The Genetic and Environmental Contributions to Oppositional Defiant Behavior: A Multi-informant Twin Study

JAMES J. HUDZIAK, M.D., ESKE M. DERKS, M.A., ROBERT R. ALTHOFF, M.D., PH.D., WILLIAM COPELAND, PH.D., AND DORRET I. BOOMSMA, PH.D.

ABSTRACT

Objective: To estimate the genetic and environmental contributions to oppositional defiant behavior (ODB) from mother, father, and teacher report using the Conners Revised Short Forms in a large twin sample. **Method:** ODB data were collected from 1,595 mothers, 1,114 fathers, and 793 teachers of 7-year-old twin pairs from the Netherlands Twin Registry in the 1990–1992 cohort with an 80% response rate. Models were fit for each informant to determine the genetic, environmental, gender, and informant influences on ODB. **Results:** Genetic analyses of the ODB quantitative scale showed additive genetic (A) by mother (55%), by father (57%), and by teacher (21% girls, 38% boys) unique environmental (E) (mother, 22%; father, 29%; teacher, 48% girls, 39% boys) and shared environmental (C) (mother, 14%; father, 23%; teacher, 31% girls, 23% boys) influences. **Conclusions:** Additive genetic and unique environmental factors account for the majority of the influences on ODB for boys and girls by all informants.
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Oppositional defiant disorder (ODD) as defined by the *DSM-IV* consists of a recurrent pattern of negativistic, defiant, disobedient, and hostile behavior toward authority figures (Loeber et al., 2000) (Table 1). Oppositional defiant behavior (ODB) and ODD are associated with a higher risk of later conduct disorder (CD), antisocial personality disorder, and substance use disorders (Burke et al., 2002). Evidence of family aggregation of oppositional and conduct problems exists, particularly when compared to other forms of child psychopathology (Szatmari et al., 1993). ODB co-occurs commonly with attention-deficit/hyperactivity disorder (ADHD) and as such is associated with severe morbidity and discrete genetic influences. Others have reported that when the genetic and environmental contributions to ADHD, ODD, and CD are studied together, there is evidence of both genetic and environmental influences, but a single shared environmental factor made the largest contribution to the covariation among the three (Burt et al., 2001). When compared with the number of genetic studies on ADHD, obsessive-compulsive disorder, and CD, it is interesting that so few studies have addressed the etiologic underpinnings of ODB. The purpose of this study is to review what is known about the genetic and environmental contributions to ODB and then to test these findings in a large twin study population. We report on the genetic architecture of ODB by informant (mother, father, and teacher) and gender while controlling for developmental confounds.

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Drs. Hudziak and Althoff are with the Department of Psychiatry, University of Vermont, Burlington; Dr. Copeland is with Duke University, Durham, NC; and Ms. Derks and Dr. Boomsma are with the Free University, Amsterdam, the Netherlands.

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Reprint requests to Dr. James Hudziak, Division of Behavioral Genetics, Department of Psychiatry, University of Vermont, Given B229, Burlington, VT 05405; e-mail: james.hudziak@uvm.edu.

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GENETIC AND ENVIRONMENTAL INFLUENCES ON ODB

For a complete review of the empirical literature on ODD, the reader is referred to Burke et al. (2002 and Loeber et al. (2000). Although more studies have focused on CD than on oppositionality (Burke et al., 2002), both oppositionality and CD have similar risk factors and may

TABLE 1
Comparison of the *DSM-IV* ODD and CRS Oppositional Items

	CRS Oppositional Items	
DSM-IV ODD Items	Parent Form	Teacher Form
Often loses temper	Loses temper	Temper outbursts; explosive, unpredictable behavior
Often argues with adults	Argues with adults	Argues with adults
Often actively defies or refuses to comply with adults' requests or rules	Actively defies and refuses to comply with adults' requests	Actively defies and refuses to comply with adults' requests
Often deliberately annoys people	Deliberately does things that annoy other people	—
Often blames others for his or her mistakes or misbehavior	—	—
Is often touchy or easily annoyed by others	Irritable	—
Is often angry and resentful	Angry and resentful	—
Is often spiteful or vindictive	—	Spiteful or vindictive
—	—	Defiant

Note: ODD = oppositional defiant disorder; CRS = Conners rating scale.

