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An Examination of the Developmental Pathways Model for Oppositional Defiant Disorder in a Twin Sample

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An Examination of the Developmental Pathways Model for

Oppositional Defiant Disorder in a Twin Sample

by

Debra L. Boeldt

B.A., University of Wisconsin, 2004

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An Examination of the Developmental Pathways Model for
Oppositional Defiant Disorder in a Twin Sample
written by Debra Lynn Boeldt
has been approved for the Department of Psychology and Neuroscience

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Date____________________

The final copy of this thesis has been examined by the signatories, and we find that both the content and the form meet acceptable presentation standards of scholarly work in the above mentioned discipline.

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An Examination of the Developmental Pathways Model for Oppositional Defiant Disorder in a Twin Sample

Thesis directed by Associate Professor Soo H. Rhee

A recent model suggested by Loeber and Burke (2011) attempts to clarify the association between ODD and the development of later internalizing and externalizing disorders. In this model, there are two separate clusters of ODD symptoms, behavioral and affective symptoms. The affective cluster of symptoms consists of ODD symptoms “touchy”, “spiteful/ vindictive”, and “angry”, and is more likely to lead to internalizing disorders. The behavioral cluster of symptoms consists of ODD symptoms “arguing”, “being defiant”, and “losing one’s temper” and is more likely to lead to externalizing disorders. The current genetically informative longitudinal study aimed to further the understanding of ODD by examining the relations between ODD symptoms assessed during early to late adolescence and MDD, GAD, and CD assessed five years later. Exploratory factor analyses suggest evidence of a two-factor model of ODD symptoms consistent with the Loeber and Burke (2011) model; however, there was also evidence of cross loadings. Findings from biometrical genetic analyses suggest a single set of genetic and nonshared environmental influences explaining the covariance among the ODD symptoms, and little evidence of shared environmental influences. The correlations between negative affect and oppositional behavior symptoms in adolescence and later MDD and GAD were significant. In contrast, the correlation between ODD symptoms and later CD was not significant. Although the overall covariance between negative affect/oppositional behavior and MDD/GAD was
significant, there was inadequate power to determine whether this covariance was due to genetic, shared environmental, and nonshared environmental influences.
CONTENTS

CHAPTER

I. INTRODUCTION ................................................................................................................. 1

II. METHODS .......................................................................................................................... 8
   Participants .......................................................................................................................... 8
   Procedure ......................................................................................................................... 9
   Analyses .......................................................................................................................... 10
   Factor Analysis .............................................................................................................. 13
   Phenotypic Correlations ................................................................................................. 14

III. RESULTS .......................................................................................................................... 20
   Factor Analyses .............................................................................................................. 20
   Correlations ................................................................................................................... 23
   Biometrical Models ......................................................................................................... 29

IV. DISCUSSION ..................................................................................................................... 37

REFERENCES ......................................................................................................................... 42
Table 1. Factor Analysis Results of ODD from Prior Studies ............................................4

2. Alternative Confirmatory Factor Analytic Models of ODD to be Tested in Current Study ..................................................................................................................7

3. Prevalence of ODD Symptoms in Males and Females .................................11

4. Percentage of Males and Females with Diagnoses ........................................12

5. Factor Loadings from Exploratory Factor Analysis ........................................21

6. Factor Loadings from Confirmatory Factor Analysis ......................................22

7. Phenotypic Correlations Among ODD Symptoms .........................................24

8. Phenotypic Correlations Among ODD Symptoms and Later Diagnoses .......25

9. Within-trait Cross-twin Correlations .................................................................27

10. Cross-trait Cross-twin Correlations .................................................................28

11. Variance Explained by $a^2$, $c^2$, and $e^2$ .....................................................34
FIGURES

Figure

1. One Factor Model ................................................................. 15
2. Two Factor Model ................................................................... 16
3. Cholesky Decomposition Example: ODD symptoms and later diagnoses.. 18
4. Cholesky Decomposition Example: ODD symptoms and comorbidity among GAD/MDD/CD ................................................................. 19
5. One Factor Independent Pathway Model ........................................ 30
6. Two Factor Independent Pathway Model ..................................... 31
7. Full Cholesky Model ................................................................ 33
8. Cholesky Model: Examining MDD ............................................... 35
9. Cholesky Model: Examining GAD ................................................ 36
CHAPTER I

INTRODUCTION

According to the Diagnostic and Statistical Manual of Mental Disorders, oppositional defiant disorder (ODD) is diagnosed during childhood and characterized by a recurrent pattern of negativistic, defiant, disobedient, and hostile behavior toward authority figures (American Psychiatric Association, 2001). The prevalence rate for ODD has been estimated to vary between 2-14% in epidemiological samples and 28-50% in clinical samples (Boylan, Vaillancourt, Boyle, Szatmari, 2007). Lifetime prevalence of ODD has been estimated to be 10.2% in a nationally representative sample from the National Comorbidity Survey Replication (Nock, Kazdin, Hiripi, & Kessler, 2007). Rates have been shown to differ by gender, with higher rates of ODD in boys in an epidemiological study (Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). Moreover, the prevalence rate of lifetime diagnosis of ODD and comorbidity with other DSM-IV diagnoses such as a mood, anxiety, impulse-control, or substance use disorder ranged from 45.7% to 68.2% among 5 to 15 year olds, illustrating the importance of understanding the etiology of ODD.

Both ODD and CD begin in early childhood (Keenan et al., 2007; Keenan et al, 2011). One drawback of much early research on ODD is the fact that ODD was not examined as a distinct disorder, but often combined with CD or left out of analyses completely (Burke et al., 2005). This failure to examine ODD as a distinct disorder may have affected our overall understanding of the etiology of child psychopathology. Although ODD has previously been viewed as less serious than CD, recent research suggests that it does cause significant impairment. The diagnosis and occurrence of ODD is less benign than previously thought and
plays a significant role in the development of child psychopathology, ranging from anxiety and depression to conduct disorder and antisocial personality disorder (Loeber, Burke, & Pardini, 2009).

