Published in final edited form as:
J Abnorm Psychol. 2010 November ; 119(4): 726–738. doi:10.1037/a0020798.

Developmental pathways in Oppositional Defiant Disorder and Conduct Disorder

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Abstract

DSM-IV specifies a developmental relationship between Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD). Evidence for this link is mixed, however, and recent studies suggest that different symptom dimensions in ODD may have different outcomes. We examined links between ODD, CD and their young adult outcomes in the Great Smoky Mountains Study; a longitudinal dataset with over 8000 observations of 1420 individuals (56% male) covering ages 9 to 21 years. ODD was a significant predictor of later CD in boys but not in girls after control for comorbid CD and sub-threshold CD symptomatology. Transitions between ODD and CD were less common than anticipated, however, particularly during adolescence. We examined characteristics and outcomes of children with pure ODD, pure CD and combined CD/ODD. Alongside many similarities in childhood and adolescent correlates, key differences were also identified: CD largely predicted behavioral outcomes, whereas ODD showed stronger prediction to emotional disorders in early adult life. Factor analysis identified irritable and headstrong dimensions in ODD symptoms that showed differential prediction to later behavioral and emotional disorders. Overall, the results underscore the utility of retaining separate ODD and CD diagnoses in DSM-V.

DSM-IV (American Psychiatric Association, 1994) specifies Oppositional Defiant Disorder (ODD) as ‘a recurrent pattern of negativistic, defiant, disobedient, and hostile behavior toward authority figures’ (p. 91). The key features of Conduct Disorder (CD) center on ‘a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated’ (p. 85). ODD has often been regarded as a milder form of CD (Rey et al., 1988) that forms an early stage in CD development. DSM-IV states that ‘all of the features of ODD are usually present in CD’ (p. 93), and precludes a diagnosis of ODD if full criteria for CD are met. As we discuss below, however, current evidence on links between the two disorders is quite limited and offers only mixed support for a developmental relationship. In addition, ODD remains a controversial diagnosis (Moffitt et al., 2008). Some critics argue that ODD resembles normal ‘rebellious’ behavior, and that oppositionality might be more usefully treated as a temperamental dimension than as a categorically defined disorder (Loeber, Burke, & Pardini, 2009). From a somewhat different perspective, commentators note that treating ODD primarily as a precursor to behavioral disorders may be to take too narrow a view. Evidence is accumulating that ODD predicts emotional as well as behavioral disorders in childhood and adolescence (see Loeber et al., 2009) and early adult life (Copeland, Shanahan, Costello, & Angold, 2009). Recent proposals to separate ‘headstrong’ and ‘irritable’ dimensions of ODD symptoms (Stringaris & Goodman, 2009b) add further complexity to the possible relationships between ODD and CD. We focus here on two key elements in these debates. First, we address developmental associations between ODD, CD and later behavioral disorders. Second, we examine the utility of a multidimensional approach to oppositionality.

Development from ODD into CD

DSM-IV organizes ODD, CD and Antisocial Personality Disorder (ASPD) hierarchically and developmentally, 'as if they reflect age-dependent expressions of the same underlying disorder' (Moffitt et al., 2008, p. 22). ODD is assumed to constitute a developmental antecedent to CD in 'a significant proportion of cases' (American Psychiatric Association, 1994), and it is noted that youth showing the Childhood-Onset sub-type of CD (though not Adolescent-Onset) may have had ODD during early childhood.

If ODD is a precursor to CD its onset should occur earlier in development. Studies in childhood samples tend to support this pattern (Loeber, Green, Lahey, Christ, & Frick, 1992). The National Comorbidity Survey Replication (NCSR), assessing childhood disorders from retrospective adult reports, also found that ODD onset pre-dated onset of a range of other disorders including CD (Nock, Kazdin, Hiripi, & Kessler, 2007). Age-trends in prevalence suggest that rates of CD diagnoses are low in childhood, but rise steeply from late childhood/the early teens, whereas rates of ODD are relatively stable from early childhood to adolescence (Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). In addition, comorbidity between CD and ODD in childhood is undoubtedly strong. In clinical samples, rates of ODD in cases of CD have been reported to be as high as 96% (Frick et al., 1992). Overlaps in general population samples are lower: approximately 60% of CD cases typically meet full criteria for ODD (Maughan et al., 2004; Rowe, Maughan, Pickles, Costello, & Angold, 2002) and others often show elevated levels of sub-threshold ODD symptoms (Rowe et al., 2002).

If ODD and CD are underlying expressions of the same disorder, we would expect them to share risk factors. Risk factors for antisocial behavior have been reported across a range of domains spanning psychosocial factors (such as neighborhood characteristics), family factors (including parenting), individual factors such as hyperactivity and neurological deficits, and genetic vulnerabilities (Loeber et al., 2009). Comparisons of risk factors between CD and ODD have generally found similarities more striking than differences (Loeber et al., 2009), though some specificities have been identified (Shanahan, Copeland, Costello, & Angold, 2008). The one clear exception to this pattern is child sex. Being male appears to be a specific risk factor for CD. Rates of ODD are fairly similar in boys and girls, with perhaps a slight male majority (Nock et al., 2007), but boys have consistently been reported to be at greater risk of CD (Loeber et al., 2009; Rowe et al., 2002). Boys and girls seem largely similar in sensitivity to psychosocial risk factors for antisocial behavior, and are also similarly exposed to potential family and environmental correlates. A number of studies indicate that boys are more exposed to individually-based risk factors however. This may in part account for their higher levels of antisocial behavior (Moffitt, Caspi, Rutter, & Silva, 2001).

Genetically informative studies indicate substantial heritability for both CD and ODD behaviors (Eaves et al., 1997) but a mixed picture of etiological similarities between the two disorders. One study suggested that both were manifestations of the same genetic liability (Eaves et al., 2000), whereas others have reported moderate degrees of shared genetic and environmental influences, along with unique effects on both ODD and CD behaviors (Dick, Viken, Kaprio, Pulkkinen, & Rose, 2005; Lahey et al., 2009; Rowe, Rijsdijk, Maughan, Hosang, & Eley, 2008; Tuvblad, Zheng, Raine, & Baker, 2009; Waldman, Rhee, Levy, & Hay, 2001). It is possible that rater effects contribute to differences in findings between twin studies (Burt, McGue, Krueger, & Iacono, 2005). A strong correlation of genetic effects does not, of course, necessarily imply that the phenotypes involved index the same underlying disorder. Strong genetic correlations have been reported for example, between internalizing and externalizing phenotypes (see e.g. Rowe et al., 2008). In addition, models

of this kind exclude the gene-environment interplay almost certainly involved in risk for externalizing disorders (e.g., Kim-Cohen et al., 2006). At this stage, then, although evidence for genetic associations between CD and ODD is suggestive of shared etiology, it is not sufficient to demonstrate whether they are manifestations of a single disorder.

Genetic effects may in part be mediated through biological factors that are beginning to be identified as shared risks for oppositionality and conduct problems (Beauchaine, Gatzke-Kopp, & Mead, 2007). However, such biological factors are likely to interact in complex ways both among themselves and with environmental risks (e.g., Rowe, Maughan, Worthman, Costello, & Angold, 2004). Much more must be learned about these effects before they can contribute to diagnostic formulations (Moffitt et al., 2008).

More direct tests of a developmental relationship between ODD and CD have also provided somewhat mixed support. In clinically referred boys followed from ages 7-12 years to age 18, ODD was predictive of later CD, with no reciprocal (i.e. CD→ODD) relationship (Burke, Loeber, Lahey, & Rathouz, 2005). In a mixed-sex general population sample, however, dimensional measures of oppositionality at ages 4-7 years showed negligible prediction to dimensional measures of later conduct problems (at ages 8-13 years) once initial levels of conduct problems were controlled (Lahey et al., 2009). The measures of oppositionality and conduct problems in this study did not cover the full DSM-IV symptom lists, however. In particular, conduct problems were measured using only 7 items, including indicators of trouble getting along with teachers and disobedience at home and school, which overlap with the construct of oppositionality.

These differing findings suggest that conclusions on ODD→CD continuity may be sensitive to a variety of factors: age/developmental stage, sample composition, measurement techniques and analytic strategies. Not dissimilar variations have emerged in past reports from the Great Smoky Mountains Study (GSMS), the sample we report on further here. Using a ‘chain’ analysis where ODD at any annual assessment was used to predict CD at the immediately succeeding wave, Costello, Mustillo, Erkanli, Keeler & Angold (2003) found no prediction from ODD to CD between ages 9 and 16 years. Rowe et al. (2002), however, found that ODD at wave 1 predicted CD at any of three subsequent waves among boys. Links with CD were stronger than with other common outcome disorders, consistent with the specific relationship between ODD and CD assumed in DSM-IV. Results showed a markedly different pattern in girls. None of the girls who met criteria for ODD but not CD at wave 1 developed CD at later waves. Instead, persistence in ODD and heterotypic continuities to depression were common outcomes for girls. In a literature still largely focused on males, these findings emphasize the need for tests of sex-specific outcomes.

