A Multivariate Twin Study of Obsessive-Compulsive Symptom Dimensions

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Context: Obsessive-compulsive disorder (OCD) is clinically heterogeneous, but it is unclear whether this phenotypic heterogeneity reflects distinct, or partially distinct, etiologic mechanisms.

Objective: To clarify the structure of the genetic and environmental risk factors for the major symptom dimensions of OCD.

Design: Self-report questionnaires and multivariate twin model fitting.

Setting: General community.

Participants: A total of 4355 female members of the TwinsUK adult twin register.

Main Outcome Measures: Scores on the Obsessive-Compulsive Inventory–Revised and 5 of its subscales (checking, hoarding, obsessing, ordering, and washing).

Results: A common pathway model did not fit the data well, indicating that no single latent factor can explain the heterogeneity of OCD. The best-fit multivariate twin model was an independent pathway model, whereby both common and unique genetic and/or environmental factors contribute to the etiology of each symptom dimension. The hoarding dimension had the lowest loading on the common factor and was more influenced by specific genetic effects (54.5% specific). With the exception of hoarding, most of the genetic variance was due to shared genetic factors (ranging from 62.5% to 100%), whereas most of the nonshared environmental variance was due to dimension-specific factors.

Conclusions: Obsessive-compulsive disorder is unlikely to be an etiologically homogeneous condition. There is substantial etiologic overlap across the different OC symptom dimensions, but dimension-specific genetic, and particularly nonshared environmental, factors are at least as important. Hoarding shares the least amount of genetic liability with the remaining symptom dimensions. The results have implications for the current deliberations regarding OCD and the inclusion of a putative hoarding disorder in DSM-5.

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THE SYMPTOMS OF OBSESSIVE-COMPULSIVE DISORDER (OCD) are heterogeneous to the extent that 2 patients with this diagnosis can display different, nonoverlapping symptom profiles. At least 4 relatively independent symptom dimensions have been identified and widely replicated: (1) contamination/cleaning, (2) forbidden thoughts, (3) symmetry/order, and (4) hoarding. Despite this phenotypic heterogeneity, the current classification systems (DSM-IV and International Classification of Diseases, Tenth Revision) regard OCD as a unitary entity. It has been suggested that this parsimony may be oversimplistic and could obscure the findings from studies attempting to understand the psychological and biological mechanisms implicated in OCD. More important, considering OCD as a unitary entity may hamper progress in developing new treatment strategies for this chronic, disabling condition.

The phenotypic heterogeneity of OCD is unquestionable and some preliminary data support the validity of its major symptom dimensions; however, it is unclear whether the observed heterogeneity reflects distinct or partially distinct etiologic mechanisms. Indeed, it is possible that a single etiologic mechanism or set of mechanisms can explain all symptoms of OCD. If this were correct, adopting a multidimensional approach may sidetrack researchers from the important common factors that may be involved in all types of OCD. It is equally plausible that both common and unique etiologic mechanisms are implicated in the major symptom dimensions of OCD, as predicted by the multidimensional model. If this were cor-
rect, some etiologic mechanisms may be common to all patients with OCD, whereas other mechanisms may be unique to each symptom dimension. Although some indirect evidence from family studies supports this hypothesis, it remains to be fully tested.

Multivariate twin modeling methods are particularly well suited to test the assumptions of the multidimensional model. Specifically, these methods can be used to estimate the extent to which the covariation between different symptom dimensions is influenced by a single phenotypic latent factor (eg, OCD), which combines the contribution of common genetic and environmental factors, or whether each symptom dimension is better explained by genetic and environmental factors common to all symptom dimensions, as well as genetic and environmental factors specific to each. To date, only 2 small twin studies8,9 have been conducted and have provided preliminary data suggesting that both common and specific genetic and environmental factors may contribute to the etiology of different obsessive-compulsive (OC) symptom dimensions. However, these studies have several methodologic shortcomings that limit their interpretation, primarily the use of instruments that lack coverage of the full range of OC symptoms, making them unsuitable for clarifying issues pertaining to the heterogeneity of OCD.