Given the importance of understanding ODD, several studies have examined ODD during childhood and later comorbid disorders, both externalizing and internalizing (Reef et al., 2010; Burke et al., 2005). For instance, children with ODD followed longitudinally for five years showed increasing comorbidity between ODD and other disorders such as ADHD, anxiety, and mood disorders (Lavigne, Cicchetti, Gibbons, Binns, Larsen, DeVito, 2001). Lavigne and colleagues (2001) also found moderate to high stability in ODD, and children who met criteria for a diagnosis of ODD in preschool were more likely to exhibit ODD comorbid with another disorder later in elementary school. Research findings also suggest that ODD is a strong predictor of early-onset CD (Burke, Waldman, Lahey, 2010). In addition, a review examining the comorbidity between ODD and internalizing disorders found that 10-20% of children with ODD during the preschool years also have comorbid internalizing disorders (Boylan, Vaillancourt, Boyle & Szatmari, 2007). Also, older youth with ODD had a higher rate of comorbidity with depression (15-46%) and anxiety (7-14%), suggesting that the rate of comorbidity may vary with age. Furthermore, internalizing disorders appeared to be more common in children who experienced persistent ODD (Boylan et al., 2007). These findings emphasize the importance of understanding the complexity of ODD, including the need to consider development when examining co-occurring internalizing and externalizing disorders.

Several theoretical models have been proposed to elucidate the trajectory of ODD from childhood to adulthood (Burke, Loeber, Lahey, & Rathouz, 2005; Loeber, Burke, Lahey, Winters, & Zera, 2000, Loeber & Burke, 2011). Loeber and Burke (2011) suggest that
examining the trajectories of ODD, CD, and internalizing behaviors will provide more clarity in understanding childhood behavior problems. A proposed developmental model suggested by Loeber and Burke (2011) attempts to clarify the association between ODD and the development of later internalizing and externalizing disorders. In this model, there are two separate clusters of ODD symptoms, behavioral and affective symptoms. ODD symptoms that occur during early childhood are risk factors for later externalizing disorders (i.e. CD and antisocial personality disorder) or internalizing disorders (i.e. anxiety and depression). The affective cluster consists of the ODD symptoms “touchy”, “spiteful/ vindictive”, and “angry”, and is more likely to lead to internalizing disorders. The behavioral cluster consists of the ODD symptoms “arguing”, “being defiant”, and “losing one’s temper” and is more likely to lead to externalizing disorders.

Several studies have examined the factor structure of the ODD symptoms, and the results of these studies have been inconsistent. A description of the samples, measures, and factor analysis results from these studies are presented in Table 1. Burke and colleagues (2005) found a two-factor model fit the data best when examining ODD in a sample of boys. In contrast, two other studies found evidence of a third factor (Burke et al., 2010, Stringaris & Goodman, 2009), with differing results. One study (Burke et al., 2010) found that the third factor consisted of “antagonistic” behaviors (e.g. annoys others and blames), whereas another study (Stringaris & Goodman, 2009) found a third factor consisting of “hurtful” behaviors (e.g. spiteful or vindictive behavior).

Despite some variability in results of studies examining the factor structure of ODD symptoms, several studies have reported evidence that the negative affective component is more
Table 1. Factor analysis results of ODD from prior studies

<table>
<thead>
<tr>
<th>Author</th>
<th>Sample &amp; Measure</th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Factor 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burke, Loeber, Lahey, &amp; Rathouz, 2005</td>
<td>Longitudinal 177 boys Diagnostic Interview Schedule for Children (ODD and ADHD, Parent Report; CD, overanxious, and depression, parent and child)</td>
<td>Behavioral Factor</td>
<td>Affective Factor</td>
<td>Argues Defies Loses Temper Touchy Angry Spiteful or Vindictive</td>
</tr>
<tr>
<td>Burke, Hipwell, &amp; Loeber, 2010</td>
<td>Longitudinal 2,451 girls Child Symptom Inventory (ODD, Parent and Teacher Report; CD, Child and Parent Report; depression, anxiety ADHD, Parent report)</td>
<td>Behavioral Factor</td>
<td>Affective Factor</td>
<td>Argues Defies Loses Temper Touchy Angry Spiteful or Vindictive Annoys Others Blames</td>
</tr>
<tr>
<td>Stringaris &amp; Goodman, 2009</td>
<td>Cross-sectional 18,415 children 5-16 year olds Development and Well-Being Assessment (Parent and Teacher Report)</td>
<td>Irritable</td>
<td>Headstrong</td>
<td>Hurtful</td>
</tr>
</tbody>
</table>
likely to lead to future internalizing disorders, whereas the oppositional behavior component is
more likely to lead to future CD (Burke, Hipwell, & Loeber, 2010; Burke & Loeber, 2010;
Burke, Loeber, Lahey, & Rathouz, 2005; Stringaris & Goodman, 2009), supporting Loeber and
Burke’s model (2011).

The examination of genetic and environmental influences can provide additional
information regarding the developmental trajectory of ODD and the later development of
internalizing and externalizing behavior. Prior research has examined the underlying genetic and
environmental factors influencing ODD and suggests both genetic and environmental influences
on ODD (Lahey, Van Hulle, Rathouz, Rodgers, D’Onofrio, & Waldman, 2009; Rowe, Rijsdijk,
Maughan, Hosang, & Eley, 2008). Similarly, a study by Dick and colleagues (Dick, Viken,
Kaprio, Pulkkinen, & Rose, 2005) found that the comorbidity among ODD, CD, and ADHD is
due to common genetic influences, although the individual disorders also have unique genetic
influences. In regard to internalizing behavior, a meta-analysis of major depression suggests both
genetic and environmental influences (Sullivan, Neale, & Kendler, 2000). Similarly, genetic
influences account for approximately 15-20% of the variability in GAD (Hettema, Prescott, &
Kendler, 2001). Moreover, the genetic risk factors underlying MDD and GAD are highly
correlated (Kendler, Gardner, Gatz, & Pedersen, 2007). However, few genetically informative
studies have examined the developmental trajectory of ODD and both internalizing and
externalizing disorders. To our knowledge, this is the first study examining the genetic and
environmental contributions in the relationship between ODD and later internalizing disorders.

Results of previous studies examining the development of ODD in both boys and girls
have been mixed. A study by Tuvblad and colleagues (2009) examined the development of co-
occurring disruptive behavior diagnoses in boys and girls, and found that the parallel
development of ODD and CD symptoms did not differ by gender (Diamantopoulou et al., 2011). In contrast, a longitudinal study examining a community sample of children from 9-16 years found ODD was a strong predictor of CD in boys but not in girls; instead, girls’ ODD was associated with later increased risk for ODD, depression, and anxiety (Rowe, Maughan, Pickles, Costello & Angold, 2002). In regards to behavioral genetics research, one study found that the heritability of CD was similar for boys and girls, but greater heritability for ODD in boys than girls (Dick et al., 2005). In summary, several studies suggest the possibility of important gender differences in the development of ODD and the importance of examining both boys and girls in studies of ODD.

