# Genetic and Environmental Contributions to Self-Report Obsessive-Compulsive Symptoms in Dutch Adolescents at Ages 12, 14, and 16

DANIËL S. VAN GROOTHEEST, M.D., MEIKE BARTELS, PH.D., CATARINA E.M. VAN BEIJSTERVELDT, PH.D., DANIËLLE C. CATH, M.D., PH.D., AARTJAN T. BEEKMAN, M.D., PH.D., JAMES J. HUDZIAK, M.D., AND DORRET I. BOOMSMA, PH.D.

**ABSTRACT**

**Objective:** To determine the contributions of genetic and environmental influences to variation in self-report of obsessive-compulsive (OC) symptoms in a population-based twin sample of adolescent boys and girls. **Method:** Self-report ratings on the eight-item Youth Self-Report Obsessive-Compulsive Scale were collected in Dutch mono- and dizygotic twin pairs who participated at age 12 ($N$ = 746 twin pairs), 14 ($N$ = 963 pairs), or 16 years ($N$ = 1,070 pairs). Structural equation modeling was used to break down the variation in liability to OC symptoms into genetic and environmental components. **Results:** At age 12, no difference in prevalence was found for OC symptoms in boys and girls. At ages 14 and 16, the prevalence was higher in girls. At all ages, genetic factors contributed significantly to variation on OC symptom liability; 27% at the age of 12, 57% at the age of 14, and 54% at the age of 16. There were no sex differences in heritability. Only at age 12, environmental factors shared by children from the same family contributed significantly (16%) to individual differences in OC symptom scores. **Conclusions:** During adolescence, OC symptoms are influenced by genetic and nonshared environmental factors. Sex differences in prevalence, but not heritability, emerge in adolescence. At age 12, shared environmental factors are of importance, but their influence disappears at later ages. This is in line with earlier research at age 12 that used parental ratings of OC symptoms. Thus, between-family factors play a significant role in explaining individual differences in OC symptoms at this age. *J. Am. Acad. Child Adolesc. Psychiatry*, 2008;47(10):1182–1188.

**Key Words:** obsessive-compulsive disorder, obsessive-compulsive symptoms, adolescence, twins.

*Accepted May 28, 2008.*

*Drs. van Grootheest, Bartels, van Beijsterveldt, and Boomsma are with the Department of Biological Psychology and Drs. Cath and Beekman are with the Department of Psychiatry, VU University Amsterdam; Dr. Hudziak is with the Vermont Center for Children, Youth, and Families, University of Vermont.*

*This article was reviewed under and accepted by Ad Hoc Editor Kenneth Towbin, M.D.*

*This research was supported by ZonMw, grant 920-03-268 and NWO, grant 400-03-330. Data collection was supported by "Genetic Basis of Anxiety and Depression" (NWO grant 904-61-090), "Bilateral Agreement" (NWO grant 463-06-001), "Database Twin Register" (NWO grant 575-25-006), "Spinozapremie" (NWO/SPI 56-464-14192), CNCR (Centre Neurogenetics Cognition Research), Center for Medical Systems Biology: Multifactorial Diseases: Common Determinants, Unifying Technologies (NWO Genomics), "Twin-Family Database for Behavior Genetics and Genomics Studies" (NWO grant 480-04-004). Dr. Bartels is financially supported by NWO (VENI grant 451-04-034). The authors are grateful to the twins for their participation.*

*Correspondence to Dr. Daniël van Grootheest, VU University Amsterdam, Department of Biological Psychology, Van der Boechorststraat 1, 1081 BT Amsterdam, the Netherlands; e-mail: Ds.van.grootheest@psy.vu.nl.*



DOI: 10.1097/CHI.0b013e3181825abd

When considering the etiology of obsessive-compulsive disorder (OCD) or obsessive-compulsive (OC) symptoms, one could regard adolescence as a natural experiment during which a wide range of developmental and environmental changes occur in a short period of time. Therefore, this episode is of special interest when studying the dynamics between genes, development, and environment. In OCD, a bimodal distribution of age at onset has been found, with one peak occurring in preadolescent childhood and another peak in adulthood.[1,2] Early age at onset of OCD is also associated with tic disorder,[3,4] and the morbidity risk of OCD in family members of OCD subjects with early-onset OCD is higher than in relatives of late-onset OCD probands.[5,6] Furthermore, adult studies found an equal representation of men and women with OCD, or a slight female preponderance, whereas in clinical studies, early age at onset of OCD is associated with male

preponderance.[7] These observations would suggest that adolescence could be a period in which genetic and environmental etiological factors in OCD change in a relatively short period of time, offering a window of opportunity to study the genetics of OCD and OC symptoms. The aim of this study is to estimate the genetic and environmental contributions to OC symptoms in the adolescent period.