The main aim of the present study was to explore the extent to which covariation between the major OC symptom dimensions is explained by genetic and environmental factors in a large sample of female monozygotic (MZ) and dizygotic (DZ) twins (N=4355). Large epidemiologic samples of unselected twins from the general population offer an excellent means to examine the relative contribution of genetic and environmental factors to behavioral traits.10 Based on the findings of previous family and twin studies (reviewed by van Grootheest et al11), we predicted that individual differences in the liability of broadly defined OCD would be influenced by genetic and nonshared environmental factors and that the influence of shared environmental factors would be negligible. Consistent with the multidimensional model of OCD, we predicted that there would be significant overlap or covariation between the different OC symptom dimensions and that these would be influenced by common as well as specific etiologic factors. Consistent with the current conceptualization of hoarding as a distinct dimension of OCD, or even a separate disorder,11,12 we predicted that there would be a greater degree of specificity for hoarding symptoms.

MEASURES

The OCI-R16 is a widely used 18-item self-report questionnaire designed to measure distress associated with OC symptoms. Each item is rated on a 5-point Likert scale ranging from 0 (not at all) to 4 (extremely), with a total scale score ranging from 0 to 72. The OCI-R consists of 3 subscales with scores ranging from 0 to 12. These subscales broadly correspond to the major symptom dimensions of OCD and are named: checking, hoarding, obsessing, order, neutralizing, and washing. The OCI-R total and its subscales have excellent psychometric properties, including test-retest reliability, and convergent/discriminant validity.26,28 Each of the OCI-R subscales correlates highly with the corresponding dimensions of the Dimensional Yale-Brown Obsessive Compulsive Scale,19 the criterion standard in symptom dimension assessment.20,21 A cutoff score of 21 on the OCI-R total scale discriminates between patients with vs those without OCD.26 Scores higher than 3 on the washing and neutralizing subscales; higher than 5 on the checking, ordering, and obsessing subscales; and higher than 7 on the ordering subscale discriminate patients with OCD who rate these symptoms as the most distressing from nonclinical controls, with sensitivity and specificity ranging from 0.83 to 1.00 and from 0.76 to 0.92, respectively.21 These cutoff scores were used to establish meaningful thresholds for model fitting (see the “Data Analyses” subsection). In the current study, an exploratory principal components analysis, followed by promax rotation (SPSS 16.0; SPSS, Inc, Chicago, Illinois) revealed a 6-factor structure, which was identical to that presented in the original article by Foa et al16 and explained 71% of the variance (cTable 1; http://www.archgenpsychiatry.com). It had good internal consistency for the total scale (Cronbach α=0.87) and all of its subscales (checking, 0.83; hoarding, 0.80; obsessing, 0.82; ordering, 0.86; and washing, 0.77). An exception was the neutralizing subscale, for which internal consistency was only modest (0.54). For this reason, and because this subscale does not correspond to any of the established symptom dimensions of OCD, it was not included in the multivariate genetic analyses. However, its inclusion did not modify the overall results (data available upon request).
Univariate maximum-likelihood model-fitting analyses of raw data were conducted to partition the variance of the OCI-R total score into additive genetic (A), shared (C), and nonshared environmental (E) factors using the structural equation modeling package Mx. As is standard procedure with analysis of raw data, the data were first fitted to a saturated model, and goodness of fit, in terms of a log-likelihood χ² statistic and Akaike information criterion, was obtained by comparing the −2 log-likelihood statistic of the saturated genetic model with that of the saturated model. Because the distribution of the OCI-R was positively skewed, polychoric correlations and genetic and environmental influences on OCD were estimated using liability threshold models. We used 3 thresholds, representing different categories of OC symptom severity: no symptoms (OCI-R,0), minimal symptoms (OCI-R,1-3), moderate symptoms (OCI-R,6-20), and severe symptoms or “caseness” (OCI-R,≥21). These provided a good representation of variability in symptom severity within our sample and ensured a sufficient number of cases within each category.

