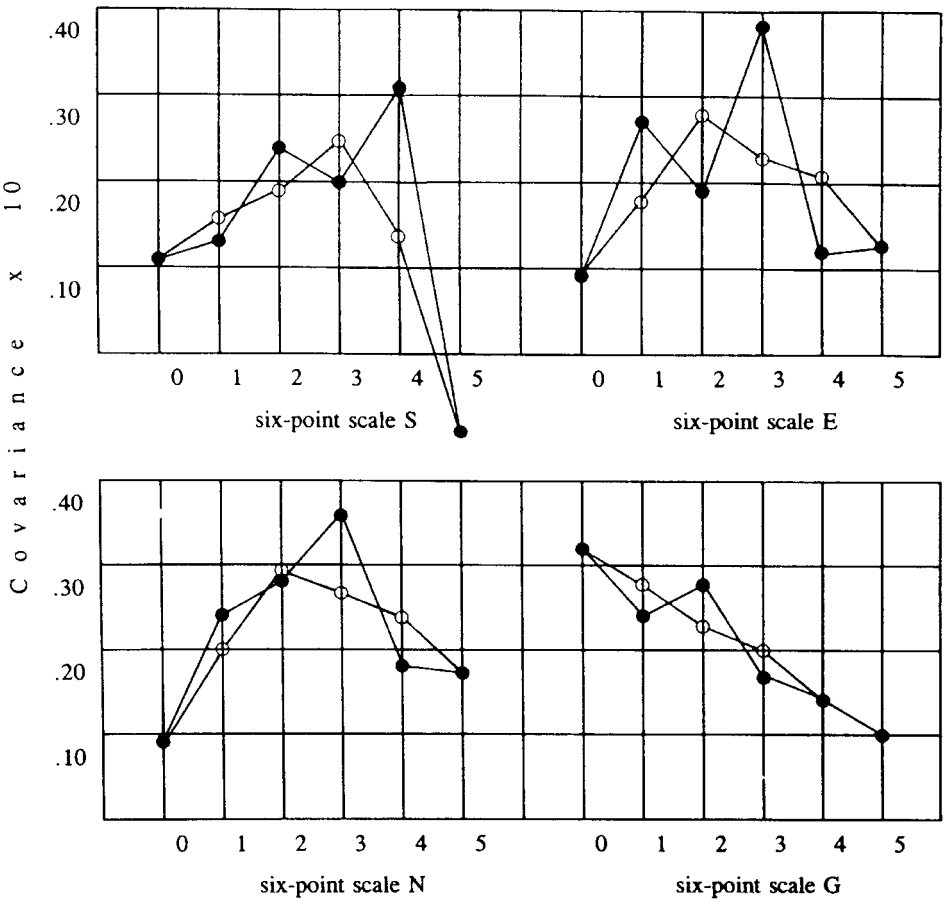

Figure 1. Mean covariances of 21 possible item pairs formed from seven 4DPT Insensitivity, seven Extraversion, seven Neuroticism, and seven Orderliness items as a function of the S, E, N, or G score on the corresponding six-point scales (unsmoothed values, ●; smoothed values, ○)

calculated on the basis of both the unsmoothed and the smoothed covariance values, are, respectively, 0.19 and 0.28 for S, 0.57 and 0.62 for E, 0.38 and 0.42 for N, and both 0.85 for G, the last value implying that the base rate for the 'non orderliness taxon' amounts to 0.15. All these values seem to be in accordance with the observed covariance patterns.

Parent–twin data and LISREL analyses

As already indicated, in this section three different parent–twin models (AE, ADE, and AIE) will be examined, estimating the effects of an additive genetic and a specific environmental factor on S, E, or N, with or without including the effects of dominance or epistasis. Although covariances will be used in the LISREL analyses, the correlations between the twins from the MZ and DZ groups and between the twins and their parents for the corrected scores on S, E, and N are also important. Table 1 mentions the intra-class correlations for all MZ and all DZ twins, as well as for the subgroups of same-sex and opposite-sex DZ pairs. It can be seen that all MZ

Table 1. Intra-class correlations of S, E, and N for MZ and DZ twin pairs (all data corrected for sex and age effects)

	S	Pairs	E	Pairs	N	Pairs
MZ twins, all	0.49***	51	0.61***	51	0.55***	52
DZ twins, all	0.08	76	0.26*	76	0.14	76
DZ twins, same sex	0.15	51	0.29*	51	0.15	51
DZ twins, oppos. sex	−0.07	25	0.16	25	0.12	25

Note: $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$.

