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Lifetime prevalence, correlates, and persistence of oppositional defiant disorder: results from the National Comorbidity Survey Replication

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Background: Oppositional defiant disorder (ODD) is a leading cause of referral for youth mental health services; yet, many uncertainties exist about ODD given it is rarely examined as a distinct psychiatric disorder. We examined the lifetime prevalence, onset, persistence, and correlates of ODD. **Methods:** Lifetime prevalence of ODD and 18 other DSM-IV disorders was assessed in a nationally representative sample of adult respondents ($n = 3{,}199$) in the National Comorbidity Survey Replication. Retrospective age-of-onset reports were used to test temporal priorities with comorbid disorders. **Results:** Lifetime prevalence of ODD is estimated to be 10.2% (males = 11.2%; females = 9.2%). Of those with lifetime ODD, 92.4% meet criteria for at least one other lifetime DSM-IV disorder, including: mood (45.8%), anxiety (62.3%), impulse-control (68.2%), and substance use (47.2%) disorders. ODD is temporally primary in the vast majority of cases for most comorbid disorders. Both active and remitted ODD significantly predict subsequent onset of secondary disorders even after controlling for comorbid conduct disorder (CD). Early onset (before age 8) and comorbidity predict slow speed of recovery of ODD. **Conclusions:** ODD is a common child- and adolescent-onset disorder associated with substantial risk of secondary mood, anxiety, impulse-control, and substance use disorders. These results support the study of ODD as a distinct disorder. Prospective and experimental studies are needed to further delineate the temporal and causal relations between ODD and related disorders. **Keywords:** Oppositional defiant disorder, conduct disorder, epidemiology, National Comorbidity Survey. **Abbreviations:** ODD: oppositional defiant disorder; CD: conduct disorder; NCS-R: National Comorbidity Survey Replication.

Oppositional defiant disorder (ODD) was first introduced as a distinct child/adolescent onset disorder in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III; APA, 1980) and currently is defined by both the DSM-IV (APA, 1994) and the ICD-10 (WHO, 1993) as a recurrent pattern of defiant, disobedient, and hostile behavior beginning in childhood or adolescence. Along with conduct disorder (CD) and attention-deficit/hyperactivity disorder (ADHD), ODD is one of the leading reasons for referral to youth mental health services (Loeber, Burke, Lahey, Winters, & Zera, 2000). However, ODD is usually considered the mildest of these three disorders both because it includes symptoms that are closest to 'normal' behavior (e.g., losing one's temper, arguing with adults) and because it is often conceptualized as merely a prodrome to CD (Lahey & Loeber, 1994; Loeber, Burke et al., 2000; Rowe, Maughan, Pickles, Costello, & Angold, 2002). Although diagnostic requirements differ slightly between the DSM-IV and ICD-10 (Rowe, Maughan, Costello, & Angold, 2005), both normalize this perception by creating a diagnostic hierarchy between ODD and CD such that ODD cannot be diagnosed in the presence of CD. As a result of this hierarchy rule, ODD often is combined with CD in empirical studies to form an 'ODD/CD' group (Angold, Costello, & Erkanli, 1999).

Recent research suggests that ODD should be the focus of more attention in its own right. This research shows that ODD can clearly be distinguished from normative child behavior (Keenan & Wakschlag, 2004; Rutter, Giller, & Hagell, 1998). Prior work also shows that although ODD and CD often are comorbid and share many risk factors (Lahey, Loeber, Burke, Rathouz, & McBurnett, 2002; Rowe et al., 2002), the majority of children with ODD do not develop CD (Lahey & Loeber, 1994; Loeber, Burke et al., 2000; Rowe et al., 2002) and ODD has somewhat different socio-environmental and genetic correlates than CD (Dick, Viken, Kaprio, Pulkkinen, & Rose, 2005; Hudziak, Derks, Althoff, Copeland, & Boomsma, 2005). Moreover, ODD is associated with increased risk of other mental disorders during childhood (Burke, Loeber, Lahey, & Rathouz, 2005; Greene et al., 2002; Lavigne et al., 2001; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004) and adulthood (Langbehn, Cadoret, Yates, Troughton, & Stewart, 1998) beyond the effects of CD. Taken together, these findings provide support for considering ODD independent of CD. Consistent with this view, in their recent review of the criteria used to define ODD and the relation between ODD and CD, Rowe and colleagues (2005) concluded that 'there is

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evidence that CD and ODD are sufficiently different from one another to be regarded as fully separate disorders' (p. 1314).

Building on this prior work, if ODD is to be studied as an independent disorder, several lingering uncertainties about the descriptive epidemiology of ODD must be resolved. Even such a basic issue as the prevalence of ODD is uncertain, as prevalence estimates in previous studies have had a wide range (2–15%) (Lahey, Miller, Gordon, & Riley, 1999; Loeber, Burke et al., 2000; Maughan et al., 2004). This variation is likely the result of relying on small, non-representative samples and using inconsistent diagnostic criteria (Lahey, Loeber, Quay, Frick, & Grimm, 1992; Loeber, Burke et al., 2000; Rowe et al., 2005). A more accurate estimate of the lifetime prevalence of ODD is needed to understand the risk of ODD in the general population and to inform scientific, clinical, and policy efforts.