Multivariate Twin Analyses of OC Symptom Dimensions

Multivariate model-fitting analyses were conducted to decompose the covariances between the different OC symptom dimensions into A, C, and E factors. Three thresholds, representing the different categories of symptom severity, were specified for each of the OCI-R subscales and ranged from no symptoms (score, 0) to caseness (eg, score, ≥5 for the checking subscale; eTable 2).

To estimate MZ and DZ twin correlations within and across each of the 5 OC symptom dimensions, the data were fitted to a constrained correlational model in Mx. The MZ and DZ correlations for each of the symptom subscales (ie, the cross-twin within-subscale correlation) provided an indication of the relative contribution of genetic and environmental factors to the liability of each dimension (eg, checking). The ratio of MZ and DZ correlations across different symptom subscales (ie, the cross-twin cross-subscale correlation) provided an indication of the contribution of genetic and environmental factors to the shared liability of different symptom dimensions (eg, washing and checking).

To formally examine the genetic and environmental etiology underlying OC symptom dimensions, the data were fitted to 3 multivariate genetic models: a fully saturated triangular Cholesky decomposition, an independent pathway (IP) model, and a common pathway (CP) model. The Cholesky decomposition estimates 1 A, 1 C, and 1 E factor for each of the OC symptom dimensions and makes no assumption regarding the genetic and environmental architecture underlying their comorbidity. As such, this model provides a basis against which the more parsimonious IP and CP models can be compared.

The IP model (eFigure, top) estimates 1 set of common genetic factors (A_s), shared environmental factors (C_s), and nonshared (E_s) environmental factors, which influence the covariance between OC symptoms through direct (independent) paths to each of the 5 OC symptom dimensions. In this model, specific genetic factors (A_{s, j}), shared environmental factors (C_{s, j}), and nonshared (E_{s, j}) environmental factors are also estimated to account for the remaining symptom-specific variance. The CP model (eFigure, bottom) is nested within the IP model and provides the most parsimonious approach to the data. In this model, the covariation between the different OC symptom dimensions is influenced by a single phenotypic latent factor (ie, OCD), which combines the shared (ie, nonindependent) contribution of common A, C, and E factors. Similarly to the IP model, A, C, and E factors specific to each OC symptom dimension are estimated to account for symptom-specific variance.

To establish the best fit for the data, the Cholesky model was compared with the increasingly more parsimonious IP and CP models. Goodness of fit was established by both likelihood ratio tests and the Akaike information criterion. Alternative submodels were then tested by systematically dropping paths from the best-fitting model, and goodness of fit was established by the likelihood ratio test.

Given the large number of matrices estimated (10 × 10), implementing the maximum-likelihood raw data approach to the multivariate analyses was too computationally intensive. For this reason, polychoric correlation and asymptotic covariance matrices were computed (PRELIS 2.3) for all twin pairs with complete data points (3454 participants, consisting of 917 MZ twin pairs and 810 DZ twin pairs) and analyzed in Mx by the method of asymptotic-weighted least squares.

### RESULTS

#### UNIVARIATE TWIN ANALYSES OF THE BROAD OCD PHENOTYPE

Polychoric correlations (with 95% confidence intervals) for the OCI-R total scale were 0.47 (0.40-0.53) for MZ twins and 0.28 (0.19-0.36) for DZ twins (all P < .001), suggesting a model consistent with genetic variation and negligible shared environmental effects.

Results of the univariate model-fitting analyses are presented in Table 1. Liability thresholds could be equated across zygosity and birth order without any loss in fit (χ² = 6.7; P = .89), indicating that the prevalence of OC symp-
toms is similar for MZ and DZ twins, as well as for first- and second-born twins. Comparisons of the ACE model with the fully saturated model showed that the genetic model fit the data well. Dropping C did not result in a significant loss in fit. In contrast, dropping A resulted in a significantly worse fit to the data, indicating that additive genetic factors are important in explaining variability in this trait. Table 1 provides the standardized parameter estimates and 95% confidence intervals for the best-fitting model. Consistent with the pattern of correlations, genetic factors accounted for 48% of the variance of OC symptoms, with nonshared environmental factors and measurement error accounting for the remainder of the variance.