To disentangle genetic and environmental factors, twin or adoption studies are needed. No adoption studies of OCD have been published. Twin studies of OCD have evolved from case studies with patients with OCD to large-scale studies of unselected subjects. In these studies, the entire distribution of OC symptoms[8] is analyzed, assuming that OC symptoms are continuous with OCD. Mathews et al.[9] substantiated this assumption by finding evidence of a heritable unidimensional symptom factor underlying obsessionality. Additional evidence of this assumption comes from the observation that family members of patients with OCD have fewer OC symptoms that their family members with OCD, but more than controls.[5]

In recent years several twin studies have been published examining OC symptoms in children. Eley et al.[10] examined 4,564 four-year-old twin pairs in a British population-based twin study and included four items to assess OC symptoms. A heritability estimate of 65% was found. The remaining variance was accounted for by nonshared environmental influence. Hudziak et al.[11] conducted a large twin study on OC behavior in children, assessed by the Child Behavior Checklist Obsessive-Compulsive Scale (CBCL-OCS),[12,13] in American and Dutch samples. OC behavior was assessed at ages 7, 10, and 12 years in the Dutch twins and at approximately 9 years in the American twins. Heritability in the Dutch sample was approximately 55% at ages 7 and 10 years and dropped to 36% at age 12. The American sample showed a heritability of 55%. Small but significant shared environmental influences were seen at age 12 in the Dutch children. Significant sex differences in heritabilities were only seen in the U.S. data. Bolton et al.[14] assessed 854 six-year old British twin pairs by maternal-informant diagnostic interviews using *DSM-IV* criteria. The effect of familial factors was estimated at 47% for subthreshold OCD. The study lacked the statistical power to distinguish between shared environment and genetic factors to explain the familial aggregation. Last, follow-up research of the Hudziak et al.[11] study investigated stability over time in twins ages 7, 10, and 12 years using a multirater design.[15] A moderate stability with phenotypic correlations of approximately 0.50 for boys and for girls was found. Stability of OC behavior was influenced by genetic factors, environmental factors shared by children growing up in the same family, and nonshared environmental factors. Shared environment was observed to be important especially at age 12.

In adults, Clifford et al.[16] analyzed the 42-item version of the Leyton Obsessional Inventory,[17] obtained in 419 adult male and female twin pairs. The heritability of the obsessive symptoms was estimated at 47%. Jonnal et al.[18] examined data from 527 pairs of female monozygotic (MZ) and dizygotic (DZ) twins from the Virginia Twin Registry, using 20 items of the Padua Inventory.[19] The best model for these data suggested heritabilities of 33% and 26% for obsessiveness and compulsiveness, respectively. van Grootheest et al.[20] obtained the Young Adult Self-Report Obsessive-Compulsive Scale (YASR-OCS) from 5,893 MZ and DZ twins and 1,304 additional siblings and found a heritability of 39% in males and 50% in females.

As far as we know, no study has examined the genetic and environmental contributions to variation in OC symptoms during adolescence. In the current cross-sectional study, Dutch twins completed a self-report on OC behavior around their 12th, 14th, or 16th birthday. We aimed to determine the genetic architecture of OC symptoms self-report items in adolescence and to address the following questions: What is the contribution of genetic and environmental influences on self-reported OC behavior in adolescence? Are there sex differences in the contributions of genetic and environmental risk factors for OC symptoms in adolescence?

## METHOD

### Participants

The study was part of an ongoing study of emotional and problem behavior in young twins who are registered with the Netherlands Twin Register.[21,22] We analyzed data from twin pairs who reported on their behavior with the YSR-OCS when they were 12, 14, or 16 years old.[22] A survey that contained the YSR-OCS was send by mail to the twins, after parents gave consent. Twins who did not return the forms within 2 months received a reminder. The overall family response rate was 56.1%.

