Waxing and Waning in Concert: Dynamic Comorbidity of Conduct Disorder With Other Disruptive and Emotional Problems Over 7 Years Among Clinic-Referred Boys

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Six waves of structured diagnostic assessments were conducted of 168 clinic-referred 7- to 12-year-olds, over 7 years. Wave-to-wave changes in the number of conduct disorder (CD) behaviors were paralleled by correlated changes in the numbers of symptoms of oppositional defiant disorder (ODD), attention-deficit/hyperactivity disorder (ADHD), depression, and anxiety. In addition, CD in Wave 1 predicted levels of ODD, ADHD, depression, and anxiety in later waves when initial levels of those symptoms were controlled, but only ODD in Wave 1 predicted CD in later waves when initial CD levels were controlled. These findings indicate a striking degree of dynamic comorbidity between CD and other types of psychopathology and provide an initial empirical framework for needed developmental models of comorbidity.

Conduct disorder (CD) co-occurs at greater than chance levels with oppositional defiant disorder (ODD), attention-deficit/hyperactivity disorder (ADHD), anxiety disorders, and depressive disorders in both clinical and population-based samples (Angold, Costello, & Erkanli, 1999; Caron & Rutter, 1991; Lahey, Miller, Gordon, & Riley, 1999; Loeber & Keenan, 1994; Moffitt, Caspi, Rutter, & Silva, 2001). Comorbid psychopathology almost certainly contributes to the distress and functional impairment experienced by youths with CD and complicates their treatment. Moreover, theories of the origins and course of CD that do not explain its high degree of comorbidity with other types of psychopathology would be incomplete. Therefore, understanding these patterns of comorbidity is essential to understanding the fundamental nature of both CD and its comorbid conditions.

Caron and Rutter (1991) and Loeber and Keenan (1994) noted the particular need for longitudinal studies of comorbidity to understand the developmental relationships among disorders. Among other things, such studies would help to determine if (a)

one type of psychopathology tends to precede another in developmental time and increase the likelihood of the latter type of psychopathology, (b) whether CD and other disorders are more likely to be comorbid at particular times in development than other times, and (c) whether CD and other disorders tend to wax and wane in concert over time. Such information would provide much of the necessary empirical framework for theories of both the development of CD and its dynamic comorbidity with other disorders. Some progress has been made in recent years in understanding comorbidity in developmental terms, but much remains to be learned (Angold et al., 1999).

In clinic samples, youths (mostly boys have been studied to date) who meet criteria for CD have been found to frequently meet criteria for ODD (Biederman et al., 1996; Lahey & Loeber, 1997; Lahey, McBurnett, & Loeber, 2000). There is currently little published evidence on the rate of comorbidity of ODD and CD in population-based samples, however, as virtually all such studies have combined ODD and CD in a single category (Angold et al., 1999; Lahey et al., 1999). There is considerable evidence that CD and ADHD are often comorbid in both clinical and population-based samples (Angold et al., 1999; Lahey et al., 1999); however, among boys, it appears that elevated concurrent rates of CD and elevated rates of subsequent CD are only found among boys with high levels of both ADHD and ODD (Lahey & Loeber, 1997; Lahey et al., 2000).

Puig-Antich (1982) cogently noted that clinic-referred children who met criteria for major depression often exhibited comorbid conduct problems and that their conduct problems improved when their depression remitted. Since then, numerous studies have demonstrated that CD co-occurs with depression at greater than chance levels in both clinic and community samples (Capaldi, 1991;

Kovacs, Paulauskas, Gatsonis, & Richards, 1988; Weissman et al., 1999). It is also clear from cross-sectional studies that CD and anxiety disorders co-occur at greater than chance rates (Zoccolillo, 1992).

A number of explanations for the co-occurrence of CD with these other types of psychopathology have been proposed. First, although CD, ODD, ADHD, and depression undoubtedly have unique causal influences, there is evidence from twin studies that CD shares common genetic influences with ADHD (Coolidge, Thede, & Young, 2000; Thapar, Harrington, & McGuffin, 2001), with ODD (Coolidge et al., 2000; Eaves et al., 2000), and with depression (O'Connor, McGuire, Reiss, Hetherington, & Plomin, 1998). Second, there is evidence that CD shares common environmental risk factors with ODD and depression, such as negativity among family members and parenting that is low in warmth and monitoring (Fergusson, Lynskey, & Horwood, 1996; Frick et al., 1992; Ge, Best, Conger, & Simons, 1996; Goodman & Gotlib, 1999; Goodman et al., 1998; Pike, McGuire, Hetherington, Reiss, & Plomin, 1996). Third, there is evidence consistent with the hypothesis that childhood CD leads to family and peer rejection and academic failure, which give rise to depressive symptoms (Capaldi, 1992; Patterson & Capaldi, 1990; Patterson & Stoolmiller, 1991). Lahey et al. (1999) similarly hypothesized that oppositional behavior during early childhood evokes maladaptive responses from parents, siblings, peers, and teachers that foster the learning of CD behaviors.

Although each of these hypotheses is based partially on data, they are each premature in the sense that the dynamic relations among CD, ODD, ADHD, and emotional symptoms over time have not yet been adequately described among children and adolescents. In order to choose among competing theoretical explanations for the comorbidity of CD with other types of psychopathology, it is first necessary to describe the temporal associations of CD with other symptoms. For example, Patterson and Capaldi's (1990) model would be disconfirmed by consistent evidence that CD behaviors do not precede and predict later depression, and Lahey et al.'s (1999) model would be falsified by consistent evidence that earlier ODD does not predict later CD behaviors. Therefore, this study maps the covariation of CD with other types of symptoms over time in a clinic-referred sample of boys to lay an initial empirical foundation for dynamic theories of comorbidity with CD.

Method

Participants

Participants were boys who were outpatients at one of three mental health clinics in Pittsburgh, Pennsylvania; Athens, Georgia; or Atlanta, Georgia, who entered the study when they were 7 to 12 years old. The sample was purposively selected to be composed of approximately 75% boys with disruptive behavior disorders and 25% boys with other disorders (Loeber, Green, Lahey, Frick, & McBurnett, 2000). Boys were ineligible for participation if they were mentally retarded, psychotic, taking medication that could not be discontinued for 2 days prior to their initial assessment, or were planning to move to another city. Eligible boys had to be living with at least one biological parent at the time of Wave 1, which was almost always the mother. Written informed consent was obtained from the parent or legal guardian, and oral assent or written consent was obtained from the boy in each wave. A total of 177 boys were assessed in Wave 1,

but the current analyses were based on the 168 boys (94.9% of the sample) who were assessed in all six waves to allow assessment of covariation in wave-to-wave changes among different types of symptoms. The 9 boys who did not participate in all assessments did not differ from the 168 boys who did in terms of age in Wave 1, or family income, maternal education, race/ethnicity, or the numbers of symptoms of ODD, anxiety, depression, or CD in any wave. Parents classified 30% of the 168 boys as African American and 70% as non-Hispanic White. One fourth of the sample reported total family incomes below $9,500, and one fourth reported incomes above $35,000. One fourth of the biological mothers had completed less that 12 years of education, and one fourth had completed more than 14 years of education. Forty-two percent of the boys' biological parents were still living together as partners in Wave 1.

