The Genetic Epidemiology of Irrational Fears and Phobias in Men

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Background: Much of our knowledge of the role of genetic factors in the etiology of phobias comes from one population-based sample of female twins. We examined the sources of individual differences in the risks for phobias and their associated irrational fears in male twins.

Methods: In personal interviews with both members of 1198 male-male twin pairs (707 monozygotic [MZ] and 491 dizygotic [DZ]) ascertained from a population-based registry, we assessed the lifetime history of agoraphobia and social, animal, situational, and blood/injury phobias as well as their associated irrational fears. Twin resemblance was assessed by means of probandwise concordance, odds ratios, tetrachoric correlations, and univariate and multivariate biometrical model fitting.

Results: The suggestive results obtained by analysis of phobias only were supported by analyzing both fears and phobias. All 5 phobia subtypes aggregate within twin-pairs. This aggregation is due largely or solely to genetic factors with heritability of liabilities ranging from 25% to 37%. Multivariate analysis revealed a common genetic factor, genetic factors specific to each subtype, and a common familial-environmental factor.

Conclusions: In male subjects, genetic risk factors, which are partially common across all subtypes and partially subtype specific, play a moderate role in the etiology of phobias and their associated irrational fears. Family environment probably has an impact on risk for agoraphobia and social phobia. The genetic liability to blood/injury phobias is not distinct from those of the more typical phobias.

Arch Gen Psychiatry. 2001;58:257-265

Family studies suggest that phobias are familial,1-4 but cannot clarify the origin of this family resemblance. Twin studies of self-reported fears consistently have suggested a significant role for genetic factors.7-11 By contrast, twin studies of clinically defined phobias have, with one exception, suffered from very small samples sizes with resultant low power to discriminate alternative models of familial transmission.12-14 This exception is our previous study of phobias in female-female twins from the Virginia Twin Registry.15-17 These reports examined 5 types of phobias—agoraphobia (AgP), social phobia (SoP), animal phobia (AnP), situational phobia (SiP), and blood/injury phobia (BiP)—and reached the following conclusions. First, all phobia subtypes are moderately familial. Second, familial aggregation of AgP, SoP, and AnP is due to genetic factors. For SiP and BiP, results based on our first assessment15,16 suggested that twin resemblance might result from familial-environmental factors. However, when our analyses included a second assessment interview,17 the added power obtained through control of measurement error suggested that the results for these phobia subtypes were similar to those for the others (ie, the familial transmission due solely to genetic factors). Third, high levels of comorbidity were seen between the phobia subtypes.15 Contrary to results from family studies,4 the best-fitting multivariate model suggested common genetic and individual-specific environmental factors that influenced risk for all phobia subtypes, as well as genetic and environmental factors unique to each subtype. However, these analyses did not include BiP, which was assessed at a later wave. Blood/injury phobia differs from typical phobias,18 where exposure to phobic stimuli (eg, snakes, heights, and public speaking) usually produces increased sympathetic activity (eg, tachycardia, increased blood pressure, sweating, and flushing). By contrast, in individuals with BiP, exposure to phobic stimuli (eg, needles and blood) usually increases parasympathetic activity (eg, bradycardia, hypotension, pallor, and fainting). Given these differences, would the genetic and environmental risk factors for BiP be distinct from those for the other phobia subtypes?
SUBJECTS AND METHODS

SAMPLE AND ASSESSMENT PROCEDURES

This report is based on data from the second wave of interviews in our study of adult twins from the Virginia Twin Registry (now part of the Mid-Atlantic Twin Registry), details of which have been outlined previously. Briefly, twins were eligible for participation if one or both members were successfully matched and if they were white, a member of a multiple birth that included at least 1 male, and born between 1940 and 1974. Of 9417 eligible individuals for the first wave, 6814 (72.4%) completed initial interviews. For those who completed the initial interview, we recontacted them to schedule a second interview at least 1 year later. Where possible, this interview was completed face-to-face (79.4% of sample). Of the eligible individuals, 3629 (82.6%) were successfully interviewed. To assess test-retest reliability, 150 members of male-male twins were reinterviewed a mean (± SD) of 4.5±1.1 weeks after their initial interview.

The current report is based on 1198 male-male pairs (707 monozygotic [MZ] and 491 dizygotic [DZ]) with complete data on irrational fears and phobias from the second-wave interview and 544 interviewed individual twins (254 from MZ and 290 from DZ pairs) with complete data whose co-twins did not complete a second-wave interview.