Zygosity was determined by DNA or blood group polymorphisms in 52.4% of the same-sex twin pairs. For the remaining same-sex twin pairs, zygosity was assessed by questionnaire items about

**TABLE 1**
YSR Items Used for the YSR-OCS

| YSR Item No. | YSR Item |
|---|---|
| 9 | I cannot get my mind off certain thoughts |
| 31 | I am afraid I might think or do something bad |
| 32 | I feel I have to be perfect |
| 52 | I feel too guilty |
| 66 | I repeat certain acts over and over |
| 84 | I do things other people think are strange |
| 85 | I have thoughts that other people would think are strange |
| 112 | I worry a lot |

*Note:* YSR = Youth Self-Report; YSR-OCS = Youth Self-Report Obsessive-Compulsive Scale.

physical similarity and frequency of confusion of the twins by family and strangers. Zygosity was correctly classified by questionnaire in 93% of the cases.[23]

## Measures

The twins completed the eight-item YSR-OCS (Table 1). The YRS-OCS was first developed and tested in young children using CBCL parental report (CBCL-OCS),[12,13] and then tested on self-report data in the YASR-OCS.[20] The YASR-OCS showed satisfactory psychometric properties with a sensitivity and specificity of 82% and 70%, respectively, in predicting OCD, when comparing an OCD group to a mixed psychiatric diagnosis group. Cronbach α was .69. The YSR-OCS makes use of the same eight items and showed a Cronbach α of .67 within the current adolescent population (all ages taken together). Both the YSR-OCS and YASR-OCS have the same format as the CBCL-OCS,[12] except that YSR and YASR items are worded in the first person. The CBCL-OCS showed high sensitivity and moderate specificity in predicting a *DSM-IV* diagnosis of OCD in children and adolescents in several studies.[12,13,24] We summarized the psychometric results of the CBCL-OCS of these studies in Table 2.

## Analyses

MZ twins share all of their genes, whereas DZ twins share on average half of their segregating genes. This different degree of genetic relatedness between MZ and DZ twins is used to estimate the genetic and environmental contributions to the variance of a trait. The total variance can be broken down into additive genetic variance (A), shared environmental variance (C), and nonshared environmental variance (E). A is due to additive effects of different alleles, C is due to environmental influences shared by members of a family, and E is due to environmental influences not shared by members of a family. E also includes measurement error and is therefore always included in the models.

A first impression of the relative importance of each component is obtained by inspecting the twin correlations. MZ correlations twice as high as DZ correlations indicate additive genetic influences on twin resemblance. DZ correlations higher than half of the MZ correlations designate shared environmental influences in addition to genetic influences. MZ correlations as high as DZ correlations indicate only shared environmental influences and no genetic sources of variance.[25] The proportion of phenotypic variance due to genetic influences is known as the heritability ($h^2$).

Structural equation modeling, as implemented in Mx,[26] was used for data analyses. Mx provides parameter estimates by maximizing the raw data likelihood. The goodness of fit of different models was evaluated by hierarchical likelihood ratio tests. Subtracting the 2 log likelihoods (−2LL) from each other yields a statistic that is asymptotically distributed as $\chi^2$ with *df* equal to the difference between the number of parameters in the two models. According to the principle of parsimony, models with fewer parameters (e.g., AE compared to ACE) were preferred if they did not give a significant deterioration in model fit. In addition, the Akaike information criterion (AIC),[27] a goodness-of-fit index that considers the rule of parsimony, was calculated. More details of genetic model-fitting analyses are reviewed elsewhere.[28]

Because the data exhibited a pronounced right skew at all ages, we used a threshold model with three thresholds. By using a threshold model, genetic analyses are carried out on an underlying continuous distribution of liability to the disorder. The number of thresholds, defining categories (e.g., unaffected, mildly affected, severely affected)[29] are chosen in such a way that the number of individuals was

**TABLE 2**
Summary of Studies Examining Psychometric Properties of the CBCL-OCS That Include the Same Eight Items as Those on the YSR-OCS

| Study | No. of Children/ Adolescents | Mean Age, y (SD) | Sensitivity (Compared to Clinical Controls) | Specificity (Compared to Clinical Controls) | Cronbach α |
|---|---|---|---|---|---|
| Nelson et al.[12] | 73 OCD patients and 73 clinical controls | 12.3 (2.8) for boys 12.0 (2.6) for girls | 75.3%–84.9% (depending on percentile scores) | 72.6%–87.7% (depending on percentile scores) | .84 |
| Hudziak et al.[13] | 61 OCD patients and 64 clinical controls (in essence, same population as used by Nelson et al.) | See Nelson et al. | 92% (using a cutoff of 5) | 67% (using a cutoff of 5) | See Nelson et al. |
| Geller et al.[24] | 64 OCD patients and 64 clinical controls | 11.2 (3.5) | 78.1%–92.2% (depending on percentile scores) | 75%–89.1% (depending on percentile scores) | .87 |

*Note*: CBCL-OCS = Child Behavior Checklist Obsessive-Compulsive Scale; YSR-OCS = Youth Self-Report Obsessive-Compulsive Scale; OCD = obsessive-compulsive disorder.

