The relationship between DSM-IV oppositional defiant disorder and conduct disorder: findings from the Great Smoky Mountains Study

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Background: We examine models of the relationship between oppositional defiant disorder (ODD) and conduct disorder (CD) in a community sample. Particular attention is paid to the generalisability of findings based on clinic-referred boys. Methods: The analyses were based on four waves of data from the Great Smoky Mountains Study covering children in the community aged 9–16 years. Child and parent reports of DSM-IV symptoms, diagnoses, and a range of family and environmental adversities were collected using the Child and Adolescent Psychiatric Assessment. Results: Cross-sectional analyses indicated that CD and ODD largely shared similar correlates, although some aspects of parenting appeared more related to CD than ODD. This pattern was broadly similar in boys and girls. Longitudinal analyses confirmed that ODD was a strong risk factor for CD in boys and there was a suggestion that ODD was a stronger risk factor for CD than for other common disorders. Atypical family structure was an important factor in the transition between ODD and CD in boys. In girls ODD provided no increased risk for later CD but was associated with increased risk for continued ODD, depression, and anxiety. Conclusions: These results are more consistent with a developmental relationship between ODD and CD in boys than girls. Keywords: Anti-social behaviour, conduct disorder, development, epidemiology, prognosis, risk-factors.

As described in DSM-IV, conduct disorder (CD) involves ‘a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated’ (American Psychiatric Association, 1994, p. 85). Oppositional defiant disorder (ODD) is described as ‘a recurrent pattern of negativistic, defiant, disobedient, and hostile behavior toward authority figures’ (American Psychiatric Association, 1994, p. 91). It is generally accepted that these two disorders are closely related, but the precise nature of the relationship remains a matter of debate.

A number of somewhat different models can be detected in recent discussions. One suggests a developmental linkage, whereby ODD forms a precursor to CD in some children (Lahey, Loeber, Quay, Frick, & Grimm, 1992; Loeber, Lahey, & Thomas, 1991). A fuller version of this model has recently been presented, including developmental sequences between disruptive disorders and comorbid conditions (Loeber, Burke, Lahey, Winters, & Zera, 2000a). A second, overlapping, but at least potentially distinct conception, views CD as a more severe form of the same underlying disorder. The ICD-10 (World Health Organisation, 1993) classification reflects a third approach, with ODD treated as a subtype of conduct disorder. And finally, some commentators have suggested that any distinction between the two disorders may be unnecessary, because many aspects of their aetiology, including genetic factors that influence both disorders, are closely similar (Eaves et al., 2000). Given the high levels of concurrent (Lahey, Loeber, Quay, Frick, & Grimm, 1997) and long-term (Maughan & Rutter, 2001) impairment associated with these diagnoses, clarifying the relationship between them is of considerable importance.

Empirical support for a developmental model has largely been derived from studies of clinic-referred boys. ODD has been reported as emerging earlier than CD in such samples, and the two disorders frequently overlap. Up to 96% (Frick et al., 1992) of referred boys with CD have been reported to meet criteria for ODD. In the Developmental Trends Study of clinically referred boys, nearly 80% of ODD cases received a diagnosis of CD in subsequent assessments (Lahey, Waldman, & McBurnett, 1999) compared with only a quarter of the remainder. Indeed Lahey, McBurnett, and Loeber (2000a) hypothesise that much of the widely documented progression from Attention Deficit Hyperactivity Disorder (ADHD) to CD may in practice reflect comorbidity between ADHD and ODD, with ODD the more important precursor to conduct problems. So far as we are aware, no data on the prognostic links between ODD and other disorders are currently available. Long-term outcomes of conduct problems appear to show some variations by sex, with risks for later externalising disorders prominent in boys but with later internalising disorder emphasised in girls (Offord & Bennett, 1994). On this basis we might also anticipate that outcomes for ODD may also differ by sex.
The differential severity model predicts that the risk factors for CD and ODD should be similar, but that associations with those risk factors should be stronger in CD than in ODD. This pattern has generally, though not universally, been supported across a range of correlates, including social disadvantage, aspects of parenting, and parental psychopathology (Frick et al., 1992; Rey et al., 1988; Schachar & Wachs, 1990). Once again, however, the main evidence on these issues comes from clinically referred samples of boys.