The final step in the developmental model posits prediction from CD to ASPD. Here, current evidence is more consistent: ASPD is typically an outcome of CD rather than ODD (Burke, in press), though one study has reported an independent relationship between ODD and ASPD (Langbehn, Cadoret, Yates, Troughton, & Stewart, 1998). Looking beyond strictly ‘antisocial’ outcomes, however, the picture is more complex. Follow-up of the GSMS sample in early adulthood (Copeland et al., 2009) showed that ODD in adolescence was independently associated with increased risks of anxiety and depression in early adult life. CD showed no similar predictions to emotional disorders. Differential prediction to emotional and behavioral outcomes has also been noted in a clinical sample of boys (Burke et al., 2005). This echoes the more diffuse patterns of comorbidity in ODD than CD reported in childhood and adolescent samples (Simonoff et al., 1997).

Predictors of development of CD from ODD

A number of studies have attempted to identify characteristics of children with ODD who do and do not go on to develop CD. Levels of sub-threshold CD symptoms have been identified as one such predictor (Rowe et al., 2002), with persistent physical fighting being perhaps of particular importance (Loeber et al., 1998). Rowe et al. (2002), however, found that effects of sub-threshold CD symptoms were not independent of family and environmental predictors. Of these, markers of family instability (including frequent house moves and step-family status) appeared most crucial in identifying cases of ODD that progressed to CD. In clinical samples, comparisons of 'pure' ODD and comorbid CD/ODD cases have largely found similar levels of environmental risk in both groups (Loeber et al., 2009). There is some evidence that parental hostility (Kolko, Dorn, Bukstein, & Burke, 2008) and lower socio-economic status (Greene et al., 2002) may be higher in comorbid CD/ODD cases.

Sub-dimensions of ODD

Links between ODD and CD may be clarified by the identification of distinct dimensions within the symptoms of ODD. Burke (in press) separated symptoms indexing negative affect (touchy, angry and spiteful) from other oppositional behaviors (loses temper, agues with adults and actively defies) using factor analysis. Negative affect predicted later symptoms of depression whereas oppositional symptoms predicted later CD. In a similar vein, Stringaris and Goodman (2009b) have proposed irritable, headstrong and hurtful dimensions of oppositionality on theoretical grounds. Despite strong inter-correlations, these dimensions show separable cross-sectional correlates. In a large national sample irritability was particularly associated with symptoms of emotional disorders, the headstrong dimension with symptoms of ADHD, and the hurtful dimension with callousness. All three dimensions were associated with CD symptoms cross-sectionally (Stringaris & Goodman, 2009b). In longitudinal analyses only the headstrong dimension predicted CD three years later, after control for baseline CD. Irritability predicted emotional psychopathology (Stringaris & Goodman, 2009a). With respect to formulating DSM-V ODD diagnostic criteria, it is possible that the identification of ODD dimensions may allow the diagnosis to be subtyped. This may in turn help to clarify the relationship of ODD and CD.

We take up these issues using the GSMS dataset. Specifically, we examine the developmental relationship between DSM-IV ODD and CD, capitalizing on longitudinal follow-ups that now cover the years from late childhood to early adulthood. We examine the hypothesis that transitions between ODD and CD may be more common in earlier developmental periods, and test whether these transitions are explained by sub-threshold CD symptomatology. We also examine the correlates and outcomes of youth who meet criteria for CD only, ODD only and mixtures of the two disorders across the course of adolescence. Finally we present a factor analysis of ODD symptoms to identify separable dimensions of oppositional behavior, and examine outcomes of these dimensions in adolescence.

Method

Sample

The GSMS is a longitudinal study of the development of psychiatric disorder based in a predominantly rural area of the southern United States. As part of an accelerated cohort (Schaie, 1965) two phase sampling design, a representative sample of 4500 9, 11and 13 year-olds resident in western North Carolina was selected using a household equal probability design from a population of approximately 20,000 children. In the screening phase parent report data addressing behavioral disorder items from the Child Behavior Checklist (CBCL, Achenbach & Edelbrock, 1983) were collected. The interview phase

included all children scoring above a pre-defined CBCL score (designed to identify the highest scoring 25% of the population) and a 10% random sample of the remainder. The GSMS target population includes an American Indian reservation and all age-appropriate American Indian children were recruited. American Indians formed 25% of the sample although they constituted 3% of the study area population. In order to generalize results back to the target population, observations were weighted proportionately to the inverse of their selection probability. Participation included 1420 individuals (56% male); 508 aged 9, 497 aged 11, and 415 aged 13. This was 80% of the target sample. Consistent with the population make-up, approximately 8% was African American and less than 1% was Hispanic.

GSMS began in 1993 with most recent data collected in 2005. Each cohort was observed annually up to age 16 with the exception that the youngest cohort was not assessed at age 13 due to funding constraints. All participants were also assessed at 19 and 21. Each study member aged 9 at initial assessment has therefore contributed up to 9 observations to the dataset; the age 11 cohort have contributed up to 8 observations and the age 13 cohort have contributed up to 6 observations. The study contains 8806 observations with 82% mean individual wave participation rate (range 94%-74%). Interviews were conducted in the home or a convenient location and timed, as far as possible, to coincide with the participants' birthdays. There is little evidence that symptom prevalence was influenced by assessment wave, cohort or differential drop out (Costello et al., 2003). Parents and children provided signed informed consent/assent and the study received ethical approval from the Duke Institutional Review Board.

Measures

At each assessment up to 16 the child and primary caretaker (usually the mother) were separately interviewed using the Child and Adolescent Psychiatric Assessment (CAPA) (Angold & Costello, 2000). The Young Adult Psychiatric Assessment (YAPA) (Angold et al., 2009) was used to interview participants at ages 19 and 21. These instruments assess psychiatric symptoms and diagnoses over the preceding 3 months using DSM-IV criteria. Age of onset was collected at each assessment with reference to each symptom endorsed. A binary variable indexing the presence of ODD using full DSM-IV diagnostic criteria, including symptom count threshold, 6-month duration and the presence of psychosocial impairment was employed here. All DSM-IV CD symptoms were measured with the exception that staying out late was not assessed at the first wave as this was conducted before DSM-IV was finalized. DSM-IV symptoms were assessed over the standard CAPA 3 month period rather than 12 months to ensure consistency across the interview. We used a binary variable indexing DSM-IV CD including requirements for both symptom severity and duration.

We also used a single binary variable, labeled depression, to index the presence of any depressive disorder (major depressive disorder, dysthymia or minor depression). Similarly, we formed a variable to indicate any anxiety disorder including generalized anxiety disorder, separation anxiety disorder, specific phobia, social phobia and panic disorder. A further binary variable indexed the presence of DSM-IV attention deficit Hyperactivity Disorder (ADHD). Substance disorder was coded as positive if any psychosocial impairment secondary to substance use was reported.

Dimensional measures of oppositionality, conduct problems, hyperactivity, anxiety and depression based on DSM-IV symptom counts were also employed. We also formed scales of aggressive conduct problems (based on the first 7 CD symptoms listed in DSM-IV) and non-aggressive conduct problems (based on the last 8 DSM-IV CD symptoms). Adult diagnoses were made according to relevant DSM-IV formulations. We used a symptom

count to index ASPD because the full disorder was very rarely endorsed (.7%). This symptom count did not include the CD history criterion to avoid predictor/outcome overlap.

Angold and Costello (1995) reported the two week test-retest reliability (kappa) of CAPA diagnoses ranged .56 to 1.0 in children aged 10-18. These reliabilities are comparable to those of other similar assessments such as the Diagnostic Assessment in Child and Adolescent Psychopathology (Shaffer, 1999). CAPA parent and child reports were combined at the symptom level using “or” logic except regarding hyperactivity as the CAPA does not ask children about this symptom area.

The CAPA also assesses a range of family and environmental correlates of disorder. We summed a number of conceptually related binary items into risk factor sub-scales. A social disadvantage scale contained six items: [1] income below federal poverty line (prevalence 20%, unweighted number of positive observations across all adolescent waves (n) =1848), [2] interviewer observed impoverished home environment (.9%, n=112), [3] one or both parents unemployed (9%, n=826), [4] one or both parents left school without graduating (18%, n=1613), [5] neighborhood (.9%, n=98) and [6] school (1%, n=101) perceived to be dangerous by parent or child. An atypical family structure scale was created from six items: [1] at least one parent a teenager at birth (18%, n=1695), [2] study child has 4 or more siblings living at home (4%, n=378), [3] one parent a step parent (13%, n=999), [4] single parent household (23%, n=1693), [5] more than 4 house moves in the previous 5 years (8%, n=591) and [6] the child has spent time in a foster home (2%, n=184). We also employed separate binary indicators of parenting difficulties; inadequate parental supervision (5%, n=512), harsh parental discipline (.6%, n=67), over-intrusive parenting (.6%, n=62), parental scape-goating of the child (.9%, n=93), and neglect (1%, n=162). Binary items also measured biological parent history of mental health treatment (26%, n=1763) and (separately) criminality (20%, n=1805), as indexed by conviction for crime.

Analyses

We categorized children according to six ‘pathways’ in diagnoses of ODD and CD across childhood and adolescence. Distinctions were made between [1] children who never met criteria for CD or ODD, [2] those who received at least one diagnosis of ODD during the study period but never met full criteria for CD, [3] those who met criteria for CD but not ODD, and three groupings of youth who met criteria for both disorders: [4] ODD preceding CD, [5] both disorders onset at the same assessment wave, and [6] CD preceding ODD. In later analyses categories 4-6 were collapsed into a single ODD/CD category.