In addition, little is known about the associations of ODD with other mental disorders independent of CD and ADHD other than evidence of high comorbidity obtained in a few studies of clinically referred youth (Burke et al., 2005; Greene et al., 2002). Temporal priorities between ODD and comorbid disorders also are largely unknown, making it impossible to know whether ODD should be conceptualized as a developmental precursor to other disorders or whether it more often occurs only in the context of pre-existing comorbid disorders. Furthermore, although considerable research exists on the adult outcomes of CD (Fergusson, Horwood, & Ridder, 2005; Kim-Cohen et al., 2003; Laub & Vaillant, 2000) and ADHD (Barkley, Fischer, Smallish, & Fletcher, 2004; Kessler, Adler et al., 2005; Rutter, Kim-Cohen, & Maughan, 2006), information regarding ODD is very limited.

The uncertainties regarding the prevalence, comorbidity, and course of ODD should ideally be addressed in a large prospective study that follows a nationally representative sample of youth into adulthood and incorporates third-party informants such as parents and teachers. Several existing prospective studies include children diagnosed with ODD (Costello et al., 1996; Loeber, Green, Lahey, Frick, & McBurnett, 2000; Moffitt, Caspi, Harrington, & Milne, 2002; Simonoff et al., 1997); although not nationally representative, these will doubtlessly continue to yield useful information about the course of ODD as the participants in these studies enter and progress through adulthood. In addition, a nationally representative survey of over 10,000 adolescents that includes an evaluation of ODD as well as many other DSM-IV disorders and incorporates parent-report is currently being carried out in conjunction with the National Comorbidity Survey Replication (NCS-R) (Kessler & Merikangas, 2004). Planned follow-ups of this sample will likely yield useful prospective information about the course of ODD. Prior to this, though, retrospective studies using adult respondents can provide preliminary answers to questions about the lifetime prevalence, correlates, and extended course of ODD. Although relying on single-informant, retrospective data may introduce problems related to recall biases (Schlesselman, 1982) and to the exclusion of parent-report of respondent behavior, such data can supply valuable, preliminary information about the prevalence, timing, and course of ODD that can inform subsequent prospective studies.

The goal of the current report is to present retrospective data of this sort on the descriptive epidemiology of ODD based on the NCS-R. We begin by estimating the lifetime prevalence, age-of-onset, and duration of DSM-IV ODD in the general US population. We then examine associations of ODD with a wide range of other DSM-IV disorders and evaluate the temporal order of onset of ODD with these comorbid disorders. We also evaluate the risk of subsequent mental disorders among respondents with ODD in the absence of CD. Finally, we examine the pattern and predictors of ODD duration and recovery.

Methods

Sample

Participants were respondents in the National Comorbidity Survey Replication (NCS-R), a face-to-face household survey of 9,282 English-speaking adults ages 18+ in the coterminous US based on a nationally representative multi-stage clustered area probability sampling design (Kessler et al., 2004). Respondents (70.9% response rate) received information about the study in an advance letter and a Study Fact Brochure and by a follow-up household informational visit prior to providing informed consent and conducting the interview. All procedures were approved by the Human Subjects Committees of Harvard Medical School and the University of Michigan.

The NCS-R was administered in two parts. Part I included demographic and diagnostic assessments administered to all 9,282 respondents. Part II included additional questions administered to respondents meeting criteria for at least one mental disorder during the Part I interview as well as a probability sub-sample of other respondents. Given concerns about recall failure among older adults in the assessment of disorders of childhood and adolescence, ODD was assessed only among the 3,199 Part II respondents 18–44 years old. This sample was weighted to adjust for the over-sampling of Part I respondents with other DSM-IV disorders as well as to correct for differential probability of selection and non-response. Additional details on the sampling and weighting procedures used in the NCS-R are presented elsewhere (Kessler et al., 2004).

Assessment

Mental disorders were assessed using the World Health Organization (WHO) Composite International Diagnos-

tic Interview (CIDI; Kessler & Ustun, 2004). The CIDI is a fully structured diagnostic interview that generates diagnoses according to the definitions and criteria of both the DSM-IV and the ICD-10 diagnostic systems. DSM-IV criteria are used in the current report. Because we were interested in examining DSM-IV ODD regardless of the presence of CD, we considered lifetime ODD to be present regardless of whether individuals also met DSM-IV criteria for lifetime CD. All respondents first provided self-report regarding the presence of each ODD symptom during their childhood or adolescence, and subsequently reported on the age-of-onset and offset of these symptoms, as well as whether or not these symptoms were present in the prior 12 months (i.e., active ODD). All other mental disorders examined were assessed in a similar manner. Overall, good concordance has been found in an NCS-R clinical reappraisal sub-sample between diagnoses of anxiety, mood, and substance use ($\kappa = .48$ to .53) disorders based on the CIDI and diagnoses based on blinded clinical reappraisal interviews using the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 2002) (see Kessler, Berglund et al., 2005). Diagnoses of impulse-control disorders, including ODD, were not validated because these diagnoses are not included in the SCID.