Two recent reviews (Keenan, Loeber, & Green, 1999; Loeber, Green, Lahey, Frick, & McBurnett, 2000b) have noted that these patterns may not be replicated in community samples, and have urged the need to examine ODD–CD associations in non-referred groups of both sexes. The few epidemiologically based findings currently available support this concern. In the Methods for Epidemiology of Child and Adolescent Disorders (MECA) sample, for example, overlaps between ODD and CD were much less marked than in clinical samples (Lahey et al., in press, cited in Keenan et al., 1999), with only 25% of girls and 33% of boys with CD showing concurrent ODD. Although these links were statistically significant, the majority of children with CD did not show strong oppositional features. In terms of prevalence, large-scale community studies show that while CD is clearly more common in boys, rates of ODD in boys and girls are quite similar (Costello et al., 1996a; Lahey et al., 2000b; Simonoff et al., 1997), providing a further hint that any developmental relationship between the two disorders may be different in boys and girls.

Community studies also offer the most appropriate basis for examining comorbidity (Angold, Costello, & Erkanli, 1999). If CD is a more severe disorder than ODD, we might expect rates of comorbidity with other disorders to be higher in CD than in ODD. Differential patterns of comorbidity have been reported for the two disorders in community samples, which do not support the severity model. While high rates of comorbidity have been found for both disorders, the pattern of relationships with other common disorders was stronger for ODD than for CD in the Great Smoky Mountains Study (GSMS) (Angold, Erkanli, Egger, & Costello, submitted) and both wider and stronger in the Virginia Twin Study of Adolescent Behavioral Development (VTSABD) (Simonoff et al., 1997). These results appear to contradict the view that CD is a more malevolent form of ODD. Furthermore, symptoms of ODD were associated with higher levels of psychosocial impairment than were symptoms of CD in the VTSABD (Pickles et al., 2001).

The work reported here provides further evidence about the relationship between CD and ODD, using data from the GSMS, a large-scale community study including both girls and boys. We examine the prevalence, phenomenology and correlates of the two disorders in a sample of 9–16-year-olds, and assess continuities from ODD to CD over four annual assessment waves. A particular focus of the analyses is to test whether models of the ODD–CD relationship found in clinically recruited boys generalise to boys and girls in the community.

Method

Population

Based in a predominantly rural area of the southern United States, the Great Smoky Mountains Study is a longitudinal study of psychiatric disorder in children and adolescents. The accelerated cohort (Schaie, 1965), two-phase sampling design and measures are described in detail elsewhere (Costello et al., 1996b). Briefly, a representative sample of 4,500 9-, 11-, and 13-year-olds resident in western North Carolina were selected using a household equal probability design. In the screening phase a parent (usually the mother) completed a questionnaire containing items regarding behavioural disorders from the Child Behaviour Checklist (Achenbach & Edelbrock, 1983). The interview phase included all children scoring above a pre-defined cut-off on this screen (designed to identify the most pathological 25% of the population), along with a 10% random sample of the remainder. All age-eligible American Indian children from the area were also recruited. Between 80% and 94% of those selected took part at each of four annual interviews, providing a data set containing 4,965 observations from 1,420 individuals (790 boys, 630 girls).

Measures

At each wave the child and primary caretaker (usually the mother) were separately interviewed using the Child and Adolescent Psychiatric Assessment (CAPA) (Angold & Costello, 2000). The CAPA assesses the child’s psychiatric status over the preceding 3 months using DSM-IV criteria (American Psychiatric Association, 1994). Kappa reliabilities were .55 for diagnoses of CD, .90 for major depression, and .64 for any anxiety disorder in a sample of 77 clinically referred children interviewed on two occasions using the child version of the CAPA (Angold & Costello, 1995). The intra-class correlation for the ODD symptom scale was .50. The standard ‘or’ rule (Costello et al., 1996b; Simonoff et al., 1997), where a symptom is endorsed if either the child or primary caretaker report meets the symptom threshold, was employed to combine reports from the two informants. Where both informants endorsed a symptom, the onset date was taken as the earlier of the two reports.