Correlates of these groupings during childhood were analyzed using the full dataset of 6674 observations age 9-16 observations in which each individual contributed up to 7 observations. Adult outcomes were analyzed in a dataset of 2132 observations collected at ages 19 and 21 years, with each individual contributing up to two observations. The survey models of Stata 10.1 were used for these analyses. These models account for the non-independence of observations from the same participant by identifying the individual as the primary sampling unit and allow inclusion of the GSMS sampling weights (StataCorp, 2007). Robust variance estimation is used to produce unbiased parameter estimates and appropriate standard errors generalizable to the original population. The majority of analyses used logistic regression to predict binary dependent variables. Ordinal logistic regression models were used for analysis of symptom counts with skewed distributions.

Exploratory factor analysis of the ODD symptoms was conducted using the categorical data models of Mplus 4.0 (Muthen & Muthen, 2006) and recognized the sampling weight. Two and 3 factor models were fitted and the promax rotated factor loadings are reported. Promax

rotation allows extracted factors to be correlated, as they are expected to be when identifying psychopathologic symptom dimensions.

Results

Is ODD a developmental precursor to CD?

Criteria for ODD or CD were met at one or more study contacts by 19.1% of boys and 9.6% of girls between the ages of 9 and 16 years: CD was diagnosed on at least one occasion in 8.6% of youth (3.7% girls, 13.2% boys), and ODD in 9.7% (7.8% girls, 11.6% boys). We used a variety of approaches to assess whether ODD constituted a developmental precursor to CD, beginning with comparisons of age at first symptom onset. We found that ODD disorder was generally reported to onset earlier in development than CD. Median age at first symptom onset for cases of ODD was 2.5 years. CD first symptom onset was a full three years later, at 5.5 years. Despite these marked differences in overall ages at symptom onset, ODD symptoms preceded CD symptoms in only 56.0% (95% Confidence Interval (CI): 43.6, 67.4) of CD cases.

Next, we tested whether ODD predicted CD at both symptom and diagnostic levels. For the symptom analyses we used ODD symptoms at wave 1 to predict maximum CD symptom count at any subsequent childhood or adolescent study wave. The mean wave 1 ODD symptom count was .8 (SD = 1.3 Range: 0 - 8), and the mean maximum CD symptom count over subsequent waves was .8 (SD = 1.2, range 0 – 9). In an ordinal logistic regression model controlling for age and sex, ODD symptom count at wave 1 was a significant predictor of later maximum CD symptom count (OR for a 1 symptom increase in ODD symptoms=1.4, 95% CI: 1.3, 1.6, p<.001). This effect remained strongly significant (OR=1.2, 95% CI: 1.1, 1.4, p=.005) when wave 1 symptom counts for CD, generalized anxiety, depression and hyperactivity were included in the model. The ODD to CD prediction was similar for boys and girls (interaction p=.512).

Parallel analyses of diagnostic categories used ODD at wave 1 as the predictor, and CD at any later childhood/adolescent wave as the outcome. Controlling for age and sex, a wave 1 ODD diagnosis predicted later CD diagnosis (OR=7.9, 95% CI: 3.6, 17.6; p<.001). After controlling for baseline levels of CD the relationships were more complicated. Whereas ODD functioned as a clear risk for later CD in boys, effects in girls appeared to be attenuated by a high rate of comorbidity between the two disorders at first assessment, and strong CD persistence. After controlling for wave 1 CD diagnosis, ODD predicted later CD in boys (OR=6.5, 95% CI: 1.8, 23.2; p=.004) but not in girls (OR=.5, 95% CI: .1, 2.3; p=.407) (interaction p=.011). Initial levels of ODD and CD were more strongly related in girls (OR=64.7, 95% CI: 19.6, 213.4; p<.001) than in boys (OR=7.6, 95% CI: 3.4, 16.8; p<.001) (interaction p=.003). Continuity from wave 1 CD to later CD was also stronger in girls (OR=54.8, 95% CI: 13.7, 218.8; p<.001) than in boys (OR=8.6, 95% CI: 3.2, 23.2; p<.001) (interaction p=.032). Controlling for ADHD at wave 1 did not substantively impact upon these results.

We extended these analyses to test whether prediction from ODD to CD in boys was affected by levels of sub-threshold CD symptoms. Confining the analysis to boys without CD at Wave 1, CD symptoms were significant additional predictors of a later CD diagnosis (OR for a 1 symptom increase: 3.0, 95% CI: 1.8, 4.9, p<.001). Despite these effects, however, ODD remained independently associated with increased risk of later CD (OR=5.7, 95% CI: 2.1, 15.2; p=.001).

The analyses to this point all suggested that ODD (whether assessed at the symptom or the diagnostic level) functioned as a precursor to, and risk factor for, CD. What they could not

demonstrate, however, was how commonly this progression occurred. To assess this, we classified each child into one of six mutually exclusive onset 'pathways' reflecting the ordering of onsets of ODD and CD across study waves. As Table 1 shows, many disruptive youth only ever met criteria for one of these disorders between late childhood and mid-adolescence, and transitions between the two disorders were less common than anticipated. In particular, of the children who met criteria for CD a substantial proportion did not have ODD at any study wave. In children with ODD no diagnosis of CD during the study was also common (60.5%, 95% CI: 49.7, 70.2). Table 1 shows that the confidence intervals around percentages of children falling into each of the comorbid pathways overlap. Concurrent CD and ODD onset was the most common configuration. ODD→CD was slightly less common than the pathway where CD was diagnosed *before* ODD. Although the confidence intervals indicate this was not significantly different, there is certainly no evidence that ODD→CD was a particularly common pathway. ODD→CD appeared especially uncommon in girls, with only 3 following this pathway.

As these findings were unexpected, we checked whether they might be an artifact, arising from differential non-response rate of children with ODD at Wave 1 at later study waves. We found no evidence for a pattern of this kind. Later participation rates were high across all the pathway groupings, and did not differ between the ODD→CD (89.7%, 95% CI: 85.4, 94.1) and ODD never CD paths (88.8%, 95% CI: 84.9, 92.7).

Next, to assess whether these transition patterns varied by age, we compared pathway classifications in the three age cohorts who entered the study at ages 9, 11 and 13 respectively. As Table 1 shows, relative to other pathways to CD, the likelihood of simultaneous CD and ODD onset was stable across cohort and a CD diagnosis with no history of ODD became more usual with increasing age. The ODD→CD pathway, by contrast, became significantly less common across age, relative to the other pathways to CD (OR for a 1 year increase in age =.6, 95% CI: .3, .9, p=.019).

The CD→ODD pathway was also unexpected, so we explored it further in longitudinal analyses. In a model predicting future ODD, wave 1 ODD (OR=7.7, 95% CI: 3.1, 19.4, p<.001) and CD (OR=2.3, 95% CI: 1.1, 4.8; p=.034) were both significant predictors. When wave 1 ODD symptom count (OR=1.8, 95% CI: 1.5, 2.0, p<.001) was included as a predictor instead of ODD diagnosis, CD diagnosis no longer predicted future ODD (OR=1.1, 95% CI: .5, 2.3; p=.819).

Characteristics of youth with pure and combined CD and ODD diagnoses

The analyses thus far suggested that, although some disruptive youth meet diagnostic criteria for both ODD and CD across the late childhood/mid-adolescent years, many present with only one of these disorders. We explored whether these groups could be distinguished on the basis of symptom patterns, selected risk factors and rates of comorbid disorders in childhood and adolescence. Because numbers in the three sub-groups meeting criteria for both ODD and CD over the study period were small, we combined these cases (ODD→CD, ODD/CD and CD→ODD) into a single grouping for these comparisons.

Table 2 shows mean levels of ODD symptoms were similar in youth with ODD only and combined ODD+CD, and mean levels of CD symptoms (total and split into aggressive and non-aggressive symptoms) differed little between cases with CD only and the combined pattern. As Table 2 also makes clear, however, youth who only ever met full diagnostic criteria for one disorder (ODD or CD) during the study period nonetheless typically showed elevated levels of sub-threshold symptomatology for the other disorder. We used the retrospectively reported age at first CD symptom to classify CD cases into childhood and adolescent onset sub-types. Onset of first CD symptom was prior to age 10 for 86.3% of CD

diagnoses (childhood onset CD) with the remaining 13.7% classified as adolescent onset CD according to DSM-IV. The mix of child and adolescent onset CD in the CD only (16.4%) and CD+ODD (10.5%) groups did not differ significantly (p=.572).

Table 2 also shows levels of selected correlates for youth in the different pathways. These were analyzed as dependent variables predicted by pathway. Boys were significantly over-represented in the CD and CD+ODD groups, while ODD only cases showed no male preponderance. In terms of other social and familial risks the diagnosed groups showed significantly elevated rates by comparison with youth with no disruptive disorders. Contrasts among disorder groupings revealed few significant differences, however. The only exception to this pattern was parental scape-goating of the child, which was specifically associated with ODD. There were some tendencies that inadequate supervision was more strongly associated with ODD than CD but the statistical comparisons among the disorder groups were not fully supportive of this relationship.

The final section of Table 2 shows levels of comorbid disorders in childhood and adolescence. Youth in all the disruptive disorder groupings were at increased risk of emotional disorders (anxiety and depression) and substance disorder relative to the no ODD/CD group, but did not differ significantly from each other. Risk for ADHD was also elevated in all disorder groups. There was some evidence that risk was particularly high in the CD+ODD group relative to the CD group, but the CD+ODD comparison with ODD group was non-significant.