Data analytic plan

The estimated prevalence of ODD was calculated using simple cross-tabulations with gender and age. Associations of ODD with other DSM-IV/CIDI disorders were examined using logistic regression analysis, controlling for sex, age, and race/ethnicity. Temporal priority of ODD in relation to comorbid disorders was examined using information obtained in retrospective age-of-onset reports. The effect of ODD in predicting the first onset of subsequent disorders was examined using discrete-time survival analysis with retrospectively reported information about the timing of onset and offset of ODD coded as time-varying variables. Information about offset of ODD was included in these analyses to investigate whether recovery from ODD is associated with a reversal of the elevated risk of secondary disorders. Logistic regression and survival coefficients and their standard errors were transformed to odds-ratios (ORs) and 95% confidence intervals (95% CIs) for ease of interpretation. All analyses were carried out using the Taylor series linearization method (Wolter, 1985), a design-based method implemented in the SUDAAN software system (SUDAAN, 2002), to adjust for the weighting and clustering of the NCS-R data. Statistical significance was evaluated using two-sided design-based .05 level tests.

Results

Prevalence, age-of-onset, and duration

The estimated lifetime prevalence of DSM-IV/CIDI ODD is 10.2% (Table 1). Estimated prevalence is not significantly different for males (11.2%) relative to females (9.2%; $z = 1.41$, *ns*). ODD is reported to have a somewhat higher lifetime prevalence among respondents in the 18–24 age range (13.4%) than older respondents (7.5–10.1%), but this could reflect differential recall or greater willingness to admit ODD symptoms among younger respondents. The median age-of-onset of ODD is 12.0 (inter-quartile range: 7.0–13.0). Self-reported onset begins at approximately age four and increases steadily into late adolescence (Figure 1). The shape of the onset curve is similar for respondents of different ages at interview, although the higher reported prevalence among the youngest respondents can be seen clearly beginning at approximately age seven. Median duration of ODD is 6 years and does not vary greatly either by sex or by age at interview. It is consistently higher among men than women in each age group, although none of these differences are statistically significant (Table 1).

Table 1 Lifetime prevalence and median duration of ODD by age and sex

	Male			Female			Total		
II. Lifetime prevalence	%	(se)	(*n*)	%	(se)	(*n*)	%	(se)	(*n*)
Age									
18–24	14.9	(2.1)	(356)	12.0	(1.6)	(442)	13.4	(1.5)	(798)
25–29	11.2	(2.9)	(232)	7.2	(1.6)	(341)	9.1	(1.3)	(573)
30–34	8.0	(2.0)	(236)	7.1	(1.2)	(332)	7.5	(1.1)	(568)
35–39	9.3	(1.7)	(322)	7.9	(1.3)	(438)	8.6	(1.1)	(760)
40–44	9.8	(1.7)	(226)	10.4	(4.2)	(272)	10.1	(2.3)	(498)
Total	11.2	(1.1)	(1372)	9.2	(.9)	(1825)	10.2	(.8)	(3197)
II. Median duration	Mdn	(IQR)	(*n*)	Mdn	(IQR)	(*n*)	Mdn	(IQR)	(*n*)
Age									
18–24	5.0	(4.0–8.0)	(70)	5.0	(3.0–9.0)	(69)	5.0	(4.0–9.0)	(139)
25–29	5.0	(2.0–8.0)	(33)	4.0	(3.0–6.0)	(30)	4.0	(2.0–7.0)	(63)
30–34	8.0	(3.0–17.0)	(31)	7.0	(5.0–14.0)	(38)	7.0	(5.0–14.0)	(69)
35–39	8.0	(4.0–16.0)	(43)	7.0	(4.0–10.0)	(50)	7.0	(4.0–13.0)	(93)
40–44	11.0	(5.0–24.0)	(30)	4.0	(3.0–11.0)	(27)	7.0	(3.0–21.0)	(57)
Total	6.0	(4.0–12.0)	(207)	5.0	(3.0–9.0)	(214)	6.0	(3.0–11.0)	(421)

Figure 1 Age-of-onset for ODD. Age-of-onset is plotted separately for 5-year cohorts based on age at the time of the NCS-R interview. Chi square test for age cohort = 6.6 ($p < .001$)

Comorbidity with other DSM-IV disorders

ODD is significantly comorbid with every one of the other lifetime DSM-IV/CIDI disorders assessed in the NCS-R, with ORs in the range of 2.1 (agoraphobia without panic disorder) to 12.6 (CD). The ORs are highest for impulse-control disorders (4.0–12.6) and lowest for anxiety disorders (2.1–4.5) (Table 2). The mood, anxiety, and substance use disorders generally considered to be the most severe within their class (i.e., bipolar disorder, obsessive-compulsive disorder, illicit drug dependence) have the highest ORs. There is also a strongly increasing relative-odds of ODD with a number of other disorders (5.3–24.7). A full 92.4% of the respondents with lifetime ODD meet criteria for at least one other lifetime disorder. Comparison of age-of-onset reports suggests that ODD is temporally primary to these comorbid conditions in the majority of cases for all disorders other than phobia, separation anxiety disorder, ADHD, and CD.