correlations are higher than the corresponding DZ correlations. In fact, the MZ:DZ ratios in all three cases are greater than two which might suggest the influence of non-additive factors. With respect to E, however, it must be noticed that the MZ correlations for male ($r_{ic} = 0.84$) and female twin pairs ($r_{ic} = 0.39$) were found to differ from each other significantly ($p < 0.01$), which contrasts with several other findings (see e.g. Loehlin, 1992), and that, therefore, the 'overall' MZ correlation for E in Table 1 might be more or less atypical. All other MZ and DZ correlations between male and female twin pairs as well as the correlations between same-sex and opposite-sex DZ pairs were not significantly different. The same conclusion of 'non additivity' might also be drawn from Table 2, which shows that nearly all father-son, father-daughter, mother-son, and mother-daughter correlations for S, E, and N (corrected scores) are very small. It is tempting to interpret the two significant—yet low—correlations for E in this table in terms of the child's (former) identification with the same-sex parent. However, if this influence exists, it represents the shared environmental influence for the same-sex co-twins, something which is clearly at odds with the consistent finding that shared environmental factors have no or negligible effects on personality development (see e.g. Plomin and Daniels, 1987). At least in this study, therefore, no further attention will be given to this interpretation. In the last row of Table 2 the 'overall' parent–child correlations are mentioned. To prevent parents' results from being replicated, these correlations were calculated by combining two randomly drawn subgroups of cases: one group of 65 cases (with complete or incomplete data) in which the father–oldest-twin pairings were combined with the mother–youngest-twin pairings, and the remaining group of 65 cases (also complete or incomplete) in which the father–youngest-twin pairings were combined with the mother–oldest-twin pairings. The overall parent–offspring groups consisted of

Table 2. Parent–offspring correlations for S, E, and N (corrected scores)

	S	*n*	f/m	E	*n*	f/m	N	*n*	f/m
Father–son	−0.01	126	(77)	0.29*	126	(77)	0.04	127	(77)
Father–daughter	0.12	127	(76)	0.09	127	(76)	0.15	127	(76)
Mother–son	0.14	126	(77)	0.06	124	(76)	0.21	125	(76)
Mother–daughter	0.15	129	(77)	0.25*	127	(76)	0.15	129	(77)
Parent–offspring	0.08	254		0.20***	252		0.16**	254	

Note: $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$.
In testing the significance of the father–son, father–daughter, mother–son, and mother–daughter correlations, the degrees of freedom are defined on the basis of the number of fathers or mothers (see columns f/m), not on the basis of the number of pairs (*n*).

127 fathers, 127 mothers, 126 sons, and 128 daughters for S and N, and 127 fathers, 125 mothers, 125 sons and 127 daughters for E. Of course, the expectation that the parent–child correlation will equal half the upper-limit of the narrow-sense heritability (see Plomin, DeFries and McClearn, 1990) results in expected h^2 values of approximately 16, 40, and 32 per cent of the phenotypic variance of S, E, and N, respectively.

Table 3 gives a summary of the main results found in the LISREL testing of the three genetic/environmental models described above. All models were examined in the total groups of MZ and DZ pairs and in the overall group of parent–offspring pairings. In the calculation of the covariance matrices, the first and second twin from each zygosity group are the youngest and the oldest twin respectively, except in the subgroup of opposite-sex DZ twins, in which twin 1 is the female twin and twin 2 the male. Prior to the LISREL testing, the father–mother correlations for S, E, and N were calculated, making use of the scores corrected for sex and age. The (low) values observed—respectively, 0.18 ($p < 0.05$), −0.06, and 0.02—are in accordance with the model assumption that the additive genetic correlation between DZ twins is 0.5 (cf. the Method section). The three models have been fitted both prior to and after normalization of the corrected scores, for it was found that especially S, but also E and N, in both twin groups, and S in the group of parents, were highly or at least moderately skewed. In MZ and DZ twins the skewness values were 0.86 and 1.21 for S, −0.76 and −0.72 for E, and 0.56 and 0.75 for N, respectively; the skewness of S in the parents group was 1.48. Although in practice the effectiveness of the normalization procedure might be limited by the number of distinct values and their distribution in the original sample (see Wilkinson, 1987, p. 232), the distributions after normalization all resulted in a skewness value of almost zero (mean absolute value = 0.01). It is clear from Table 3 that for all scales all models provide a satisfactory fit to the data, with all p values associated with χ^2 well beyond 0.05. In fact, five p values of 1.00 even suggest a (nearly) perfect fit, a situation which seems to be partly dependent on the highly effective normalization procedure. Moreover, all parameter estimates were found to depart significantly from zero (p at least < 0.05). Regarding the non-normalized scores, only the differences in χ^2 between the nested models for S (AE and ADE, or AE and AIE) proved to be significant ($p < 0.05$), attesting that the fit of the ADE or AIE model was statistically superior to the fit of the AE model. The only changes in χ^2 that approached significance ($p < 0.10$) in the case of the nonnormalized scores were those observed for the ADE and AIE models for N compared to the AE model; the ADE and AIE models proved to fit slightly better here. However, as regards the normalized scores, both the ADE and AIE models for both S and N were found to result in a significant decrease in χ^2 ($p < 0.05$) compared to the χ^2 obtained for an AE model, which suggests that an ADE or AIE model for S and N might perhaps be a better choice. A choice between the two nonadditive models for S and N does not seem possible. With respect to E, no significant differences in χ^2 were observed, and, therefore, a choice between the AE, ADE, and AIE models for E remains undecided. However, because the results obtained in our taxometric study strongly support the existence of nonadditive factors in the etiology of S, E, and N (and of G), it seems best to select the ADE or AIE models for all three dimensions, decomposing the trait variance observed into additive genetic, nonadditive (dominant or epistatic), and nonshared environmental components. With this choice, and limiting our description merely to the LISREL estimates based on normalized scores, the narrow heritability,