### MULTIVARIATE ANALYSES OF OC SYMPTOM DIMENSIONS

Phenotypic correlations between the OCI-R subscales were computed across all individual twins and are given in Table 2. The pattern of correlations indicates significant covariation across all combinations of symptom categories. The largest correlation is seen between the ordering and washing subscales \( r = 0.55 \), followed by the checking and washing subscales \( r = 0.53 \). The lowest correlation is seen between the hoarding and washing subscales \( r = 0.38 \).

Cross-twin within-subscale correlations for MZ and DZ twins are shown on the diagonal of Table 3. The pattern of correlations suggests meaningful genetic bases to the liability of each of the 5 symptom dimensions. Of note, twin correlations for the hoarding subscale \( 0.50 \) for MZ twins and \( 0.27 \) for DZ twins) are remarkably similar to those obtained with another measure of hoarding symptoms (0.52 for MZ twins and 0.27 for DZ twins) in a previous report.27

The cross-twin cross-subscale correlations (shown on the off-diagonal) are consistently lower for DZ twins than for MZ twins, suggesting that genetic factors are at least partly responsible for the overlap of each of the individual combinations of subscales (Table 3). The moderate MZ correlations across the different subscales \( r = 0.15-0.31 \) also indicate that common nonshared environmental factors are likely to be important in explaining correlations across different symptoms.

Results of the multivariate model-fitting analyses are shown in Table 4. The fully saturated Cholesky decomposition model yielded a nonsignificant \( \chi^2 \) value and a large and negative Akaike information criterion value, indicating a good fit to the data. The more parsimonious IP model (2) provided an excellent fit to the data, as indicated by a significant improvement in Akaike information criterion. However, the more restrictive CP model (3) produced a significantly worse fit. The IP model was therefore retained as the model of choice, and all subsequent models were compared with it.

In the next step, a series of nested submodels (4-10) were fitted to the data to test the importance of specific parameters. In these models, the genetic and environmental liabilities of each symptom dimension were either forced to be entirely independent (models 4-7) or entirely shared (models 8-10). In model 4, dropping the common genetic factor from the full IP model and forcing all genetic risk to be symptom specific produced a significantly worse fit to the data, suggesting that some of the genetic liability is shared among the different OC symptom dimensions. Similarly, forcing all environmental risk to be symp-

### Table 2. Phenotypic Correlations Between the 5 Obsessive-Compulsive Symptom Dimensions

<table>
<thead>
<tr>
<th>Symptom Dimension</th>
<th>Checking</th>
<th>Hoarding</th>
<th>Obsessing</th>
<th>Ordering</th>
<th>Washing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Checking</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hoarding</td>
<td>0.48</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obsessing</td>
<td>0.46</td>
<td>0.45</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ordering</td>
<td>0.49</td>
<td>0.45</td>
<td>0.43</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Washing</td>
<td>0.53</td>
<td>0.38</td>
<td>0.42</td>
<td>0.55</td>
<td>1.00</td>
</tr>
</tbody>
</table>

*All correlations were significant at \( P < .001 \)."