The current study aims to further the understanding of ODD by examining the relations between ODD symptoms assessed during early to late adolescence and MDD, GAD, and CD assessed five years later using the longitudinal data collected by the Colorado Center for Antisocial Drug Dependence (CADD; DA011015; PI: Hewitt). We will address two primary aims and three secondary aims examining genetic and environmental influences.

First, we aim to replicate the proposed two-factor structure for ODD (specifically, the distinction between the affective and behavioral dimensions in ODD), testing alternative models based on factor analyses results from prior studies examining the factor structure of ODD (see Table 2). We hypothesize that the ODD symptoms will consist of two factors. More specifically, the factor structure will be consistent with prior studies (e.g. affective and behavioral factors) by Burke and colleagues (2010).

Second, we aim to examine the associations between ODD assessed at baseline (wave 1) and CD, MDD and GAD assessed five years later (wave 2). We hypothesize that the affective dimension of ODD will be significantly associated with the development of depression or
Table 2. Alternative confirmatory factor analytic models of ODD to be tested in current study

<table>
<thead>
<tr>
<th>Models</th>
<th>Variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Factor</td>
<td>Argues Defies Loses Temper Touchy Angry Spiteful Annoys Others Blames</td>
</tr>
<tr>
<td>2 Factor Burke et al., 2005</td>
<td>Argues Defies Loses Temper Touchy Angry Spiteful</td>
</tr>
<tr>
<td>3 Factor Burke et al., 2010</td>
<td>Argues Defies Loses Temper Touchy Angry Spiteful Annoys Others Blames</td>
</tr>
<tr>
<td>3 Factor Stringaris &amp; Goodman, 2009</td>
<td>Loses Temper Touchy Angry Argues Defies Annoys Others Blames Spiteful</td>
</tr>
</tbody>
</table>
anxiety, whereas the behavioral dimension is significantly associated with the development of CD later in adolescence.

Third, we aim to examine the factor structure of genetic and environmental influences on ODD. Regardless of the phenotypic factor structure of the ODD symptoms, we will conduct alternative biometrical genetic analyses (i.e., single underlying latent factor vs. affective/behavioral dimensions vs. affective/behavioral/antagonistic dimensions) examining the genetic and environmental influences on ODD symptoms, as the results may differ for the phenotypic factor structure and the genetic/environmental influences. This is the first study examining alternative models for the factor structure of genetic influences on ODD; we do not have specific hypotheses regarding the best-fitting model.

Fourth, we aim to examine the comorbidity between ODD in wave 1 and GAD, MDD, and CD in wave 2. Although genetically informative studies of ODD and the association between ODD and other disorders have been conducted (Burke & Loeber, 2002; Dick, Viken, Kaprio, Pulkkinen, & Rose, 2005; Nadder, Rutter, Silberg, Maes, & Eaves, 2002), studies incorporating new evidence regarding the separate dimensions within ODD have not been conducted yet, and we do not have specific hypotheses regarding the amount of covariance between ODD and GAD/MDD/CD that are accounted for by genetic and environmental factors.

Fifth, we aim to examine sex differences in the development of externalizing behaviors. Given evidence suggesting possible sex differences in the trajectory of ODD, alternative models will be conducted to examine whether results differ in males and females. These are exploratory analyses, and we do not have any specific hypotheses regarding gender differences.

Methods

Participants
The current study used data obtained from The Center for Antisocial Drug Dependence (CADD), which is an ongoing, multi-component, collaborative study at the Institute for Behavioral Genetics (IBG) at the University of Colorado-Boulder and University of Colorado-Denver. The sample consisted of 2746 adolescents from the CADD, and includes 1373 twin pairs. More specifically, there are 361 female and 296 male MZ twin pairs, 222 female and 220 male DZ twin pairs, and 274 opposite sex DZ pairs. There were 2746 adolescents assessed during wave 1 (mean age= 14.55; ranging from 11 to 19 years old) and 2434 adolescents assessed during wave 2 (mean age=20.30; ranging from 16-29 years old). Samples are from two community based samples: the Colorado Longitudinal Twin Sample (LTS) and Community Twin Sample (CTS). Rhea et al., (2006) provides further detailed information regarding recruitment and additional description of the samples. The participants from the LTS sample have been studied since birth, whereas, the participants from the CTS samples were recruited for the first time by the CADD. Males (48%) and females (52%) are similarly represented among the adolescence.

Procedure

The zygosity of twin pairs was determined through genotyping of 11 highly informative short tandem repeat polymorphisms. Twins with identical genotype markers were classified as MZ pairs, whereas twins who were not similar on these markers were classified as DZ pairs. DNA was extracted through epithelial cells obtained via cheek swabs. The extraction, storage, and genotyping were completed by IBG faculty for the CTS and LTS samples.

The present study examined internalizing disorders (MDD and GAD) and externalizing disorders (ODD and CD) assessed via adolescent interviews with the Diagnostic Interview Schedule for Children-IV (DISC-IV; Shaffer et al, 2000). During wave 1, ODD was assessed via
self-report using the DISC-IV at age 11 to 19 years. Table 3 presents the sample sizes for each of the individual ODD symptoms and the percentage in comparison to the entire sample size for males and females. We examined wave 1 ODD as risk factors for wave 2 diagnoses of Generalized Anxiety Disorder (GAD), Major Depressive Disorder (MDD), or Conduct Disorder (CD) assessed via self-report using the DISC or DIS approximately five years later (age 17 to 22). The DISC-IV is a structured psychiatric interview used to assess DSM-IV (American Psychiatric Association, 1994) symptoms and diagnoses for Axis I disorders. The assessment examines both lifetime and past year prevalence of disorders. The DISC-IV has shown moderate to good validity (Schwab-Stone et al, 1996) and reliability (Shaffer et al., 1996). Ehringer et al. (2006) reported the prevalence rates for MDD, SAD, GAD, ADHD, CD, and ODD in a subset of the present sample; these rates were similar to the rates reported in the Methods for the Epidemiology of Child and Adolescent Mental Disorders Study (Shaffer et al., 1996). During wave 2, participants who were older than age 18 were assessed via the Diagnostic Interview Schedule (DIS; Robins et al, 1989) instead of the DISC. The DIS has also shown good validity in general population samples (Anthony et la., 1985; Helzer et al., 1985, Robins et al., 1981). The DISC-IV and DIS were administered by trained research assistants. Computer algorithms were developed to determine presence or absence of symptoms and diagnosis for disorders according to the instrument’s authors. The individuals were categorized as having no symptoms, one or more symptoms, or a diagnosis of a disorder during the past year and lifetime (Table 4).