Table 2 Lifetime comorbidity and temporal priority of ODD with other DSM-IV disorders

					Temporal priority						
					ODD first		ODD second		Same year		
	%[1]	(se)	OR[2]	(95% CI)	%	(se)	%	(se)	%	(se)	(*n*)
I. Mood disorders											
Bipolar disorder	20.2	(2.0)	6.5*	(4.7–8.9)	82.5	(4.0)	11.5	(3.3)	6.0	(1.6)	(90)
Major depression	39.1	(2.6)	2.9*	(2.2–3.7)	77.6	(2.8)	15.2	(3.0)	7.1	(1.9)	(185)
Dysthymia	10.4	(1.6)	3.7*	(2.5–5.4)	75.3	(7.8)	17.2	(6.2)	7.5	(4.6)	(47)
Any mood disorder	45.8	(2.8)	3.4*	(2.7–4.3)	75.9	(3.1)	16.6	(3.2)	7.5	(1.5)	(212)
II. Anxiety disorders											
Agoraphobia without panic	2.4	(.8)	2.1*	(1.1–4.1)	73.9	(9.8)	16.8	(8.1)	9.3	(6.9)	(13)
Panic disorder	10.9	(1.7)	2.7*	(1.8–4.0)	81.2	(5.6)	12.4	(4.8)	6.4	(3.3)	(57)
PTSD	19.7	(2.2)	4.5*	(3.3–6.2)	66.9	(6.6)	23.9	(5.2)	9.2	(3.1)	(94)
GAD	15.5	(2.0)	2.7*	(1.8–4.2)	77.7	(4.8)	15.5	(4.4)	6.8	(3.1)	(81)
Specific phobia	24.7	(3.1)	2.4*	(1.6–3.5)	20.6	(5.0)	71.7	(4.9)	7.7	(2.1)	(124)
Social phobia	31.4	(2.7)	3.3*	(2.4–4.5)	44.6	(5.9)	43.0	(4.6)	12.3	(3.4)	(138)
OCD	2.8	(.7)	5.2*	(2.5–10.6)	95.1	(4.8)	.0	(.0)	4.9	(4.8)	(15)
SAD	12.5	(2.0)	3.3*	(2.3–4.7)	16.4	(4.5)	77.7	(5.2)	6.0	(3.0)	(58)
Any anxiety disorder	62.3	(2.3)	4.3*	(3.4–5.5)	36.1	(4.5)	55.2	(4.8)	8.7	(1.5)	(280)
III. Impulse-control disorders											
IED	29.0	(2.7)	4.0*	(3.0–5.2)	55.1	(5.0)	32.6	(4.9)	12.3	(3.9)	(130)
ADHD	35.0	(2.3)	10.4*	(7.6–14.2)	19.8	(4.4)	61.5	(5.0)	18.7	(2.5)	(146)
Conduct disorder	42.3	(2.3)	12.6*	(9.5–16.7)	46.3	(5.8)	24.4	(4.2)	29.3	(3.9)	(183)
Any impulse-control disorder	68.2	(2.4)	11.0*	(8.7–14.1)	25.3	(2.9)	51.1	(2.9)	23.6	(2.3)	(298)
IV. Substance use disorders											
Alcohol abuse	40.5	(2.8)	4.8*	(3.7–6.3)	93.2	(1.9)	1.7	(1.1)	5.1	(1.4)	(178)
Alcohol dependence	21.5	(2.2)	5.4*	(3.8–7.8)	93.0	(2.2)	.8	(.8)	6.1	(2.1)	(99)
Drug abuse	35.8	(3.1)	5.8*	(4.2–8.2)	92.3	(2.1)	1.6	(1.0)	6.1	(1.9)	(154)
Drug dependence	18.6	(2.3)	7.2*	(4.5–11.6)	90.9	(3.3)	2.3	(2.2)	6.8	(2.8)	(82)
Any substance	47.2	(3.0)	5.6*	(4.2–7.4)	89.3	(2.6)	3.3	(1.7)	7.4	(1.7)	(202)
V. Any disorder											
Any disorder	92.4	(2.3)	13.3*	(6.9–25.6)	24.4	(2.9)	60.8	(3.9)	14.8	(1.8)	(399)
Exactly 1 disorder	15.1	(2.3)	5.3*	(2.4–11.7)	–	–	–	–	–	–	
Exactly 2 disorders	13.5	(1.6)	8.9*	(4.4–18.3)	–	–	–	–	–	–	
3 or more disorders	63.8	(2.5)	24.7*	(13.1–46.6)	–	–	–	–	–	–	

*Significant at the .05 level, two-sided test.
[1]The prevalence of the comorbid disorder among respondents who have LT ODD ($n = 421$).
[2]Controlled for age at interview (5-year intervals) sex, and race-ethnicity in every model. Sample included respondents whose age at interview was less than or equal to 44 ($n = 3197$). The last model had predictors 'exactly one,' 'exactly two' and 'three or more' disorders in one model. All the other models have one DSM-IV disorder at a time as predictor of LT ODD.
PTSD = Post traumatic stress disorder, GAD = Generalized anxiety disorder, OCD = Obsessive compulsive disorder, SAD = Separation anxiety disorder, IED = Intermittent explosive disorder, ADHD = Attention deficit/hyperactivity disorder.