Table 3. Maximum-likelihood parameter estimates, percentages of variance, and χ^2 for several models based on 3DPT data from MZ and DZ twins and their parents

		LISREL estimates										
Model	Scale	*h*	*e*	*d*	*i*	h^2	e^2	d^2	i^2	χ^2	df	*p*
AE	S[a]	0.55	0.84	—	—	0.30	0.70	—	—	7.20	7	0.41
ADE	S[a]	0.36	0.72	0.60	—	0.13	0.51	0.36	—	2.98	6	0.81
AIE	S[a]	0.39	0.71	—	0.59	0.15	0.50	—	0.35	2.31	6	0.89
AE	E[a]	0.72	0.69	—	—	0.52	0.48	—	—	7.62	7	0.37
ADE	E[a]	0.63	0.63	0.44	—	0.40	0.40	0.20	—	5.64	6	0.46
AIE	E[a]	0.65	0.63	—	0.41	0.43	0.40	—	0.17	6.05	6	0.42
AE	N[a]	0.66	0.74	—	—	0.44	0.56	—	—	7.27	7	0.40
ADE	N[a]	0.54	0.65	0.50	—	0.30	0.44	0.26	—	4.44	6	0.62
AIE	N[a]	0.56	0.65	—	0.50	0.32	0.43	—	0.25	4.13	6	0.66
AE	S[b]	0.59	0.77	—	—	0.37	0.63	—	—	6.84	7	0.45
ADE	S[b]	0.43	0.64	0.59	—	0.20	0.43	0.37	—	1.68	6	0.95
AIE	S[b]	0.44	0.63	—	0.60	0.20	0.42	—	0.38	0.49	6	1.00
AE	E[b]	0.69	0.68	—	—	0.51	0.49	—	—	2.29	7	0.94
ADE	E[b]	0.61	0.62	0.43	—	0.40	0.41	0.20	—	0.54	6	1.00
AIE	E[b]	0.62	0.62	—	0.42	0.41	0.41	—	0.19	0.33	6	1.00
AE	N[b]	0.63	0.74	—	—	0.42	0.58	—	—	4.59	7	0.71
ADE	N[b]	0.50	0.64	0.54	—	0.26	0.43	0.31	—	0.58	6	1.00
AIE	N[b]	0.52	0.63	—	0.53	0.29	0.42	—	0.30	0.43	6	1.00

Note: [a]Scores corrected for sex and age, but not normalized.
[b]Scores corrected for sex and age and normalized.

h^2, which refers to that part of the genetic variation that is transmissible across generations, amounts to 20%, 40–41%, and 26–29% of the variance for S, E, and N respectively (see Table 3). Our estimates of the broad heritability ($h^2 + d^2$ or $h^2 + i^2$), which refers to the effects of all genetic influences on S, E, and N, are 57–58%, 60%, and 57–59% respectively. Finally, in the ADE or AIE models, the proportions of variance attributable to nonshared environmental factors (e^2) are 42–43%, 41%, and 42–43% for S, E, and N respectively.

