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Obsessive Compulsive Symptom Dimensions and Neuroticism: An Examination of Shared Genetic and Environmental Risk

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Individuals with obsessive compulsive disorder can display diverse and heterogeneous patterns of symptoms. Little is known about the relationship between obsessive-compulsive symptom (OCS) dimensions and normal personality traits, particularly those that increase risk for other internalizing disorders. In this study of 1,382 individuals from female–female twin pairs, we examined the relationship between self-report OCS dimensions derived from the Padua Inventory and Eysenck's personality traits neuroticism and extraversion. We conducted factor analysis to determine their phenotypic structure followed by twin analyses to determine their genetic and environmental sources of covariation. A three-factor solution, with dimensions corresponding to checking, aggressive obsessions, and contamination, was the best fit for the Padua OCS items. These dimensions were significantly and somewhat variably associated with neuroticism but negligibly associated with extraversion. The genetic correlations between neuroticism and these three OCS dimensions were moderate to high (0.66 with checking, 0.89 with aggressive obsessions, and 0.40 with contamination). However, the estimated genetic correlation between neuroticism and a unified latent OCS construct was smaller (0.32). Overall this study suggests that genetic, and to a smaller extent environmental, factors underlying neuroticism may act differentially as risk factors for OCS dimensions.

Key words: obsessive compulsive disorder; personality; twin study; factor analysis

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INTRODUCTION

Obsessive compulsive disorder (OCD), with a lifetime prevalence of 1–2% [Karno et al., 1988; Weissman et al., 1994], is as common a psychiatric condition as schizophrenia. Like many psychiatric disorders, while OCD is moderately heritable [Hettema et al., 2001; van Grootheest et al., 2005], gene-finding efforts have been fraught with difficulties [Pauls et al., 2014]. One particular complexity is that obsessive compulsive symptoms (OCS) are quite diverse and heterogeneous, with one patient's symptom profile looking very different from the next. This has potentially important implications for genetic studies of OCD [Miguel et al., 2005].

Previous research has explored the potential dimensional structure of OCS in order to understand and refine its phenotypic characteristics for clinical and genetic studies. In factor analytic studies using the 13 categories from the Yale-Brown Obsessive-Compulsive Symptom Checklist (YBOCS SC), four replicable factors emerge: (Factor I) Forbidden thoughts–aggression, sexual, religious, and somatic obsessions and checking compulsions; (Factor II) Symmetry–symmetry obsessions and repeating, ordering, and counting compulsions; (Factor III) Cleaning–cleaning and contamination; and (Factor IV) Hoarding–hoarding obsessions and compulsions [Bloch et al., 2008; Hasler et al., 2007]. In an item-

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level factor analysis of all YBOCS SC items, five factors emerged: Taboo thoughts, Symmetry/Ordering, Hoarding, Contamination/Cleaning, and Doubt/Checking [Pinto et al., 2008].

Several twin studies have examined the risk structure of OCS dimensions. In our prior study of female twins, we reported that three dimensions ("ruminations", "checking", and "contamination") adequately accounted for associations among OCS items from the Padua Inventory. These symptom dimensions were found to covary due to shared genetic and environmental influences via a common latent obsessive-compulsive phenotype [van Grootheest et al., 2005]. A study of 517 adolescent male and female twins reported differential heritability by sex and shared genetic risk for the three dimensions they analyzed [Moore et al., 2010]. Iervolino and colleagues conducted a female-only twin study in which genetic risk for five subscales of the Obsessive–Compulsive Inventory-Revised covaried due to a shared genetic factor [Iervolino et al., 2011].

Along with the different identifiable dimensions that characterize OCS, normal personality traits may also reflect individual differences in patients with OCD. Individuals meeting full OCD diagnostic criteria [Bienvenu et al., 2004] as well as those will subclinical OCD [Fullana et al., 2004] tend to score higher on measures of neuroticism than healthy controls. A large family study reported higher neuroticism scores and a higher prevalence of obsessive-compulsive personality disorder in relatives of individuals with OCD compared to control relatives [Samuels et al., 2000]. In a pediatric twin study, OCS shared underlying genetic factors with neuroticism (genetic correlation $r_g = 0.44$) and extraversion ($r_g = -0.17$) [Hur, 2009]. A study of adult twins reported that two measures related to neuroticism were genetically correlated with six OCS scales [Taylor et al., 2011]. These findings suggest that risk factors underlying personality traits and OCS or OCD co-aggregate in families, in part due to overlapping genetic mechanisms. This is important to understand in greater depth, given that neuroticism differentially increases risk across anxiety and depressive disorders due to shared genetic and environmental factors [Hettema et al., 2006].