At the second-wave interview (1994 through 1998), subjects were aged 20 to 58 years (mean±SD, 36.8±9.1 years). Interviewers had a master's degree in a mental health–related field or a bachelor's degree in this area plus 2 years of clinical experience. Members of a twin pair were interviewed by different interviewers unaware of clinical information about the co-twin. Zygosity diagnosis was performed using a discriminant function analysis based on 6 standard zygosity questions. The algorithm was developed on 227 twin pairs who underwent genotyping using 8 or more highly polymorphic DNA markers.

We assessed a lifetime history of phobias with an adaptation of the phobic disorders section of the Diagnostic Interview Schedule (DIS) Version III-A. The 22 specific individual fears that were assessed are outlined in Table 1. We also asked respondents “Is there anything else you’ve been unreasonably terrified to do or be near?” If any phobia described in response to this question best belonged with 1 of the 5 specific subtypes, it was so treated. Other phobias mentioned in response to this question (eg, fear of darkness) were included in our analysis of any phobia, but were not counted as belonging to a specific subtype.

In the DIS, to be considered a phobia, the fear must result in seeing a physician, taking medications, or reporting that the fear or its avoidance “interfered with life or activities a lot.” Given the low and variable rates of treatment seeking for phobias, we defined phobias solely through a modification of the third criterion, objective impact of the fear on respondent behavior. In contrast to the DIS, where the respondent makes the judgment about fear–associated interference, in our interview, the interviewer made this assessment.

STATISTICAL ANALYSIS

The univariate and multivariate twin models used in this report are described elsewhere. We assume that variation in liability to phobias results from the following 3 sets of factors: (1) additive genetic (A), which contribute twice as much to the correlation in MZ twins as DZ twins; (2) family or common environment (those familial factors such in the direction opposite that predicted. Controlling for zygosity, twin pairs with more similar childhood environments were significantly less likely to be concordant for AnP ($\chi^2 = 5.55; P = .02$) and SiP ($\chi^2 = 4.10; P = .04$). Those in frequent adult contact were significantly more likely to be concordant for SiP ($\chi^2 = 5.27; P = .02$).

Short-term test-retest reliability for irrational fears and phobias, as assessed by the coefficient and the tetrachoric correlation, were modest to moderate (Table 2). With the exception of social fears/SOP, irrational fears were more reliably reported than phobias. The reliability of the assignment of a phobia given an irrational fear was particularly modest. Consistent with our previous results, individuals may be more reliable at recalling irrational fears than they are at reporting their behavioral consequences.

DESCRIPTION BY INDIVIDUAL FEAR

Many more individuals endorsed an irrational fear than met criteria for an associated phobia (Table 1). However, this ratio differed widely. For example, although 46% of individuals who feared being in crowds met criteria for AgP, the parallel figures for fears of needles, giving a speech, and snakes were 18%, 15%, and 14%, respectively. Table 1 also presents, within each of the phobia subtypes, the proportion of individuals who met crite-

RESULTS

TEST FOR BIASES AND RELIABILITY

We performed 12 analyses predicting twin concordance for each phobia subtype and any phobias from the similarity of their childhood environment and the frequency of adult contact. Three were significant at the 5% level; 2,
as parental attitudes that are shared by members of a twin pair (C), which contribute equally to the correlation in MZ and DZ twins; and (3) individual specific environment (E), which reflect environmental experiences not shared by members of a twin pair and therefore contribute to differences between them in their phobia histories.

Our multivariate twin analyses decompose into genetic and environmental sources the variance in phobia liability and the covariance in liability among different phobia subtypes. We attempt, in these analyses, to explain the correlations between the phobia subtypes as resulting from a small number of latent factors. Multivariate genetic analysis goes beyond traditional factor analysis in providing insight into the causes of resemblance among variables.

Two alternative multivariate models that describe how genetic and environmental factors influence covariance are tested. Genetic and environmental factors can influence covariance through a single common pathway.23 By contrast, in the independent pathway model, genes and the environment contribute to covariance through separate genetic and environmental latent factors.

Having 5 phobia subtypes, two factors were potentially identifiable. However, we focused on single common factor models, because this was the approach used previously.15 The relationship between the common genetic factor and BiP was evaluated by setting the connecting path to 0 and examining the change in fit.

To use complete twin pairs and twins whose co-twin was not interviewed, we used an Mx option24 to fit models by maximum likelihood. During optimization, trial values of the parameters are used to generate a predicted covariance matrix and a matrix of thresholds. For any particular observation, the overall matrix is filtered to create a submatrix matching those observations that are present.