Measures and Procedure

The assessments were conducted annually, except for the 5th year of the study, when the assessment was omitted because of limitations in funding. The boys and one of their parents were interviewed separately using the National Institute of Mental Health Diagnostic Interview Schedule for Children (DISC; Costello, Edelbrock, Kalas, & Dulcan, 1984), which queried for symptoms of ADHD, ODD, CD, dysthymia, major depression, and anxiety occurring during the last 6 months, according to criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (3rd, revised 3rd, & 4th editions; *DSM–III, DSM–III–R, & DSM–IV*, respectively; American Psychiatric Association, 1980, 1987, 1994). The anxiety module of this version of the DISC covered only overanxious disorder and separation anxiety disorder. The DISC has acceptable test–retest reliability (Edelbrock, Costello, Dulcan, Kalas, & Conover, 1985), discriminates clinic-referred youths from those without need of treatment (E. J. Costello, Edelbrock, & Costello, 1985), and correlates substantially with standardized parent ratings (Edelbrock & Costello, 1988).

The DISC queried both informants about *DSM–III–R* symptoms of CD and the *DSM–IV* symptom of bullying, but only the parent was asked about the *DSM–IV* symptom of staying out late without permission. The "or rule" was used to combine reports of CD behaviors, with each behavior considered to be present if reported by either the parent or the youth (Piacentini, Cohen, & Cohen, 1992). A continuous sum of *DSM–IV* CD behaviors and the diagnosis of *DSM–IV* CD were generated by computer algorithm, except that the *DSM–III–R* symptom of frequent lying was used instead of the more restrictive *DSM–IV* symptom of lying to con others. Among the 168 boys with complete data in all waves, 69 met criteria for CD in Wave 1, and a total of 114 boys met criteria for CD during at least one wave. Among the 69 boys who met criteria for CD in Wave 1, 65 (94.2%) were reported to have had at least one CD behavior with an age of onset before 10 years of age. Of the 114 boys who met criteria for CD in any wave, 110 (96.5%) were reported to have age of onset before 10 years of age, indicating that nearly all boys who met criteria for CD met *DSM–IV* criteria for the childhood-onset type.

Depression symptoms were measured by summing the number of non-overlapping symptoms of *DSM–III–R* major depression and dysthymia reported by either the parent or the youth. Anxiety symptoms were assessed by summing the number of symptoms of *DSM–III–R* overanxious disorder and separation anxiety disorder reported by either the parent or the youth. Because preliminary analyses indicated that the association of CD with symptoms of overanxious disorder and separation anxiety disorder were qualitatively similar, albeit with somewhat stronger associations between CD and separation anxiety than with overanxious symptoms, the number of symptoms of these two types of anxiety were summed. The correlations of the numbers of overanxious and separation anxiety symptoms during Waves 1–7 ranged from .21 to .43 (*Mdn* = .32, all *ps* < .01).

DSM–III–R criteria were applied to create diagnoses of dysthymia and major depression using a computer algorithm based on the combination of parent and youth reports of symptoms. On the basis of evidence that youths

are unreliable reporters of symptoms of ODD and ADHD in DISC interviews (Hart, Lahey, Loeber, & Hanson, 1994), only parent-reported ODD and ADHD symptoms were used in the analyses. Counts of the boys' numbers of symptoms of ODD were created by summing the number of the *DSM–IV* symptoms reported by the parent. Counts of ADHD symptoms were created by first dividing the *DSM–III–R* symptoms assessed in this version of the DISC into separate dimensions of inattention and hyperactivity—impulsivity on the basis of the *DSM–IV* model. Six symptoms of inattention involving difficulties with sustained attention, following instructions, listening to adults, losing things, distractibility, and shifting from one unfinished task to another were summed. Similarly, seven symptoms of hyperactivity–impulsivity involving difficulty remaining seated, awaiting one's turn, playing quietly, talking excessively, interrupting others, fidgeting and squirming in seat, and remaining seated were summed. The *DSM–III–R* symptom of "gets into dangerous situations without considering the consequences" was deleted from the list of ADHD symptoms because the *DSM–IV* field trials showed that this symptom is more closely linked to CD than ADHD (Frick et al., 1994). For this reason, including it in the list of ADHD symptoms may have inflated estimates of the association between ADHD and CD over time. Because preliminary analyses of the two dimensions of ADHD symptoms revealed no differences in their association with CD over time, they were combined in a single count of ADHD symptoms. The correlations of the numbers of inattention and hyperactivity–impulsivity symptoms during Waves 1–7 ranged from .56 to .75 (*Mdn* = .71, all *p*s < .0001).

During Wave 1, interobserver agreement for the report of each symptom was assessed by having a second interviewer observe 25% of the parent and child interviews from behind a one-way mirror. As reported earlier (Lahey et al., 1990), the median interobserver kappa for agreement for the report of individual symptoms of CD, ODD, ADHD, anxiety, and depression in this highly structured protocol ranged from .87 for ADHD symptoms to 1.00 for CD symptoms. The values for alpha coefficients across Waves 1–7 for each type of symptoms was as follows: CD: range = .60–.72, *Mdn* = .65; ODD: range = .76–.87, *Mdn* = .84; ADHD: range = .85–.90, *Mdn* = .88; depression: range = .81–.90, *Mdn* = .86; anxiety: range = .71–.82, *Mdn* = .78.

Data Analysis

The response variables in the present analyses were counts of CD, ODD, ADHD, depression, and anxiety symptoms during each assessment wave. As is typical of such symptom data, each distribution was highly skewed, with modal values being at or near zero. When collapsed across waves, the median number of CD symptoms (range = 0–11) was between 1 and 2, with 28.4% of the counts being 0 and 75% being 3 or less. The median number of ODD symptoms (range = 0–9) was 4, with 16.9% of the counts being 0 and 75% being 6 or less. The median number of ADHD symptoms (range = 0–13) was 5, with 16.4% of the counts being zero and 75% being 8 or less. The median number of anxiety symptoms (range = 0–15) was 2, with 13.8% of the counts being 0 and 75% being 5 or less. The median number of depression symptoms (range = 0–11) was 1, with 41.3% of the counts being 0 and 75% being 2 or less.