Due to the limited knowledge about the sources of shared risk between normal personality and individual OCS dimensions, we expanded our prior twin analysis to examine this question. The aims of the current study were to jointly analyze OCS dimensions and personality traits using both phenotypic and twin structural models to investigate and estimate the extent of overlap of genetic, common environmental, and unique environmental factors between neuroticism and each OCD symptom dimension.

MATERIALS AND METHODS

Participants

Participants in this study were from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders, which has been described previously [Kendler and Prescott, 1999]. Only twins from the female–female pairs were assessed for OCS symptomatology and included in the current analyses. These analyses used personality data collected at the Wave 1 assessment (mean age = 29.3 ± 7.7 years) and OCS data collected at the Wave 4 assessment (mean age = 36.3 ± 8.2 years) conducted approximately 7 years later. Wave 1 included 2,270 individuals from female–female twin pairs and Wave 4 included 1,942 of these. Zygosity was determined by a combination of standard questions [Eaves et al., 1989], photographs, and DNA analysis [Kendler and Prescott, 1999]. Approval of the local Institutional Review Board was obtained prior to the study and informed consent was obtained from all subjects prior to data collection.

Measures

Neuroticism and extraversion were assessed with 12 and 8 dichotomously scored items (yes = 1, no = 0), respectively, from the shortened form of the Eysenck Personality Questionnaire-Revised [Eysenck and Eysenck, 1975]. Total scores for each were obtained by summation (ranges 0–12, 0–8, respectively).

OCS were assessed using a self-report questionnaire made up of 21 items from the Padua Inventory [Sanavio, 1988]. The mailed questionnaire was completed by 1,382 twins of the Wave 4 sample (331 MZ and 193 DZ pairs plus 334 twins without co-twin data). Each twin was asked to respond either "yes" or "no" to each of the 21 items listed in Table I according to "Are you the type of person…?". As described previously [Jonnal et al., 2000], these items were selected based upon their loadings on four primary factors derived from the full instrument.

Statistical Analyses

Item-level analyses were carried out in all twin subjects using exploratory factor analysis (EFA) and exploratory structural equation modeling (ESEM). All models were fitted using Mplus version 6.0 [Muthen and Muthen, 2010]. The weighted least squares mean- and variance-adjusted robust estimator was used for optimization, and non-independence of the data due to the inclusion of cotwins was handled using the Mplus COMPLEX option in which a sandwich estimator is implemented to adjust standard errors and fit indices. For the EFA applied to the OCS data, Items 1 and 21 were combined to form a composite variable for use in the analyses, due to their strong content overlap. One to four factors were extracted from the EFA modeling based upon prior results in our data.

ESEM methodology [Marsh et al., 2010] was used to expand the analyses of the OCS data to simultaneously include information from the neuroticism and extraversion scales. ESEM combines both EFA and confirmatory factor analysis (CFA) approaches to allow structural relationships to be estimated between the two. A model was fitted that estimated the factor structure of the OCS items as an EFA block while simultaneously including single-factor CFA models for the 12 neuroticism and eight extraversion items. Specifically, the OCS dimensional factors obtained from the EFA were regressed onto the neuroticism and extraversion factors with the inclusion of age and age^2 as covariates.

Classical twin modeling [Neale and Cardon, 1992] was conducted using OpenMx version 1.3.1 [Boker et al., 2011] to decompose the sources of individual differences for the outcome phenotypes. Additive genetic factors [(A), correlated 100% between monozygotic (MZ) cotwins and, on average, 50% between

TABLE I. 21 Padua Inventory Items Used to Assess Obsessive Compulsive Symptoms (OCS)