Analyses

The analyses examining the structure of ODD, association between ODD and later CD, MDD, and GAD, as well as the multivariate genetic analyses were conducted in Mplus (Muthen and Muthen, 1998-2004). The data were analyzed with the assumption that a normal continuous
Table 3. Prevalence of ODD symptoms in males and females

<table>
<thead>
<tr>
<th>ODD Symptoms</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(％ of 1566 individuals)</td>
<td>(％ of 1712 individuals)</td>
</tr>
<tr>
<td>Loses Temper</td>
<td>97(6)</td>
<td>138 (8)</td>
</tr>
<tr>
<td>Argues with Adults</td>
<td>121(8)</td>
<td>186(12)</td>
</tr>
<tr>
<td>Refuse to Comply</td>
<td>77(5)</td>
<td>89 (7)</td>
</tr>
<tr>
<td>Easily Annoyed</td>
<td>68(4)</td>
<td>113(7)</td>
</tr>
<tr>
<td>Angry and Resentful</td>
<td>83(5)</td>
<td>103(6)</td>
</tr>
<tr>
<td>Spiteful/Vindictive</td>
<td>49(3)</td>
<td>27(2)</td>
</tr>
<tr>
<td>Deliberately annoys others</td>
<td>62(4)</td>
<td>43(3)</td>
</tr>
<tr>
<td>Blames others</td>
<td>10(1)</td>
<td>12(1)</td>
</tr>
</tbody>
</table>
Table 4. % of males and females with MDD, GAD, and CD symptoms and diagnoses

<table>
<thead>
<tr>
<th></th>
<th>MDD (% of 1159 males and 1266 females)</th>
<th>GAD (% of 1161 males and 1266 females)</th>
<th>CD (% of 1159 males and 1266 females)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 symptoms</td>
<td>At least 1 symptom</td>
<td>DSM-IV Criteria</td>
</tr>
<tr>
<td>Male</td>
<td>963(83)</td>
<td>144(12)</td>
<td>52(4)</td>
</tr>
<tr>
<td>Female</td>
<td>941(74)</td>
<td>197(16)</td>
<td>128(10)</td>
</tr>
</tbody>
</table>
liability distribution underlies the ordinal variables (i.e., 0=no symptoms, 1= one or more symptoms, and 2=diagnosis) since the DSM-IV psychiatric symptoms are highly skewed in the community sample. There are several statistical advantages of the normality assumptions such as preserving underlying liability, retaining an explicit mapping between the underlying liability and observed data, and providing accurate measures of the underlying correlation and parameter estimates (Stallings et al., 2001; Derks et al, 2004).

Factor Analysis

An exploratory factor analysis (EFA) was conducted in Mplus to determine whether the symptoms of ODD loaded on affective and behavioral factors. Given the inconsistent findings presented in Table 1 and to further understand the factor structure of ODD, we also conducted confirmatory factor analyses to test alternative models to determine the best fitting model for ODD (see Table 2). The percentage of individuals with each ODD symptom in males and females is presented in Table 3. We intended to examine one-, two-, and three-factor models suggested by previous results. For the two-factor model, we planned to examine behavioral (argues, defies, loses temper) and affective (touchy, angry, spiteful or vindictive) factors (Burke et al., 2005). The three-factor model consisted of behavioral (argues, defies, loses temper), affective (touchy, angry, spiteful or vindictive), and antagonistic (annoys others, blames) factors (Burke et al., 2010). For a second three-factor model, we planned to examine irritable (loses temper, touchy, angry), headstrong (argues, defies, annoys others, blames), and hurtful (spiteful or vindictive) factors (Stringaris & Goodman, 2009). We examined whether factor loadings could be equated across sexes by testing measurement noninvariance models (where factor loadings are free to vary across sexes) and measurement invariance models (where factor loadings are equated across sexes). For females, there was non-convergence of the data in the
three-factor models, which prevented the further examination of the three-factor models in the analyses. Non-convergence of the data in the three-factor model may have resulted from the small number of ODD variables (eight) in the model.

**Phenotypic Correlations**

Phenotypic correlations among the negative affective and oppositional behavior symptoms of ODD at wave 1 and symptoms/diagnoses of MDD, GAD, and CD at wave 2 were calculated. The percentage of males and females with no symptoms, at least one symptom, or diagnosis of MDD, GAD, and CD are presented in Table 4. The within-trait cross-twin correlations (i.e., correlations between twin 1 and twin 2 for the same variable) and cross-trait cross twin correlations (i.e., correlations between twin 1 and twin 2 for different variables) were calculated for MZ and DZ twin pairs for affective ODD symptoms, behavioral ODD symptoms, MDD, GAD, and CD. The covariation among MDD, GAD, and CD was also examined.

**Multivariate Genetic Models**

In addition to phenotypic factor analyses of ODD symptoms, multivariate genetic analyses testing alternative models regarding the structure of genetic, shared environmental, and nonshared environmental influences on the ODD symptoms were tested. Table 2 presents the proposed models given prior research; however, we were unable to test the three-factor models. Also, we could not include the “angry” variable due to bivariate missingness in the data in analyses testing the one- and two-factor models depicted in Figures 1 and 2. That is, because the prevalence of ODD symptoms was low in the community sample, there were several cases of missing cells (e.g. there were no twin pairs where the “angry” symptom was endorsed by both DZ twin 1 and DZ twin 2). Figure 1 depicts the adjusted one-factor model assuming a single underlying genetic or environmental factor influences of the five ODD symptoms. Figure 2
Figure 1. One-Factor Model. For the sake of simplicity, only the genetic influences are depicted.
Figure 2. Two-Factor Model. $A_{NA} =$ genetic influences of affective factor, $A_{OB} =$ genetic influences of behavioral factor. For the sake of simplicity, only the genetic influences are depicted.
suggests two separate genetic or environmental factors for affective or behavioral symptoms of ODD, but does not include the “angry” variable.