All DSM-IV ODD and CD symptoms were assessed at each of the first three assessment waves, with the exception that staying out late (CD) was not measured at the first wave because this was conducted before DSM-IV was finalised. At wave four, a screened version of the CAPA was used, in which three ODD and three CD symptoms (chosen as the most informative in identifying caseness) were asked of all participants. Assessment of the remaining CD and ODD symptoms
was completed only if the intensity criterion was reached on at least one of these screen symptoms. All DSM-IV criteria for ODD and CD were imposed, including the requirement that ODD was not diagnosed in the presence of CD. This allowed the formation of three exclusive groups: those without either ODD or CD (referred to as the no diagnosis group), those with ODD, and those with CD. DSM-IV symptoms and diagnoses of other common psychiatric disorders were also assessed using the CAPA. In this paper we consider binary variables indicating the presence of any anxiety disorder (generalised anxiety disorder, separation anxiety disorder, specific phobia, social phobia, or panic disorder), and any depressive disorder (major depression, dysthymia, or minor depression).

A wide range of measures of family and environmental correlates of disorder was also assessed in the CAPA. For ease of interpretation, conceptually related indicators were summed into sub-scales for the present study. A social disadvantage sub-scale was constructed from six items: 1) income below the federal poverty line; 2) interviewer-observed impoverished home environment; 3) one or both parents unemployed; 4) one or more parents left school without graduating; 5) neighbourhood and 6) school perceived to be dangerous by parent or child. An atypical family structure sub-scale was formed from six items: 1) at least one parent a teenager at the child’s birth; 2) large family (the study child had four or more siblings living in the home); 3) one parent a step-parent; 4) single parent household; 5) frequent family moves (more than four moves in the previous five years); and 6) the child had spent some time in a foster home. These scales were standardised to have a weighted mean of 50 and a standard deviation of 10. Individual binary variables were also constructed to reflect a series of characteristics of the child’s biological parents: a history of personal psychiatric hospital; and report of convictions for a criminal offence.

Variables indicating the presence of any anxiety disorder (generalised anxiety disorder, separation anxiety disorder, specific phobia, social phobia, or panic disorder), and any depressive disorder (major depression, dysthymia, or minor depression). Diagnostic group was treated as a categorical predictor in all cross-sectional analyses. The relationship between diagnostic group and age was assessed using ordinary regression. Associations between diagnostic group and sex, and parental characteristics were assessed by logistic regression. The scales of family/environmental adversities were treated as ordered categories, and analysed using ordinal logistic regression. Ordinal logistic regression models calculate proportional odds ratios (POR), which can be interpreted as the increase in the odds of crossing any particular threshold on the dependent variable given a single unit increase in the independent variable. The relationships between an ODD diagnosis at wave 1 and outcome disorders at later waves were assessed using logistic regression.

Results
Prevalence, age of onset, and phenomenology

Ninety-six boys (weighted prevalence 2.0%) and 70 girls (1.5%) met ODD criteria and 132 boys (3.1%) and 47 girls (1.1%) met CD criteria. ODD was as common in girls as in boys ($p = .4$), but CD was substantially more common in boys ($p = .008$), although the contrast between ODD and CD did not reach significance ($p = .1$). Children meeting criteria for CD were significantly older than those in the no diagnosis group (mean age of 13.0 years, by contrast with 12.3 years; $p = .02$) and those with ODD (12.2 years; $p = .04$). There was no age difference between the ODD and no diagnosis groups ($p = .7$).