These highly skewed count variables clearly do not meet the distributional assumptions of most classical approaches to longitudinal data analysis, such as repeated-measures analysis of variance (ANOVA) and hierarchical linear models. Although the Poisson distribution may be used as a working model for such data, each distribution of symptom counts is "overdispersed," as the variance exceeds the mean in each case. Therefore, even the more recently developed generalized linear mixed models, which can handle data with true Poisson distributions, are not appropriate because of the overdispersion of the present data.

An alternative statistical approach is to model mean numbers of ODD, ADHD, depression, and anxiety symptoms in longitudinal log-linear regression models, specifying working models of the response distributions

based on either Poisson or negative binomial distributions in generalized estimating equations (GEE; Zeger & Liang, 1986). GEE is a data-driven strategy that models the average value of the response variable for each subset of individuals who share the same value of the predictor variable and it accounts for within-subject correlations in the response variable over waves. Because GEE estimates averages, and not the entire distribution of values, GEE is less restricted by distributional assumptions than other approaches to longitudinal analysis. That is, GEE can be used when the distributions of values of response variables do not conform to the distributions (normal, Poisson, etc.) required by other longitudinal models. All statistical tests in the present GEE analyses used robust ("empirical") standard errors because an adjustment for overdispersion is automatically included in the calculation of the robust standard error, and its use reduces concern about correct specification of the within-subject covariance structure. Thus, GEE allows unambiguous presentation and interpretation of counts of symptoms when the distributions take the shape of the present data, which means that it is an appropriate option for many similar studies of psychopathology. All present analyses were conducted specifying Poisson working distributions, but virtually identical results were obtained when the same GEE models were fitted specifying negative binomial working distributions.

In a preliminary set of GEE analyses, mean response was modeled simply as a function of increasing time, adjusting for the age of the boys in Wave 1. These analyses of developmental change provided a background for the interpretation of the subsequent longitudinal analyses of symptom covariation. In these initial analyses, time (waves) was treated as a linear covariate with values of 1, 2, 3, 4, 6, and 7. Analyses (not shown) modeling the effect of time, using five dichotomous indicator variables for waves, were performed with very similar results.

The next longitudinal GEE analyses assessed temporal covariation of CD symptoms with the other types of symptoms. Two complementary statistical approaches to assessing temporal covariation among symptoms were used, referred to as Model A and Model B. Model A analyses assessed the cross-sectional association between numbers of CD symptoms and other types of symptoms within each wave over time. Hence, any temporal associations between symptoms detected in Model A reflect both between-persons effects (some youths tend to have more or less of both types of symptoms over time than other youths) and correlated within-person changes in symptoms over time (as the number of CD behaviors increases or decreases from one wave to the next, the number of the other type of symptoms correspondingly increases or decreases, controlling for any developmental trends). The number of CD symptoms in each wave was treated as a time-varying covariate in Model A, with the response variables in separate analyses being the number of ODD, ADHD, depression, or anxiety symptoms in each wave. Model A analyses are provided primarily to aid in the interpretation of Model B analyses, which focus on the central issue of correlated, within-person changes in CD and other types of symptoms over time (waves). Model B analyses assessed the association of the number of CD behaviors in Wave $t + 1$ with the number of each other type of symptom in Wave $t + 1$ (over Waves 2–7), controlling for the number of that type of symptom in Wave t and the number of CD behaviors in Wave t. In doing so, Model B analyses allow an assessment of the degree to which CD behaviors and the various types of comorbid symptoms increase or decrease together in the same person from wave to wave.

A final set of prospective statistical models used log-linear regression in GEE of ODD, ADHD, depression, and anxiety symptom counts over time to ask, for example, whether early levels of CD (in Wave 1) are a predictor of later levels of depression (over Waves 2–7), controlling for depression in Wave 1, and vice versa. These analyses allow us to assess asymmetries in the prospective association between CD and depression—in which early CD predicted later depression but not vice versa, identified in previous studies (Capaldi, 1992; Patterson & Capaldi, 1990; Patterson & Stoolmiller, 1991)—and to determine if these asymmetric prospective relations extend to other types of symptoms.

In all of the log-linear models described above, the regression coefficient, beta, represents the log-relative mean number of symptoms for a 1-unit difference in each independent variable. For example, in the model assessing changes in ODD symptoms over time (adjusting for age in Wave 1), $\beta = -0.07$ indicates that the mean number of ODD symptoms in each wave (adjusted for age in Wave 1) was an average of 93% of the number of ODD symptoms in the previous wave because $\exp(-0.07) = .93$. Interpreting beta as an estimate of effect size in this sense requires recognition that the range of scores for each predictor variable differs. As a result, a smaller beta for a predictor with a broader range of scores could indicate a stronger association than a larger beta for a predictor with a narrower range of scores.

Results

Changes in Disruptive, Emotional, and CD Symptoms Over Waves

Preliminary longitudinal log-linear GEE regression models specifying an autoregressive correlation structure and controlling for age in Wave 1 revealed that there was not a significant change across Waves 1–7 in the mean number of depression symptoms, $\beta = -.01$, $z = -0.23$, $p = .82$, or the number of ADHD symptoms, $\beta = -0.02$, $z = -1.69$, $p = .09$; however, there were significant declines over time in the numbers of symptoms of ODD, $\beta = -0.07$, $z = -6.68$, $p < .0001$, and anxiety, $\beta = -0.13$, $z = -8.93$, $p < .0001$. The mean number of CD behaviors did not change significantly across the six assessments waves, $\beta = -0.01$, $z = -1.04$, $p = .30$; however, this lack of change in the mean masked a marked degree of individual differences in the numbers of CD behaviors over time: Some boys exhibited relatively stable low or high numbers of CD behaviors over time, whereas others exhibited high (or low) levels of CD in Wave 1 but showed decreases (or increases) over subsequent waves.

For the sole purpose of preparing the graphic illustrations of the data presented in Figures 1–3, six arbitrary subgroups of boys were distinguished on the basis of their initial levels of CD in Wave 1 and their mean levels of CD in Waves 4–7. Among the 168 boys who participated in all assessment waves, 69 boys met diagnostic criteria for CD during Wave 1 and 99 boys did not. As shown in Figure 1, nearly half of the boys who met criteria for CD in Wave 1 (left panel) continued to engage in an average of 3 or more CD behaviors in Waves 4–7, whereas the remainder showed moderate to strong improvement in CD behaviors over time. Conversely, among the 99 boys who did not meet criteria for CD in Wave 1, most of these boys consistently exhibited low to moderate numbers of CD behaviors during Waves 4–7; however, 15 of the boys who did not meet criteria for CD in Wave 1 showed an increase to an average of 3 or more CD behaviors during Waves 4–7. These marked individual differences in the course of CD behaviors over time (illustrated in Figure 1) and their association with individual differences in other types of symptoms (illustrated in Figures 2–3) are the topic of the present analyses. Note, however, that no statistical analyses were based on the illustrative subgroups used in Figures 1–3.