The choice of genetic models examining the relations between ODD and later MDD, GAD, and CD was dependent on the results of the genetic models examining ODD symptoms. The two-factor model fit the data best in the EFA and CFA analyses; therefore, a Cholesky decomposition model examining the role of affective and behavioral symptoms of ODD during wave 1 on MDD, GAD, and CD during wave 2 was examined. In the model shown in Figure 3, the first variable is affective symptoms of ODD, the second variable is behavioral symptoms of ODD, and the third variable is later diagnosis of MDD, GAD, or CD. The first factor influences affective symptoms, behavioral symptoms, and later diagnoses, the second factor only influences behavioral symptoms and later diagnoses, and the third factor only influences later diagnoses. The variance of later diagnosis (i.e. MDD, GAD, or CD) is divided into the variance shared in common with both affective and behavioral symptoms, the variance shared in common with only behavioral symptoms, and variance specific to the later diagnosis. Given high comorbidity among psychopathology diagnoses, the model in Figure 4 aims to examine whether common underlying genetic or environmental influences on ODD symptoms explain the comorbidity among GAD, MDD, and CD. As in Figure 3, the number of genetic, shared environmental, and non-shared environmental influences are equal to the number of variables in the model. For the sake of simplicity, only the genetic components are shown in Figures 3 and 4.

We were unable to compare and contrast models allowing parameters to be free across males and females to models fixing the parameters to be equal across gender due to bivariate missingness issues that resulted when the sample was divided into MZ and DZ groups and twin 1
Figure 3. Cholesky Decomposition Model MDD= Major Depressive Disorder, GAD= Generalized Anxiety Disorder, and CD= Conduct Disorder. The covariance of affective symptoms (NA) on behavioral symptoms (OB) of ODD with a later diagnoses of MDD, GAD, or CD. For the sake of simplicity, only the genetic influences are depicted.
Figure 4. Cholesky Decomposition Model MDD= Major Depressive Disorder, GAD= Generalized Anxiety Disorder, and CD= Conduct Disorder. The covariance of affective symptoms (NA) on behavioral symptoms (OB) of ODD with a later diagnoses of MDD, GAD, or CD. Due to space the genetic model of the ACE model is depicted.
and twin 2. This issue affected all of the multivariate biometrical models. The biometrical analyses did include age as a covariate.

Results

Factor Analyses

Table 5 presents the factor loadings from the EFA. When examining all eight ODD variables, the results did not converge for the three-factor model in females. Therefore, we focused on the one- and two-factor models of the six variables examined by Loeber and Burke (2011). The results examining the one- and two-factor models including the six variables proposed by Burke et al. (2005) did not suggest significant sex differences (one-factor, $\chi^2(4) = 7.120$, $p = .13$); two-factor, $\chi^2(6) = 9.926$, $p = .13$). The difference in the fit of the models comparing the one-factor and two-factor measurement invariance models examining the six variables was significant, ($\chi^2(8) = 47.395$, $p < .01$), suggesting that the two-factor model fits the data better than the one-factor model. The factor loadings suggested that the negative affect items had significant loadings on one factor, and the oppositional behavior items had significant loadings on another factor, although there were also significant cross-loadings. Overall, EFA findings support prior research suggesting a two-factor structure (Burke and et al., 2005).

CFA models were conducted examining both a single factor and two factor models consistent with the variables examined by Burke et al. (2005). The CFA results are presented in Table 6. The model constraining parameters to be equal across genders did not result in a decrement in fit for both the one-factor ($\chi^2(5) = 7.345$, $p = .1962$) and two-factor ($\chi^2(4) = 7.347$, $p = .1186$) models. Therefore, results from the measurement invariance model are presented. Both the one factor and two factor models examining the Loeber & Burke (2011) variables fit adequately (one factor, $\chi^2(23) = 66.435$, $p < .01$; CFI = .99, RMSEA = 0.034; two factor, $\chi^2(20)$
Table 5. Factor loadings from exploratory factor analysis of Loeber ODD symptoms

<table>
<thead>
<tr>
<th></th>
<th>1 Factor</th>
<th></th>
<th>2 Factor</th>
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<tr>
<td></td>
<td></td>
<td>Factor 1</td>
<td>Factor 2</td>
<td></td>
</tr>
<tr>
<td>Loses Temper</td>
<td>.87**</td>
<td>.72**</td>
<td>.21*</td>
<td></td>
</tr>
<tr>
<td>Argues with Adults</td>
<td>.91**</td>
<td>.55**</td>
<td>.43**</td>
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<tr>
<td>Refuse to Comply</td>
<td>.90**</td>
<td>.001</td>
<td>1.1**</td>
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<tr>
<td>Easily Annoyed</td>
<td>.77**</td>
<td>.91**</td>
<td>-.012</td>
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<tr>
<td>Angry and Resentful</td>
<td>.85**</td>
<td>.88**</td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td>Spiteful/Vindictive</td>
<td>.73**</td>
<td>.39*</td>
<td>.39**</td>
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</table>

*p < .10; *p < .05; **p < .01
Table 6. Factor loadings from confirmatory factor analysis of Loeber ODD symptoms

<table>
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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Factor 1 (NA)</td>
<td>Factor 2 (OB)</td>
</tr>
<tr>
<td>Loses Temper</td>
<td>.87**</td>
<td>.79**</td>
</tr>
<tr>
<td>Argues with Adults</td>
<td>.90**</td>
<td>.80**</td>
</tr>
<tr>
<td>Refuse to Comply</td>
<td>.90**</td>
<td>.90**</td>
</tr>
<tr>
<td>Easily Annoyed</td>
<td>.77**</td>
<td>.88**</td>
</tr>
<tr>
<td>Angry and Resentful</td>
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</tr>
<tr>
<td>Spiteful/Vindictive</td>
<td>.75**</td>
<td>.91**</td>
</tr>
</tbody>
</table>

*p < .10; * p < .05; ** p < .01
=57.288, p<.01; CFI=.99, RMSEA=0.034). The chi-square difference between the one- and two-factor model was significant ($\chi^2 (3) =10.528$, p=.01) suggesting the one-factor model fits significantly worse than the two-factor model. For the two-factor model, the correlation between the negative affect factor and the oppositional behavior factor was significant ($r=.90$, $p<.01$), suggesting that negative affect symptoms are highly correlated with oppositional behavior symptoms. Since the factor analysis results suggest that a two-factor model with the negative affect symptoms loading on one factor and the oppositional behavior symptoms loading on another factor fits significantly better than the one-factor model, negative affect and oppositional behavior are examined separately in subsequent analyses. Each ODD symptom was coded 0 or 1, for absent or present. The NA factor was created by combining the score from the three symptoms (touchy, angry, spiteful/vindictive; i.e., a score of 0 to 3). Similarly, the OB factor was created by combining the three proposed variables (argues, defies, loses temper; i.e., a score of 0 to 3).