### Table 3. Cross-Twin Within-Subscale (Diagonal) and Cross-Twin Cross-Subscale (Off-Diagonal) Correlations for MZ and DZ Female Twins

<table>
<thead>
<tr>
<th>Symptom Dimension</th>
<th>Checking</th>
<th>Hoarding</th>
<th>Obsessing</th>
<th>Ordering</th>
<th>Washing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Checking</td>
<td>0.38 (MZ)</td>
<td>0.22</td>
<td>0.29</td>
<td>0.26</td>
<td>0.28</td>
</tr>
<tr>
<td>Hoarding</td>
<td>0.10</td>
<td></td>
<td>0.29</td>
<td>0.21</td>
<td>0.15</td>
</tr>
<tr>
<td>Obsessing</td>
<td>0.09</td>
<td>0.13</td>
<td>0.49 (MZ)</td>
<td>0.29 (DZ)</td>
<td>0.26</td>
</tr>
<tr>
<td>Ordering</td>
<td>0.16</td>
<td>0.15</td>
<td>0.10</td>
<td>0.42 (MZ)</td>
<td>0.31</td>
</tr>
<tr>
<td>Washing</td>
<td>0.17</td>
<td>0.09</td>
<td>0.02</td>
<td>0.15</td>
<td>0.51 (MZ)</td>
</tr>
</tbody>
</table>

Abbreviations: DZ, dizygotic; MZ, monozygotic.

*Cross-twin cross-subscale correlations for MZ are given in the shaded area above the diagonal and for DZ twins in the clear area below the diagonal."

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symptom specific, by dropping the shared and nonshared common environmental factors in models 5 and 6, led to a significant deterioration in fit, suggesting that some of the environmental liability is also shared. Fitting a model that assumes that all the genetic risk is shared and that there are no symptom-specific genetic influences (8) also provided a significantly worse fit to the data, suggesting that the different OC symptom dimensions are not exclusively influenced by the same genetic factors. A poor fit was also obtained by constraining all the nonshared environmental influence (and measurement error) to be the same across all OC symptom dimensions (10), suggesting that, although some of the nonshared environmental risk factors are shared, some are also unique. Dropping symptom-specific shared environmental factors from the model did not contribute to a worsening in fit (9), suggesting that these factors are not important in explaining individual differences in liability to the different OC symptom dimensions (Table 4).

The best-fitting model to the data, therefore, was model 9, in which the covariation between the 5 OC symptom dimensions is explained by a set of common A, C, and E factors, and the remainder of the variance is explained by independent A and E factors specific to each dimension. Parameter estimates for each subscale for the best-fitting model are presented in Table 5 and Figure 1. Consistent with the pattern of correlations, genetic factors accounted for 38% to 44% of the variance of the individual OCI-R subscales, with nonshared environmental accounting for the remainder of the variance.

Although the common shared environment factor could not be dropped from the model without deterioration in fit, this factor accounted for a negligible percentage of the overall variance (0.05%-11%) and contributed significantly only to the hoarding, obsessing, and washing subscales. That is, fixing the path from the common C factor to 0 for the checking and ordering dimensions did not worsen the fit of the model. Genetic influences that are specific to washing symptoms could also be dropped from the model without a worsening in fit, suggesting that genetic influence on this OC symptom dimension is entirely shared (asymptotic-weighted least squares $\chi^2 = 76.45$, $P = .80$; Akaike information criterion $= -99.53$, compared with model 9: $\Delta \chi^2 = 3.11$; $P = .37$; Figure 1).

Excluding the hoarding dimension from the analyses did not change the pattern of results, with the IP model still fitting the data best (data available upon request).

The extent to which genetic and environmental influence on each of the 5 symptom dimensions is shared (vs unshared) with other OC symptom dimensions can be cal-