Covariation of CD With Other Types of Psychopathology Over Time

The covariation of CD behaviors with the symptoms of other disorders over time was evaluated using the analytic strategies defined above as Model A and Model B. When the number of CD behaviors in each wave was treated as the time-varying covariate and the number of the other type of symptoms in the same wave was treated as the dependent variable (controlling for time and the boys' age in Wave 1), the number of CD behaviors was associated with the numbers of symptoms of ODD, ADHD, depression, and anxiety. The results of these analyses are presented on the left side of Table 1 (labeled Model A) and the Spearman correlations between the number of CD symptoms and the numbers of each
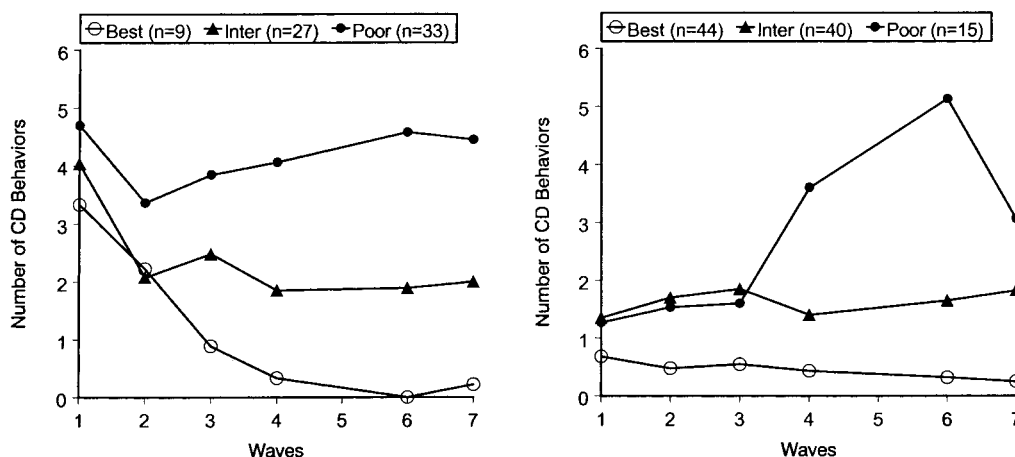

Figure 1. Individual differences in the course of conduct disorder (CD) behaviors over waves among 69 boys who met criteria for CD in Wave 1 (left panel) and 99 boys who did not meet criteria for CD in Wave 1 (right panel). For the purpose of graphic illustration only, the mean number of CD behaviors in each wave is presented for three arbitrary subgroups on the basis of outcomes of CD in each panel: those with (a) the best outcomes (mean of <1.0 CD behaviors during Waves 4–7), (b) intermediate (Inter) outcomes (mean of 1.0–2.99 CD behaviors during Waves 4–7), and (c) the poorest outcomes (mean of ≥3.0 CD behaviors in Waves 4–7).

Table 1
Associations Over Time Between Conduct Disorder Behaviors and Symptoms of Other Types of Psychopathology

Measure	Model A: Covariation between and within persons			Model B: Correlation of within-person changes over time		
	β	z	p	β	z	p
Response variable: ODD behaviors in Waves $t + 1$ (Waves 2–7)						
Age in Wave 1	−.03	−1.23	.22	−.03	−2.11	.04
Time (waves)	−.05	−4.11	.0001	−.02	−2.57	.01
CD in Wave $t + 1$	.12	9.66	.0001	.11	9.43	.0001
CD in Wave t				−.03	−2.67	.01
ODD in Wave t				.15	13.13	.0001
Response variable: ADHD behaviors in Waves $t + 1$ (Waves 2–7)						
Age in Wave 1	−.12	−5.34	.0001	−.05	−3.99	.0001
Time (waves)	−.06	−5.69	.0001	−.01	−1.40	.16
CD in wave $t + 1$	.08	7.18	.0001	.07	5.51	.0001
CD in Wave t				−.03	−2.28	.02
ADHD in Wave t				.13	16.36	.0001
Response variable: Depression symptoms in Waves $t + 1$ (Waves 2–7)						
Age in Wave 1	.05	1.22	.22	.03	1.04	.30
Time (waves)	−.01	−0.30	.76	−.01	−0.47	.64
CD in Wave $t + 1$	.17	10.14	.0001	.15	7.27	.0001
CD in Wave t				.02	0.61	.54
DEP in Wave t				.12	7.17	.0001
Response variable: Anxiety behaviors in Waves $t + 1$ (Waves 2–7)						
Age in Wave 1	−.07	−2.76	.006	−.04	−2.55	.01
Time (waves)	−.13	−7.18	.0001	−.08	−4.80	.0001
CD in Wave $t + 1$	.10	7.05	.0001	.09	6.26	.0001
CD in Wave t				.00	0.19	.85
ANX in Wave t				.10	9.21	.0001

Note. ODD = oppositional defiant disorder behaviors; CD = conduct disorder behaviors; ADHD = attention-deficit/hyperactivity disorder behaviors; DEP = symptoms of depression; ANX = symptoms of anxiety.

other type of comorbid symptoms within each assessment wave are presented in Table 2 to provide an index of these within-wave associations in the familiar metric of correlation coefficients.

Model B analyses focused on correlated changes within persons in CD behaviors and the symptoms of ODD, ADHD, depression,

Table 2
Cross-Sectional Correlations of CD Behaviors in Each Wave With Each Type of Comorbid Symptoms in the Same Wave

Measure	Wave						
	1	2	3	4	5	6	7
ODD	.59	.53	.53	.54		.59	.59
Depression	.27	.38	.26	.41		.42	.35
Anxiety	.17	.30	.27	.37		.26	.26
ADHD	.24	.35	.13	.21		.34	.35

Note. No assessment was conducted during the 5th year. For conduct disorder (CD), oppositional defiant disorder (ODD), depression, and anxiety, they each represent the sum of the symptoms of the disorder. $N = 168$ for all correlations (Spearman's rhos). All coefficients are significantly different from zero at $p < .05$. ADHD = attention-deficit/hyperactivity disorder.

and anxiety (see the right side of Table 1). Controlling for the boys' age in Wave 1 and time, the number of CD behaviors in Wave $t + 1$ (controlling for the number of CD behaviors in the previous wave) was significantly associated with the number of the other type of symptoms in Wave $t + 1$ (controlling for the number of that type of symptom in the previous wave) in each Model B analysis. For each type of comorbid psychopathology, the beta coefficient in Model B (which focuses on within-person covariation in symptoms over time) for CD symptoms in Wave $t + 1$ is about 90% of the corresponding beta coefficient in Model A (which combines within- and between-persons components of the covariation in symptoms over time). Although it may not be possible to fully isolate within-person temporal covariation in the Model B analyses, this comparison suggests that correlated within-person changes in symptoms over time account for a substantial degree of the association of CD with other types of psychopathology over time, and between-persons differences account for less of these associations.