**TABLE 3**
Number of Complete Twin Pairs and Twin Correlations at Ages 12, 14, and 16 Years

| | Age 12 | | | Age 14 | | | Age 16 | | |
|---|---|---|---|---|---|---|---|---|---|
| Zygosity | Complete Twin Pairs | Incomplete Twin Pairs | Twin Correlations | Complete Twin Pairs | Incomplete Twin Pairs | Twin Correlations | Complete Twin Pairs | Incomplete Twin Pairs | Twin Correlations |
| MZM | 140 | 3 | 0.50 | 134 | 5 | 0.57 | 175 | 13 | 0.45 |
| DZM | 138 | 2 | 0.38 | 128 | 7 | 0.17 | 130 | 16 | 0.30 |
| MZF | 162 | 3 | 0.45 | 222 | 9 | 0.60 | 209 | 17 | 0.58 |
| DZF | 124 | 6 | 0.36 | 144 | 10 | 0.30 | 189 | 13 | 0.33 |
| DOS | 162 | 6 | 0.21 | 272 | 32 | 0.22 | 240 | 68 | 0.22 |

*Note*: MZM = monozygotic male; MZF = monozygotic female; DZM = dizygotic female; DOS = dizygotic opposite sex.

roughly similar in each of the categories without the presence of empty cells (e.g., category not including any person).

We started with a saturated model, in which all thresholds and all twin correlations were estimated freely. Because a threshold model was used, polychoric correlations were obtained, which represent the resemblance between twins on the liability for OC symptoms. We tested whether thresholds were the same for first-born and second-born twins, for MZ and DZ twins to examine effects on the prevalence of OC symptoms, and for boys and girls to examine sex differences in the prevalence of OC symptoms. The saturated model provides a baseline model against which genetic models were compared. In the genetic models, the significance of sex differences in the estimates for the influences of A, C, and E were tested. The significance of the contributions of additive genetic influences and shared environmental influences was tested by assessing the deterioration in model fit after each component was constrained at zero in the full model.

## RESULTS

In the saturated model, no effect of birth order or zygosity was detected at any age ($p$ values > .05) on the thresholds. There was no sex effect on the thresholds at age 12 ($\chi^2_3 = 2.16.$, $p = .54$). However, at ages 14 and 16, the thresholds were significantly lower for girls ($\chi^2_3 = 43.94$, $p < .001$ and $\chi^2_3 = 42.57$, $p < .001$, respectively), indicating that girls score higher than boys on the YSR-OCS at the ages of 14 and 16.

Polychoric twin correlations are presented in Table 3 as a function of zygosity and age. At age 12, shared environmental factors seem to be of importance because MZ correlations are less than twice the DZ correlations. Except for boys at age 16, with MZ correlations smaller than twice the DZ correlations, at ages 14 and 16, MZ correlations are clearly about twice the DZ correlations, indicating the influence of genetic factors, but not shared environment. The twin correlations in opposite-sex twin pairs were not attenuated compared to the correlations in same-sex dizygotic twin pairs ($p$ values > .05) at three different ages. This means that there is no indication for sex-specific genes influencing variance in YSR-OCS scores.

Model-fitting results are given in Table 4. Starting from the saturated threshold model, the full ACE threshold model with sex differences in variance components fits the data well at all ages ($p$ values > .05). From the full model we constrained the estimates for additive genetic, shared environmental, and non-shared environmental factors to be equal across the sexes. At all ages, this model did not worsen the fit ($p$ values > .05), meaning that the relative effects of these components were the same in boys and girls.

At age 12, the ACE model without sex differences gave a standardized estimate of .27 for genetic variance and .18 for shared environmental variance. We fitted both an AE and a CE model. Both models fit the data, although the AE model ($\chi^2_1 = 2.5$, $p = .12$) fit slightly better than the CE model ($\chi^2_1 = 3.5$, $p = .06$). A closer look at the Akaike information criterion fit index showed that the ACE model without sex differences fit the data the best, suggesting that both genetic and shared influences play a role in individual differences in OC symptoms, regardless of sex.