Correlations

The phenotypic correlations among the individual ODD symptoms were highly significant (Table 7). Only five of the six variables are presented due to bivariate missingness for the “angry” variable in models examining twin data. The phenotypic correlations among the ODD symptoms and MDD, GAD, and CD variables are presented in Table 8. The correlation between the negative affect symptoms (not including angry) and oppositional behavior symptoms of ODD was high and significant (.77, $p<.01$). The correlation between negative affect symptoms and MDD/GAD was moderate and significant (.24 and .32), whereas negative affect was not significantly correlated with CD (.04). Similarly, oppositional behavior symptoms were moderately correlated with MDD (.25) and GAD (.31), but not CD (.01). Among the
Table 7. Phenotypic Correlations among oppositional behavior symptoms

<table>
<thead>
<tr>
<th></th>
<th>Temper</th>
<th>Argues</th>
<th>Refuse</th>
<th>Easily</th>
<th>Even</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temper</td>
<td>1</td>
<td>.76**</td>
<td>.76**</td>
<td>.67**</td>
<td>.70**</td>
</tr>
<tr>
<td>Argues</td>
<td>1</td>
<td>.84**</td>
<td>.59**</td>
<td>.61**</td>
<td></td>
</tr>
<tr>
<td>Refuse</td>
<td>1</td>
<td>.50**</td>
<td>.70**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easily</td>
<td>1</td>
<td></td>
<td>.45**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Even</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < .10; * p < .05; ** p < .01
Table 8. Phenotypic Correlations among negative affect and oppositional behavior symptoms of ODD, MDD, GAD, and CD

<table>
<thead>
<tr>
<th></th>
<th>Negative Affect</th>
<th>Oppositional Behavior</th>
<th>MDD</th>
<th>GAD</th>
<th>CD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative Affect</td>
<td>1</td>
<td>.77**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oppositional Behavior</td>
<td>1</td>
<td>.25**</td>
<td>.49**</td>
<td>.12+</td>
<td></td>
</tr>
<tr>
<td>MDD</td>
<td>1</td>
<td>.49**</td>
<td></td>
<td></td>
<td>-.03</td>
</tr>
<tr>
<td>GAD</td>
<td>1</td>
<td></td>
<td>.32**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < .10; * p < .05; ** p < .01
diagnoses, MDD and GAD were moderately correlated (.49), MDD was not significantly correlated with CD (-.03), but CD and GAD were modestly correlated (.12). Given the low and non-significant phenotypic correlations between NA/OB and CD, we decided to not examine CD in subsequent biometrical modeling analyses and only examine the association between NA/OB and MDD and GAD.

The within-trait cross-twin and cross-trait cross-twin correlations were conducted for MZ and DZ twin pairs for negative affect, oppositional behavior, MDD, GAD, and CD. The results are presented in Tables 9 and 10. The within-trait cross-twin correlation examines the correlation between twin 1 and twin 2 for the same variable and the cross-trait cross-twin correlation examines the correlation between twin 1 and twin 2 for different variables. The within-trait cross-twin correlation for the negative affect and oppositional factors were greater in MZ than in DZs, suggesting possible genetic influences. The correlations were more similar between MZ and DZ twins in MDD, GAD, and CD, but still higher in MZ than DZ twins, suggesting genetic influences. The cross-trait cross-twin correlations are similar to the within-trait cross-twin correlations for negative affect. Overall, cross-trait cross-twin MZ correlations were higher than DZ correlations for negative affect and MDD/GAD. The MZ and DZ correlations were similar for the cross-trait cross-twin correlations between oppositional behavior and MDD, suggesting shared environmental influences. In contrast, the cross-trait cross-twin correlation between oppositional behavior and GAD was higher in MZ than DZ twins, suggesting genetic influences. The cross-trait cross-twin correlation between MDD and GAD and the cross-trait cross-twin correlation between GAD and CD was higher in MZ than DZ twins, suggesting common genetic influences.
Table 9. Within-trait cross-twin correlations

<table>
<thead>
<tr>
<th></th>
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<th>DZ</th>
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</thead>
<tbody>
<tr>
<td>Negative Affect</td>
<td>.36**</td>
<td>.07</td>
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<tr>
<td>Oppositional</td>
<td>.43**</td>
<td>.18*</td>
</tr>
<tr>
<td>MDD</td>
<td>.33**</td>
<td>.25*</td>
</tr>
<tr>
<td>GAD</td>
<td>.39**</td>
<td>.29*</td>
</tr>
<tr>
<td>CD</td>
<td>.57**</td>
<td>.46**</td>
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</table>

*p < .10; *p < .05; **p < .01
Table 10. Cross-trait cross-twin correlations

<table>
<thead>
<tr>
<th></th>
<th>MZ</th>
<th>DZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>NA-OB</td>
<td>.30**</td>
<td>.10</td>
</tr>
<tr>
<td>NA-MDD</td>
<td>.21**</td>
<td>.14*</td>
</tr>
<tr>
<td>NA-GAD</td>
<td>.32**</td>
<td>-.02</td>
</tr>
<tr>
<td>NA-CD</td>
<td>.05</td>
<td>-.09</td>
</tr>
<tr>
<td>OB-MDD</td>
<td>.21*</td>
<td>.21**</td>
</tr>
<tr>
<td>OB-GAD</td>
<td>.38**</td>
<td>.03</td>
</tr>
<tr>
<td>OB-CD</td>
<td>.02</td>
<td>-.10</td>
</tr>
<tr>
<td>MDD-GAD</td>
<td>.32**</td>
<td>.19*</td>
</tr>
<tr>
<td>MDD-CD</td>
<td>.06</td>
<td>-.06</td>
</tr>
<tr>
<td>GAD-CD</td>
<td>.19*</td>
<td>.09</td>
</tr>
</tbody>
</table>

*p < .10; * p < .05; ** p < .01
Biometrical Models

Next, alternative models were conducted to determine whether there are one or two factors underlying genetic and environmental influences on the ODD symptoms. First, a one-factor model was fit to the data (Figure 5). The model included the five symptoms (i.e. touchy, spiteful, temper, argues, refuse), and excludes “anger” due to bivariate missingness in the twin data. The model fit well ($\chi^2 (97) =90.807$, p.6577; CFI=1.00, RMSEA=0.00). All genetic and nonshared environmental influences were statistically significant, but there were no significant shared environmental influences. A two-factor model, with separate but correlated genetic influences on the NA symptoms (touchy and spiteful) and OB symptoms (temper, argues, and refuse), was also examined (Figure 6). In order for the model to converge, we had to fix the correlation between shared environmental influences on NA and OB to 1, and this model fit well ($\chi^2 (94) =88.197$, p.5416; CFI=1.00, RMSEA=0.00). The results of the two-factor model are difficult to interpret because the shared environmental influences on the NA and OB factors was also very high ($r = .99$, p < .01). A chi-square difference test could not be conducted to compare the one- and two-factor models because the two-factor model includes a constraint group (i.e., the DZ correlation between genetic influences influencing NA in one twin and OB in the other twin is constrained to be half that of the corresponding MZ correlation). However, the model fit statistics for both models are very similar. Overall, the results suggest a single set of genetic, shared environmental, and nonshared environmental influences rather than separate sets of influences on NA and OB symptoms.