To graphically illustrate these patterns of temporal covariation of comorbid symptoms with CD behaviors within persons, we plotted the mean numbers of symptoms of each type of comorbid symptoms across waves, depicted in Figure 2 for boys who met
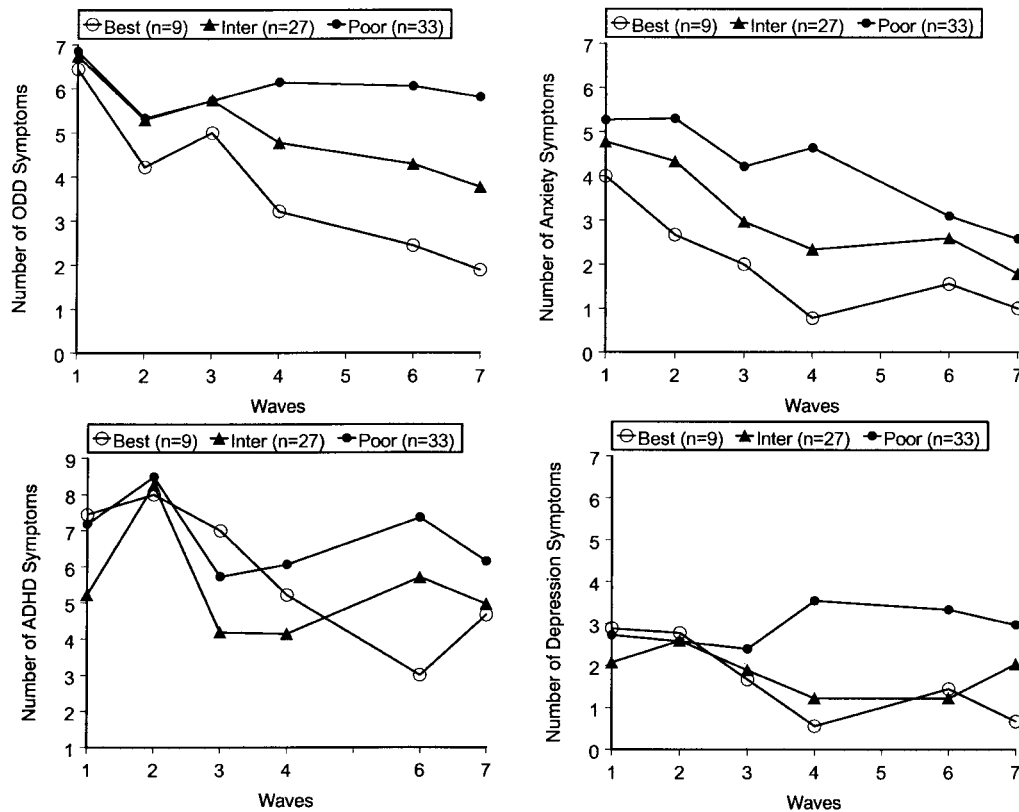
Figure 2. Illustration of the temporal association between changes in conduct disorder (CD) behaviors over waves, and changes in other types of psychopathology among the 69 boys who met criteria for CD in Wave 1, by plotting the mean number of oppositional defiant disorder (ODD) behaviors (upper left), anxiety symptoms (upper right), attention-deficit/hyperactivity disorder (ADHD) symptoms (lower left), and depression symptoms (lower right), against wave for three arbitrary subgroups of boys on the basis of their CD outcomes: those with (a) the best outcomes (mean of <1.0 CD behaviors during Waves 4–7), (b) intermediate (Inter) outcomes (mean of 1.0–2.99 CD behaviors during Waves 4–7), and (c) the poorest outcomes (mean of ≥3.0 CD behaviors in Waves 4–7).

criteria for CD in Wave 1 and depicted in Figure 3 for boys who did not meet criteria for CD in Wave 1, using the same three illustrative subgroups based on the course of CD over time that were used in Figure 1. These figures illustrate temporal covariation at the level of subgroups of similar persons rather than at the individual level. Nevertheless, the temporal covariation of CD behaviors with each other type of psychopathology can be seen by comparing each panel of Figure 1 with the corresponding panels of Figures 2 and 3.

Because of concerns about the reliability of child reports of symptoms among younger youths, all of the models reported in Table 1 were also estimated using (a) only boys who were younger than 9 years of age in Wave 1 and (b) only boys who were 9 years of age or older in Wave 1. All results were qualitatively and quantitatively quite similar in each case to the results for the full sample. This suggests that the combined parent and youth reports of symptoms used in these analyses were sufficiently reliable and valid for boys who were 7 or 8 years of age in Wave 1 to exhibit essentially the same patterns of covariation for youths who were older in Wave 1.

Asymmetric Prospective Associations Between CD and Other Types of Symptoms Across Waves