Prediction of temporally secondary disorders

The strength of association between temporally primary ODD and the subsequent onset of secondary disorders was evaluated with discrete-time survival analysis. Distinctions were made between active and remitted ODD and between ODD in the absence versus presence of CD. Both active (2.4–15.7) and remitted (1.2–5.0) ODD were found to be associated with significantly increased odds of virtually all other disorders in the total sample (Table 3), although the ORs are significantly higher for active compared to remitted ODD in 13 of 21 comparisons. Surprisingly, the ORs are generally higher in the absence of CD than in the presence of CD. As with the cross-sectional ORs reported in Table 2, the ORs of ODD with subsequent secondary disorders are generally highest in predicting impulse-control disorders and weakest in predicting anxiety disorders. ODD generally has stronger predictive relationships with the most severe mood (bipolar disorder), anxiety (obsessive-compulsive disorder), and substance use (illicit drug dependence) disorders than with other disorders in the same class.

Patterns and predictors of recovery

Offset of ODD occurs most often prior to age 18 years, with more than 70% of respondents who report a lifetime history of ODD no longer having symptoms by age 18. Age-of-offset curves are steepest for individuals with later onset and survival analysis confirms that early onset (i.e., age-of-onset $\leqslant$7 years) is strongly and inversely related to speed of recovery (OR: .5; 95% CI: .3–.8). In other words, those with earlier onset experience a longer duration of ODD. A number of comorbid mood, anxiety, impulse-control, and substance use disorders also significantly predicted slower speed of recovery, with ORs in the range of .4–.7. Interestingly, comorbid CD is not significantly related to speed of recovery of ODD (OR: .9; 95% CI: .7–1.1). In contrast, the presence of agoraphobia is a strong and significant predictor of recovery from ODD (OR = 15.9, $p < .05$). (Detailed results are available in Appendix Table 1.)

Discussion

The results of this study should be interpreted in the context of several important limitations. The most significant limitation is the use of retrospective self-report data, as respondents may have forgotten events, made errors in the timing of events, or been biased by their mood state at the time of the interview (Schacter, 1999). Inaccuracies are especially likely in reported ages-of-onset (Angold & Costello, 1996; Kazemian & Farrington, 2005), although it is noteworthy that the age-of-onset distribution found here is quite consistent with the distributions found in prior prospective studies (Lahey et al., 1999; Maughan et al., 2004), indirectly suggesting that recall bias might not have been of great importance in this regard. Notably, systematic reviews on the use of retrospective surveys have revealed that despite the limitations mentioned above, participants in retrospective studies are able to recall experiences from childhood and adolescence, particularly those that are well operationalized, with sufficient accuracy to provide accurate and useful information (Brewin, Andrews, & Gotlib, 1993; Hardt & Rutter, 2004). Thus, although the current study is limited by the use of retrospective data, these data provide useful information about ODD that can inform prospective studies and other scientific, clinical, and policy efforts.

Another important limitation is that the diagnosis of ODD in the current study relied on a single informant in each case. This is a departure from most prior studies as well as from clinical work with this population, in which the diagnosis of ODD is made during childhood or adolescence incorporating clinical information provided by parents and teachers. Parents and teachers can provide particularly valuable information about oppositional and defiant behavior given they often are the ones attempting to manage the child. In addition, when assessed, parents of children with ODD often report that symptoms have always been present (Angold & Costello, 1996) and the absence of such a perspective may result in a later estimated age-of-onset. In this study we did not obtain clinical information from parents or teachers, nor was the presence of ODD symptoms validated using follow-up clinical reappraisal interviews in the same way most other NCS-R diagnoses were. As a result, we are unable to evaluate the validity of ODD diagnoses or associated age-of-onsets. These differences should be borne in mind when interpreting the obtained results. Finally, although the NCS-R assessed a wide range of comorbid disorders, it would have been instructive to expand the analysis to consider a wider range of outcomes associated with ODD, such as later role impairment, criminality, and mortality.

These limitations notwithstanding, this study provides important, preliminary information about the lifetime prevalence, correlates, and persistence of ODD. Prior studies have reported the prevalence of ODD at a given point in time (Lahey et al., 1999; Loeber, Burke et al., 2000; Maughan et al., 2004), and this study is the first to provide an estimate of the lifetime prevalence of this disorder. The lifetime prevalence estimate of 10.2% found here approximates the cumulative prevalence estimate for ODD for those up to 16 years (11.3%) reported in a prior study (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). This is not surprising given our finding that ODD occurs primarily during childhood and adolescence and rarely beyond age 18 years, suggesting most cases will have been identified in prior studies

Table 3. Temporally primary active and remitted DSM-IV ODD predicting the subsequent first onset of other DSM-IV disorders in the total sample and separately in the sub-samples of respondents with and without conduct disorder[1]