TWIN RESEMBLANCE

Twin resemblance was seen for all 5 of the phobia subtypes in MZ twins, with the odds ratios (ORs) ranging from 2.30 for SoP to 4.64 for AnP (Table 3). In DZ twins, twin resemblance was seen for AgP, SoP, and AnP, but not for SiP or BiP. For all phobia subtypes as well as for any phobia, the OR and tetrachoric correlations in MZ twins exceeded those seen in DZ twins, suggesting the importance of genetic risk factors.

Table 4 shows the results of model fitting for each of the phobia subtypes and for any phobia. For AgP and SoP, the full ACE model suggested that twin resemblance resulted from both genetic and familial-environmental factors. When we tested the simpler AE and CE models in these 2 phobias, they produced nearly identical fits. Table 4 presents results for the AE model. The CE model produced the following estimates for $c^2$ and $e^2$: $-0.29$ and $0.71$, respectively, for AgP, and $0.17$ and $0.83$, respectively, for SoP. The E-only model, by contrast, fit more poorly for both phobias. Although AgP and SoP aggregated within twin pairs, we had no power to distinguish the degree to which this was due to genetic and/or environmental mechanisms.

Likewise, the corresponding subset of the matrix of thresholds is created. These matrices are then used to compute the log-likelihood of the observation in question. The log-likelihoods of all cases are summed to obtain the log-likelihood of the sample, which is maximized using numerical optimization software. An advantage of this method is that it reduces the possible impact of cooperation bias by using information on potential differences in the prevalence of phobias in those twins with vs those without a co-twin participating to obtain a better estimate of true prevalences.

Because maximum likelihood analysis of raw ordinal data does not provide an overall test of goodness of fit, we compare relative fits against the full model. Twice the difference in log-likelihood between the full model and submodel tested yields a statistic that is asymptotically $\chi^2$, with degrees of freedom equal to the difference in the number of parameters in both models. Alternative models are evaluated for the optimal balance between explanatory power and parsimony, which is operationalized by Akaike's Information Criterion (AIC).25,26 Because unreasonable fears are more common than phobias, analysis including fears would have increased power.27 Such analysis would be appropriate if fears and phobias resulted from a single continuum of liability, a hypothesis testable by the multiple-threshold model.28 For our analyses, P indicates phobias only, and FP, fears and phobias.

We tested the equal environment assumption that exposure of MZ and DZ twin pairs to environmental risk factors for fears and phobias was equally correlated by predicting twin concordance for phobias, controlling for zygosity by means of the similarity of environmental experiences of the twins in childhood29 and adulthood.
We began by testing with the multiple threshold model the assumption that irrational fears and phobias represented differing points on a single continuum of liability. Although none of the results for the 5 phobia subtypes, examined separately in MZ and DZ twins, were statistically significant, the results for any phobia were significant in both twin groups (MZ, χ² = 80.0; P < .001; DZ, χ² = 67.7; P < .001). Therefore, we proceeded with model fitting using the trivariate classification of unaffected, fear only, and phobia for the 5 specific subtypes of fears and phobias only. The test-retest reliability for this tripartition is shown in Table 2.

The magnitude of the polychoric correlations for fears/phobias in MZ and DZ twins (Table 3) are generally similar to those seen with phobias only (although there are some exceptions, eg, AgP). However, as expected, the SEs of these correlations are substantially smaller than those seen with phobias alone.

The pattern of modeling results for fears and phobias (Table 5) differed from that found with phobias alone in 3 important ways. First, the full or ACE model was the best-fit model, with estimates of the heritability of liability ranging from 0.24 for SoFP to 0.43 for AgFP. Second, the statistical power with which we could reject alternative models was substantially greater. Using the χ² difference test, we could reject the CE model against the ACE model at or near the 5% level for the following 4 of the 5 fear/phobia subtypes: AgFP (χ² = 3.64; P = .06), AnFP (χ² = 11.87; P < .001), SiFP (χ² = 8.09; P = .004), and BiFP (χ² = 3.21; P = .07). For all fears/phobias, we could reject the E only (or no familial transmission model) with confidence, ie, AgFP (χ² = 21.49; P < .001), SoFP (χ² = 22.97; P < .001), AnFP (χ² = 42.17; P < .001), SiFP (χ² = 34.32; P < .001), and BiFP (χ² = 25.30; P < .001).