Are you the type of person who?
1. nearly always thinks about all the facts in detail before you make a decision, even when other people demand a quick decision?
2. tends to keep on checking things more often than necessary?
3. after doing something carefully, still has the impression it is either done badly or is not finished?
4. has to wash their hands more often and longer than necessary?
5. finds it difficult to touch garbage or dirty things?
6. sometimes feels a need to break or damage things for no reason?
7. has unpleasant thoughts that come into your mind against your will, and which you cannot get rid of?
8. finds it difficult to touch an object which has been touched by strangers or certain people?
9. has to do things several times before thinking they are done properly?
10. invents doubts and problems about most of the things you do?
11. sometimes has to wash or clean yourself because you think you may be dirty or "contaminated"?
12. has to return home to check doors, windows, drawers etc., to make sure they are properly shut?
13. when looking down from a bridge or very high window, feels an impulse to throw yourself into space?
14. imagines that catastrophic consequences may result from absent-mindedness or minor errors you have made?
15. likes to think about things for a long time before you make a decision?
16. avoids using public toilets because of fear of disease and contamination?
17. has to keep on checking forms, documents, checks etc. in detail, to make sure they have been filled out correctly?
18. when a train approaches, sometimes thinks of throwing yourself under the wheels?
19. checks and rechecks gas burners, water faucets, and light switches after turning them off?
20. 'while driving' sometimes feels an impulse to drive the car into someone or something?
21. usually thinks about all the facts in detail before you make a decision?

dizygotic (DZ) cotwins], common environmental factors [(C), correlated 100% within pairs regardless of zygosity], and unique environmental factors [(E), not shared within a pair] were estimated for each OCS dimension and the personality scales. The E variance component also includes occasion-specific measurement error. For all analyses, raw data from all individual twins, including those without an interviewed cotwin, were used, and models were fitted using normal theory maximum likelihood estimation.

Like phenotypic factor analysis, multivariate genetic analysis seeks to account for covariation among multiple variables; however multivariate genetic analysis further decomposes the covariance into genetic and environmental sources. For the OCS dimensional phenotypes, first we applied the Cholesky decomposition, a saturated model in which all latent sources of A, C, and E variances and covariances are estimated. This step estimates the genetic and environmental correlations between the OCS dimensions and neuroticism and extraversion, respectively.

The more hypothesis-driven common pathway (CPM) and independent pathway (IPM) models were also estimated and compared to the Cholesky decomposition to determine the best-fitting covariance structure for the OCS dimensions. In the CPM, shared A, C, and, E are assumed to generate variation in, and covariation between, the outcome phenotypes in a coordinated fashion through a common latent phenotypic factor. Factor loadings estimate the magnitude to which each phenotype is related to the common factor. In addition, residual (specific) A, C, and E factors are also estimated that influence each specific phenotype independent of the common factor. In the IPM, shared A, C and E factors directly influence all variables (plus variable-specific A, C, and E factors). Models were compared using a bootstrapped likelihood ratio test.

RESULTS

Omnibus fit indices for the four-factor EFA solution for the OCS items were very good (CFI = 0.99, TLI = 0.99, RMSEA = 0.01), but this solution revealed that the new composite item (constructed from items 1 and 21) and item 15 formed a separate factor that was poorly identified in the sample. As a result, these items were not retained, and the resulting Geomin-rotated three-factor solution (CFI = 0.98, TLI = 0.96, RMSEA = 0.03)] was selected as the best fitting model. Factor loadings for this three-factor solution (dimensions reflecting "doubt/checking", "contamination/cleaning", and "aggressive obsessions") are listed in Table II.

Most items loaded predominantly on only one of the three factors, conforming to a simple structure organization. Four exceptions were noted (items 7, 10, 12, and 14) that showed additional complexity as evidenced by sizable cross-loadings (>0.3). In addition, inter-factor correlations were relatively small except for the correlation between "checking" and "contamination" (r = 0.413).

ESEM analyses provided further support for the results obtained by EFA. The three-factor structure identified in the EFA was retained when the 18 OCS items were estimated and rotated in the presence of the neuroticism and extraversion item factor blocks while simultaneously regressing the OCS factors on the neuroticism and extraversion factors. Factor loadings showed some attenuation compared with those from the EFA, but the overall factor loading pattern was similar. Neuroticism had significant ($p < 0.005$), posi-

TABLE II. Three Factor Geomin Rotated EFA Factor Loadings for Final 18 Obsessive Compulsive Symptoms (OCS) Included in Analyses