At ages 14 and 16, the estimates of the shared environmental variance were zero. An AE model was the best fitting model for the data. Individual differences in OC symptoms were explained by additive genetic influences (57% and 54% at ages 14 and 16, respectively) and nonshared environmental effects (43% and 46% at ages 14 and 16, respectively). Next, we tested whether genetic and nonshared environmental influences were of the same magnitude for adolescents at 14 and 16 years of age. At both ages, genetic influences accounted for 55% of the variation in OC symptoms

**TABLE 4**
Model Fitting Results for YSR-OCS Scores

| Study Sample | Type of Model | −2LL | $\chi^2$ | $\Delta df$ | $p$ | AIC | Compared With Model | Parameter Estimates: Boys $a^2$ | Boys $c^2$ | Boys $e^2$ | Girls $a^2$ | Girls $c^2$ | Girls $e^2$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Age 12 y | 1. Fully saturated | 3,939.5 | | | | | | | | | | | |
| | 2. ACE sex | 3,954.8 | 15.3 | 13 | .29 | 1,030.8 | 1 | 0.23 | 0.27 | 0.51 | 0.10 | 0.33 | 0.57 |
| | **3. ACE no sex** | **3,958.0** | **3.2** | **2** | **.20** | **1,030.0** | **2** | **0.27** | **0.18** | **0.54** | **0.27** | **0.18** | **0.54** |
| | 4. AE no sex | 3,960.5 | 2.5 | 1 | .12 | 1,030.5 | 3 | 0.49 | 0.00 | 0.51 | 0.49 | 0.00 | 0.51 |
| | 5. CE no sex | 3,961.6 | 3.6 | 1 | .06 | 1,031.6 | 3 | 0.00 | 0.38 | 0.62 | 0.00 | 0.38 | 0.62 |
| Age 14 y | 1. Fully saturated | 4,923.4 | | | | | | | | | | | |
| | 2. ACE sex | 4,935.7 | 12.3 | 13 | .50 | 1,229.7 | 1 | 0.54 | 0.01 | 0.46 | 0.50 | 0.09 | 0.41 |
| | 3. ACE no sex | 4,936.9 | 1.2 | 2 | .27 | 1,226.9 | 2 | 0.57 | 0.00 | 0.43 | 0.57 | 0.00 | 0.43 |
| | **4. AE no sex** | **4,936.9** | **0.0** | **1** | **.99** | **1,224.9** | **3** | **0.57** | **0.00** | **0.43** | **0.57** | **0.00** | **0.43** |
| Age 16 y | 1. Fully saturated | 5,246.2 | | | | | | | | | | | |
| | 2. ACE sex | 5,260.7 | 14.5 | 13 | .34 | 1,254.7 | 1 | 0.45 | 0.05 | 0.50 | 0.55 | 0.04 | 0.42 |
| | 3. ACE no sex | 5,262.7 | 2.0 | 2 | .16 | 1,252.7 | 2 | 0.54 | 0.00 | 0.46 | 0.54 | 0.00 | 0.46 |
| | **4. AE no sex** | **5,262.7** | **0.0** | **1** | **.99** | **1,250.7** | **3** | **0.54** | **0.00** | **0.46** | **0.54** | **0.00** | **0.46** |

*Note*: Boldface type represents the best fitting model for that sample. $a^2$ , $c^2$ , $e^2$ = the proportion of variance of A, C, and E, respectively, and is calculated by squaring the parameters a, c, and e and dividing them by the total variance. YSR-OCS = Youth Self-Report Obsessive-Compulsive Scale; LL = log likelihood; AIC = Akaike information criterion; A = additive genetic effects; C = shared environmental effects; E = nonshared or individual specific effects.

and nonshared environmental influences were estimated to account for 45% ($\chi^2_1$ = .253, $p$ = .61).

## DISCUSSION

To our knowledge, this is the first twin study of OC symptoms in adolescents, revealing that individual differences in OC symptoms are heritable throughout puberty, with shared environmental influences only playing a role at the beginning of adolescence. No sex differences in heritability estimations were found, and individual differences in OC symptoms are influenced by the same additive genetic factors in boys and girls. Female adolescents scored higher on the OCS than males at the ages of 14 and 16, but not at the age of 12.