DISCUSSION

Although many models (both monogenic and polygenic) have been postulated to account for the specific genetic mechanisms involved in the transmission of schizophrenia, the type of model most often supported seems to be polygenic, which assumes the influence of a large number of underlying genes whose effects are (roughly) additive (Faraone and Tsuang, 1985). Besides arguments such as the fact that schizophrenia is not rare in the general population, or the fact that schizophrenia is a graded disease, ranging from mild to severe (see, e.g. Gottesman, 1991, p. 88), the most prominent finding supporting this view seems to be that the rates of schizophrenia observed among monozygotic co-twins and among first-, second-, and third-degree relatives of schizophrenic probands can be neatly predicted under a multifactorial polygenic threshold model (see e.g. McGue, Gottesman and Rao, 1983; 1985), whereas a single-locus model, whether assuming a dominant or a recessive gene, has repeatedly failed to account for this (exponential) pattern of familial risk figures (McGue and Gottesman, 1989). In this paper, therefore, the working hypothesis that the mode of genetic transmission in schizophrenia is basically polygenic has been accepted. Notwithstanding this, our model is in some ways at variance with the usual type of polygenic model applied to schizophrenia. Typical for these models is that all genetic and environmental causes of schizophrenia are said to be components of one single, unobservable, and continuously distributed variable termed liability or predisposition to schizophrenia (see e.g., Reich, Cloninger and Guze, 1975). In our model, though, the single vulnerability factor has been replaced by three independent factors, referring to the personality dimensions S or Insensitivity, (low) E or Extraversion, and N or Neuroticism respectively. We have done this not only on the basis of our criticisms on Eysenck's Psychoticism model (Van Kampen, 1993), but also in line with Claridge's (1990) assertion that the predisposition to schizophrenia manifests itself in personality variations that, in moderate degree, are quite normal. Moreover, the fact already mentioned (see the Introduction) that it is precisely the personality dimensions S, E, and N, and not G, that we found to correlate with symptoms of schizotypy, also adds credibility to our vulnerability model. In fact, the larger number of vulnerability factors we postulate may be not surprising, for in other polygenic diseases, such as coronary heart disease, numerous contributory risk factors have been identified, each with its own genetic and environmental background (Vogel and Motulsky, 1986). Consequently, the usual assumption of only one liability factor in schizophrenia seems not justified, and might, perhaps, be attributed to the unspecified nature of the schizophrenia diathesis in present-day formulations. The second departure from the usual type of polygenic model for schizophrenia consists in the introduction in our model of dominance or multiple-gene epistasis, i.e. the explicit acknowledgement that

a particular combination of many alleles at one locus (dominance) or at different chromosomal loci (epistasis) has a specific influence on the phenotypic variation in S, E, or N, in ways that cannot be explained by the additive genetic effects mentioned above. For the affirmation of these gene interaction effects, we especially referred to the results of a series of MAXCOV–HITMAX analyses, because on the basis of our LISREL parent–twin study alone no 'definite' choice could be made between three models for S, E, or N, that allow for, respectively, additive gene action plus nonshared environmental effects (AE model), the same influences plus dominance (ADE model), and the same influences plus multiple-gene epistasis (AIE model). In fact, all models did provide a satisfactory fit to the data, although the inclusion of the *d* or *i* parameter in the models for S and N significantly improved the fit compared to the AE model. The ADE or AIE model yielded also the best fit for E, although this time the inclusion of the parameters *d* or *i* did not give a significantly better fit. However, the results of our MAXCOV–HITMAX analyses did suggest very convincingly that in every instance the ADE or AIE model had to be chosen, because both for the personality dimensions S, E, and N, and for the personality dimension G or Orderliness, the influence of a latent class variable could be demonstrated. To the best of our knowledge, this seems to be the first time that such an influence has been revealed for personality factors that are clearly similar to four of the Big-Five dimensions (Van Kampen, 1997). However, it must be noted that the sample in the MAXCOV–HITMAX study is much older than the sample of MZ and DZ twins. Since quantitative genetic parameters could change developmentally, one could question our assumption that the results of our taxometric study might be used as an argument when choosing between alternative models in the LISREL analyses. Moreover, a nonadditive genetic interpretation of the MAXCOV–HITMAX results does not seem to be the only interpretation possible. The demonstration of a latent class, for instance, could also support a major-gene hypothesis or a 'threshold effect' in a polygenic model (Korfine and Lenzenweger, 1995). All one can say is that the results here reported converge and that they most likely seem to point to a model that also allows for non-additive effects. Of course, the ADE and AIE models also emphasize the influence of nonshared environmental factors, which is in accordance with, for instance, the observation that the concordance rate for schizophrenia in monozygotic twin pairs is far from complee (see e.g. Gottesman and Shields, 1972), and with the demonstration in adoption studies of schizophrenia that common environmental factors are not important (see, for instance, Kety, Rosenthal, Wender and Schulsinger, 1968). Moreover, in twin studies of normal personality, nonshared environmental factors have also been found to be most influential (see e.g. Plomin and Daniels, 1987). With all these findings at least consistent with our three-dimensional ADE or AIE model, it might perhaps be emphasized once more that it is only the specific personality constellation consisting of high S (or low Agreeableness), high N, and low E scores that seems to be more or less exclusively related to the development of schizophrenia. For these factors, when considered more individually, have also been found to correlate with several measures for personality disorders other than schizotypal personality disorder, and even with measures for disorders normally not subsumed in the schizophrenic spectrum (Soldz, Budman, Demby and Merry, 1993; Trull, 1992). However, the fact that the aforementioned configuration of personality scores seems to be at least characteristic for schizotypal personality disorder lends support to our model too.