Next, univariate models were examined for MDD and GAD to determine genetic, shared environmental, and nonshared environmental influences for the specific disorders. The model fit well for MDD ($\chi^2(12)=7.986$, p.7862; CFI=1.00, RMSEA=0.00). For MDD, both the shared
Figure 5. Results of one factor independent pathway model. Temper, loses temper; argues, often argues with adults; refuse, refuse to comply; touchy, easily annoyed; spiteful, spiteful and vindictive. + p < .10; * p < .05; ** p < .01
Figure 6. Results of two factor independent pathway model with separate factors for negative affect ODD symptoms and oppositional behavior ODD symptoms. Temper, loses temper; argues, often argues with adults; refuse, refuse to comply; touchy, easily annoyed; spiteful, spiteful and vindictive. + p < .10; * p < .05; ** p < .01
(p<.05) and nonshared (p<.01) environmental influences were statistically significant (a^2=.13, c^2=.17, e^2=.64). The univariate model also fit well for GAD (\(\chi^2\)(12)=5.942, p.9190; CFI=1.00, RMSEA=0.00). In contrast to MDD, only the nonshared environmental influences (p<.01) on GAD (a^2=.19, c^2=.19, e^2=.61) were statistically significant.

A Cholesky decomposition model examining the role of the negative affect (NA) symptoms and oppositional behavior (OB) symptoms (Wave 1) on MDD and GAD (Wave 2) simultaneously was conducted. This model (see Figure 7) fit the data well (\(\chi^2\)(2)=76.888, p.6088; CFI=1.00, RMSEA=0.00). The variance components influencing MDD and GAD were calculated by squaring the paths in the models presented in Figure 7. The genetic, shared environmental, and nonshared environmental covariance between both NA and OB with MDD and GAD was not statistically significant (although overall, there is a statistically significant covariance between NA/OB and MDD/GAD). However, there is little variance of MDD and GAD that is not shared in common with NA and OB, with the exception of nonshared environmental influences specific to the disorder (see Table 11). There is also little evidence of genetic and shared environmental influences shared in common with MDD and GAD after controlling for those influencing NA and OB also (see Table 11).

Additional Cholesky models examining the association between NA and OB with later diagnosis of either MDD or GAD were conducted. Figure 8 presents the model examining NA, OB, and MDD (\(\chi^2\)(55)=49.777, p=6739; CFI=.1.00, RMSEA=0.00), and Figure 9 presents the model examining NA, OB, and GAD (\(\chi^2\)(55)=51.207, p.6203; CFI=1.00, RMSEA=0.00). Results were similar to those from the full model examining MDD and GAD simultaneously; there is no significant genetic, shared environmental, or nonshared environmental covariance between NA/OB and MDD/GAD, but little evidence of disorder-specific variance not shared in
Figure 7. Results of full cholesky pathway model. NA, negative affect ODD symptoms; OB, oppositional behavior ODD symptoms; MDD, major depressive disorder; GAD, generalized anxiety disorder. \( ^* p < .10; ^* * p < .05; ^* * * p < .01 \)
Table 11. Variance explained by $a^2$, $c^2$, and $e^2$ for NA, OB, MDD and GAD

**Quadrivariate Model**

<table>
<thead>
<tr>
<th>Variance shared in common with both NA and OB</th>
<th>Variance shared in common with OB (not NA)</th>
<th>Variance specific to MDD / shared in common with only MDD</th>
<th>Variance specific to GAD</th>
<th>Variance explained by Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>$a^2$</td>
<td>$c^2$</td>
<td>$e^2$</td>
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<td>$c^2$</td>
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<td>GAD</td>
<td>.31</td>
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<td>.02</td>
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**Trivariate Model**

<table>
<thead>
<tr>
<th>Variance shared in common with both NA and OB</th>
<th>Variance shared in common with OB (not NA)</th>
<th>Variance specific to MDD</th>
<th>Variance specific to GAD</th>
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<tr>
<td>GAD</td>
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<td>.00</td>
<td>.07</td>
</tr>
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</table>
Figure 8. Results of full cholesky pathway model examining MDD. NA, negative affect ODD symptoms; OB, oppositional behavior ODD symptoms; MDD, major depressive disorder. 

+ p < .10; * p < .05; ** p < .01
Figure 9. Results of full cholesky pathway model examining GAD. NA, negative affect ODD symptoms; OB, oppositional behavior ODD symptoms; GAD, generalized anxiety disorder. + p < .10; * p < .05; ** p < .01
common with NA and OB, with the exception of nonshared environmental influences.

Although the genetic, shared, and nonshared environmental covariance between NA/OB and later MDD/GAD was not significant, the genetic and shared environmental covariance between NA and later MDD and between NA and later GAD were moderate, suggesting that the non-significant findings may be due to inadequate power.

Discussion

Research suggests that children exhibiting ODD during childhood may later develop internalizing or externalizing disorders later in adulthood (Reef et al., 2010; Burke et al., 2005). The current study is one of the first longitudinal, genetically informative, and prospective studies to examine the development of ODD at a symptom level. The present study took advantage of multiple assessments available in the CADD, including ODD assessments in later childhood or early in adolescence and the development of MDD, GAD, or CD five years later.

We examined the factor structure of ODD, with the aim of replicating the evidence for two- and three-factor models suggested by previous findings (Burke, Hipwell, & Loeber, 2010; Burke & Loeber, 2010; Burke, Loeber, Lahey, & Rathouz, 2005; Stringaris & Goodman, 2009). We then examined the genetic, shared environment, and nonshared environmental influences underlying ODD. Lastly, we assessed whether the association between the negative affect factor and oppositional factor of ODD and the later development of MDD and GAD was due to genetic, shared environment, or nonshared environmental influences.