The finding of equal prevalence of OC symptoms in boys and girls at age 12 is in line with the study of Hudziak et al.,[11] who found no sex differences in scores at ages 7, 10, and 12. The prevalence of OC symptoms in boys and girls within community samples seems to be more similar than in clinical samples with OCD, where boys outnumber girls. Interestingly, at the ages of 14 and 16, girls showed a higher prevalence of OC symptoms than boys. This is in line with a recent study using the YASR-OCS in adults, which found significantly higher prevalence for women and also with clinical and epidemiological findings of a slight preponderance in the prevalence of OC symptoms in women.[20] These results strongly suggest that sex differences in prevalence develop in early puberty and that these sex differences persist until adulthood.

The finding of shared environmental influences of 18% in this study at age 12 is remarkably similar to that of 16% found in the study of Hudziak et al.,[11] who used essentially the same sample as the present study. Note that the study of Hudziak et al.[11] presented mother ratings, whereas the present study used self-reports. van Grootheest et al.[15] also found shared environmental influences to be important (approximately 30%) at age 12 using a design with both maternal and paternal ratings of OC symptoms. This means that the same results (i.e., no sex differences in prevalence and significant shared environmental influences) were found in a variety of studies using different ratings and raters, underscoring the relevance of these shared influences at age 12. Recently, we also found shared environmental influences at age 12 to be important for loneliness[30] and depression/anxiety.[31] This could mean that shared environmental influences are not specific for OC symptoms, but indicate an age-dependent family

environment factor to exacerbate internalizing psychopathology in general at that age. The question remains as to which factors cause the emergence of shared environmental influences at this age. Although somewhat speculative, the age of 12 is the key age for the transition from primary to secondary school in the Netherlands, a well-known stressful event.[32] When leaving the old school to enter the new, children leave their old peer group to build a new one and therefore rely more on family life than before. Considering the demands in this transitional period, families may differ in dealing with these stressful events. This conclusion also has clinical consequences. Given that cognitive-behavioral therapy could clearly be seen as a potential environmental mediator of OC symptoms, involving family members in behavioral treatment of OC symptoms may be best aimed at the very young when shared environment is in play.[33]

A closer look at the heritability estimates over time reveals that OCS is remarkably stable, with heritability estimates of approximately 55% at younger ages,[11] with a decrease at age 12, in which part of the variance was accounted for by shared environmental influences, continuing to 55% at ages 14 and 16, and 45% in adulthood.[15] This decrease in heritability between children/adolescents and adults may reflect the bimodal distribution found in the clinical OCD literature,[1,2] which found early-onset OCD to be associated with a higher genetic load compared to late-onset OCD.

By demonstrating that genetic factors are influencing OC symptoms also in adolescence, gene finders are given a strong signal to pursue. However, a relatively stable heritability does not automatically imply that the same genes are involved at different ages. Only longitudinal data can elucidate whether the genes associated in childhood[15] also persist in adolescence and even in adulthood. As the Netherlands Twin Register grows and the twins are getting older, future studies will focus on longitudinal analyses of OC symptoms from childhood to adolescence to adulthood.

The results of this study should be interpreted in the context of several potential limitations. First, the genetic and environmental contributions presented in this report reflect YSR-OCS scores, not clinical measures of *DSM-IV* OCD. Because of the relatively low prevalence of OCD, twin studies rely on dimensional measures with the underlying assumption that OCD reflects the end of a normal distribution, whereas OC symptoms represent a milder form of the latter.[18,34,35] Because the current twin analyses are based on a liability threshold model, it should make no difference if the studied variable is dimensional as long as it reflects the same underlying liability as the categorical diagnosis.[36]

Second, the use of twin models requires several assumptions, including the absence of assortative mating, the equal environment assumption, and the absence of gene–environment interaction and correlation. van Grootheest et al.[37] found that small, but significant assortative mating existed for OC symptoms but concluded that the bias in twin studies caused by the small amount of assortment is negligible. Jonnal et al.[18] tested the equal environment assumption for OC symptoms and concluded that the equal environment assumption was not violated. Gene–environment interaction could affect twin similarity in either direction depending on whether both twins are exposed to the specific environmental factor in question; to our knowledge, gene–environment interaction and/or correlation have yet to be demonstrated for the phenotype studied here.