Item	F1 Checking	F2 Contamination	F3 Aggression
2	0.695	0.047	0.000
3	0.717	−0.007	0.290
9	0.767	0.065	0.196
10	0.496	−0.098	0.504
12	0.604	0.342	−0.028
14	0.600	−0.001	0.368
17	0.692	0.187	0.026
19	0.688	0.282	−0.030
4	0.055	0.725	−0.015
5	−0.054	0.693	0.085
8	−0.005	0.779	−0.065
11	0.188	0.640	0.126
16	0.135	0.658	0.013
6	0.186	0.122	0.510
7	0.443	−0.011	0.477
13	0.045	0.000	0.670
18	−0.015	0.081	0.887
20	−0.029	0.034	0.843

Inter-Factor Correlations	F1	F2	F3
F1	1.00		
F2	0.413	1.00	
F3	0.087	0.127	1.00

tive regression coefficients with all OCS dimensions (checking: 0.489, contamination: 0.222, aggression: 0.435) whereas extraversion was not significantly associated with any of these.

Because the ESEM indicated that only neuroticism was phenotypically related to the three OCS dimensions, extraversion was excluded from the subsequent twin analyses.[1] Within- and cross-trait twin correlations are displayed in Table III separately by zygosity (MZ above the diagonal, DZ below). MZ correlations are typically a bit less than twice the DZ correlations, suggesting that a combination of A and C accounts for twin resemblance. Non-zero cross-twin, cross-trait correlations indicate shared familiar effects between neuroticism and OCS.

Model comparison statistics for the Cholesky, IPM and CPM analyses are provided in Table IV. While the Cholesky decomposition fits significantly better than the IPM or CPM, the more restrictive CPM does not fit worse than the IPM. Therefore, according to the principle of parsimony, a single latent common OCS factor accounts for the covariance between the OCS dimensions better than allowing separate genetic and environmental factors. The atheoretical Cholesky decomposition, however, fits significantly better than the CPM, suggesting that there is more covariation between the dimensions of OCS than accounted for by the CPM and IPM.

From the Cholesky decomposition, the estimated heritability for neuroticism was 0.39, aggressive obsessions 0.27, checking 0.40, and contamination 0.41. The results of the Cholesky analysis are displayed in Table V in terms of genetic and environmental correlations between measures. As can be seen in the top panel of Table V, there are sizable and statistically significant genetic correlations among the OCS dimensions and between the dimensions and neuroticism. The genetic correlations between neuroticism and aggressive obsessions and checking were high, 0.89 and 0.66, respectively. A moderate genetic correlation of 0.30 was estimated between neuroticism and contamination, consistent with the phenotypic correlation. The common environmental correlations between the dimensions are not statistically significant and can be ignored. Finally, the unique environmental correlations are weaker in magnitude than the genetic correlations, but the overall pattern is generally consistent with the genetic correlations.

[1]The twin analyses from the extraversion-neuroticism models confirmed that there are no interesting genetic or environmental correlations.

DISCUSSION

In this study, we examined the phenotypic and genetic vs. environmental correlations (i) among three OCS dimensions derived from items from a shortened version of the Padua Inventory and (ii) between these and Eysenck's neuroticism and extraversion scales. Analyses were conducted in data from 1,937 twins from female–female pairs using phenotypic EFA, ESEM, and biometric twin models. Three interpretable factors were found reflecting aggressive obsessions, doubt/checking, and contamination/washing dimensions. These dimensions were substantially correlated with neuroticism but not with extraversion.

To further explore the relationship between OCS and neuroticism, we fit a Cholesky decomposition, Independent pathway model (IPM), and Common Pathway Model (CPM). The Cholesky decomposition allowed us to separately estimate the correlations between neuroticism and each OCS dimension. The genetic correlations between neuroticism and these three OCS dimensions were moderate to high (0.66 with checking, 0.89 with aggressive obsessions, 0.40 with contamination). As an overall model of OCS, the more parsimonious CPM provided a better global fit to the data than the IPM although not as good as the Cholesky decomposition. However, the estimated genetic correlation between neuroticism and the unified latent OCS construct in the CPM was much smaller (0.32) than those for neuroticism and the individual dimensions. i.e., the CPM only accounted for a fraction of the covariance between the OCS items and neuroticism, suggesting that there is likely a substantial degree of residual genetic correlation between the separate OCS items and neuroticism that is not captured by the latent OCS construct. Based on these findings, it appears that joint studies of the genetics of neuroticism with OCD might depend upon whether one uses a unitary obsessive-compulsive construct or examines individual dimensions separately.