evince a common genetic underpinning (Eaves et al., 2000). Eaves et al. examined 1,376 twin pairs and examined the relationship between gender, rater contrast, and comorbidity among child- and parent-rated ODD, CD, and ADHD. They found that there were rater contrast effects influencing the heritability estimates of ADHD and concluded that this was likely because of differences within the raters themselves rather than sibling imitation. This was not present for ODD or CD. In addition, they found that the influences on CD and ODD were additive genetic and unique environmental with no shared environmental components. This is in contrast to Burt et al. (2001), who showed in their sample of 753 twin pairs a common shared environmental factor contributing to ADHD, ODD, and CD that accounted for between 47% and 59% of the variance when using parent and child reports.

The differences between the two may reflect differences in the sample, differences in the modeling, or differences in the ages of the samples. Eaves et al. (2000) used twins ages 6–18, whereas Burt et al. (2001) limited their study to the 11-year-old age group. This difference is more important given the recent work of Moffitt et al. (2002), who describe an early-onset aggressive CD (associated with ODD) and a later-onset delinquent CD. Moffitt et al. hypothesize that these are two types of CD, with varying genetic and environmental influences. Others have shown, using the Child Behavior Checklist, that measures of aggression (AGG) are highly influenced by genetic factors, accounting for between 51% and 72% of the variance in twin studies, which remain stable over the course of development (Bartels et al., 2003; Hudziak et al., 2003; van Beijsterveldt et al., 2003). In addition, the more delinquent set of behaviors, rule-breaking behavior, is associated with genetic influences in the 30%–79% range, with lower numbers in girls and discrete and greater environmental influences (Bartels et al., 2003; van den Oord et al., 1994). It is interesting that AGG is correlated with a diagnosis of ODD, whereas AGG and rule-breaking behavior are correlated with a diagnosis of CD (Hudziak et al., 2004). Thus, a twin study of ODB in young children may yield valuable information on the magnitude of the genetic and environmental influences on ODD and its developmental outcomes such as CD. In addition, neither Burt et al. (2001) nor Eaves et al. (2000) found any differences in the models between genders; however, neither of these studies included teacher report data. A study using teacher data and again investigating these differences seems warranted. This idea is supported by experts in the field who recommend that more research is needed and further that these studies aim to disentangle the contributions of informants, gender, and age to better estimate the genetic and environmental estimates on ODB (Burke et al., 2002; Loeber et al., 2000).

Taxonomic Approaches

The purpose of this study was to attempt to control for some of these obstacles by studying a group of young,

same-aged twins using a taxonomic approach that used *DSM-IV* ODD symptoms, but it was also developed to provide informant, gender, and age norms. Here, we aim to add to what is known about ODD by providing cross-sectional analyses of ODB data on a large group of 7-year-old twins using mother, father, and teacher reports. These data allow us to perform informant-specific analyses in which gender effects are included and in which age affects are controlled.

METHOD

Subjects and Procedure

The study was part of an ongoing twin-family study of health-related characteristics, personality, and behavior in the Netherlands. The subjects were all part of the Netherlands Twin Registry (NTR) (DiMaio et al., 2003). The NTR currently has data on more than 25,000 twin pairs. For this study, we assessed a sample of Dutch twin pairs whose parents and teachers reported on their behavior when they were 7 years old. The twins at age 7 were representative of Dutch 7-year-old children with respect to their scores on measures such as the Child Behavior Checklist (van den Oord et al., 1995). The socioeconomic status of the parents of the twins was somewhat higher than that of the general Dutch population (Rietveld et al., 2004). Mothers, fathers, and teachers are sent a Conners Rating Scale-Revised (CRS-R) form in the mail and are asked to return it to the NTR by mail. Parents who do not return the form within 2 months are sent a reminder and those who do not respond after 4 months are phoned by an NTR research assistant, yielding a 80% participation rate. Table 2 describes the number of pairs by zygosity, gender, and informant.

Zygosity was determined by questionnaire items about physical similarity and frequency of confusion of the twins by family and strangers. The classification of zygosity was based on a discriminant analysis, relating the questionnaire items to zygosity based on blood/DNA typing in 634 same-sex twin pairs. According to this analysis, the zygosity was correctly classified by questionnaire data in nearly 95% of the cases (Rietveld et al., 2003). The possible misclassification of 5% of the sample has been shown to have relatively little impact on estimates of heritability with samples this large (Rietveld et al., 2000), although the possibility exists that it may slightly increase estimates of the shared environmental component at the expense of the additive genetic estimate.