Early adult outcomes

Psychiatric outcomes at ages 19 and 21 years are shown in Table 3. ASPD symptoms showed stronger associations with a history of childhood and adolescent CD than with prior ODD. Both the CD and CD+ODD groups had significantly higher levels of ASPD symptoms than youth with no history of CD/ODD, and did not differ from each other. Youth on the ODD only pathway, by contrast, were not at significantly increased risk. Early adult substance disorder appeared more common in the CD and CD+ODD groups relative to the ODD only and no CD/ODD groups, although the only significant difference was between the CD+ODD and the no CD/ODD groups.

In contrast, increased risk for anxiety disorders in early adulthood was confined to youth with a history of ODD. Both the ODD and ODD+CD groups were at significantly higher risk of anxiety than the no CD/ODD and CD only groups. Youth on the CD only pathway showed significantly *lower* rates of early adult anxiety disorders than those with no history of disruptive behavior problems. A somewhat similar pattern emerged for depression, although contrasts between the disordered groups were non-significant.

Symptom dimensions in ODD

In the final stage of the analyses we explored whether there were separable symptom dimensions within the ODD symptoms. In an exploratory factor analysis of ODD symptoms in the full sample at Wave 1, a two factor solution provided an acceptable fit to the data (RMSEA=.021). Computation of a three factor model failed, showing a negative estimated residual variance for the spiteful symptom which may indicate that too many factors had been specified. Factor loadings from the two factor model are shown in Table 4, following Promax rotation. The two factors correlated at .55, and high loading items were similar to the irritable and headstrong dimensions proposed by Stringaris and Goodman (2009b); the symptom of spitefulness loaded clearly on the headstrong factor in this solution.

We next examined whether pure cases of irritable and headstrong ODD could be identified within the DSM-IV diagnostic framework. The factor analysis identified 3 symptoms

loading onto the irritability factor, and 5 symptoms loading onto the headstrong factor. Because DSM-IV criteria for ODD require 4 symptoms to be present, at least one headstrong symptom must be endorsed in all cases to reach the diagnostic threshold. Across the full 9-16 age-range, almost all cases that met ODD criteria also showed at least some level of irritability: only 3.3% had no irritability symptoms, 18.8% had one, 52.9% had two and 25.0% had three. These figures suggest few cases of 'pure' headstrong ODD exist within the DSM framework; most youth reaching the diagnostic threshold showed a mixed symptom pattern.

Given these findings, we took a dimensional approach to examine the predictive utility of the irritability/headstrong distinction. We summed items loading onto each factor at Wave 1 and examined the extent to which these two dimensions were differentially related to risk for behavioral and emotional disorders at subsequent childhood and adolescent study waves. Both symptom dimensions were standardized and age, sex and the Wave 1 level of the outcome variable were included as predictors.

Beginning with behavioral disorders, Table 5 shows the headstrong dimension significantly predicted later CD. Prediction from irritability fell only a little below significance, and there was no evidence that effects of the two dimensions differed. In further analyses we examined prediction to aggressive and non-aggressive CD symptom counts. An ordinal logistic regression model showed that the irritability (OR=1.3, 95% CI 1.1, 1.6; p=.01) and headstrong (OR=1.4, 95% CI: 1.2, 1.7; p<.001) dimensions predicted maximum aggressive CD symptom count over future observations. Non-aggressive CD symptoms were also predicted by headstrongness (OR=1.4, 95% CI: 1.2, 1.6; p<.001) while irritability (OR=1.2, 95% CI: 1.0, 1.5; p=.058) fell just below significance. Given the overlap in the confidence intervals, however, there was no convincing evidence of differential prediction of aggressive and non-aggressive CD symptoms.

Links between the dimensions and later full ODD diagnosis were also very similar for the two dimensions. The headstrong dimension did, however, predict later substance disorder significantly more strongly than irritability (which had an OR below 1 in relation to substance disorder risk). We ran a further model including Wave 1 CD diagnosis as an additional predictor of substance disorder: headstrong symptoms remained highly significant predictors even with prior CD controlled (OR=1.4, 95% CI: 1.1, 1.7; p=.004) and CD diagnosis was non-significant (OR=1.3, 95% CI: .6, 2.7; p=.484). An opposite pattern emerged in relation to anxiety: irritability predicted risk for later anxiety diagnoses while headstrongness did not. Depression was significantly predicted by headstrongness; prediction from irritability fell short of significance, but comparisons between the two dimensions were non-significant.

Discussion

This paper examined the relationship of DSM-IV CD and ODD with a view to informing the revision process of the disruptive behavior disorders specification in DSM-V. The GSMS is well placed for this purpose, and has already produced a number of relevant papers (Angold & Costello, 1996; Rowe, Maughan, Costello, & Angold, 2005; Rowe et al., 2002). Strengths of the study include the collection of parent and child interview data from a large-scale community sample of boys and girls, and a longitudinal design spanning ages 9-21 years. Data from community samples are important for many of the issues being considered in the revision process, as clinical samples may be subject to referral biases. Four limitations must also be noted. First, the GSMS sample is substantially rural: replication of our results in urban samples and samples covering a wider range of ethnic backgrounds will be important to ensure generalizability. Second, the inherently low prevalence of psychiatric disorders

and the less than ideal reliability of psychiatric interviews reduced statistical power to some extent. Set against this concern, the GSMS provides a large sample and a number of our analyses aggregated diagnoses across waves of assessment, increasing the frequency of outcome. We also supplemented our analyses of diagnoses with more powerful analyses based on continuous counts of psychiatric symptoms. Third, during the course of analyses we have conducted a number of statistical tests, taking alpha as .05 for each analysis. This increase the possibility that Type I errors may have been made in statistical inference. This highlights the importance of replication in research of this kind. Fourth, our parenting variables were measured with single items that may not have fully captured the dimensions of risk present in the population. This may limit the conclusions that can be made about relationships with these constructs.

Developmental links between ODD and CD

Approximately one in five boys and one in ten girls in GSMS met criteria for ODD and/or CD at some point between the ages of 9 and 16 years – cumulative prevalence estimates close to those reported retrospectively by adults in the NCSR (Nock, Kazdin, Hiripi, & Kessler, 2006; Nock et al., 2007). Our first aim was to examine developmental associations between these two disorders, and in particular, to evaluate DSM-IV's assumption that in '… a significant proportion of cases…' (p. 92) ODD will constitute a developmental precursor to CD.

We approached this issue in a variety of ways. First, like other studies (Loeber et al., 1992; Nock et al., 2007), we confirmed that retrospectively reported first ODD symptom onsets in the GSMS sample occurred very early in development, well before CD symptom criteria typically began to be met. We note, however, that earlier onset is a necessary condition for the developmental model, but is far from sufficient. Next, we confirmed that ODD symptoms at the first assessment wave were clear predictors of later CD symptoms in boys and girls, and found that wave 1 ODD diagnoses independently predicted later diagnoses of CD in boys but not girls. Finally, we evaluated the relative *frequency* of this particular sequence of onsets (that is, ODD preceding CD), given the range of potential onset sequences between two disorders that could, in principle, be observed.

Taking this last approach, we found associations between the two disorders far less marked than might have been anticipated. The majority (55%) of CD cases never received a diagnosis of ODD. Less surprisingly, many children with ODD never met full criteria for CD. However, youth in these 'pure' diagnostic categories had higher levels of sub-threshold symptoms of the other diagnosis than children who were never diagnosed with CD or ODD. Viewed from another perspective, of all the CD cases only 9.2% had shown ODD at an earlier study wave. In general, however, the overall pattern of our findings was consistent with results from other epidemiological studies (Loeber et al., 2009) in showing that in community samples CD and ODD are less strongly associated than suggested by data from referred groups.

Among youth who did show both disorders, the retrospective age of onset data showed just more than half reported that an ODD symptom emerged prior to their first CD symptom. This result must, of course, be interpreted in the context of known inaccuracies in dating symptom onsets retrospectively (Angold, Erkanli, Costello, & Rutter, 1996). Our pathway analyses showed that the assumed transition from full disorder ODD to CD diagnosis was not common. It was no more common than concurrent onset or the pathway where CD onset prior to ODD. We considered a number of GSMS design features that might have contributed to this pattern of results. First, we checked whether selective attrition might have underestimated ODD development to CD but found no evidence to support this effect. Participation rates were high across all study waves, and did not differ in the ODD never CD

and ODD→CD pathways. Second, the CAPA uses a three-month reporting period. As a result, annual assessments may miss some periods of disorder and therefore underestimate prevalence in the population. This may be particularly relevant for CD as DSM-IV requires symptoms to be present at any point in a 12 month timeframe, so long as one is present within the previous 6 months. In addition, year-on-year fluctuations (whereby cases fall just above and just below diagnostic thresholds) have been reported even in clinical samples (Burke, in press). Each of these factors could have contributed to an underestimation of rates of disorder. To the extent that cases of ODD were missed we may have overestimated the proportion of cases in the CD only pathway. To the extent that cases of CD were missed, we may have overestimated proportions in the ODD only group. In practice, however, we know of no evidence to suggest that the CAPA substantially underestimates either ODD or CD diagnoses. Comparative studies show no systematic difference between prevalence estimates derived from the CAPA and from other diagnostic approaches (Maughan et al., 2004). It is possible that measurement artifacts may have led to some underestimation of the prevalence of ODD and CD but any such effect was likely to have been minimal.