Third, the eight-item YSR-OCS is not suitable for examining OC symptom dimensions. There is more and more evidence that OCD or OC symptoms appear to encompass at least four consistent and temporally stable symptom dimensions.[38] By considering these OC symptom dimensions as quantitative components of a more complex OC phenotype, a dimensional approach could provide a more powerful approach for the detection of genes or environmental risk factors that contribute to OC behavior.[39] We recently found evidence of specific genetic and environmental factors underlying the contamination dimension,[40] underscoring the potential value of OC symptom dimensions in this field of research.[41]

In summary, the present study suggests that heritability estimates of OC symptoms in adolescence are similar (55%) to the heritability estimates in children, with a decrease around the age of 12. At 12 years, a clear contribution of shared environment was found to the variation of OC symptoms. Sex differences in scores on OC symptoms were found, with girls scoring higher on OC symptoms at the ages of 14 and 16, but not at age 12. The present study underscores the importance of conducting more research on OC symptoms in the adolescent period.

*Disclosure: The authors report no conflicts of interest.*

## REFERENCES


1. Delorme R, Golmard JL, Chabane N, et al. Admixture analysis of age at onset in obsessive-compulsive disorder. *Psychol Med*. 2005;35:237–243.
2. Chabane N, Delorme R, Millet B, Mouren MC, Leboyer M, Pauls D. Early-onset obsessive-compulsive disorder: a subgroup with a specific clinical and familial pattern? *J Child Psychol Psychiatry*. 2005;46: 881–887.
3. Swedo SE, Rapoport JL, Leonard H, Lenane M, Cheslow D. Obsessive-compulsive disorder in children and adolescents. Clinical phenomenology of 70 consecutive cases. *Arch Gen Psychiatry*. 1989;46(4):335–341.
4. Rosario-Campos MC, Leckman JF, Mercadante MT. Adults with early-onset obsessive-compulsive disorder. *Am J Psychiatry*. 2001;158:1899–1903.
5. Pauls DL, Alsobrook JP, Goodman W, Rasmussen S, Leckman JF. A family study of obsessive-compulsive disorder. *Am J Psychiatry*. 1995; 152:76–84.
6. Nestadt G, Samuels J, Riddle M, et al. A family study of obsessive-compulsive disorder. *Arch Gen Psychiatry*. 2000;57:358–363.
7. Geller DA, Biederman J, Faraone SV, et al. Disentangling chronological age from age of onset in children and adolescents with obsessive-compulsive disorder. *Int J Neuropsychopharmacol*. 2001;4:169–178.
8. van Grootheest DS, Cath DC, Beekman AT, Boomsma DI. Twin studies on obsessive-compulsive disorder: a review. *Twin Res Hum Genet*. 2005; 8:450–458.
9. Mathews CA, Greenwood T, Wessel J, et al. Evidence for a heritable unidimensional symptom factor underlying obsessionality. *Am J Med Genet B Neuropsychiatr Genet*. 2007. DOI: 10.1002/ajmg.b.30660.
10. Eley TC, Bolton D, O'Connor TG, Perrin S, Smith P, Plomin R. A twin study of anxiety-related behaviours in pre-school children. *J Child Psychol Psychiatry*. 2003;44:945–960.
11. Hudziak JJ, van Beijsterveldt CEM, Althoff RR, et al. Genetic and environmental contributions to the Child Behavior Checklist Obsessive-Compulsive Scale: a cross-cultural twin study. *Arch Gen Psychiatry*. 2004; 61:608–616.
12. Nelson EC, Hanna GL, Hudziak JJ, Botteron KN, Heath AC, Todd RD. Obsessive-compulsive scale of the child behavior checklist: specificity, sensitivity, and predictive power. *Pediatrics*. 2001;108:E14.
13. Hudziak JJ, Althoff RR, Stanger C, et al. The Obsessive Compulsive Scale of the Child Behavior Checklist predicts obsessive-compulsive disorder: a receiver operating characteristic curve analysis. *J Child Psychol Psychiatry*. 2006;47:160–166.
14. Bolton D, Rijsdijk F, O'Connor TG, Perrin S, Eley TC. Obsessive-compulsive disorder, tics and anxiety in 6-year-old twins. *Psychol Med*. 2007;37:39–48.
15. van Grootheest DS, Bartels M, Cath DC, Beekman AT, Hudziak JJ, Boomsma DI. Genetic and environmental contributions underlying stability in childhood obsessive-compulsive behavior. *Biol Psychiatry*. 2007;61:308–315.