Measures

Mothers and fathers of all participants completed the Parent CRS-Revised Short Form, and teachers completed the Teachers CRS-Revised Short Form. The questionnaire consists of 27 (Parent scale) or 28 (Teacher scale) items rated on a four-point Likert scale for symptom severity (i.e., 0 = not true at all, 1 = just a little true, 2 = pretty much true, 3 = very much true). The items are summarized on four scales: Oppositional, Cognitive Problems/Inattention, Hyperactivity, and the ADHD Index. Three of these scales, Oppositional, Cognitive Problems/Inattention, and Hyperactivity, were originally derived from the Conners Rating Form: Long Form. To provide brief versions of these scales, only items loading the highest from an exploratory factor analysis of the factor scale items on the long form were used (loadings ≥0.40). This study specifically used the Oppositional subscale, which consisted of six items on the parent form and five items on the teacher form (Table 1). The internal consistency coefficient for both scales was greater than 0.80 for males and females and the test-retest reliability coefficients for scales were between 0.63 and 0.85 during a period of 6 to 8 weeks (Conners, 2001).

Data Analyses

Distribution of Oppositional Scores. Means and twin correlations were calculated using the statistical software program Mx (Neale, 1997), a statistical software package designed for conducting genetic analyses using an approach that is standard in structural equation modeling (Bollen, 1989). Because the Oppositional scale from the Conners Revised Form was not normally distributed (maternal data: skewness = –0.288, kurtosis = –0.271; paternal data: skewness = –0.310, kurtosis = –0.444; teacher data: skewness = 1.605, kurtosis =

TABLE 2
Twin Correlations and Number of Complete Twin Pairs by Gender, Zygosity, and Informant

	Mother		Father		Teacher	
Zygosity	No. of Twin Pairs	Corr.	No. of Twin Pairs	Corr.	No. of Twin Pairs	Corr.
MZm	245 (7[a])	0.71	186 (4)	0.76	247 (37)	0.60
DZm	266 (10)	0.41	184 (5)	0.57	249 (49)	0.36
MZf	289 (2)	0.69	210 (0)	0.78	294 (44)	0.55
DZf	255 (2)	0.46	159 (2)	0.50	232 (41)	0.33
DOS	493 (17)	0.41 m 0.47 f	353 (8)	0.48 m 0.48 f	482 (85)	0.21 m 0.10 f
Unknown	9 (0)		3 (0)		27 (3)	
Total zygosity known	1,548 (38)		1,092 (19)		1,504 (256)	
Total	1557 (38)		1095 (19)		1531 (259)	

Note: Corr. = twin-twin correlation; MZm = monozygotic mal; DZm = dizygotic male; MZf = monozygotic female; DZf = dizygotic female; DOS = opposite sex.

[a] Number of incomplete twin pairs, e.g., those for whom only one form is available.

1.576), the data were square-root transformed to approximate normal distribution.

Model Fitting. Genetic and environmental influences on ODB were estimated using structural equation modeling in Mx (Neale, 1997). The influence of the relative contributions of genetic and environmental factors to individual differences in ODB can be inferred from the different level of genetic relatedness of monozygotic (MZ) and dizygotic (DZ) twins (Neale et al., 1992). Figure 1 summarizes the fundamental univariate genetic model that underlies our analyses. The variance may be the result of additive genetic (A) factors, common or shared environment (C) factors, or nonshared environment (E) effects. The genetic factors are correlated 1.0 in MZ twins because they are genetically identical. For DZ twins, the additive genetic factors are correlated 0.5 because DZ twins share on average half of their genes. The environment shared by a twin pair is assumed not to depend on the zygosity, and thus the shared environmental factors correlate 1.0 in both MZ and DZ twins. E or nonshared environment is, by definition, uncorrelated. All uncorrelated error is also absorbed in the E term. The parameters a, c, and e are loadings of the observed phenotype on the latent factors A, C, and E and indicate the degree of relationship between the latent factors and the observed phenotype. The proportion of the variation accounted for by heritability or environmental influences is calculated by squaring the parameters a, c, and e and dividing them by the total variance ($a^2 + c^2 + e^2$). In addition, in the univariate model, the effects of sibling interaction (path = *s*) are also considered. The sibling interaction is characterized by the behavior of one twin having an effect on the behavior of the other twin. The interaction effect may also be the result of bias in parent reports when parents rate their children's behavior in comparison with each other. When the path *s* is positive, then high parental ratings on twin 1 for ODB will lead to high parental rating on twin 2's ODD; however, when *s* is negative, high ratings on ODB for twin 1 will lead to lower ratings on ODD for twin 2.