Third, the CIs around the parameter estimates were considerably narrower when we considered both fears and phobias in the model fitting compared with phobias alone.

**COMORBIDITY AMONG PHOBIA SUBTYPES AND MULTIVARIATE MODEL FITTING**

Substantial comorbidity exists among all combinations of the phobia subtypes (Table 6). As measured by OR or tetrachoric correlations, the highest comorbidity is seen between AgP and SoP, followed by SoP and BiP. The pattern of comorbidity of BiP is similar to that seen with more “typical” phobias.

Because of greater statistical power and stability of estimates, we performed multivariate model fitting with fears and phobias, the results of which are outlined in Table 7. Model 1 was the full independent pathway model with the A, C, and E common factors as well as the A, C, and E factors specific to each phobia subtype. When we tried to simplify this to a common pathway model, the fit deteriorated substantially, and the AIC increased. Therefore, in subsequent models, we set to 0, in turn, the C common factor (model 3), the A common factor (model 4), the E common factor (model 5), the phobia-specific C paths (model 6), and the phobia-specific A paths (model 7). The AIC improved only for model 6. Indeed, the fit of the model was unchanged when constraining to zero C paths unique to each phobia.

We then tested the relationship between the genetic risk factors for BiFP and the more typical phobias. Starting with model 6, we constrained to 0 the path from the common genetic factor to BiFP, thereby forcing all genetic risk for BiFP to be independent of the other phobia subtypes. This model fit substantially more poorly than model 6, with a deterioration of 6.5 χ² units (P < .01).

Parameter estimates for the best-fit model 6 are seen in the Figure and Table 8. Five results are noteworthy. First, contrary to the hypothesis that the genetic risk factors for BiFP are distinct from those for the more typical phobias, BiFP actually had the highest loading on the common genetic factor. Second, the best-fit model also contained genetic risk factors specific to each phobia subtype. These genetic-specific factors were most important for SoFP, AnFP, and SiFP. Third, unlike the univariate analysis for fears/phobias (but more like the univariate analyses for phobias suggested for all 5 phobia subtypes that the familial aggregation of phobia susceptibility was due solely to genetic factors. For each phobia/fear, AE was the best-fit model, with estimates of the heritability of liability ranging from 0.24 for SoFP to 0.43 for AgFP. Second, the statistical power with which we could reject alternative models was substantially greater. Using the χ² difference test, we could reject the CE model against the ACE model at or near the 5% level for the following 4 of the 5 fear/phobia subtypes: AgFP (χ² = 3.64; P = .06), AnFP (χ² = 11.87; P < .001), SiFP (χ² = 8.09; P = .004), and BiFP (χ² = 3.21; P = .07). For all fears/phobias, we could reject the E only (or no familial transmission model) with confidence, ie, AgFP (χ² = 21.49; P < .001), SoFP (χ² = 22.97; P < .001), AnFP (χ² = 42.17; P < .001), SiFP (χ² = 34.32; P < .001), and BiFP (χ² = 25.30; P < .001).

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alone), we found evidence of the impact of shared environment, but only in the form of a common factor. This common factor significantly affected only AgFP and SoFP. Fourth, the best-fit model contained one common E factor. That is, some environmental experiences that were unique to individual twins influence the general risk for fears/phobias. This factor had highest loadings on AgFP and SoFP. Finally, substantial specific E loadings were seen for all subtypes, which would represent an admixture of measurement error and environmental experiences that predisposed uniquely to individual phobia subtypes.

We addressed 3 questions about the genetic epidemiology of irrational fears and phobias in male-male twin pairs from a population-based registry. We examine these questions in turn.

Individual phobia subtypes and any phobia aggregated within twin pairs. For AnP, SiP, BiP, and any phobia, the best-fit model suggested that familial aggregation was due solely to genetic factors with modest heritabilities. For AgP and SoP, model fitting could not distinguish between genetic and familial-environmental sources of twin resemblance. For all the phobia subtypes, power was limited, and CIs for the parameter estimates were broad.

Many twins in our sample reported irrational fears without objective impact on their lives. Given our need for greater statistical power to resolve sources of individual differences and the low reliability with which individuals recalled whether the fears were impairing, we applied the multiple-threshold model to a trichotomous classification of all individuals into unaffected, fear only, and phobia. Results from these analyses indicated that for all the phobia subtypes, the pattern of results within MZ and DZ twins pairs were consistent with the
hypothesis that fears were a milder manifestation of the same liability dimension that produced clinical phobias. We found similar results in our female twins.17

We repeated our analysis using this trichotomous classification, with results that were substantially clearer than those obtained for phobias alone. With the increased statistical power, evidence of familial-environmental effects on AgP and SoP disappeared. The best-fit model for all the phobia subtypes suggested that familial resemblance was due solely to genetic factors, again with modest heritability estimates, but now with considerably smaller CIs.