As expected from previous reports, the great majority of boys with CD also showed oppositional features. Only 5% had no symptoms and 26% met full criteria for ODD. Among girls, overlap between the two disorders was stronger; 54% of girls with CD also met full ODD criteria, and only 2% showed no ODD symptoms whatsoever. The majority of children with ODD (57% of boys and 55% of girls) also showed sub-diagnostic levels of CD symptomatology. Retrospective reports suggested that children with both disorders had shown signs of antisocial behaviour from early in childhood. The median age of onset of any antisocial symptom (CD or ODD) was 4.5 years in both the ODD and CD groups. Boys with ODD had earlier onsets than girls (2.5 years vs. 5.5 years). In the CD group, both boys and girls reported median ages of onset of 4.5 years.

Family/environmental adversities

Mean scores on the scales of social/material disadvantage and atypical family structure in each diagnostic group are shown in Table 1. Initial analyses tested for interactions between sex and diagnostic group. Where such an interaction was absent, further tests for differences between diagnostic groups were conducted in single predictor models. As expected, rates of social and family adversity were
considerably elevated in both diagnostic groups by contrast with the no diagnosis group. Children with ODD faced similarly adverse backgrounds to those with CD in terms of social disadvantage and atypical family structures, and these patterns of group differences were similar for girls and boys.

**Parental characteristics**

Rates of parental mental health problems/deviance are also shown in Table 1. Of the biological parental history measures, only a history of mental health problems showed a different pattern of relationships between the sexes ($p < .03$). In boys this variable did not relate to diagnostic status ($p = .6$; no diagnosis 25.8%, ODD 27.9%, CD 31.9%). In girls, by contrast, both the CD (52.9%; Odds Ratio (OR) $= 3.0$; 95% Confidence Interval (CI): 1.4, 6.1; $p = .004$) and ODD (63.9%; OR $= 4.6$; 95% CI: 1.8, 12.0; $p = .004$) groups had higher rates than the no diagnosis group (27.6%) but did not differ from each other (OR $= .6$; 95% CI: .2, 2.0; $p = .4$). Rates of parental criminality were significantly higher in the ODD group than in the no diagnosis group. There was also a tendency for raised levels in the CD group ($p = .09$) relative to the no diagnosis group, and the ODD and CD groups did not differ ($p = .3$). In terms of parental drug and alcohol problems the CD group showed significantly higher rates than the no diagnosis group and those with ODD fell between the two, differing significantly from neither. Current caretaker depression did not differ between ODD and CD and was elevated in both these groups relative to the no diagnosis group.

**Parenting behaviours**

The rates of adverse parenting behaviours are shown in Table 2. Initial analyses examined whether boys and girls differed in their exposure to these adverse parenting behaviours. There were no sex differences regarding harsh discipline, overintrusive parenting, or scapegoating but there was a tendency for inadequate supervision to be more common in boys (OR $= 1.4$; 95% CI: 1.0, 2.2; $p = .08$). There was, however, evidence that the associations between some of these parenting behaviours and diagnostic group differed by sex. Regarding harsh discipline, there was a significant interaction between diagnostic group and sex ($p = .04$). Among boys, exposure to harsh discipline occurred at similar rates in the ODD and no diagnosis groups (OR $= 1.3$; 95% CI: .4, 4.1; $p = .6$). The rate in the CD group was elevated relative to the no diagnosis group (OR $= 4.3$; 95% CI: 1.6, 11.5; $p = .004$), but the comparison with ODD fell short of significance (OR $= 3.2$; 95% CI: .8, 13.7; $p = .1$). In girls rates of harsh discipline were elevated in both ODD (OR $= 7.7$; 95% CI: 2.7, 21.7; $p < .001$) and CD (OR $= 14.4$; 95% CI: 4.0, 52.3, $p < .001$), relative to the no diagnosis group, with no significant difference between the ODD and CD groups (OR $= 1.9$; 95% CI: .4, 8.2; $p = .4$).