All model fitting was performed on raw data with Mx (Neale, 1997). The basic model was an ACE model with gender effects in means and variances. The possible presence of an interaction component was tested by equating the variances between MZ and DZ twins. If these variance differences are nonsignificant, then the presence of sibling interaction or rater bias is not plausible-an important distinction, given that sibling interaction effects have been postulated in ADHD (Bartels et al., 2003; Hudziak et al., 2000; van den Oord et al., 1994) and CD (Simonoff et al., 1995) but are not consistently found (Eaves et al., 2000). The significance of the A and C factors or sibling interaction was tested by dropping the variance components, using the χ^2 difference test. The χ^2 statistic is computed by subtracting the –2 log-likelihood for the full model from that for a reduced model. The degrees of freedom (*df*) for this test is the difference in the number of estimated parameters between the full and the reduced model. We also computed likelihood-based 95% confidence intervals (Neale, 1997; Neale and Miller, 1997). More technical details of genetic model-fitting analyses are reviewed elsewhere (Neale et al., 1992).

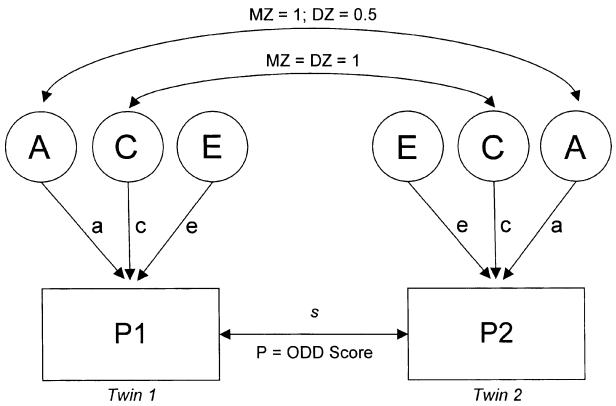

Fig. 1 Genetic model.

RESULTS

Means and Correlations

The means of the square-root–transformed Oppositional scores are shown in Table 3. We conducted 2 × 3 univariate analyses of variance to test differences in mean ODD scores between groups based on informant status (i.e., mother report, father report, teacher report) and gender of child. Analyses of variance were computed separately for older and younger twins. Main effects for gender and informant status were significant for first-born twins ($F_{1,2,542} = 34.30$, $p < .001$ and $F_{2,2,541} = 274.60$, $p < .001$, respectively). Post hoc Tukey tests were calculated between informant groups. Means were not significantly different between mother and father report, whereas both parent report groups were significantly higher than the teacher group ($p < .001$ for all sets). This is perhaps not surprising because there are six CRS ODD items in the parent checklist and only five CRS ODD items in the teacher checklist. Analysis of variance results for the younger twin were similar to those for the elder twin.

The Pearson twin correlations for the Oppositional scores are shown in Table 2. In both boys and girls, the MZ correlations were larger than were the DZ correlations, indicating the influences of genes. The MZ and DZ correlations were not different across genders with the exception of teacher reports. In addition, the opposite-sex correlations equaled the same-sex correlations for mothers and fathers, suggesting that the same genes and environmental influences play a role in boys and girls. The teacher reports revealed gender differences and remarkably lower opposite-sex correlations than same-sex correlations.

Model Fitting

A summary of the model fitting results is given in Table 4. The difference in χ^2 indicates the goodness of fit of the model as compared with a saturated model.