A further – and possibly more salient – issue concerns the age-range covered by our observations. The 9-16 age range covered in GSMS is a crucial period for the development of antisocial behavior, but clearly does not cover the full window of risk. Many children meet criteria for disruptive behavior disorders at much younger ages, beginning in the pre-school years (Egger & Angold, 2006). This could have a number of implications for our study. First, DSM-IV indicates that ODD may be a stronger risk factor for childhood-onset (first symptom onset prior to age 10) than adolescent onset CD. Although most GSMS observations were of children older than 10, first symptom onsets were typically reported to have occurred much earlier in childhood. Applying the DSM-IV age 10 ‘rule’ the vast majority of CD cases in the sample were classified as childhood-onset. Though we did not directly observe our cases at these earlier ages, this makes it unlikely that our relatively low rate of ODD→CD progression could be explained by a predominance of adolescent-onset cases of CD.

A rather different possibility is that we underestimated ODD→CD because some of the children with CD in our dataset had a history of ODD in early childhood that had resolved prior to entry into GSMS. We have no way of testing this possibility directly. We did examine whether ODD→CD progression was more common at younger ages within the GSMS timeframe, however. Developmental variation was highlighted as the youngest cohort of GSMS (entering age 9) showed a stronger ODD→CD link than did youth who entered the study at older ages (age 11 or 13). Even in the youngest cohort, less than 1 in 5 cases of CD was preceded by ODD. Therefore we found some evidence that ODD→CD progression is more common earlier in the GSMS age range. To our knowledge this is the first analysis to test whether ODD→CD transition varies by age. Younger samples will be required to test whether this pattern extends earlier into childhood. If replicable this might indicate that ODD is a stronger precursor to full disorder CD that onsets in childhood rather than adolescence. This possibility should be addressed in further research.

Despite the relative scarcity of children following the ODD→CD pathway, we found that a diagnosis of ODD at wave 1 was a significant risk factor for a later CD diagnosis independent of CD continuity for boys, but not girls. This finding was highlighted in our analyses of the first four waves of GSMS (Rowe et al., 2002), and was confirmed over the more extended observation period available here. As noted in the introduction, Lahey et al. (2009) recently reported negligible prediction from questionnaire measures of ODD symptoms to later CD symptoms once baseline levels of CD symptoms were controlled. We extended our analyses of ODD→CD in boys to control for sub-threshold CD symptoms at baseline in a parallel way. Adding this covariate reduced associations between ODD and

later CD, but still showed that prior ODD was a significant predictor. In part, these discrepant findings seem likely to reflect the more severe difficulties indexed by a diagnostic measure than by questionnaire approaches. In addition, as outlined earlier, the measures used by Lahey et al. may not have provided the most effective separation of oppositional and conduct problem constructs.

Our own symptom count based analyses found ODD symptoms at wave 1 predicted later CD symptoms but did not identify the sex difference indicated in the diagnosis based analyses. ODD symptoms at wave 1 were similarly predictive of later CD symptoms for boys and girls even after control of wave 1 CD symptoms. This discrepancy between diagnosis and symptom based analyses may be relevant to the debates on the utility of sex-specific diagnostic thresholds for CD (Zoccolillo, 1993). A dimension of CD severity is implied by the DSM-IV specification of mild, moderate and severe CD. Increased prominence for a dimensional approach has been considered for DSM-V (Moffitt et al., 2008) and there is increased recognition that diagnostic thresholds may be arbitrary (e.g., Pickles et al., 2001). In clinical practice, however, dichotomous decision making will continue to be required, for example in deciding whether and when to provide treatment. Therefore dichotomous diagnosis is likely to remain a prominent feature of DSM-V (Angold & Costello, 2009). Identification of thresholds that are effective for both boys and girls is an important challenge for research.

The identification of a small group of children who met criteria for DSM-IV CD prior to a diagnosis of ODD was unexpected. When explored further in longitudinal analyses, CD at initial contact did not predict future onset of ODD once initial ODD symptomatology was controlled. This indicates that sub-threshold ODD symptomatology comorbid with CD was responsible for the prediction to future ODD.

Differentiating ‘pure’ and combined/comorbid cases

Our pathway analyses suggested that in community samples youth meeting criteria for ODD and/or CD in late childhood and adolescence fall into one of three broad groupings: those who only ever meet full criteria for ODD, those who only ever meet full criteria for CD and those who meet full criteria for both disorders at some point between late childhood and middle adolescence. We compared these three groups in terms of exposure to a range of potential risk factors, comorbidities and psychiatric outcomes in early adult life. All three groups were clearly differentiated from youth who did not meet criteria for ODD/CD on almost all of these indicators. These results clearly highlight that ODD is a disorder worthy of clinical recognition rather than a feature of normal child development. In addition, we identified a number of features (largely consistent with evidence from past reports) that distinguished ODD and CD. By contrast, comparisons between ‘pure’ and ‘combined’ disorder patterns did not highlight substantial differences.

Risk factors, histories of parental antisocial behavior and mental health problems were closely similar in CD and ODD, as were elevated levels of exposure to social adversity and problems in family functioning. Although few studies have compared the correlates of ODD and CD, this pattern of findings is consistent with previous reports based on GSMS (Rowe et al., 2002) and other datasets (Kolko et al., 2008), and runs counter to the view that ODD is associated with lower levels of such risks as compared with CD. There were two exceptions to this pattern. First, boys were at greater risk of CD (whether alone or in combination with ODD), but there were no sex differences in rates of ‘pure’ ODD. This finding is also largely consistent with reports from other studies, although many have reported at least some tendency for more boys to meet criteria for ODD than girls. We suspect that these study differences may largely reflect differing treatments of ODD/CD comorbidity: by definition, our ‘ODD only’ group excluded cases with comorbid CD,

whereas studies examining overall prevalence rates frequently do not. Second, parental use of the child as a scapegoat was elevated in ODD (whether comorbid with CD or not) but not in CD as was also highlighted in other analyses of the GSMS (Shanahan et al., 2008). It is possible that this represents a parental response to child oppositonality rather than a specific risk factor for ODD.

Our final between-group contrasts centered on comorbidity with other disorders in childhood and adolescence and prediction to psychiatric outcomes in early adult life. Across all childhood and adolescent observations, rates of comorbidity were largely similar. Early adult outcomes provided a more differentiated picture. Increased risks of ASPD symptoms in adulthood were largely confined to cases with prior histories of CD. In contrast, a history of ODD was associated with some increased risk for depression, and with markedly increased risk for anxiety in early adulthood. Strikingly, cases in the CD only group were at significantly *lower* risk for anxiety disorders than youth with no prior history of ODD or CD. Copeland et al.'s (2009) analyses of the GSMS also provide evidence of poor adult psychiatric functioning following ODD. Taken together, these findings on longer-term outcomes provide less support for a model of ODD as a milder form of CD, and point more in the direction of the two disorders representing distinct forms of psychopathology. This indicates, therefore, that the hierarchical relationship between ODD and CD specified in DSM-IV should be abandoned. Our final set of analyses was designed to test whether distinguishable dimensions of disorder could be identified within ODD.

Symptom dimensions in ODD

Recent evidence has suggested that different ODD behavioral dimensions may have different correlates and outcomes (Burke, in press; Stringaris & Goodman, 2009a, 2009b). We examined whether there was empirical support for sub-factoring ODD symptoms that may in turn have implications for understanding links with CD. Our findings supported a two factor model with close similarities to the dimensions proposed by Stringaris and Goodman (2009b) on a priori theoretical grounds. Our irritability factor contained the same items as their irritability dimension, and our headstrong factor also contained all the items they proposed as indexing 'headstrongness'. In addition, however, our headstrong factor also included the spiteful/vindictive symptom that Stringaris and Goodman treat as a separate dimension. In the GSMS spiteful/vindictiveness is indexed by a single symptom (following the DSM-IV specification), whereas in Stringaris and Goodman's work separate items index spitefulness and vindictiveness. It is possible that a separate third factor would have been identified here if that approach had been taken. Future studies would benefit from including a greater range of items designed to measure each hypothesized dimension. Our factor analytic results also overlapped with Burke's (in press) analysis. Our irritability factor was similar to Burke's negative affect factor, with the exception that our results included temper tantrums and excluded spitefulness. Our headstrong factor was also similar to Burke's oppositionality factor except that ours included spitefulness and also added annoying and blaming. The latter two symptoms were excluded from Burke's factors due to cross-loadings. Differences in analytic approaches and our focus on a community rather than a referred sample may have contributed to these variations.

Prognostic links between irritable and headstrong factors at wave 1 and outcomes up to age 16 were largely consistent with Stringaris and Goodman's (2009a) results. Prediction to later DSM-IV ODD and CD, including aggressive and non-aggressive CD symptoms, was very similar for the irritable and headstrong dimensions, and did not differ significantly regarding depression. The headstrong dimension uniquely predicted the substance disorder outcome, however, while irritability uniquely predicted anxiety. These results also have some similarities with Burke's (in press) findings; negative affect predicted later depression specifically, whereas the oppositional factor predicted later CD.