### Table 1

<table>
<thead>
<tr>
<th>Scales of disadvantage</th>
<th>Mean level (scales) or percentage endorsing (binary items)</th>
<th>PORs (scales) and ORs (binary items). 95% CIs shown in brackets**</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diagnosis ODD CD</td>
<td>ODD vs. no dx CD vs. no dx CD vs. ODD</td>
<td></td>
</tr>
<tr>
<td>Social adversity scale</td>
<td>49.8 55.4 57.1 2.7 (1.7, 4.4) 3.5 (2.1, 5.9) 1.3 (7.2, 4.1)</td>
<td></td>
</tr>
<tr>
<td>Atypical family structure scale</td>
<td>49.7 55.6 58.1 2.7 (1.4, 5.3) 4.1 (2.5, 6.6) 1.5 (7.3, 3.3)</td>
<td></td>
</tr>
</tbody>
</table>

* Evidence that the relationship between this variable and diagnostic group differed by sex is discussed in the text.

** Coefficients significant at $p < .05$ shown in bold.

### Table 2

<table>
<thead>
<tr>
<th>Boys</th>
<th>Girls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No diagnosis ODD CD Overall</td>
</tr>
<tr>
<td></td>
<td>Harsh discipline 2.7 3.6 10.7 3.0</td>
</tr>
<tr>
<td></td>
<td>Over-intrusive parenting 1.0 3.6 1.2 1.1</td>
</tr>
<tr>
<td></td>
<td>Inadequate supervision 6.5 27.6 42.3 8.0</td>
</tr>
<tr>
<td></td>
<td>Scapegoating 1.5 22.8 10.4 2.2</td>
</tr>
<tr>
<td></td>
<td>Mean parenting problem count .1 .6 .6 .1</td>
</tr>
</tbody>
</table>
Overintrusive parenting also showed a significant interaction between diagnostic group and sex \((p = .02)\). In boys overintrusiveness was reported at similar, low levels across all groups. In girls, however, overintrusive parenting was reported more frequently in the CD group than in both the no diagnosis \((\text{OR} = 31.6; 95\% \text{ CI}: 4.8, 209.9; p < .001)\) and ODD \((\text{OR} = 14.2; 95\% \text{ CI}: 1.3, 159.0; p = .03)\) groups.

Neither inadequate supervision \((p = .4)\) nor scapegoating \((p = .2)\) showed significant interactions between sex and diagnostic group. Inadequate supervision was more common in the ODD \((\text{OR} = 5.1; 95\% \text{ CI}: 2.7, 9.4; p < .001)\) and CD \((\text{OR} = 13.4; 95\% \text{ CI}: 7.8, 23.1; p < .001)\) groups than the no diagnosis group, and in the CD group relative to the ODD group \((\text{OR} = 2.7; 95\% \text{ CI}: 1.1, 6.2; p = .02)\). Scapegoating was also more common in the ODD \((\text{OR} = 10.4; 95\% \text{ CI}: 3.5, 31.2; p < .001)\) and CD groups \((\text{OR} = 6.4; 95\% \text{ CI}: 2.7, 14.9; p < .001)\) compared to the no diagnosis group but did not differ significantly between CD and ODD \((\text{OR} = .6; 95\% \text{ CI}: .2, 2.3; p = .5)\).

We formed a scale by summing the number of endorsed parenting items in order to examine the effect of the presence of multiple risk factors, as shown in Table 2. There was a suggestion that the relationship between this scale and diagnostic group differed by sex \((p = .1)\). As before, rates of adverse parenting were substantially higher in the diagnosed groups relative to the no diagnosis group. In addition, however, parenting problems were significantly more common in CD than ODD in girls \((\text{POR} = 5.0; 95\% \text{ CI} 1.5, 16.8; p = .009)\) but not in boys \((\text{POR} = 1.3; 95\% \text{ CI}: .5, 3.3; p = .6)\).

### Prognostic relations between ODD and other disorders

To explore later outcomes of ODD we examined risks for CD, ODD, depression, and anxiety at waves 2–4 of the study for children with and without a diagnosis of ODD at wave 1. The outcome disorders were treated as present if a diagnosis was made at any of waves 2, 3, or 4. Sixty-three per cent of children with ODD at wave 1 met criteria for one or more of the target disorders in at least one subsequent study wave. Rates of each subsequent disorder are shown in Table 3. The relationships were modelled using logistic regressions with comorbid depression and anxiety at wave 1 included as covariates.