	Total sample				Without conduct[2]				With conduct[3]			
	Active ODD		Remitted ODD		Active ODD		Remitted ODD		Active ODD		Remitted ODD	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
I. Mood disorders												
Bipolar disorder	8.5*	(6.3–11.6)	4.1*#	(2.2–7.5)	6.9*	(4.1–11.6)	3.2*	(1.5–7.2)	4.0*	(2.1–7.7)	2.3*	(1.3–4.4)
Major depression	3.5*	(2.8–4.4)	2.0*#	(1.5–2.7)	3.9*	(2.8–5.5)	1.9*#	(1.2–3.1)	1.6*	(1.0–2.6)	1.5	(.9–2.5)
Dysthymia	5.6*	(3.2–9.8)	2.8*	(1.5–5.0)	6.3*	(3.0–13.0)	2.3*#	(1.2–4.5)	2.0	(.7–6.0)	1.8	(.6–5.7)
Any mood disorder	4.2*	(3.2–5.5)	2.1*#	(1.6–2.8)	4.3*	(2.9–6.3)	2.1*	(1.3–3.3)	2.0*	(1.2–3.2)	1.4	(.8–2.5)
II. Anxiety disorders												
Agoraphobia without panic	3.8*	(1.6–9.0)	1.7	(.5–5.9)	6.6*	(2.6–16.7)	3.2	(.9–11.6)	–[4]	–	–[4]	–
Panic disorder	3.3*	(2.1–5.2)	2.4*	(1.4–4.2)	2.8*	(1.3–6.1)	1.9	(.8–4.4)	1.3	(.7–2.5)	1.3	(.7–2.6)
PTSD	6.5*	(4.0–10.6)	2.8*#	(1.8–4.5)	7.5*	(3.7–15.3)	2.4*#	(1.3–4.6)	2.0*	(1.0–3.9)	1.4	(.6–3.2)
GAD	3.8*	(2.4–5.8)	2.0*	(1.3–3.2)	3.8*	(2.3–6.2)	1.5#	(.9–2.4)	1.8	(.9–3.4)	1.7	(.8–3.4)
Specific phobia	2.4*	(1.5–3.9)	3.0*	(1.4–6.7)	2.7*	(1.3–5.6)	2.3	(.7–7.9)	1.2	(.7–2.4)	1.9	(.8–4.8)
Social phobia	3.8*	(2.9–5.1)	1.9*#	(1.1–3.6)	4.1*	(2.7–6.2)	2.6*	(1.3–5.2)	1.6	(1.0–2.7)	.5#	(.1–1.5)
OCD	7.5*	(3.0–18.6)	5.0*	(1.4–18.3)	6.3*	(2.0–19.4)	7.8*	(1.7–36.2)	5.5	(.6–53.1)	.9	(.1–13.0)
SAD	2.5*	(1.5–4.3)	1.4	(.2–10.3)	3.6*	(1.9–7.0)	–[4]	–	.6	(.2–1.6)	1.1	(.2–7.2)
Any anxiety disorder	4.3*	(3.0–6.1)	2.7*	(1.9–3.6)	4.6*	(2.5–8.5)	2.7*	(1.8–4.3)	1.9*	(1.2–2.9)	1.1	(.6–2.2)
III. Impulse-control disorders												
IED	4.4*	(3.1–6.2)	2.1*	(1.1–4.2)	3.7*	(2.4–5.8)	2.6*	(1.0–6.4)	1.9*	(1.1–3.3)	.7	(.3–1.7)
ADHD	15.7*	(10.3–24.2)	1.2#	(.1–10.4)	12.8*	(6.3–25.9)	–[4]	–	8.2*	(5.3–12.9)	1.0	(.1–9.9)
Conduct disorder	15.0*	(11.8–19.2)	1.9#	(.7–5.0)	–	–	–	–	–	–	–	–
Any impulse-control disorder	11.5*	(8.8–14.9)	2.3*#	(1.0–5.1)	6.4*	(4.0–10.2)	2.4	(.9–6.6)	6.5*	(3.9–10.9)	1.3#	(.7–2.3)
IV. Substance use disorders												
Alcohol abuse	6.1*	(4.7–7.9)	3.1*#	(2.4–4.0)	5.4*	(3.9–7.6)	2.7*#	(1.9–3.9)	2.1*	(1.3–3.5)	1.4	(.8–2.4)
Alcohol dependence	8.3*	(5.6–12.3)	4.0*#	(2.7–5.8)	8.4*	(5.0–14.2)	3.7*#	(2.3–6.1)	2.2*	(1.2–4.0)	1.4	(.7–2.7)
Drug abuse	7.0*	(5.0–9.7)	3.8*#	(2.7–5.4)	6.1*	(3.9–9.6)	2.5*#	(1.6–3.8)	2.4*	(1.5–3.9)	2.2*	(1.3–3.6)
Drug dependence	11.5*	(7.5–17.7)	3.7*#	(2.1–6.4)	10.6*	(5.3–20.9)	3.1*#	(1.6–5.7)	3.0*	(1.7–5.3)	1.3#	(.6–2.9)
Any substance	6.0*	(4.6–7.9)	3.6*#	(2.7–4.7)	5.6*	(4.0–7.7)	2.9*#	(2.0–4.3)	2.0*	(1.3–3.2)	1.7	(.9–3.1)
V. Any disorder												
Any disorder	8.0*	(5.2–12.2)	3.3*#	(2.1–5.3)	6.1*	(3.3–11.4)	3.4*	(2.0–5.6)	6.4*	(3.6–11.5)	1.1#	(.5–2.2)

*Significant at the .05 level, two-sided test.

#OR for remitted ODD is significantly different (at .05 level) from the OR for active ODD.