We note that our OCS dimensional results are similar, but not identical to, those reported in an earlier analysis using these same data [van Grootheest et al., 2005]. Minor differences likely stem from our use of an updated EFA method implemented in MPlus that, among other improvements, allowed us to use data from all the twins. For example, in both analyses we found a three-factor solution best explained the patterns of OCS correlations, but their item content and loadings varied somewhat. Similarly, both identified a common latent factor via CPM consisting of shared genetic

TABLE III. Within- and Cross-trait Twin Correlations (Standard Errors) by Zygosity: MZ Above Diagonal, DZ Below.

	Twin 1				Twin 2			
	Checking	Contamination	Aggressive Obsessions	Neuroticism	Checking	Contamination	Aggressive Obsessions	Neuroticism
Twin 1								
Checking	1	**0.41** (.03)	**0.44** (.04)	**0.42** (.03)	**0.39** (.06)	**0.25** (.05)	**0.23** (.06)	**0.24** (.04)
Contamination	**0.41** (.03)	1	**0.25** (.05)	**0.22** (.04)	**0.25** (.05)	**0.43** (.08)	**0.17** (.07)	**0.14** (.05)
Aggressive obsessions	**0.44** (.04)	**0.25** (.05)	1	**0.39** (.04)	**0.23** (.06)	**0.17** (.07)	**0.38** (.09)	**0.30** (.05)
Neuroticism	**0.42** (.03)	**0.22** (.04)	**0.39** (.04)	1	**0.24** (.04)	**0.14** (.05)	**0.30** (.05)	**0.41** (.05)
Twin 2								
Checking	**0.20** (.08)	−0.07 (.07)	−0.11 (.09)	**0.13** (.05)	1	**0.41** (.03)	**0.44** (.04)	**0.42** (.03)
Contamination	−0.07 (.07)	−0.04 (.12)	0.04 (.10)	0.07 (.07)	**0.41** (.03)	1	**0.25** (.05)	**0.22** (.04)
Aggressive obsessions	−0.11 (.09)	0.04 (.10)	−0.28 (.18)	−0.01 (.08)	**0.44** (.04)	**0.25** (.05)	1	**0.39** (.04)
Neuroticism	**0.13** (.05)	0.07 (.07)	−0.01 (.08)	**0.15** (**.07**)	**0.42** (.03)	**0.22** (.04)	**0.39** (04)	1

Significant ($p < 0.05$) Parameters are Bolded.

TABLE IV. Model Comparison Statistics for Cholesky, Independent Pathway and Common Pathway analyses

Full Model	Restricted Model	Δdf	Mean χ^2	$\chi^2_{95\%\ CI}$	p-value
Cholesky decomposition	Independent Pathway Model	5	45.06	(20.7, 80.38)	0.001
Cholesky decomposition	Common Pathway Model	9	59.13	(33.1, 95.7)	0.000
Independent pathway model	Common Pathway Model	4	12.83	(4.08, 30.18)	0.278

Note: The Common Pathway Model is nested within the Independent Pathway Model which is nested within the Cholesky Decomposition, making the Likelihood Ratio Test (LRT) the most appropriate test statistic for these analyses, with Δdf = the difference in degrees of freedom. All of the LRTs are based on the bootstrapped analyses, resulting in empirical χ^2 95% confidence intervals (CI) and associated *p*-values.

and unique environmental influences on the OCS dimensions, also with modestly differing loadings.

Our findings are broadly consistent with those from other genetically informative studies of OCS dimensions, with some caveats deriving from the symptom items available for analysis or types of models tested. A family study of OCS dimensions identified a five-factor solution using data from the full YBOCS SC (9). Those factors included the three we identified plus ones for symmetry/ordering and hoarding, items of which were not included in the Padua Inventory. The authors reported significant familial

TABLE V. Median Bootstrapped Genetic and Environmental Correlations Between the Obsessive Compulsive Symptom (OCS) Dimensions and Neuroticism (empirical 95% confidence intervals)