We conducted some initial exploration of whether categorical diagnoses of ODD could be sub-typed on the basis of irritable and headstrong symptoms. Using current diagnostic guidelines this was far from straightforward. Cases of ODD purely involving irritability cannot exist with a four symptom diagnostic threshold, and cases with only headstrong symptoms were exceedingly rare. In practice, most youth who met criteria for ODD showed a mix of these two symptom patterns. Person-centered approaches such as latent class analyses might provide a complimentary perspective to our findings and more complex approaches to sub-typing warrant further investigation. For example the recent DSM-V proposal for Temper Dysregulation Disorder with Dysphoria (TDD, DSM-5 Childhood and Adolescent Disorders Work Group, 2010) would identify the most irritable cases of ODD as suffering a mood disorder. The links found here and elsewhere between irritability and affective disorder are consistent with the suggested mood diagnosis. Further research will be required to identify whether the correlates and outcomes of TDD differ from the current specification of ODD and to identify the clinical characteristics of children with ODD who do not meet the TDD criteria. A growing body of evidence also indicates that callous and unemotional traits may usefully sub-type CD (Frick & White, 2008; Rowe et al., 2010). It is unclear whether similar sub-typing may be useful for ODD as well.

Recommendations for DSM-V

The formulation of the relationship between ODD and CD in DSM-IV was largely based on findings from clinical samples. Findings from community studies such as GSMS show a somewhat different pattern. Although there are strong overlaps between CD and ODD and developmental continuity at least in boys, these links are much weaker than those found in clinically referred samples. At least in late childhood and adolescence, many cases of CD will emerge without marked histories of ODD, and many cases of ODD will not progress to CD. As we have seen, however, this in no sense implies that cases meeting full criteria for ODD represent benign or transient disorders. Indeed, as our and other findings are increasingly underlining, ODD may be ‘in a class by itself’ (Copeland et al., 2009) in indexing risk for both emotional and behavioral pychiatric disorders later in development. Further studies are needed to clarify the processes underlying these links. At this stage, however, our findings point clearly to the utility of treating CD and ODD as fully separate disorders in DSM-V. As noted by Burke (in press) much past research has combined CD and ODD in epidemiological studies, hindering development of an evidence base on their differences. Fully separating the disorders for the latest revision will therefore improve the information available for the formulation of DSM-VI.

References

Achenbach, TN.; Edelbrock, CS. Manual for the child behavior checklist and revised profile. University of Vermont; Burlington: 1983.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th edition. American Psychiatric Association; Washington DC: 1994.

Angold A, Costello EJ. A test-retest reliability study of child reported psychiatric symptoms and diagnoses using the Child and Adolescent Psychiatric Assessment (CAPA-C). Psychological Medicine 1995;25:755–762. [PubMed: 7480452]

Angold A, Costello EJ. Toward establishing an empirical basis for the diagnosis of oppositional defiant disorder. Journal of the American Academy of Child and Adolescent Psychiatry 1996;35(9): 1205–1212. [PubMed: 8824064]

Angold A, Costello EJ. The Child and Adolescent Psychiatric Assessment (CAPA). Journal of the American Academy of Child and Adolescent Psychiatry 2000;39(1):39–48. [PubMed: 10638066]

Angold A, Costello EJ. Nosology and measurement in child and adolescent psychiatry. Journal Of Child Psychology And Psychiatry 2009;50(1-2):9–15. [PubMed: 19175818]

Angold, A.; Cox, A.; Prendergast, M.; Rutter, M.; Simonoff, E.; Costello, EJ., et al. The Young Adult Psychiatric Assessment (YAPA): Version 2.0.3. Duke University Medical Center; Durham, NC: 2009.

Angold A, Erkanli A, Costello EJ, Rutter M. Precision, reliability and accuracy in the dating of symptom onsets in child and adolescent psychopathology. Journal of Child Psychology and Psychiatry and Allied Disciplines 1996;37(6):657–664.

Beauchaine TP, Gatzke-Kopp L, Mead HK. Polyvagal Theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence. Biological Psychology 2007;74(2):174–184. [PubMed: 17045726]

Burke, JD. The Relationship between Conduct Disorder and Oppositional Defiant Disorder and their Continuity with Antisocial Behaviors: Evidence from Longitudinal Clinical Studies. In: Shaffer, D.; Leibenluft, E.; Rohde, LA., editors. Externaliizing Disorders of Childhood: Refining the Research Agenda for DSM-V. American Psychiatric Association; Arlington, VA: (in press)

Burke JD, Loeber R, Lahey BB, Rathouz PJ. Developmental transitions among affective and behavioral disorders in adolescent boys. Journal of Child Psychology and Psychiatry 2005;46(11): 1200–1210. [PubMed: 16238667]

Burt SA, McGue M, Krueger RF, Iacono WG. Sources of covariation among the child-externalizing disorders: informant effects and the shared environment. Psychological Medicine 2005;35(8): 1133–1144. [PubMed: 16116939]

Copeland WE, Shanahan L, Costello EJ, Angold A. Childhood and Adolescent Psychiatric Disorders as Predictors of Young Adult Disorders. [Article]. Archives Of General Psychiatry 2009;66(7): 764–772. [PubMed: 19581568]

Costello EJ, Mustillo S, Erkanli A, Keeler G, Angold A. Prevalence and development of psychiatric disorders in childhood and adolescence. Archives of General Psychiatry 2003;60(8):837–844. [PubMed: 12912767]

Dick DM, Viken RJ, Kaprio J, Pulkkinen L, Rose RJ. Understanding the covariation among childhood externalizing symptoms: Genetic and environmental influences on conduct disorder, attention deficit hyperactivity disorder, and oppositional defiant disorder symptoms. Journal of Abnormal Child Psychology 2005;33(2):219–229. [PubMed: 15839499]

DSM-5 Childhood and Adolescent Disorders Work Group. Justification for Temper Dysregulation Disorder with Dysphoria. [Retrieved 13 May 2010]. 2010 from http://www.dsm5.org/Proposed%20Revision%20Attachments/Justification%20for%20Temper%20Dysregulation%20Disorder%20with%20Dysphoria.pdf

Eaves LJ, Rutter M, Silberg JL, Shillady L, Maes H, Pickles A. Genetic and environmental causes of covariation in interview assessments of disruptive behavior in child and adolescent twins. Behavior Genetics 2000;30(4):321–334. [PubMed: 11206087]

Eaves LJ, Silberg JL, Meyer JM, Maes HH, Simonoff E, Pickles A, et al. Genetics and developmental psychopathology: 2. The main effects of genes and environment on behavioral problems in the Virginia Twin Study of Adolescent Behavioral Development. Journal of Child Psychology and Psychiatry 1997;38(8):965–980. [PubMed: 9413795]

Egger HL, Angold A. Common emotional and behavioral disorders in preschool children: presentation, nosology, and epidemiology. [Review]. Journal Of Child Psychology And Psychiatry 2006;47(3-4):313–337. [PubMed: 16492262]

Frick PJ, Lahey BB, Loeber R, Stouthamerloeber M, Christ MAG, Hanson K. Familial Risk-Factors to Oppositional Defiant Disorder and Conduct Disorder - Parental Psychopathology and Maternal Parenting. Journal of Consulting and Clinical Psychology 1992;60(1):49–55. [PubMed: 1556285]

Frick PJ, White SF. Research Review: The importance of callous-unemotional traits for developmental models of aggressive and antisocial behavior. Journal of Child Psychology and Psychiatry 2008;49(4):359–375. [PubMed: 18221345]

Greene RW, Biederman J, Zerwas S, Monuteaux MC, Goring JC, Faraone SV. Psychiatric comorbidity, family dysfunction, and social impairment in referred youth with oppositional defiant disorder. American Journal of Psychiatry 2002;159(7):1214–1224. [PubMed: 12091202]

Kim-Cohen J, Caspi A, Taylor A, Williams B, Newcombe R, Craig IW, et al. MAOA, maltreatment, and gene-environment interaction predicting children's mental health: new evidence and a meta-analysis. [Article]. Molecular Psychiatry 2006;11(10):903–913. [PubMed: 16801953]

Kolko DJ, Dorn LD, Bukstein OG, Burke J. Clinically Referred ODD Children with or without CD and Healthy Controls: Comparisons Across Contextual Domains. Journal of Child Family Studies 2008;17:714–734.

Lahey B, Hulle C, Rathouz P, Rodgers J, D'Onofrio B, Waldman I. Are Oppositional-Defiant and Hyperactive-Inattentive Symptoms Developmental Precursors to Conduct Problems in Late Childhood? Genetic and Environmental Links. Journal Of Abnormal Child Psychology 2009;37(1):45–58. [PubMed: 18648930]

Langbehn DR, Cadoret RJ, Yates WR, Troughton EP, Stewart MA. Distinct contributions of conduct and oppositional defiant symptoms to adult antisocial behavior. Archives of General Psychiatry 1998;55:821–829. [PubMed: 9736009]

Loeber R, Burke J, Pardini DA. Perspectives on oppositional defiant disorder, conduct disorder, and psychopathic features. Journal of Child Psychology and Psychiatry 2009;50(1-2):133–142. [PubMed: 19220596]

Loeber R, Green SM, Lahey BB, Christ MA, Frick PJ. Developmental sequences in the age of onset of disruptive child behaviors. Journal of Child Family Studies 1992;1:21–41.