[1]Controlled for age at interview (5-year = intervals) sex, race-ethnicity and person-years. Sample included respondents whose age at interview was $\leqslant 44$ ($n = 3197$). A separate equation was estimated for each row of the table. Each equation included controls plus dummy variables for active and remitted ODD as time-varying predictors of the subsequent first onset of the outcome disorder in a discrete-time survival equation with person-year the unit of analysis.

[2]The results in this column are based on the subsample of 2812 respondents who did not have a lifetime history of conduct disorder.

[3]The results in this column are based on the subsample of 385 respondents with a lifetime history of conduct disorder.

[4]Insufficient precision to estimate parameter.

PTSD = Post traumatic stress disorder, GAD = Generalized anxiety disorder, OCD = Obsessive compulsive disorder, SAD = Separation anxiety disorder, IED = Intermittent explosive disorder, ADHD = Attention deficit/hyperactivity disorder.

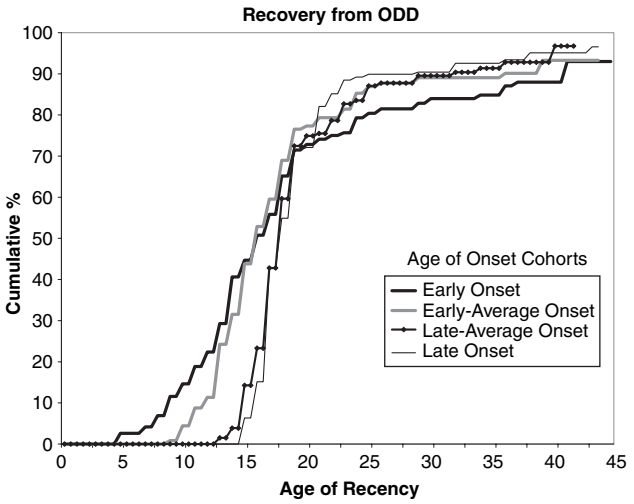

Figure 2 Recovery from ODD. Cases were divided into four categories of roughly equal size based on their age-of-onset. Early-onset cases were those with ages of onset $\leqslant 7$, while early-average, late-average and late-onset were defined as onsets in the age ranges 8–11, 12–13 and 14+. Chi square test for age-of-onset cohort = 6.7 ($p < .001$)

of only children and adolescents. Prior studies of CD and ADHD have demonstrated that these disruptive behavior disorders are more prevalent among males than females (Costello et al., 1996; Nock, Kazdin, Hiripi, & Kessler, 2006; Simonoff et al., 1997). Our findings show a similar pattern for ODD, but one that is much less pronounced, with the prevalence of this disorder consistently higher for males than females, but at a rate that is not statistically significant – a finding in line with prior epidemiologic studies of ODD (see Maughan et al., 2004; Rowe et al., 2002).

The result that the reported lifetime prevalence of ODD is highest among the youngest respondents is consistent with reports suggesting that child–adolescent oppositional and aggressive behaviors may be increasing in recent cohorts (Loeber & Farrington, 1998; Rutter & Smith, 1995). This result should be interpreted with caution; however, as these results were not consistently statistically significant (perhaps due to insufficient power), we cannot rule out the possibility that the pattern is due to reporting artifacts. Examination of trends in ODD in prospective studies remains an important area for future research.

Our finding that only a minority of respondents with ODD develop CD (42.3%) or ADHD (25.0%) is consistent with results from recent reports using clinical samples (Biederman et al., 1996; Lahey & Loeber, 1994). Several studies have reported that ODD also is associated with elevated rates of mood and anxiety disorders (Burke et al., 2005; Greene et al., 2002); however, it has been suggested that only about one quarter of those with ODD actually meet criteria for one of these disorders during childhood or adolescence (Angold & Costello, 1996). Our study revealed that during their lifetime 92.4% of those with ODD meet criteria for another mental disorder. More than half have a comorbid anxiety disorder and almost half have a mood and/or substance use disorder. The higher rate of comorbidity observed in this study relative to previous studies in this area may be due to our focus on lifetime comorbidity rather than the shorter time frames used in prior studies as well as to our evaluation of a much broader range of comorbid conditions.

Our study also offers new data on the temporal relations between ODD and a wide range of comorbid disorders that occur into adulthood, whereas previous studies have examined the relations between ODD and other mental disorders over relatively short time periods (typically 4 to 6 years) (August, Realmuto, Joyce, & Hektner, 1999; Biederman et al., 1996; Burke et al., 2005; Lavigne et al., 2001). Our examination of the timing of ODD in relation to these comorbid disorders revealed that ODD is typically secondary to phobias, SAD, and ADHD. This is consistent with research showing an earlier age-of-onset for these disorders (Dadds & Barrett, 2001; Lahey & Loeber, 1994; Loeber, Green et al., 2000). In contrast, ODD typically is temporally primary to all other comorbid disorders. Survival analyses showed that this temporal priority is related to consistently significant associations between ODD and the subsequent onset of a wide range of secondary disorders. These results are striking and suggest that the presence of ODD substantially increases the risk of developing a full range of other psychiatric disorders. It is noteworthy that the elevated risk of secondary disorders exists both in the presence and absence of CD and effects are equally strong for all comorbid disorders. For instance, the presence of ODD is associated with a four- to six-fold increase in the odds of a secondary mood, anxiety, impulse-control, and substance use disorder among those who never even develop CD.