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APPENDIX

Selected 4DPT items for the MAXCOV–HITMAX analyses (the original Dutch items are given in italics)

S—Insensitivity

3. Are you a rather ambitious person (*Bent u een nogal eerzuchtig iemand*)?
15. Have you ever taken real advantage of someone (*Hebt u wel eens van iemand misbruik gemaakt*)?
19. Are you often quite obstinate (*Bent u vaak een echte stijfkop*)?
27. Do you deliberately react in such a manner as to keep other people at a distance (*Reageert u dikwijls opzettelijk zó, dat u andere mensen op een afstand kunt houden*)?
47. Do you often lie so convincingly that other people simply believe you (*Vertelt u een onwaarheid vaak zó goed, dat andere mensen er zonder meer in geloven*)?
55. Do you easily criticize other people's ideas (*Hebt u vaak snel kritiek op andermans ideeën*)?
63. Do you strongly tend to follow your own will without being too concerned about other people (*Bent u sterk geneigd uw eigen wil te volgen zonder u veel van andere mensen aan te trekken*)?

E—Extraversion

1. Are you a talkative person (*Bent u een spraakzaam iemand*)?
5. Can you easily get some life into a rather dull party (*Kunt u gemakkelijk een nogal vervelend feestje wat op gang brengen*)?
9. Do you easily make new friends (*Maakt u gemakkelijk nieuwe kennissen*)?
33. Are you capable of cheering people up quickly (*Bent u in staat andere mensen snel weer op te monteren*)?
37. Do you usually enjoy yourself very much at parties and the like (*Amuseert u zich vaak kostelijk op feestjes en dergelijke*)?
53. Do you think other people consider you very lively (*Vinden andere mensen u naar uw gevoel erg levendig*)?
61. Do you rather easily get enthusiastic about something (*Raakt u nogal gemakkelijk enthousiast voor iets*)?

N—Neuroticism

2. Do disappointments often tend to weigh so heavily that you are unable to get rid of them (*Neemt u uw teleurstellingen vaak zo zwaar op dat u ze niet van u af kunt zetten*)?
22. Do you rather frequently doubt yourself (*Twijfelt u nogal vaak aan uzelf*)?
30. Do you often feel highly agitated (*Voelt u zich dikwijls erg gejaagd*)?
34. Do you easily get upset when others criticize you (*Raakt u vaak snel van streek als anderen kritiek op u hebben*)?
38. Would you call yourself tense or highly strung (*Zou u zich gespannen of overgevoelig noemen*)?
50. Do you worry about awful things that might happen (*Maakt u zich zorgen over afschuwelijke dingen die zouden kunnen gebeuren*)?
54. Do you panic rather quickly (*Raakt u nogal snel in paniek*)?

G—Orderliness

8. Are you often a real perfectionist in your work (*Bent u in uw werk vaak een echte perfektionist*)?
16. Do you enjoy to plan your activities carefully beforehand (*Houdt u ervan om van tevoren uw aktiviteiten zorgvuldig te plannen*)?
24. Do you usually keep to rather firm habits (*Houdt u er doorgaans nogal vaste gewoonten op na*)?
28. Do you value tidiness and cleanliness very much (*Stelt u netheid en zindelijkheid erg op prijs*)?
32. Do you often do many things on the spur of the moment or on an off-chance (*Doet u veel dingen vaak zomaar of op goed geluk*)?
44. Do you always return the things you have borrowed on time (*Brengt u de dingen die u geleend hebt steeds op tijd terug*)?
60. Do you usually put away your belongings in exactly the same place (*Bergt u uw spullen meestal op precies dezelfde plaats op*)?

*Direction of scoring reversed.

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