On the basis of previous findings of an asymmetric prospective association between CD and depression (Capaldi, 1992; Patterson & Capaldi, 1990; Patterson & Stoolmiller, 1991), we conducted an additional series of analyses using the 168 boys with complete data to examine prospective associations between CD behaviors and each of the other types of comorbid symptoms, and vice versa. In each case, the predictor was measured in Wave 1 (e.g., CD behaviors in Wave 1) and the outcome was measured over the next 6 years (e.g., depression symptoms over Waves 2–7). As shown in Table 3, in analyses that controlled for age and time, the number of CD behaviors in Wave 1 significantly predicted the number of ODD, ADHD, anxiety, and depression symptoms during Waves 2–7, even when the number of each of those symptoms in Wave 1 was controlled. In contrast, only the number of ODD symptoms in Wave 1 predicted the number of CD behaviors during Waves 2–7 when the number of CD behaviors in Wave 1 was controlled. The associations between the number of CD behaviors
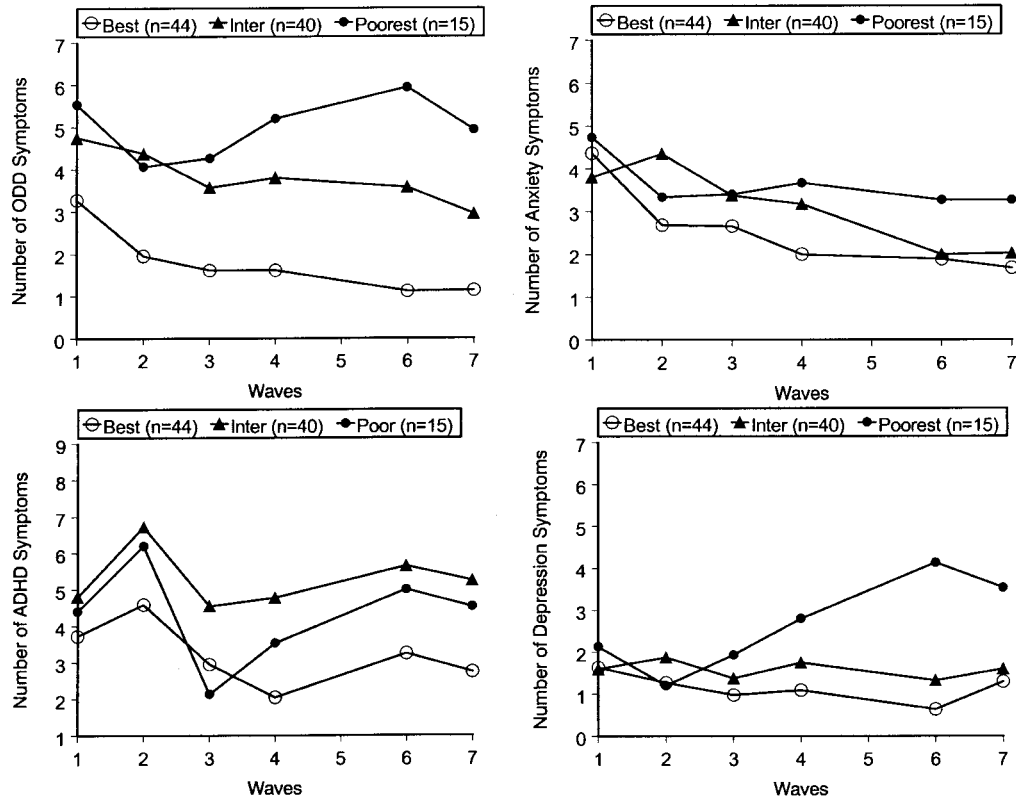
Figure 3. Illustration of the temporal association between changes in conduct disorder (CD) behaviors over waves, and changes in other types of psychopathology among the 99 boys who did not meet criteria for CD in Wave 1, by plotting the mean number of oppositional defiant disorder (ODD) behaviors (upper left), anxiety symptoms (upper right), attention-deficit/hyperactivity disorder (ADHD) symptoms (lower left), and depression symptoms (lower right), against wave for three arbitrary subgroups of boys on the basis of their CD outcomes: those with (a) the best outcomes (mean of <1.0 CD behaviors during Waves 4–7), (b) intermediate (Inter) outcomes (mean of 1.0–2.99 CD behaviors during Waves 4–7), and (c) the poorest outcomes (mean of ≥3.0 CD behaviors in Waves 4–7).

in Wave 1 and the number of each type of comorbid symptoms in Waves 2–7 are presented in Figure 4. The graphed data are not adjusted for the number of the same type of symptoms in Wave 1 because the adjustments had very little effect on the curves and it is not possible to show both the number of each type of comorbid symptoms in Wave 1 as a point of reference (for judging increases or decreases from baseline) and adjust for the number of the same type of symptoms in Wave 1. In each case, boys with higher levels of CD in Wave 1 tended to exhibit somewhat higher levels of each type of comorbid symptoms across Waves 2–7.

CD and the Diagnosis of Depression

Unlike other types of comorbid psychopathology, the diagnoses of major depression and dysthymic disorder are not based on simple counts of symptoms but require particular combinations of symptoms that have persisted for specific durations. As a result, the relation of numbers of symptoms of depression to the diagnosis of depression is less clear than for the other types of psychopathology. For this reason, additional analyses were conducted to examine the relation of individual differences in the course of CD

to the boys' meeting *DSM–III–R* criteria for either major depression or dysthymia.

As a follow-up to analyses of the asymmetric prospective relation between numbers of symptoms of CD and depression, the number of CD behaviors in Wave 1 was found to predict the diagnosis of depression during Waves 2–7, $\beta = .18$, $z = 2.41$, $p < .02$, controlling for age in Wave 1, time, and levels of depression symptoms in Wave 1. When the interaction of CD in Wave 1 and time was added to the model, however, it was significant, $\beta = -.07$, $z = -2.10$, $p < .04$, indicating that CD in Wave 1 predicted the diagnosis of depression better in subsequent waves nearer to Wave 1 in time than in later waves.

When the number of CD behaviors in Wave $t + 1$ was treated as a time-varying covariate in logistic regression, with autocorrelation for the response variable of the diagnosis of depression in each wave across Waves 2–7 (comparable to Model A in Table 1), there was a significant association of CD behaviors with the diagnosis of depression in the same waves, $\beta = 0.33$, $z = 7.46$, $p < .0001$, when age in Wave 1 and time were controlled. When the interaction of CD in Wave $t + 1$ and time was added to the

Table 3
Prospective Associations Between Each Disorder in Wave 1 and Other Disorders in Waves 2–7

Measure	Predicting ODD from CD			Predicting CD from ODD		
	β	z	p	β	z	p
Age in Wave 1	−.07	−3.29	.001	−.03	−1.03	.30
Time (waves)	−.04	−2.99	.003	.03	1.45	.15
CD in Wave 1	.05	2.81	.005	.17	5.35	.0001
ODD in Wave 1	.18	9.98	.0001	.10	3.81	.0001

Measure	Predicting ADHD from CD			Predicting CD from ADHD		
	β	z	p	β	z	p
Age in Wave 1	−.04	−1.76	.08	−.03	−0.73	.47
Time (waves)	−.05	−4.76	.0001	.03	1.46	.14
CD in Wave 1	.03	2.30	.02	.22	7.50	.0001
ADHD in Wave 1	.10	10.00	.0001	.00	0.07	.94

Measure	Predicting anxiety from CD			Predicting CD from anxiety		
	β	z	p	β	z	p
Age in Wave 1	−.06	−2.24	.02	−.03	−0.89	.37
Time (waves)	−.12	−6.86	.0001	.03	1.46	.14
CD in Wave 1	.05	2.17	.03	.22	7.57	.0001
Anxiety in Wave 1	.08	5.62	.0001	.00	0.05	.96

Measure	Predicting depression from CD			Predicting CD from depression		
	β	z	p	β	z	p
Age in Wave 1	.03	0.77	.44	−.03	−0.94	.35
Time (waves)	.00	0.20	.84	.03	1.46	.14
CD in Wave 1	.10	3.10	.002	.22	7.24	.0001
Depression in Wave 1	.11	4.06	.0001	.01	0.54	.59

Note. All predictions of numbers of symptoms of disorders are made during Waves 2–7 from the number of symptoms of disorders in Wave 1. CD = conduct disorder; ODD = oppositional defiant disorder; ADHD = attention-deficit/hyperactivity disorder.

model, it was not significant ($p = .98$). Note that this model was based on Waves 2–7 (like Model A in Table 1) to allow direct comparisons with the model reported later, which is based on Model B in Table 1. This finding is illustrated in Figure 5, which shows that within each wave, the proportion of boys who met criteria for depression increased as the number of CD behaviors in that wave increased. Although the proportion of boys who met criteria for depression in each wave was always below 30% even among boys with the highest numbers of CD behaviors, the proportion of boys with high levels of CD over time who met criteria at least once for major depression or dysthymia was higher. For example, 63.2% of boys who exhibited an average of 3 or more CD behaviors across Waves 2–7 met criteria for major depression or dysthymia at least once during Waves 2–7, which was more than five times that of boys who exhibited a mean of less than 1 CD behavior over waves 2–7 (11.8%) and nearly double that of boys who exhibited an average of 1–2.99 CD behaviors (35.4%).