16. Clifford CA, Murray RM, Fulker DW. Genetic and environmental influences on obsessional traits and symptoms. *Psychol Med*. 1984;14: 791–800.
17. Cooper J. The Leyton Obsessional Inventory. *Psychol Med*. 1970;1: 48–64.
18. Jonnal AH, Gardner CO, Prescott CA, Kendler KS. Obsessive and compulsive symptoms in a general population sample of female twins. *Am J Med Genet*. 2000;96:791–796.
19. Sanavio E. Obsessions and compulsions: the Padua Inventory. *Behav Res Ther*. 1988;26:169–177.
20. van Grootheest DS, Cath DC, Beekman AT, Boomsma DI. Genetic and environmental influences on obsessive-compulsive symptoms in adults: a population-based twin-family study. *Psychol Med*. 2007;37: 1635–1644.
21. Boomsma DI, de Geus EJ, Vink JM, et al. Netherlands Twin Register: from twins to twin families. *Twin Res Hum Genet*. 2006;9:849–857.
22. Bartels M, van Beijsterveldt CE, Derks EM, et al. Young Netherlands Twin Register (Y-NTR): a longitudinal multiple informant study of problem behavior. *Twin Res Hum Genet*. 2007;10:3–11.
23. Rietveld MJ, Der Valk JC, Bongers IL, Stroet TM, Slagboom PE, Boomsma DI. Zygosity diagnosis in young twins by parental report. *Twin Res*. 2000;3:134–141.
24. Geller DA, Doyle R, Shaw D, et al. A quick and reliable screening measure for OCD in Youth: Reliability and Validity of the Obsessive Compulsive Scale of the Child Behavior Checklist. *Compr Psychiatry*. 2006;47:234–240.
25. Boomsma D, Busjahn A, Peltonen L. Classical twin studies and beyond. *Nat Rev Genet*. 2002;3:872–882.
26. Neale MC, Boker SM, Xie G, Maes HM. Mx: Statistical Modeling. 6th ed. Richmond, VA: Department of Psychiatry; 2003.
27. Akaike H. Factor analysis and AIC. *Psychometrika*. 1987;52:317–332.
28. Neale MC, Cardon LR. *Methodology for Genetic Studies of Twins and Families*. Dordrecht, the Netherlands: Kluwer Academic; 1992.
29. Derks EM, Dolan CV, Boomsma DI. Effects of censoring on parameter estimates and power in genetic modeling. *Twin Res*. 2004;7:659–669.
30. Bartels M, Cacioppo JT, Hudziak JJ, Boomsma DI. Genetic and environmental contributions to stability in loneliness throughout childhood. *Am J Med Genet Part B* 2008;147:385–391.
31. Boomsma DI, van Beijsterveldt CEM, Bartels M, Hudziak JJ. Genetic and environmental influences on anxious/depression. In: Hudziak JJ, ed. *Developmental Psychopathology and Wellness*. Washington, DC: American Psychiatric Publishing; 2008:185–219.
32. Sirsch U. The impending transition from primary to secondary school: challenge or threat? *Int J Behav Dev*. 2003;27:385–395.
33. Renshaw KD, Steketee G, Chambless DL. Involving family members in the treatment of OCD. *Cogn Behav Ther*. 2005;34:164–175.
34. van den Oord EJ, Pickles A, Waldman ID. Normal variation and abnormality: an empirical study of the liability distributions underlying depression and delinquency. *J Child Psychol Psychiatry*. 2003;44: 180–192.
35. Kendler KS. Psychiatric genetics: a methodologic critique. *Am J Psychiatry*. 2005;162:3–11.
36. Reichborn-Kjennerud T, Czajkowski N, Torgersen S, et al. The relationship between avoidant personality disorder and social phobia: a population-based twin study. *Am J Psychiatry*. 2007;164: 1722–1728.
37. van Grootheest DS, van den Berg SM, Cath DC, Willemsen G, Boomsma DI. Marital resemblance for obsessive-compulsive, anxious and depressive symptoms in a population-based sample. *Psychol Med*. 2008;27:1–10.
38. Mataix-Cols D, do Rosario-Campos MC, Leckman JF. A multidimensional model of obsessive-compulsive disorder. *Am J Psychiatry*. 2005;162:228–238.
39. Miguel EC, Leckman JF, Rauch S, et al. Obsessive-compulsive disorder phenotypes: implications for genetic studies. *Mol Psychiatry*. 2005;10: 258–275.
40. van Grootheest DS, Boomsma DI, Hettema JM, Kendler KS. Heritability of obsessive-compulsive symptom dimensions. *Am J Med Genet B Neuropsychiatr Genet*. 2008;147:473–478.
41. Leckman JF, Rauch SL, Mataix-Cols D. Symptom dimensions in obsessive-compulsive disorder: implications for the *DSM-V*. *CNS Spectr*. 2007;12:376–400.