TABLE 3
Mean Square-Root Transformed ODD Scores and Standard Deviations by Gender, Zygosity, and Informant

	Mean Score (SD)		
Zygosity	Mother	Father	Teacher
MZm	1.95 (0.91)	1.84 (0.91)	0.45 (0.83)
DZm	1.94 (0.96)	1.88 (1.01)	0.55 (0.83)
MZf	1.68 (0.94)	1.66 (0.99)	0.28 (0.64)
DZf	1.69 (0.94)	1.70 (0.94)	0.30 (0.64)
DOSm	1.86 (0.97)	1.80 (0.97)	0.44 (0.74)
DOSf	1.81 (1.00)	1.74 (0.99)	0.45 (0.77)
Total males	1.93 (0.97)	1.85 (0.97)	0.56 (0.84)
Total females	1.70 (0.94)	1.68 (0.97)	0.29 (0.64)

Note: MZm = monozygotic male; DZm = dizygotic male; MZf = monozygotic female; DZf = dizygotic female; DOSm = opposite sex, male; DOSf = opposite sex, female.

First, variance differences between MZ and DZ twins were tested. The fit of a model that constrained the variances to be equal was compared with the fit of a fully saturated model in which all variances and covariances were freely estimated. The variances were not significantly different; therefore, the presence of sibling interaction or rater bias is not plausible. Parent ratings of ODB showed significant influences of A, C, and E. These influences were similar in boys and girls. For mother reports, A accounted for 57% of the variance, E accounted for 29% of the variance, and C accounted for 14% of the variance. Analyses of father reports varied little from those of mothers, with A accounting for 55%, E accounting for 22%, and C accounting for 23% of the variance. Analysis of the teacher reports were best fit by an ACE model, but here gender differences could not be discounted. For boys, A accounted for 38%, C accounted for 23%, and E accounted for 39% of the variance. For girls, the estimates were 21%, 31%, and 48% for A, C, and E, respectively.

DISCUSSION

Despite the common perception among the lay public that ODB is caused by poor parenting and multiple sources of information for the lay public on the Internet that include only learning theory and disordered development as the probable causes of ODD, we have found that models from mother, father, and teacher reports all point to the influence of additive genetic factors in both boys and girls. These data support the idea that ODB is highly influenced by genetic as well as environmental factors. Genetic modeling also revealed slight differences between parent and teacher ratings. Parent reports did not reveal gender differences in estimates of A, C, and E, whereas teacher reports showed different estimates in boys and girls. Teachers rate ODB as more heavily influenced by genetic factors in boys than in girls (38% in boys as compared with 21% in girls), with more of the variance attributed to environmental effects in girls. Analyses of teacher ratings reveal that ODB symptoms were mainly influenced by environmental factors, with approximately 62%–79% of the variance attributed to either shared or unique environmental factors as compared with 43%–45% by parent report. These data on the environmental contributions to oppositional behavior in school will be the focus of future work by our team. One alternate reason for the differences in ratings for parent versus teacher models could be that some twins were in separate classrooms and were, therefore, rated by different teachers. There were no significant findings of rater contrast or bias in the teacher models, however, and these differences would have been evident in that part of the model had they been significant.

Clinical Implications

These data on etiologic contributions to ODB may assist both research scientists and clinicians in their approaches. This study provides evidence that the construct of oppositionality as measured by the Conners Short Forms is similar in its heritability to other measures of ODD, thus supporting its use in measuring oppositionality in clinical practice. Further study of the relationship between the construct of oppositionality as measured by the Conners forms, the AGG scale of the CBCL, and the *DSM-IV* ODD symptoms seems warranted and is a current emphasis of our group's work.

Future work combining the relative utility of combining all informants in phenotypic definition will be a focus for study by our group as our data set grows. These data may be helpful to clinicians as they appear to support a robust gender difference in the rates of ODB that will be identified. Furthermore, this work argues that much more work on the etiologic contributions to ODB needs to be done.