When the number of CD behaviors in Wave t and the number of depression symptoms in Wave t were added to the model above to focus on within-person temporal covariation (comparable to Model B in Table 1), there was still a significant association of CD behaviors and the diagnosis of depression, $\beta = 0.31$, $z = 6.06$, $p < .0001$. When the interaction of CD in Wave $t + 1$ and time was added to the model, it was not significant ($p = .92$). As when depression was treated as the continuous number of symptoms, comparison of the beta weights for these two models suggests that most of the association between CD and the diagnosis of depression reflects correlated within-person changes in CD and depression.

Discussion

In this clinic-referred sample of boys, within-person changes in CD behaviors over time were paralleled by correlated within-person changes in the numbers of symptoms of ODD, ADHD, depression, and anxiety. That is, greater or lesser increases (or decreases) in CD over time were accompanied by correspondingly greater or lesser changes in levels of comorbid symptoms. These patterns of temporal covariation are illustrated in Figures 1–3. The specific pattern of temporal covariation of each type of symptoms with CD varied (a) as a function of the overall developmental trend for each type of comorbid symptoms to increase, decrease, or remain stable over time, and (b) whether or not the prospective associations between earlier and later symptoms of different types were symmetrical. Anxiety, depression, and ADHD showed asymmetric prospective associations with CD in that early CD predicted later anxiety, depression, and ADHD when early levels of these symptoms were controlled, but not vice versa. As shown in Figure 4, these prospective associations between CD and the four types of comorbid symptoms reflect relatively enduring between-persons differences that are superimposed on the substantial degree of correlation between within-person changes in CD and each type of comorbid symptoms. The analyses in which depression was treated as a diagnosis, however, raise the possibility that the prospective association between early CD behaviors and later depression that is severe enough to warrant a diagnosis may be less enduring than indicated by average numbers of symptoms of depression.

Only ODD behaviors showed a symmetrical pattern of prospective association with CD. As shown in Figure 2, boys with higher levels of CD in Wave 1 who engaged in persistently high levels of CD over the next 6 years exhibited relatively stable and high levels of ODD. More to the point, Figure 3 shows that boys who exhibited lower levels of CD in Wave 1 but showed increasing levels of CD over time also exhibited high and relatively stable levels of ODD behaviors across Waves 1–7. Consistent with the hypothesis that childhood ODD is a developmental precursor to CD in at least some boys (Lahey et al., 2000; Loeber, Green, Keenan, & Lahey, 1995), this reveals that boys who engaged in high levels of CD during adolescence showed high levels of ODD during the early waves of the study, even if they did not engage in high levels of CD until later waves in the study. That is, among these boys with high levels of conduct problems only during the later waves of the study, high early levels of ODD preceded their upsurge in CD behaviors. In contrast, higher levels of ADHD, depression, and anxiety symptoms did not presage later increases in CD. These findings shed additional light on developmental sequences from
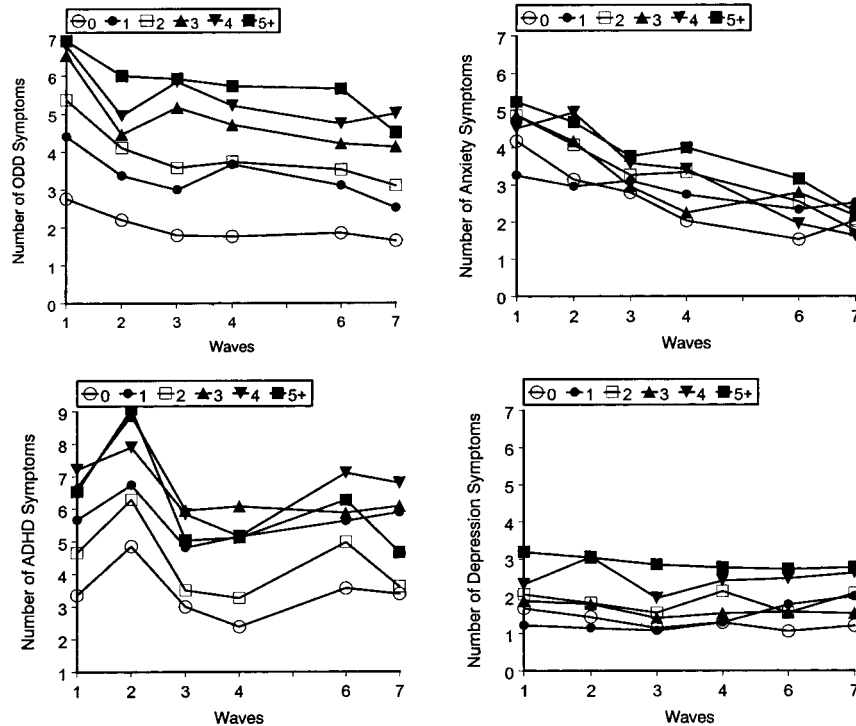
Figure 4. Prospective associations between the number of conduct disorder (CD) behaviors in Wave 1 and the number of symptoms of oppositional defiant disorder (ODD), anxiety, attention-deficit/hyperactivity disorder (ADHD), and depression in Waves 2–7 (with the number of each type of comorbid symptom in Wave 1 presented to provide a point of reference) are shown by plotting the number of each type of symptom in each wave for boys with 0 ($n = 34$), 1 ($n = 27$), 2 ($n = 38$), 3 ($n = 24$), 4 ($n = 19$), and 5 or more ($n = 26$) CD behaviors in Wave 1.

one type of psychopathology to another, or what Angold et al. (1999) termed *successive comorbidity.*

Although it is not novel to demonstrate that higher levels of ODD often precede increases in CD behaviors, the present data shed new light on the relation between CD and ODD when levels of CD decline over time. Before the present study, one might have assumed that factors that led to reductions in high levels of CD would leave underlying levels of ODD unchanged. For example, changes in environmental conditions (such as moving to a new neighborhood or an improvement in parental supervision) that led to reductions in stealing, vandalism, and fighting might not have influenced youths' irritability and argumentativeness. The present findings indicate, however, that boys with higher initial levels of CD who showed declining levels of CD over time also showed declining levels of ODD over time (compare Figure 1 with Figure 2). Indeed, the most striking and novel aspect of these findings is that boys who improved from initially higher levels of CD showed declining levels of all types of comorbid symptoms (they improved in everything).