There was some suggestion of a particularly strong relationship between ODD and later CD in boys, as the odds ratio for CD was over twice the size of those for other disorders. A multivariate outcome regression model was used as a formal test of whether the strength of prediction differed between the various outcome disorders. There was some evidence of heterogeneity in the strength of relationships with different later disorders \((F(3, 1253) = 1.79, p = .1)\). Specific comparisons showed that prediction to CD was stronger than prediction to later anxiety \((p = .02)\) but did not differ from the prediction to ODD \((p = .4)\) or depression \((p = .2)\).

In girls the pattern of associations was markedly different. Of the 21 girls with ODD at wave 1, none developed CD at later waves, but prediction to continued ODD, depression, and anxiety was substantial. Omitting the CD outcome, a multivariate analysis showed that there were significant differences between the strengths of prediction to different disorders \((F(2, 1254) = 4.19, p = .02)\). In particular, prediction to continued ODD was stronger than prediction to depression \((p = .06)\) and anxiety \((p = .04)\).

Because the dissociation between ODD and later CD in girls was unexpected, we tested whether it would be repeated in the somewhat larger group of girls \((n = 39)\) showing ODD at either of waves 1 or 2, predicting outcomes at waves 3 and 4. In this case just one girl (2.1%) went on to develop CD, the same rate as in the non-disordered comparison group (14 cases). We also tried relaxing the definition of ODD at wave 1 to include DSM-IV ODD Not Otherwise Specified (2 or more symptoms of ODD and some impairment) which provided a group of 68 girls with ODD at wave 1. Again the rate of subsequent CD was only 2.4% compared to 1.7% in the remainder.

### Factors associated with the development of CD in boys

As Table 3 shows, over 40% of the boys with ODD at wave 1 showed CD at one of the following three assessments. Although the numbers involved were small, we undertook a series of exploratory analyses.

**Table 3** Common psychiatric disorder at any of waves 2–4 in those with and without ODD at wave 1. ORs account for presence of comorbid depression, and anxiety at wave 1

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Boys No ODD at wave 1 N = 658</th>
<th>Boys ODD at wave 1 N = 36</th>
<th>OR* (95% CI)</th>
<th>Girls No ODD at wave 1 N = 541</th>
<th>Girls ODD at wave 1 N = 21</th>
<th>OR* (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CD</td>
<td>5.4</td>
<td>42.7</td>
<td><strong>12.9 (3.7, 45.7)</strong></td>
<td>1.8</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>ODD</td>
<td>3.4</td>
<td>17.4</td>
<td><strong>5.6 (1.9, 16.7)</strong></td>
<td>2.5</td>
<td>59.8</td>
<td><strong>58.0 (13.6, 246.8)</strong></td>
</tr>
<tr>
<td>Depression</td>
<td>3.0</td>
<td>10.8</td>
<td><strong>3.7 (1.0, 14.1)</strong></td>
<td>3.5</td>
<td>41.6</td>
<td>19.4 (3.1, 121.7)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>5.0</td>
<td>3.4</td>
<td><strong>6 (1.4, 9.9)</strong></td>
<td>4.4</td>
<td>19.3</td>
<td>5.0 (1.3, 18.8)</td>
</tr>
</tbody>
</table>

*Coefficients significant at \(p < .05\) shown in bold.
to identify factors associated with this progression. Rates of each factor in boys who did and did not develop CD are shown in Table 4, along with bivariate odds ratios for these associations. Within the ODD group, sub-threshold CD symptomatology (0, 1, or 2 symptoms) at wave 1 was associated with increased risk for a later CD diagnosis, but initial level of ODD symptomatology (beyond the diagnostic threshold of 4 symptoms) was not. All the scales of family/environmental adversity showed significant links with the development of CD. In this analysis the parenting scale was transformed to have a weighted mean of 50 and standard deviation of 10 to make it comparable with the other scales. None of the biological parent characteristics emerged as significant predictors. In a joint model the atypical family structure scale remained significantly associated with risk for CD (OR for 1 SD increase) = 3.1 95%; CI: 1.0, 9.1; p = .05) and the parenting scale was of borderline significance (OR = 2.7 95%; CI: 9, 7.8; p = .07); neither the social disadvantage scale (OR = 1.4 95%; CI: .6, 3.6; p = .5) nor the extent of sub-threshold CD symptomatology at wave 1 (OR = 2.6 95%; CI: .6, 11.6; p = .2) contributed significantly to prediction.