### Table 4. Model-Fitting Results for Phobias in Male-Male Twin Pairs

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<tr>
<th>Phobia</th>
<th>Fit of Models†</th>
<th>Parameter Estimates</th>
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<td>Full</td>
<td>Best-Fit</td>
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<td></td>
<td>a²</td>
<td>c²</td>
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<tr>
<td>Agoraphobia‡</td>
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<td>AIC</td>
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<td>AIC</td>
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<td>AIC</td>
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<td>Animal‡</td>
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<td>Situational</td>
<td>χ²</td>
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<tr>
<td>Blood/injury</td>
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<td>Any</td>
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* A indicates additive genetic factors; C, family or common environment factors; E, individual specific environment factors; CI, confidence interval; and AIC, Akaike’s Information Criterion.25 Analyses were based on 1198 complete twin pairs and 544 single twins.
† Degrees of freedom were 2937 for ACE, 2938 for AE and CE, and 2939 for E.
‡ For these phobias, we present parameters from the AE model as the best fit, but the CE model also fit nearly as well, results of which are presented in the “Univariate Model Fitting to Phobias Only” subsection of the “Results” section.
§ Best-fit models by means of AIC.

### Table 5. Model-Fitting Results for Fears and Phobias in Male-Male Twin Pairs

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* Abbreviations and degrees of freedom for fit of models are given in the first and second footnotes to Table 4. Analyses were based on 1198 complete twin pairs and 544 single twins. Degrees of freedom for fit of models were as follows: ACE, 2936; AE, 2937; CE, 2937; and E, 2938.
† Best fit model(s) by means of AIC.25

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2 waves of interviews from which we can derive expected heritability from a single interview: for AgFP, 0.47; for SoFP, 0.31; for AnFP, 0.27; for SiFP, 0.26; and for BiFP, 0.32. These results, well within the 95% CIs obtained in the male sample, suggest that the role of genetic factors in liability to irrational fears and phobias are probably similar in men and women.

Similar to other community samples, substan-
tial comorbidity was seen between phobia subtypes. Multivariate twin modeling is a powerful method to examine the contributions of genetic and environmental factors to observed patterns of comorbidity. Given the higher test-retest reliability and greater power and stability of parameter estimates associated with adding of information on irrational fears, we included these along with phobias in our multivariate analyses. The best-fit model contained 3 common factors (reflecting genetic and shared and unique environments) that influenced liability to all forms of phobia. In addition, each phobic subtype had evidence of genetic and unique environmental factors specific to that phobia.

The pattern of results we obtained was similar to that in our previous multivariate analysis in women (which contained only 4 phobia subtypes) in 2 important ways. In both sexes, certain genetic factors influenced risk for all phobia subtypes, whereas others specifically influenced risk for individual phobic subtypes. In both sexes, the loadings of AnP on this common genetic factor were higher than those for the other typical phobias of AgP, SoP, and SiP. We also saw evidence that in men and women, certain individual environmental experiences increased risk nonspecifically for all phobic subtypes, whereas others were phobia-subtype specific in their impact. However, 2 differences are noteworthy. First, we found evidence in our multivariate analysis in men but not in women of shared environmental effects that had a significant impact on AgP and SoP. These results suggest that twin siblings shared some environmental experiences in their family or community that influence risk specifically for AgP and SoP. Second, the pattern of loadings for phobia-specific genetic factors differed between the sexes. For example, we found in women much larger contributions of phobia-specific genetic influences for AgP than AnP, whereas in men the findings were reversed. Further research will be required to determine if these