Loeber, R.; Keenan, K.; Russo, MF.; Green, SM.; Lahey, BB.; Thomas, C. Secondary data analyses for DSM-IV on the symptoms of oppositional defiant disorder and conduct disorder. In: Widiger, TA.; Frances, AJ.; Pincus, HJ., editors. DSM-IV Sourcebook. Vol. Vol. 4. American Psychiatric Association; Washington, DC: 1998. p. 465-490.

Maughan B, Rowe R, Messer J, Goodman R, Meltzer H. Conduct disorder and oppositional defiant disorder in a national sample: developmental epidemiology. Journal of Child Psychology and Psychiatry 2004;45:609–621. [PubMed: 15055379]

Moffitt TE, Arseneault L, Jaffee SR, Kim-Cohen J, Koenen KC, Odgers CL, et al. Research Review: DSM-V conduct disorder: research needs for an evidence base. Journal of Child Psychology and Psychiatry 2008;49:3–33. [PubMed: 18181878]

Moffitt, TE.; Caspi, A.; Rutter, M.; Silva, PA. Sex differences in antisocial behaviour: Conduct disorder, delinquency, and violence in the Dunedin Longitudinal Study. Cambridge University Press; Cambridge: 2001.

Muthen, LK.; Muthen, BO. Mplus User's Guide. Fourth Edition. Muthen & Muthen; Los Angeles, CA: 2006.

Nock MK, Kazdin AE, Hiripi E, Kessler RC. Prevalence, subtypes, and correlates of DSM-IV conduct disorder in the national comorbidity survey replication. Psychological Medicine 2006;36(5):699–710. [PubMed: 16438742]

Nock MK, Kazdin AE, Hiripi E, Kessler RC. Lifetime prevalence, correlates, and persistence of oppositional defiant disorder: Results from the National Comorbidity Survey Replication. Journal Of Child Psychology And Psychiatry 2007;48(7):703–713. [PubMed: 17593151]

Pickles A, Rowe R, Simonoff E, Foley D, Rutter M, Silberg J. Child psychiatric symptoms and psychosocial impairment: relationship and prognostic significance. British Journal of Psychiatry 2001;179:230–235. [PubMed: 11532800]

Rey JM, Bashir MR, Schwarz M, Richards IN, Plapp JM, Stewart GW. Oppositional defiant disorder: Fact or fiction? Journal of the American Academy of Child and Adolescent Psychiatry 1988;27(2): 157–162. [PubMed: 3360716]

Rowe R, Maughan B, Costello EJ, Angold A. Defining oppositional defiant disorder. Journal of Child Psychology and Psychiatry 2005;46(12):1309–1316. [PubMed: 16313431]

Rowe R, Maughan B, Moran P, Ford T, Briskman J, Goodman R. The role of callous and unemotional traits in the diagnosis of conduct disorder. Journal of Child Psychology and Psychiatry 2010;51(6): 688–695. [PubMed: 20039995]

Rowe R, Maughan B, Pickles A, Costello EJ, Angold A. The relationship between DSM-IV oppositional defiant disorder and conduct disorder: findings from the Great Smoky Mountains Study. Journal of Child Psychology and Psychiatry and Allied Disciplines 2002;43(3):365–373.

Rowe R, Maughan B, Worthman CM, Costello EJ, Angold A. Testosterone, conduct disorder and social dominance in boys: Pubertal development and biosocial interaction. Biological Psychiatry 2004;55:546–552. [PubMed: 15023584]

Rowe R, Rijsdijk FV, Maughan B, Hosang GM, Eley TC. Heterogeneity in antisocial behaviours and comorbidity with depressed mood: a behavioural genetic approach. Journal Of Child Psychology And Psychiatry 2008;49(5):526–534. [PubMed: 18400059]

Schaie KW. A general model for the study of developmental problems. Psychological Bulletin 1965;64:92–107. [PubMed: 14320080]

Shaffer, D. Diagnostic Assessment in Child and Adolescent Psychopathology. Guilford Press; New York: 1999.

Shanahan L, Copeland W, Costello EJ, Angold A. Specificity of putative psychosocial risk factors for psychiatric disorders in children and adolescents. Journal Of Child Psychology And Psychiatry 2008;49(1):34–42. [PubMed: 18181879]

Simonoff E, Pickles A, Meyer JM, Silberg JL, Maes HH, Loeber R, et al. The Virginia Twin Study of Adolescent Behavioral Development. Influences of age, sex, and impairment on rates of disorder. Archives of General Psychiatry 1997;54:801–808. [PubMed: 9294370]

StataCorp. Stata Statistical Software: Release 10.1. Stata Corporation; College Station, TX: 2007.

Stringaris A, Goodman R. Longitudinal Outcome of Youth Oppositionality: Irritable, Headstrong, and Hurtful Behaviors Have Distinctive Predictions. Journal Of The American Academy Of Child And Adolescent Psychiatry 2009a;48(4):404–412. [PubMed: 19318881]

Stringaris A, Goodman R. Three dimensions of oppositionality in youth. Journal Of Child Psychology And Psychiatry 2009b;50(3):216–223. [PubMed: 19166573]

Tuvblad C, Zheng M, Raine A, Baker LA. A Common Genetic Factor Explains the Covariation Among ADHD ODD and CD Symptoms in 9-10 Year Old Boys and Girls. Journal Of Abnormal Child Psychology 2009;37(2):153–167. [PubMed: 19015975]

Waldman, I.; Rhee, SH.; Levy, F.; Hay, DA. Causes of the overlap among symptoms of ADHD, oppositional defiant disorder, and conduct disorder. In: F., L.; Hay, DA., editors. Attention, genes and ADHD. Brunner-Routledge; New York: 2001.

Zoccolillo M. Gender and the development of conduct disorder. Development and Psychopathology 1993;5:65–78.

Table 1

Ordering of ODD and CD diagnoses across the 9-16 age range.

In all participants						**In participants with CD**		
Pathway	**Statistic**	**Overall**	**Males**	**Females**	**Cohorts combined**	**Age 9 cohort1**	**Age 11 cohort*1***	**Age 13 cohort*1***
Never CD or ODD	Percentage	85.6	80.9	90.4	-	-	-	-
	95% CI	82.8, 88.0	76.5, 84.6	86.8, 93.1	-	-	-	-
	Unweighted N	1083	558	525	-	-	-	-
ODD never CD	Percentage	5.9	5.9	5.8	-	-	-	-
	95% CI	4.4, 7.8	4.0, 8.7	3.8, 8.9	-	-	-	-
	Unweighted N	134	74	60	-	-	-	-
CD never ODD	Percentage	4.7	7.5	1.8	55.0	40.0	51.9	71.6
	95% CI	3.3, 6.6	5.2, 10.9	.7, 4.0	43.2, 66.2	24.7, 57.5	31.2, 72.0	55.8, 83.4
	Unweighted N	99	81	18	99	28	36	35
ODD→CD	Percentage	.8	1.4	.2	9.2	19.8	7.3	2.2
	95% CI	.4, 1.7	.6, 3.2	.0, .5	4.3, 18.6	6.8, 45.7	3.4, 14.9	.5, 8.8
	Unweighted N	21	18	3	21	8	11	2
CD and ODD onset	Percentage	2.1	2.8	1.4	24.9	24.4	27.7	22.2
together	95% CI	1.4, 3.2	1.7, 4.4	.6, 3.3	16.7, 35.5	14.7, 37.7	12.2, 51.4	12.6, 36.2
	Unweighted N	57	40	17	57	20	14	23
CD→ODD	Percentage	.9	1.5	.4	10.9	15.8	13.1	4.0
	95% CI	.5, 1.8	.7, 3.2	.2, .8	5.7, 19.9	8.6, 27.3	3.6, 37.8	1.4, 10.7
	Unweighted N	26	19	7	26	12	9	5

[1]Cohort age at entering study

Table 2

Symptomatology, risk factors and comorbidity of pure CD, pure ODD and children who displayed both CD and ODD (CD+ODD)