Domain	Measure	Checking	Contamination	Aggression	Neuroticism
Genetic correlations	Checking	—			
	Contamination	0.85 (0.44, 1.00)	—		
	Aggression	0.83 (0.51, 0.98)	0.48 (0.07, 0.84)	—	
	Neuroticism	0.66 (0.33, 0.93)	0.40 (0.09, 0.71)	0.89 (0.55, 1.00)	—
Shared environmental correlations	Checking	—			
	Contamination	−0.98 (−1.00, 1.00)	—		
	Aggression	−0.97 (−1.00, 1.00)	0.82 (−1.00, 1.00)	—	
	Neuroticism	0.92 (−1.00, 1.00)	−0.55 (−1.00, 1.00)	−0.93 (−1.00, 1.00)	—
Unique environmental correlations	Checking	—			
	Contamination	0.34 (0.18, 0.48)	—		
	Aggression	0.39 (0.25, 0.53)	0.14 (−0.05, 0.34)	—	
	Neuroticism	0.29 (0.17, 0.42)	0.14 (−0.03, 0.28)	0.21 (0.02, 0.38)	—

resemblance for the three dimensions we examined, with sib–sib correlations in the range 0.16–0.22. Moore and colleagues, in a study of 517 adolescent male and female twins assessed using the Short Leyton Obsessional Inventory-Children's Version, reported significant heritability for two of the three dimensions they derived (Obsessions/Incompleteness, Numbers/Luck) only in the males, with a strong genetic correlation between them ($r = 0.95$). For the third dimension (Cleanliness), they found significant genetic risk for females only, with an estimated heritability (37%) similar to what we found for the analogous contamination dimension (11). Notably, their estimate for heritability of this dimension in males, while not significant, was the same as that for females. Iervolino and colleagues conducted a large ($N = 4355$) female-only twin study that examined five subscales of the Obsessive-Compulsive Inventory-Revised: checking, hoarding, obsessing, ordering, and washing. Here an independent pathway model best fit this higher dimensional data, in which the genetic variance for each OCS dimension was largely due to genetic components shared with the others, with the exception of hoarding, in which the common and specific genetic variances were nearly equal (12).

To our knowledge, only two other twin studies examined the sources of variance between OCS and normal personality traits. Hur assessed OCS using the Maudsley Obsessive Compulsive Inventory (MOCI) in 752 Korean pediatric twin pairs of both sexes. Unlike in our sample, the phenotypic correlation between their MOCI total score and extraversion was significantly different from zero ($r = -0.10$). They reported underlying genetic factors shared between MOCI items and neuroticism ($r_g = 0.44$) and extraversion ($r_g = -0.17$) (16). Such differences might derive from the instrument used and age or ancestry differences between study subjects. A study in a small community sample of adult twins jointly analyzed three subscales from the Dimensional Assessment of Personality (DAPP) with six OCS scales from the Obsessive Compulsive Inventory-Revised. Two of the DAPP scales assessed negative emotionality (trait anxiety and affective lability) and the third OC personality traits of perfectionism and rigidity. They reported significant genetic and environmental correlations among all six OCS dimensions and between each of these and the two negative emotionality measures (17).

The interpretation of our results is subject to the following limitations. First, only data from Caucasian female subjects was available, limiting the generalizability of these findings. Second, our measurement of OCS dimensions was constrained by the instrument used and the items selected from it. In particular, our items were assessed by self-report and not in subjects with OCD. Studies using more comprehensive instruments, such as the YBOCS SC, identified more than three dimensions, so our results are limited to the dimensions we obtained. Third, our sample was moderately sized, providing somewhat limited power to differentiate additive genetic (A) from common familial (C) sources of resemblance. Finally, these findings are predicated on the assumptions inherent in structural equation modeling of twin data, namely, independence and additivity of the latent variables, absence of assortative mating, and equal correlation in MZ and DZ twins for environmental experiences of relevance to the trait under study [Neale and Cardon, 1992].

In summary, we replicated and extended findings from prior genetically informative studies of OCS and personality traits. First, we confirmed that OCS dimensions are familial and largely due to the effects of genetic risk factors. Second, there is not an isomorphic relationship between OCS dimensions and underlying genetic factors, but rather, a portion of that risk is shared between them and the rest is dimension-specific. Third, similar to other internalizing symptoms [Jardine et al., 1984] and disorders [Hettema et al., 2006], genetic factors contributing to individual differences in the personality trait neuroticism are variably shared with genetic liability for OCS dimensions. One prior twin study has reported shared genetic risk between OCS and other anxiety disorders [Tambs et al., 2009], but the role of neuroticism and other personality traits have not been clarified in these relationships. A recent review of the relationship between neuroticism and overall psychopathology found neuroticism to be an efficient but non-specific risk marker of common mental disorders, with various models partially and variably explaining these associations [Ormel et al., 2013]. OCS/OCD was not included in that examination, so the current study adds to that literature.

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