Limitations

The major limitation of this report is that the Conners Oppositional scale does not include all eight of the

TABLE 4
Univariate Model Fitting of Square-Root–Transformed Ratings of Conners Oppositional Scale With Parameter Estimates and 95% Confidence Interval for Best-Fitting Models

	–2 ll	No. of Param	Compared to Model	Δdf	$\Delta\chi^2$	p	A	C	E
Maternal									
Fully saturated	7,962.28	30	—	—	—	—			
Equal variances MZ/DZ	7,966.65	26	1	4	4.36	.359			
ACE, with sex differences	7,994.47	18	1	12					
AE, with sex differences	8,002.62	16	3	2	8.157	.017			
CE, with sex differences	8,075.11	16	3	2	80.65	.000			
ACE, no sex differences	7,998.73	15	3	3	4.26	.235	0.57 (0.46–0.69)	0.14 (0.04–0.24)	0.29 (0.26–0.33)
Paternal									
Fully saturated	5,519.92	30	—	—	—	—			
Equal variances MZ/DZ	5,525.67	26	1	4	5.76	.218			
ACE, with sex differences	5,530.59	18	1	12	10.67	.557			
AE, with sex differences	5,545.67	16	3	2	15.09	.001			
CE, with sex differences	5,609.19	16	3	2	78.60	.000			
ACE, no sex differences	5,531.27	15	3	3	0.68	.877	0.55 (0.44–0.67)	0.23 (0.12–0.33)	0.22 (0.19–0.26)
Teacher									
Fully saturated	6,889.11	30	—	—	—	—			
Equal variances MZ/DZ	6,894.24	26	1	4	5.13	.274			
ACE, with sex differences	6,908.65	18	1	12	19.54	.076	Male 0.38 (0.20–0.55) Female 0.21 (0.3–0.38)	Male 0.23 (0.9–0.37) Female 0.31 (0.17–0.46)	Male 0.39 (0.32–0.47) Female 0.48 (0.41–0.56)
AE, with sex differences	7,012.32	16	3	2	103.67	.000			
CE, with sex differences	6,986.50	16	3	2	77.855	.000			
ACE, no sex differences	7,050.39	15	3	3	141.74	.000			

Note: –2 ll = –2 log likelihood; No. of param = number of estimated parameters; A = additive genetic; C = shared environmental; E = unique environmental.

DSM-IV ODD criteria. Therefore, despite the fact that the ODD checklist began with all eight items and factor analyses done by Conners (2001) yielded evidence that the five or six items retained were the most highly loaded, we cannot argue that we collected data or fit genetic models on all eight of the *DSM-IV* ODD data. We are collecting *DSM-IV* interview ODD data in an older population of twins that can be compared with CRS

Oppositional data to test the relationship between the two in a large general population sample. In addition, although it is likely that the construct of ODD as measured by the Conners Teacher forms and the Parent forms are similar, given that they were constructed to detect a specific *DSM-IV* phenotype of ODD, they are not identical. The scales contain different items because children have the opportunity to express different oppositional behavior in the classroom than they do at home. This leads to the possibility that the findings of differences in the genetic models are a result of different items on the scales. One alternate possibility for the higher ratings of oppositionality by the parents is that parents may have more occasions to request compliance from children, although these data cannot be related to that issue.

Another limitation of the study is that we are unable in a basic twin design to investigate parent–child interaction effects. It is, of course, possible that interactional temperamental effects may play a role in the expression and reporting of oppositional behavior. For example, our group has shown that the combination of the temperamental measure of high novelty seeking in a child with high novelty seeking in the mother is associated with child externalizing problems (Rettew et al., 2003). Further work with a twin and family study design with measurement of child, parental, and possibly teacher temperamental factors would be necessary to resolve this complicated issue. Finally, we did not include information about cooccurring ADHD in this sample. As the sample size grows with continued recruitment, we will be able to generate bivariate models that will include information about ADHD and oppositionality and the genetic and environmental influences that they have in common.

This study was done in the Netherlands, which has an ethnic (and therefore genetic) composition that is different from that of the United States and where cultural attitudes toward child behavior may lead to differing child-rearing practices. Because the parental socioeconomic status was somewhat higher than in the general Dutch population, it is likely higher than in the general U.S. population. In addition, the child-rearing environment of twins is likely to differ from that of singletons.

Of course, genetic models of childhood psychopathology are only as good as the phenotype being studied (Hudziak, 1996, 2001). Here, we examine a commonly used phenotype for oppositional behavior, but further refinement of the phenotype using alternate diagnostic criteria (Angold and Costello, 1996), alternate measurement tools, and alternate analytic tools such as latent class analysis alone and in combination will be important to accurately understand the genetics of this common and important condition.

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