					Odds ratio (95% Confidence Intervals)					
					Comparison to no CD/ODD			Comparison between disorders		
No CD/ODD N=5043[3]		**ODD N=688[3]**	**CD N=435[3]**	**CD+ODD N=508[3]**	**ODD**	**CD**	**CD+ODD**	**CD vs ODD**	**CD+ODD vs ODD**	**CD+ODD vs CD**
CD symptoms[1]	.2	.5	1.7	1.6	**2.9 *** (1.9, 4.4)**	**18.0 *** (10.7, 30.4)**	**12.5 *** (8.6, 18.3)**	**6.2 *** (3.3, 11.6)**	**4.3 *** (2.6, 7.2)**	.7 (.4, 1.3)
ODD Symptoms[1]	.4	2.0	1.2	2.3	**9.0 *** (6.0, 13.6)**	**4.6 *** (3.5, 5.9)**	**11.4 *** (6.8, 19.3)**	**.5 ** (.3, .8)**	1.3 (.7., 2.3)	**2.5 ** (1.5, 4.3)**
Aggressive CD symptoms[1]1	.0	.1	.5	.5	**3.1 *** (2.0, 4.7)**	**11.3 *** (7.1, 17.9)**	**14.0 *** (9.5, 20.7)**	**3.6 *** (2.2, 6.1)**	**4.5 *** (2.9, 7.1)**	1.2 (.8, 2.0)
Non-aggressive CD symptoms[1]	.1	.3	1.3	1.1	**2.7 *** (1.8, 4.2)**	**13.7 *** (8.7, 21.8)**	**9.2 *** (6.7, 12.6)**	**5.0 *** (2.8, 8.9)**	**3.4 *** (2.1, 5.4)**	.7 (.4, 1.1)
Risk factors/Parenting Measures										
Sex[2] (% male)	48.2 (2562)	50.3 (382)	80.8 (355)	74.5 (370)	1.1 (.6, 2.1)	**4.6 ** (1.7, 12.2)**	**3.2 ** (1.4, 6.9)**	**4.2 * (1.3, 13.2)**	**2.9 * (1.1, 7.7)**	.7 (.2, 2.4)
Bio P psych problem[2] (%)	24.1 (1158)	39.7 (266)	34.1 (155)	35.4 (184)	**2.1 ** (1.3, 3.4)**	1.8 (1.0, 3.4)	**1.8 * (1.1, 2.9)**	.9 (.4, 1.8)	.9 (.5, 1.6)	1.0 (.5, 2.1)
Bio P arrest[2] (%)	18.8 (1232)	31.0 (225)	29.5 (173)	24.1 (175)	**2.0 ** (1.3, 3.1)**	**2.1 ** (1.3, 3.2)**	1.3 (.7, 2.4)	1.0 (.6, 1.8)	.7 (.3, 1.3)	.6 (.3, 1.3)
Economic problems[1]	.5	.6	.8	.9	**1.7 * (1.1, 2.7)**	**2.5 *** (1.6, 3.8)**	**2.5 *** (1.6, 3.9)**	1.4 (.8, 2.5)	1.5 (.8, 2.6)	1.0 (.6, 1.7)
Family structure[1]	.6	1.0	1.1	1.0	**2.4 ** (1.3, 4.3)**	**3.0 *** (1.7, 5.3)**	**2.5 * (1.2, 5.1)**	1.3 (.6, 2.8)	1.1 (.4, 2.6)	.8 (.3, 2.0)
Inadequate supervision[2] (%)	3.5 (259)	13.1 (110)	9.8 (45)	16.6 (98)	**4.3 *** (2.5, 7.2)**	**2.6 ** (1.3, 5.3)**	**5.6 *** (3.5, 8.8)**	.6 (.3, 1.3)	1.3 (.7, 2.3)	**2.1 * (1.0, 4.4)**
Harsh discipline[2] (%)	.5 (32)	1.0 (11)	1.2 (10)	2.0 (14)	2.0 (.8, 5.3)	**2.8 * (1.1, 7.2)**	**3.9 ** (1.5, 10.2)**	1.4 (.5, 3.9)	1.9 (.7, 5.3)	1.4 (.5, 3.8)
Over-protective[2] (%)	.5 (47)	.5 (6)	.2 (2)	1.0 (7)	.9 (.3, 2.6)	.3 (.1, 1.8)	1.5 (.5, 4.5)	.3 (.1, 2.2)	1.6 (.4, 5.8)	4.6 (.7, 29.4)
Scape-goating[2] (%)	.7 (45)	2.8 (17)	.6 (6)	3.4 (25)	**4.5 ** (1.7, 11.7)**	1.1 (.3, 3.3)	**5.9 *** (2.6, 13.5)**	**.2 * (.1, .9)**	1.3 (.5, 3.7)	**5.5 ** (1.7, 18.3)**
Neglect[2] (%)	.9 (68)	3.9 (33)	4.1 (16)	6.4 (45)	**4.7 *** (2.4, 9.6)**	**4.0 ** (1.5, 10.5)**	**7.4 *** (3.6, 14.9)**	.8 (.3, 2.3)	1.6 (.7, 3.4)	1.9 (.7, 4.9)

					Odds ratio (95% Confidence Intervals)					
					Comparison to no CD/ODD			Comparison between disorders		
No CD/ODD N=5043[3]		**ODD N=688[3]**	**CD N=435[3]**	**CD+ODD N=508[3]**	**ODD**	**CD**	**CD+ODD**	**CD vs ODD**	**CD+ODD vs ODD**	**CD+ODD vs CD**
Comorbidity										
Anxiety[2] (%)	1.7 (114)	6.2 (47)	5.5 (16)	5.8 (38)	**3.9 *** (1.9, 7.9)**	**4.4 ** (2.0, 9.7)**	**4.2 *** (2.1, 8.4)**	1.1 (.5, 2.8)	1.1 (.5, 2.4)	1.0 (.4, 2.2)
Depression[2] (%)	1.0 (66)	10.8 (52)	9.3 (13)	8.1 (48)	**14.1 *** (6.6, 29.8)**	**15.9 *** (5.3, 47.4)**	**13.3 *** (7.3, 24.3)**	1.1 (.4, 3.5)	.9 (.5, 1.9)	.8 (.3, 2.3)
Substance disorder[2] (%)	1.7 (84)	3.9 (33)	9.8 (52)	6.9 (43)	**2.4 * (1.1, 5.4)**	**5.9 *** (2.7, 13.0)**	**5.1 *** (2.4, 10.6)**	2.5 (1.0, 6.1)	2.1 (.9, 5.0)	.9 (.4, 1.9)
ADHD[2] (%)	.5 (39)	3.3 (25)	1.5 (10)	6.1 (39)	**6.9 *** (3.0, 16.0)**	**2.9 * (1.0, 8.4)**	**10.3 *** (4.3, 25.0)**	.4 (.1, 1.2)	1.5 (.6, 3.7)	**3.5 * (1.2, 10.2)**

[1] Continuous scales analyzed with ordinal logistic regression

[2] Binary measures (unweighted N shown in brackets).

[3] Ns vary slightly due to occasional missing values All models control for age and sex. Bold coefficients indicate p<.05.

* p<.05

** p<.01

*** p<.001

Table 3

Adult outcomes of pure CD, pure ODD and children who displayed both CD and ODD (CD+ODD)

					Odds ratio (95% Confidence Intervals)					
					Comparison to no CD/ODD			Comparison between disorders		
Adult Outcome	**No CD/ODD N=1631[3]**	**ODD N=211[3]**	**CD N=133[3]**	**CD+ ODD N=157[3]**	**ODD**	**CD**	**CD+ODD**	**CD vs ODD**	**CD+ODD vs ODD**	**CD+ODD vs CD**
ASPD symptoms[1]	.3	.4	.8	.7	1.6 (.9, 2.9)	**2.9 ** (1.5, 5.6)**	**2.1 * (1.2, 3.7)**	1.8 (.8, 4.0)	1.3 (.6, 2.7)	.7 (.3, 1.6)
Substance disorder[2]	24.0 (348)	29.0 (58)	43.2 (56)	41.9 (60)	1.3 (.7, 2.5)	1.8 (.8, 4.1)	**1.8 * (1.1, 3.2)**	1.4 (.5, 3.9)	1.4 (.6, 3.2)	1.0 (.4, 2.6)
Anxiety[2]	4.3 (57)	11.9 (18)	.8 (3)	18.4 (15)	**3.0 * (1.2, 7.6)**	**.2 * (.1, .9)**	**5.8 ** (1.8, 19.5)**	**.1 ** (.0, .4)**	1.9 (.5, 8.0)	**27.0 *** (4.8, 151.7)**
Depression[2]	2.0 (46)	6.3 (16)	5.6 (5)	11.8 (9)	**3.3 * (1.3, 8.6)**	4.1 (.9, 19.3)	**8.9 *** (3.0, 25.9)**	1.2 (.2, 6.5)	2.7 (.8, 9.2)	2.2 (.4, 11.4)

All models control for age and sex

[1] Continuous scale analyzed with ordinal logistic regression

[2] Binary measures (unweighted N shown in brackets)

[3] Ns vary slightly due to occasional missing values. Bold coefficients indicate p<.05

* p<.05

** p<.01

*** p<.001

Table 4

Promax rotated factor loadings from an exploratory factor analysis of the ODD symptoms at wave 1.

Symptom	Irritable[1]	Headstrong[1]
Temper tantrums	**.67**	.04
Argues with adults	.15	**.50**
Defies adult's requests	.00	**.70**
Deliberately annoys people	−.14	**.77**
Blames others	.05	**.51**
Touchy or easily annoyed	**.79**	−.11
Angry or resentful	**.57**	.30
Spiteful or vindictive	.16	**.52**

[1] Loadings >.40 are shown in bold

Table 5

Prediction from a 1 SD increase in irritable and headstrong ODD symptom dimensions at wave 1 to later psychiatric outcomes up to age 16.

	Odds Ratio (95% Confidence Interval)[1]		
Outcome up to age 16	**Irritable**	**Headstrong**	**Irritable vs Headstrong**
CD	1.2 (1.0, 1.6)	**1.3** * **(1.1, 1.6)**	1.1 (.7, 1.5)
ODD	**1.6** *** **(1.2, 2.0)**	**1.5** *** **(1.2, 1.9)**	1.0 (.6, 1.5)
Substance disorder	.9 (.7, 1.2)	**1.4** ** **(1.2, 1.7)**	**1.6** * **(1.1, 2.3)**
Anxiety	**1.6** ** **(1.2, 2.1)**	1.0 (.8, 1.3)	**.6** * **(.4, 1.0)**
Depression	1.2 (.9, 1.5)	**1.5** ** **(1.2, 1.9)**	1.3 (.9, 1.9)

All models control for age and sex Bold coefficients indicate p<.05

* p<.05

** p<.01

*** p<.001

[1] Initial level of outcome, age and sex are included as covariates