More detailed analyses of the constituent items of the atypical family structure scale showed that having a parent who was a teenager at child’s birth (OR = 10.6 95%; CI: 1.0, 111.6; p = .05), having a step parent (OR = 41.8 95%; CI: 2.5, 699.4; p = .01), and having moved house often (OR = 34.8 95%; CI: 2.0, 619.2; p = .02) all increased risk of developing CD. The remaining items (single parent family, large family, and having spent some time in a foster home) did not predict the development of CD significantly (all ps > .4). In a model including the three significant predictors, teenage parenthood dropped to non-significance (OR = 1.1 95%; CI: .1, 7.9; p = .9) while both step-family status (OR = 14.6 95%; CI: .7, 294.8; p = .08) and frequent house moves (OR = 10.2, 95%; CI: .8, 133.7; p = .08) were of borderline significance.

### Table 4: The relationship between vulnerabilities at wave 1 and development of CD at later waves in boys with ODD at wave 1

<table>
<thead>
<tr>
<th>Wave 1</th>
<th>No CD N = 25**</th>
<th>CD N = 11**</th>
<th>OR***</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychiatric status</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD symptoms*</td>
<td>.7</td>
<td>1.8</td>
<td>5.6</td>
<td>1.3, 24.2</td>
</tr>
<tr>
<td>ODD symptoms*</td>
<td>4.8</td>
<td>4.3</td>
<td>.6</td>
<td>.2, 1.7</td>
</tr>
<tr>
<td>Scales of disadvantage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parenting scale*</td>
<td>53.1</td>
<td>68.6</td>
<td>3.7</td>
<td>1.2, 11.3</td>
</tr>
<tr>
<td>Atypical family structure scale*</td>
<td>49.0</td>
<td>66.4</td>
<td>4.6</td>
<td>1.5, 14.1</td>
</tr>
<tr>
<td>Social adversity scale*</td>
<td>52.3</td>
<td>66.3</td>
<td>3.4</td>
<td>1.0, 11.3</td>
</tr>
<tr>
<td>Biological parent characteristics (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of mental health problems</td>
<td>24.8</td>
<td>7.7</td>
<td>.3</td>
<td>.03, 2.4</td>
</tr>
<tr>
<td>Criminality</td>
<td>42.0</td>
<td>29.3</td>
<td>.6</td>
<td>.1, 4.2</td>
</tr>
<tr>
<td>History of drug/alcohol problems</td>
<td>2.9</td>
<td>15.5</td>
<td>6.1</td>
<td>.6, 62.0</td>
</tr>
</tbody>
</table>

**ORs for 1 SD increase. **Ns vary slightly between predictors due to missing data. ***Coefficients significant at p < .05 are shown in bold.
poverty and social disadvantage, maternal depression, and a history of criminality in biological parents, were associated with similar levels of risk for ODD in most cases. The main exception to this pattern was in relation to the measures of adverse parenting. Inadequate supervision was more strongly associated with CD than ODD in both boys and girls, while harsh discipline was specifically associated with CD in boys but with both disorders in girls. Overintrasuive and overprotective parenting was associated with CD only in girls, and not associated with diagnostic status in boys. This may suggest that these aspects of parenting are more likely to contribute to risk for CD rather than ODD. However, because these analyses were cross-sectional we cannot be certain of the direction of the effects involved. Disruptive behaviours are known to evoke, as well as form a reaction to, different styles of parenting (Lytton, 1990). It is possible that children with particular types of problem evoke adverse parenting to a greater or lesser extent.