### Table 4. Model-Fitting Results for the Cholesky, IP, and CP Models

<table>
<thead>
<tr>
<th>Model</th>
<th>Additive Genetic Factors</th>
<th>Shared Environmental Factors</th>
<th>Common Specific Total</th>
<th>Comparison of Nested Models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Common Factors</td>
<td>Specific Factors</td>
<td>AWLS $\chi^2$</td>
<td>$df$</td>
</tr>
<tr>
<td>1 Cholesky, saturated</td>
<td>ACE</td>
<td>ACE</td>
<td>57.39</td>
<td>70</td>
</tr>
<tr>
<td>2 IP</td>
<td>ACE</td>
<td>ACE</td>
<td>70.37</td>
<td>80</td>
</tr>
<tr>
<td>3 CP</td>
<td>ACE</td>
<td>ACE</td>
<td>106.31</td>
<td>88</td>
</tr>
<tr>
<td>4 IP</td>
<td>CE</td>
<td>ACE</td>
<td>124.74</td>
<td>85</td>
</tr>
<tr>
<td>5 IP</td>
<td>AE</td>
<td>ACE</td>
<td>99.96</td>
<td>85</td>
</tr>
<tr>
<td>6 IP</td>
<td>AC</td>
<td>ACE</td>
<td>215.38</td>
<td>85</td>
</tr>
<tr>
<td>7 IP</td>
<td>E</td>
<td>ACE</td>
<td>309.71</td>
<td>85</td>
</tr>
</tbody>
</table>

### Table 5. Parameter Estimates for the Best-Fitting Independent Pathway Model

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Common</th>
<th>Specific</th>
<th>Total</th>
<th>Common</th>
<th>Specific</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Checking</td>
<td>0.27 (0.18-0.34)</td>
<td>0.11 (0.05-0.16)</td>
<td>0.38 (0.29-0.45)</td>
<td>0.0006b (0-0.06)</td>
<td>0.27 (0.20-0.35)</td>
<td>0.35 (0.27-0.43)</td>
</tr>
<tr>
<td>Hoarding</td>
<td>0.20 (0.08-0.31)</td>
<td>0.24 (0.15-0.30)</td>
<td>0.44 (0.29-0.54)</td>
<td>0.05 (0-0.17)</td>
<td>0.24 (0.17-0.31)</td>
<td>0.27 (0.19-0.34)</td>
</tr>
<tr>
<td>Obsessing</td>
<td>0.29 (0.15-0.47)</td>
<td>0.18 (0-0.28)</td>
<td>0.47 (0.32-0.57)</td>
<td>0.05 (0-0.17)</td>
<td>0.14 (0.08-0.19)</td>
<td>0.34 (0.27-0.42)</td>
</tr>
<tr>
<td>Ordering</td>
<td>0.29 (0.19-0.37)</td>
<td>0.15 (0.08-0.21)</td>
<td>0.44 (0.32-0.51)</td>
<td>0.0005b (0-0.09)</td>
<td>0.21 (0.15-0.27)</td>
<td>0.35 (0.27-0.42)</td>
</tr>
<tr>
<td>Washing</td>
<td>0.35 (0.09-0.54)</td>
<td>0.04b (0-0.21)</td>
<td>0.39 (0.13-0.55)</td>
<td>0.11 (0.003-0.33)</td>
<td>0.25 (0.15-0.36)</td>
<td>0.25 (0.10-0.35)</td>
</tr>
</tbody>
</table>

**Abbreviations:** A, additive genetic factor; AIC, Akaike information criterion; AWLS, asymptotic-weighted least squares; C, shared environmental factor; CP, common pathway; E, nonshared environmental factor; IP, independent pathway; OC, obsessive-compulsive.

**a**Best-fitting model (in boldface type) based on AIC.

**b**Parameters can be dropped without any loss in fit.
A consistent body of research indicates that OCD is a highly heterogeneous condition. A key question in our field is whether this phenotypic heterogeneity also reflects etiologic heterogeneity. The present study tested one of the key assumptions of the multidimensional model of OCD, namely, that both common and specific etiologic factors are implicated in each of its major symptom dimensions.

GENETIC AND ENVIRONMENTAL INFLUENCES ON THE BROAD OCD PHENOTYPE

Univariate model-fitting analyses showed that the best-fitting model for the data comprised additive genetic and nonshared environmental factors. Under this model, additive genetic factors accounted for approximately 50% of the variance of OC symptoms, with nonshared environmental and measurement error accounting for the remainder. Our estimate of heritability is comparable to that obtained in other samples of adult twins (i.e., 26%-55%) and somewhat lower than that obtained in samples of child and adolescent twins (i.e., 47%-61%). In line with previous research, environmental factors shared by siblings growing up in the same family did not influence individual variation in the liability to OCD, further suggesting that these factors may be less important in the etiology of the disorder.