In exploratory factor analyses examining all eight ODD symptoms, the three-factor model did not converge for females. Therefore, subsequent analyses focused solely on the one- and two-factor models (Burke, Hipwell, & Loeber, 2010; Stringaris & Goodman, 2009). The two-factor model included a negative affect factor (touchy, spiteful, and angry) and an
oppositional behavior factor (temper, argues, and refuse), and fit the data better than a one-factor model. In addition to significant loadings on the respective negative affect and oppositional behavior factors, there were significant cross-loadings. Findings from the confirmatory factor analyses were consistent with the exploratory factor analyses. In addition, the NA and OB factors were highly and significantly correlated in the two-factor model. In both the EFA and CFA results, there was evidence of measurement invariance, suggesting similar factor structure of ODD symptoms between males and females.

Although the factor analyses of ODD suggested a two-factor model fit better than the one-factor model, the results of the exploratory factor analyses suggested that there were several significant cross-loadings. Also, the biometrical modeling results suggest a single underlying factor of genetic and nonshared environmental influences (there was little evidence of shared environmental influences on ODD symptoms). These results are somewhat inconsistent with those of prior research (Burke et al., 2005), which found stronger evidence for two separate NA and OB factors. The difference in the results may be due to differences in the sample, with Burke et al. (2005) examining a male clinical sample and the current sample examining both males and females in a community sample.

Since the two-factor model fit the data better than the one-factor model in both the EFA and CFA, however, NA and OB factors were examined separately in subsequent analyses of the association between ODD and later psychopathology. Symptoms of ODD at baseline (wave 1) were examined as predictors of MDD, GAD, and CD five years later in development (wave 2). Both MDD and GAD were significantly correlated with both the NA and OB scores. In addition, MDD and GAD were also significantly correlated. However, neither NA nor OB was significantly correlated with CD.
Given results of prior studies (Burke et al., 2005) and the model proposed by Loeber & Burke (2011), we expected to see a stronger association between the NA factor and later development of MDD and GAD than between the OB factor and MDD/GAD. However, MDD and GAD’s associations with the NA and OB factors were similar in magnitude. The greater similarity in the associations in the present study makes sense given the significant cross-loadings in the EFA and a high correlation between the two factors in the CFA in the present study. This finding may be influenced by method covariance in the present study; twins reported on their own symptoms at both wave 1 and wave 2. Prior studies used a range of different measures to assess psychopathology (Burke et al., 2005; Burke et al., 2010; Stringaris & Goodman, 2009), which may explain the inconsistent findings across studies.

We also expected to see an association between the OB factor and later development of CD, but found that CD was not associated with the NA or OB factor and was only correlated with GAD. The number of cases of individuals exhibiting past year CD symptoms or diagnoses in the current study was very low, decreasing the possibility of finding any associations with the NA and OB factors. Since CD is a childhood disorder, it is likely that the low prevalence rate of CD in later adolescence/early adulthood impacted these findings, as the prevalence of CD and antisocial behavior declines after adolescence (Moffitt, 1993).

The biometrical models were fit to determine the magnitude of genetic, shared environmental and nonshared environmental influences on ODD symptoms and the association between ODD symptoms and the development of later psychopathology. There was a significant covariation between NA and OB and the later development of MDD and GAD. However, we were unable to determine whether the covariation among NA/OB and MDD/GAD was due to genetic, shared environmental, or nonshared environmental influences. Although we were
unable to determine the source of the covariation, the results do suggest that there was little
genetic and shared environmental variance of MDD or GAD that is not explained by NA or OB.
The only significant disorder-specific variance for MDD and GAD was nonshared environmental
influences. There was also little genetic or shared environmental covariance between MDD and
GAD that was not explained by influences shared in common with NA or OB.

The current study had several strengths. The present study was a prospective
examination of ODD and later psychopathology. We were able to examine ODD at wave 1 and
the possible development of MDD, GAD, and CD five years later in wave 2. This is the first
genetically informative study to examine the association between ODD and the later
development of internalizing psychopathology. In addition, it is the first prospective study to
examine the association between ODD and later psychopathology in both males and females.
The only other study examining ODD and later development of externalizing and internalizing
disorders in both sexes was a cross-sectional study (Stringaris & Goodman, 2009).

The results of the present study should be interpreted while considering the following
limitations. A main limitation of the current study was low power. Although the sample size of
the present study was large, the prevalence of psychopathology was very low in this community
sample. The analysis of MDD consisted of ordinal coding of the data (0=no symptoms, 1=one or
more symptoms, and 2=diagnosis) due to the low prevalence; however, one symptom of MDD is
not considered depression. The analyses do capture individuals who may be experiencing
depressive symptoms, but are not severe enough to meet criteria for diagnosis. The low
prevalence of psychopathology symptoms also resulted in several problems with bivariate
missingness when analyzing MZ and DZ twins and twin 1 and twin 2 separately. Because of the
bivariate missingness problem, we were unable to examine all six variables proposed by Loeber
and Burke (2011) and unable to examine potential sex differences in the parameters in the biometrical modeling analyses. Lack of power was also a problem in the biometrical modeling examining the association between NA/OB and MDD/GAD. Although we found significant covariances between NA/OB and MDD/GAD, we were unable to distinguish genetic, shared environmental, and nonshared environmental influences on this covariance.

The symptoms of ODD, MDD, GAD, and CD were assessed via self-report. This was a limitation, as the covariation between the NA and OB factors with later MDD and GAD symptoms may be due to method covariance. Self-report may not be the most accurate assessment of ODD symptoms, whereas parent and teacher report may provide a more accurate depiction of child behavior. In addition, the ODD assessment in wave 1 was retrospective, with participants aged 12 to 19 being asked to recall lifetime ODD symptoms.

These limitations suggest several future directions. Future studies should examine the prospective relationship between the NA and OB factors in childhood and later measures of Antisocial Personality Disorder in adulthood. Studies employing a multi-method approach (such as a combination of self, parent, teacher, and observational measures) for assessing ODD symptoms and the later development of psychopathology are also needed. In addition, larger studies with higher prevalence rates of psychopathology and ODD symptoms are needed, given the problems with low power in our community sample and the small sample size of the study supporting the developmental pathway model of ODD (Burke et al., 2005). Moreover, future research should examine the underlying liability for psychopathology through the use of dimensional measures of internalizing and externalizing symptoms. Also, additional genetically informative studies examining ODD and later psychopathology, especially internalizing psychopathology, are needed.
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of the NIMH Diagnostic Interview Schedule for Children Version 2.3 (DISC-2.3). 