The fact that both active and remitted ODD are significant predictors of later disorders suggests that ODD is as much a risk marker as a risk factor. That is, a history of ODD is indicative of vulnerability to a wide range of later mental disorders that is not necessarily related to the continued presence of ODD. This important finding suggests that the secondary disorders are not necessarily the direct result of ODD but may instead be indirect consequences of ODD. For instance, engaging in oppositional defiant behavior may lead to long-term interpersonal, academic, or legal difficulties (e.g., lack of supportive relationships, limited educational or occupational opportunities), which may in turn lead to increases in the risk of anxiety, mood, and substance use problems, even after symptoms of ODD remit. Such a model could be tested by examining whether the impact of remitted ODD varies as a function of duration, or more directly by testing potential mediators

such as failure to complete high school or get married and the experience of legal difficulties. It is also possible that ODD and comorbid disorders are related via shared genetic effects. Such analyses are beyond the scope of this initial report; however, our findings and those of previous studies in this area (Dick et al., 2005; Moffitt, 2005; Patterson & Stoolmiller, 1991; Rutter et al., 2006) suggest these are important directions for future research.

Although on one hand ODD continues to be associated with increased risk of subsequent disorders even after remission, it is important to highlight the significant decrease in risk associated with remission of ODD. The fact that the risk of secondary disorders is significantly lower after remission of ODD suggests that successful treatment of ODD might reduce the risk of later disorders (Kessler & Price, 1993). However, an alternative explanation is that the reduced risk of subsequent disorders in remitted ODD is the result of such cases being of lesser clinical severity. A definitive evaluation of this question would require implementation of an effectiveness trial, ideally using one of the evidence-based treatments currently available (Kazdin & Weisz, 2003; Nock, 2003). Nevertheless, our findings underscore the potential importance of early intervention for ODD especially given the enormous percentage of children with ODD who develop a comorbid disorder. Interventions aimed directly at reducing symptoms of ODD may decrease the risk of these secondary disorders. In addition, research on factors that mediate the indirect relations between ODD and secondary disorders, such as those outlined in the previous paragraph, could elucidate additional targets for early intervention programs beyond ODD symptoms (e.g., academic assistance, social support).

Our study also provides new information about the persistence of ODD. Prior prospective studies have demonstrated that ODD is fairly stable over a four- to five-year period (August et al., 1999; Lavigne et al., 2001), but have not examined the longer course of ODD so the actual duration of this disorder has remained unknown. Our study revealed a median duration of six years and found that ODD remitted by 18 years of age for approximately 70% of those with the disorder. Of course, it is possible that this significant drop-off in ODD diagnosis reflects a number of ODD symptoms no longer being appropriately assessed among adults (e.g., refusing to comply with adults' requests), rather than the presence of an important developmental transition. In contrast, although ODD is considered a disorder of childhood and adolescence, it is interesting to note that 30% of those with the disorder continued to report symptoms into adulthood. It would be instructive in future studies to more closely examine possible changes in the form these symptoms take over time (e.g., arguing with one's boss rather than parents).

It also is interesting to note that persistence is significantly longer for males and for those with early onset ODD, and that even after accounting for age-of-onset of ODD, the presence of comorbid disorders also predicts greater persistence (i.e., non-recovery). It may be that these factors are indicative of more severe ODD, and it is this greater severity that leads to longer persistence. Given we did not assess symptom severity this remains a key question for future studies. These results require replication in a longitudinal design but suggest a vicious cycle in which the presence of ODD increases the risk of subsequent mental disorders, and the presence of such disorders increases the persistence of ODD. A notable exception is agoraphobia, which is present in only 2.4% of those with ODD and is the only disorder whose presence significantly predicts recovery. This intriguing finding suggests that specific forms of anxiety, such as agoraphobia, actually may have a protective effect by limiting the performance of oppositional or aggressive behaviors.

ODD has received attention primarily in the context of other disruptive behavior disorders. The present results convey that ODD is a disorder associated with significant long-term comorbidity in its own right. Even when the disorder remits, the likelihood of a subsequent disorder is high. Moreover, ODD relates in important ways to mood, anxiety, and substance use disorders apart from its more familiar relation to CD and ADHD. Given the significance of the course and predictive features of ODD, much more attention to this disorder is warranted. Although ODD is clearly associated with subsequent disorders, important questions remain regarding whether ODD itself *causes* subsequent disorders, and if so what mechanisms are involved, or whether ODD and subsequent disorders are caused by some other common factors (e.g., shared genetic and/or environmental factors). The answers to these key questions are of course beyond the scope of this retrospective study and require the use of natural experiments (e.g., Costello, Compton, Keeler, & Angold, 2003), intervention studies (Kazdin, 2000; Nock, 2003), and behavioral-genetic research designs (Moffitt, 2005), all of which should aim at improving the understanding and ability to prevent and treat this prevalent and costly behavior problem.

Supplementary material

The following supplementary material is available for this article:

Appendix Table 1.

This material is available as part of the online article from: http://www.blackwell-synergy.com/doi/abs/10.1111/j.1469-7610.2007.01733.x

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