GENETIC AND ENVIRONMENTAL INFLUENCES ON THE OC SYMPTOM DIMENSIONS

As expected, there was significant phenotypic overlap across the different OC symptom dimensions. We used multivariate genetic analyses to explore the extent to which this overlap was influenced by genetic and environmental factors. Specific hypotheses about the genetic and environmental structure of OC symptoms were explored by fitting the data to a CP model and an IP model. The CP model, which assumes that the genetic and environmental causes of all OC symptom dimensions have the same underlying structure, provided a poor fit to the data. This suggests that, although the different dimensions are etiologically related to some extent, there is no underlying unitary OCD construct that can account for their covariation. Instead, we found that an IP model, which allows genetic and environmental factors to influence each dimension directly, best described our data. This was true even after the hoarding dimension was excluded from the analyses. The best-fitting model comprised additive ge-
nentic and shared and nonshared environmental factors common to all OC symptom dimensions, and genetic and unique environmental factors specific to each symptom dimension. Covariation across the different dimensions was largely explained by genetic and nonshared environmental factors; although significant shared environmental effects were found, these tended to be negligible.

Additive genetic factors made a significant contribution to all the OC symptom dimensions. Heritability estimates ranged from 38% to 47% and, although broadly similar across dimension, these were somewhat greater for the obsessing (47%), hoarding (44%), and ordering (44%) subscales. These findings are consistent with those of previous research, showing that different symptom dimensions, in particular hoarding and obsessions/checking, tend to cluster in families. They further suggest that the pattern of familiality observed in the previous studies is partly due to genetic factors.

Although we found evidence for overlap, genetic and environmental factors specific to each dimension were at least as important. The relative contribution of common vs specific genetic factors varied across dimension. In particular, we found that some dimensions were either completely (washing) or largely (ordering, checking, obsessing) influenced by common genetic factors. In contrast, hoarding had the lowest loading on the common factor and was mostly influenced by specific genetic effects (54.5% specific). If this finding were to be replicated, hoarding and was mostly influenced by specific genetic effects (54.5% specific). That study was limited by the use of an instrument that lacks coverage of all the full range of OC symptoms, and further research is needed to replicate our findings.

We hope that our results will encourage further research into specific biological and environmental mechanisms underlying specific OC symptom dimensions. For example, studies of MZ twins discordant for specific symptom dimensions could be used to explore dimension-specific nonshared environmental risk/protective factors.

LIMITATIONS

The current study has some limitations. First, our sample was 90% female and there was insufficient power to test quantitative and qualitative sex differences in the genetic and environmental risk factors to the broad OCD phenotype and its symptom dimensions. Second, because OC symptoms were measured at a single time point, it was not possible to estimate measurement error and adjust heritability and nonshared environment estimates accordingly. Studies clarifying the extent to which “pure” nonshared environmental factors contribute to these traits are needed. Third, OC symptoms were measured by self-report and were not validated by clinical interview. However, the OCI-R subscales are valid measures of OCD symptom subtypes in both clinical and nonclinical populations and correlate highly with clinician-administered measures.

Nonshared environmental factors and measurement error were also important in explaining the liability of the different OC symptom dimensions. The contribution of these factors ranged from 48% to 62% and was somewhat higher for the checking subscale. The relative contribution of common vs specific environmental factors suggested that a greater proportion of the variance was attributable to specific environmental factors to each dimension, ranging from 52% to 71%. This raises the intriguing possibility that hoarding aside a set of (largely) common genetic factors may predispose individuals to develop broadly defined OCD, but (largely) specific environmental risk factors may shape the specific symptomatic expression of the disorder. However, because of the cross-sectional design of this study, it was not possible to measure the extent to which nonshared environmental influences reflect the effect of instrumentation.
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Online-Only Material: The eTables and eFigure are available at http://www.archgenpsychiatry.com.

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