There was only one other analysis which suggested that the correlates of CD and ODD may differ between boys and girls; a biological parent history of mental health problems was associated with increased risk of both CD and ODD in girls but not in boys. Although we do not know the exact nature of the parents’ difficulties, one interpretation of this finding is that antisocial disorders (at least as currently operationalised) may have a stronger genetic loading in girls than in boys. Though few studies have examined this possibility to date, Eley, Lichtenstein, and Stevenson (1999), analysing parent and teacher questionnaire scores, found that genetic effects on non-aggressive antisocial behaviour were greater for girls than for boys. However, in the VTSABD a number of measures of conduct and oppositional symptoms did not suggest sex differences in heritability (Eaves et al., 1997). We must await further studies using genetically informative designs to clarify this picture.

Overall, findings from the cross-sectional analyses were generally consistent with the hypothesis that ODD and CD are closely related, and may share a developmental relationship. First, almost all the children with CD showed some ODD behaviours (and many reached clinical thresholds for ODD), while a substantial proportion of those with ODD did not show any CD symptoms. Second, in this sample of 9–16-year-olds, children with CD were on average older than those with ODD. And third, correlates of the two disorders were closely similar, consistent with the possibility that both form part of the same underlying disorder. In all these respects a developmental model, initially proposed on the basis of studies of boys, seemed equally appropriate for girls.

Our longitudinal analyses, however, highlighted some rather different features. In boys, as expected from results in clinical samples, there was a clear tendency for ODD to be a stronger risk factor for CD than for other disorders, although (possibly as a result of low statistical power) this did not receive conclusive support from the significance testing. As outlined earlier, very high rates of progression from ODD to CD have been reported in referred samples of boys, suggesting that ODD is a key developmental precursor to CD; indeed Lahey et al. (2000a) have speculated that the high rates of progression to CD noted in samples of boys with ADHD may largely reflect comorbidity between ADHD and ODD. Because ADHD has an onset early in childhood, developmental studies beginning before age 6 are needed to evaluate this important hypothesis, and our sample – where the youngest children were aged 9 years and the majority of observations focused on early adolescence – was not well placed to address it. We were, however, able to evaluate ODD to CD progression across this age range. In this community sample almost a third of boys with ODD at wave 1 had no DSM-IV disorder in the three following years, and just over 40% received a diagnosis of CD. Lahey et al. (1999) suggested that oppositionality (viewed as a temperamental feature) might constitute a key element in risk for more directly antisocial behaviour in boys when combined with other adverse temperamental features, and in certain environmental contexts. Of the environmental predictors available within GSMS, only the family structure scale indicated an independently significant relationship with progression to CD. Analyses of the items that make up the family structure scale indicated that teenage parenthood, step-family status, and frequent house moves were significantly related to progression to CD. However, in a joint analysis, teenage parenthood did not have a significant independent relationship with progression, suggesting the effects of teenage parenthood may be mediated by subsequent family instability.

By contrast, none of the girls with ODD at wave 1 went on to develop CD at later waves. Our numbers, of course, were small in these analyses. Relaxing the ODD definition and reorganising the analysis to combine cases of ODD at waves 1 and 2 did increase the number of girls followed longitudinally but confirmed that ODD was not an important risk factor for CD in the girls studied here. ODD in girls did, however, appear to constitute a strong risk factor for continuity to later ODD, and also for the development of depression and (to a lesser extent) anxiety.

When assessing the results reported here, a number of potential limitations should be considered. Despite the large overall sample size (of almost 5,000 observations for cross-sectional analyses), some analyses may have suffered from low power as the number of cases was relatively low, reflecting the prevalence of ODD and CD in the population. In addition, although the study covered the core childhood and early adolescent years when disruptive disorders are common, it did not include direct measures on the early childhood period when
antisocial behaviour often begins, nor on the later teenage period. As in most psychiatric epidemiological studies of childhood, this study focused on a specific community, which was predominantly rural in our case. The extent to which our findings will generalise to other sections of the general population can only be identified with further research. Notwithstanding these limitations, it appears that the results reported confirm a number of previously reported findings and extend knowledge on a number of key issues.

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