<table>
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<th>Phobia</th>
<th>Agoraphobia</th>
<th>Social Phobia</th>
<th>Animal Phobia</th>
<th>Situational Phobia</th>
<th>Blood/Injury Phobia</th>
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<td>Agoraphobia</td>
<td>12.78 (8.51-19.18)</td>
<td>5.37 (3.32-8.70)</td>
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<td>4.59 (3.01-7.01)</td>
<td>3.83 (2.68-5.46)</td>
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<td>3.81 (2.59-5.59)</td>
<td>5.20 (3.39-7.96)</td>
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<tr>
<td>Situational</td>
<td>0.33 (0.06)</td>
<td>0.36 (0.05)</td>
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<td>4.40 (3.07-6.31)</td>
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<tr>
<td>Blood/injury</td>
<td>0.29 (0.07)</td>
<td>0.45 (0.05)</td>
<td>0.41 (0.06)</td>
<td>0.39 (0.05)</td>
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* Data in lower-left triangle are given as tetrachoric correlations (asymptotic SE); in upper-right triangle, odds ratios (95% confidence intervals).
and other differences seen in both sexes are substantive in nature or due to stochastic fluctuations in patterns of phobia resemblances in twin pairs.

Our multivariate analyses also permitted us to evaluate the hypothesis that the genetic risk factors for BiP are distinct from those for the more typical phobia subtypes. Our results were inconsistent with this hypothesis. Indeed, BiP had the highest loading on the common genetic factor, and we could not set that path to 0 without a substantial deterioration in fit. The more typical phobias and BiP probably share common early fear pathways that then diverge in their outflow to the hypothalamus and autonomic pathways. These findings suggest that genetic risk factors for phobias in men act largely on the individual differences in the sensitivity of those early fear pathways shared by BiP and the more typical phobias.

These analyses should be considered in the context of 2 potential methodological limitations. First, our criteria for phobias differ from those proposed in recent DSM editions. For example, our interviewer-based assessment of impairment, although more objective, may produce a lower threshold for interference than the self-report measure used in the DIS. Our definition may also be broader than that proposed in DSM-III-R criteria in that we require objective impact on respondent behavior rather than the significant interference with normal routine of DSM-III-R. However, DSM-III-R would include an unreasonable fear that produced marked distress, whereas we did not. The Epidemiological Catchment Area screened for a more limited number of phobic diagnoses. These findings suggest that genetic risk factors for phobias in men act largely on the individual differences in the sensitivity of those early fear pathways shared by BiP and the more typical phobias.

Table 8. Sources of Variance in Liability to Individual Phobia Subtypes From Best-Fit Fear/Phobia Multivariate Model

<table>
<thead>
<tr>
<th>Fear/Phobia</th>
<th>Genetic</th>
<th></th>
<th></th>
<th>Shared Environment,</th>
<th></th>
<th></th>
<th>Unique Environment</th>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Common</td>
<td>Specific</td>
<td>Total</td>
<td>Common</td>
<td></td>
<td></td>
<td>Unique</td>
<td></td>
<td></td>
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<td>----------</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>0.11</td>
<td>0.02</td>
<td>0.13</td>
<td>0.23</td>
<td>0.25</td>
<td>0.39</td>
<td>0.64</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social</td>
<td>0.05</td>
<td>0.13</td>
<td>0.18</td>
<td>0.06</td>
<td>0.05</td>
<td>0.35</td>
<td>0.77</td>
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<td></td>
</tr>
<tr>
<td>Animal</td>
<td>0.21</td>
<td>0.15</td>
<td>0.36</td>
<td>0.01</td>
<td>0.12</td>
<td>0.57</td>
<td>0.69</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Situational</td>
<td>0.13</td>
<td>0.18</td>
<td>0.31</td>
<td>0.02</td>
<td>0.21</td>
<td>0.50</td>
<td>0.71</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood/injury</td>
<td>0.22</td>
<td>0.06</td>
<td>0.28</td>
<td>0.01</td>
<td>0.21</td>
<td>0.50</td>
<td>0.71</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Genetic proportion of reliable variance (0.28) would be considerably higher than those reported herein.

Accepted for publication October 10, 2000.

This work was supported by grants MH/AA-49492 and MH-54150 and Research Scientist awards MH-01277 (Dr Kendler) and MH-01458 (Dr Neale) from the National Institutes of Health, Bethesda, Md. We acknowledge the contribution of the Virginia Twin Registry, now part of the Mid-Atlantic Twin Registry, to ascertainment of subjects for this study. The Mid-Atlantic Twin Registry, directed by Linda Corey, PhD, and Lenn Murrelle, PhD, has received support from the National Institutes of Health, the Carman Trust (Richmond, Va), and the WM Keck (Los Angeles, Calif), John Templeton (Radnor, Pa), and Robert Wood Johnson (Princeton, NJ) Foundations.

These data were collected under the direction of Patsy Waring, Sarah Woltz, MA, and Frank Butera, MS.

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REFERENCES