Why does CD display these patterns of prospective and dynamic comorbidity with other types of symptoms over time? Findings that the comorbidity of CD with ODD, ADHD, and depression may each be partly explained by sharing some of the same genetic influences may point us in the right direction. More remains to be learned, however, about the reasons for the substantial degree of wave-to-wave changes in each type of symptom and the factors behind the correlated nature of these changes. That is, in order to completely explain comorbidity, competing models must be tested in terms of their ability to explain the dynamic patterns of comorbidity in changing levels of symptoms over time. One implication of the present findings is that simple genetic models of comorbidity might not be sufficient by themselves to explain the dynamic patterns of covariation. Clearly, it will be important to evaluate the possible role of time-varying environmental influences, such as life events and changing social environments, that simultaneously influence changes in levels of both CD and each type of comorbid symptoms. On the other hand, genetic influences are often not static. Indeed, there is evidence of genetic influence on the degree of change in both intellectual ability and conduct problems in youths over time (O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998; Wilson, 1986). This raises the possibility that genetic influences (which may be direct or indirect, such as genetic influences on sensitivity to environmental influences) might account for some or all of the correlated changes in CD and comorbid symptoms. It is important to note, however, that the one study that provided evidence of genetic influences on change in CD over time did not find evidence of genetic influences on change in depression, even though much of the genetic influences on CD and depression were found to be shared (O'Connor et al., 1998).
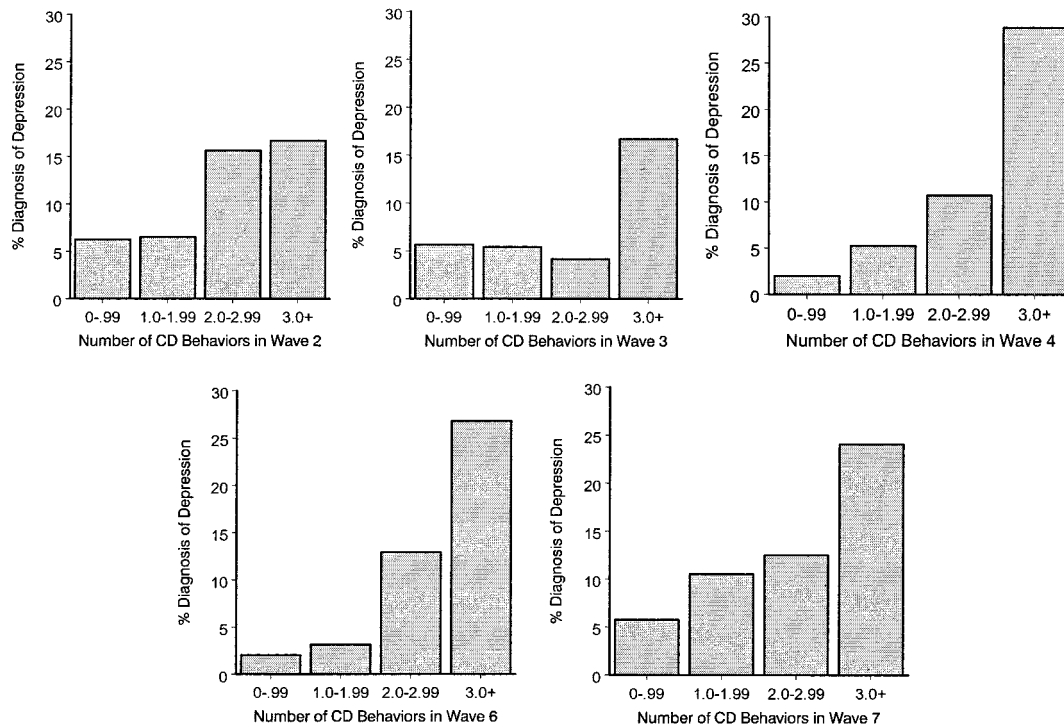
Figure 5. The proportion of boys who concurrently met *DSM–III–R* diagnostic criteria for major depression or dysthymia during Waves 2–7 as a function of the number of conduct disorder (CD) behaviors in the same wave.

Clearly, we need to learn more about genetic and environmental influences on correlated changes in CD and comorbid symptoms.

Why do early levels of CD predict future levels of ODD, ADHD, depression, and anxiety? Patterson and Capaldi (Capaldi, 1992; Patterson & Capaldi, 1990; Patterson & Stoolmiller, 1991) hypothesized that CD behaviors indirectly influence the likelihood of future depression by causing academic failure and rejection by family and peers. The present findings present two challenges to their hypothesis, however. First, the level of CD in Wave 1 predicted future levels of all four types of comorbid symptoms, suggesting that the prospective association between earlier CD and later psychopathology is not specific to depression. Second, the associations between early CD and later symptoms are not consistent with the pattern of early CD leading to later increases in depression implied by Patterson and Capaldi's model. Rather, as shown in Figure 4, higher levels of CD in Wave 1 are accompanied by higher average levels of depression symptoms in Wave 1 that remain relatively stable across Waves 2–7. This is perhaps not surprising because depression symptoms do not increase in prevalence with age across childhood and adolescence among boys in the general population, even though they increase from childhood to adolescence in girls (Hankin & Abramson, 2001). Indeed, the present findings suggest that when depression does increase from one wave to the next, it is accompanied by concurrent increases in CD rather than preceded by increases in CD as the Patterson and Capaldi model would suggest.

It is possible that childhood-onset CD and other types of behavioral and emotional symptoms are dynamically linked concurrently and prospectively because they each partially reflect a common underlying psychological process that is relatively stable but can fluctuate over time. One candidate for such a general process is the dimension referred to as "negative emotionality," "negative affectivity," or "neuroticism" in childhood and adulthood (Eisenberg, Fabes, Guthrie, & Reiser, 2000; McCrae et al., 2000; Watson, Clark, & Carey, 1988), which has been found to be correlated with symptoms of depression, anxiety, and conduct problems both concurrently and prospectively in children and adolescents (Eisenberg et al., 1996; Goodyer, Ashby, Altham, Vize, & Cooper, 1993; Rende, 1993). If the boys' general tendency to respond to their social environments with negative emotion is not fully stable but waxes or wanes over time in some boys, their levels of CD and other types of psychopathology may wax or wane along with it. Such an explanation is consistent with the close temporal association of changing levels of CD and other types of symptoms over time found in the present study. Additionally, it is possible that the prospective association of CD with future levels of other types of psychopathology indicates that childhood CD is a particularly good indicator of this common underlying process. These and other hypotheses must be explored in the future to fully understand the nature and course of both CD and its comorbid conditions.

It is important to note that the striking degree of dynamic association between CD and other syndromes of psychopathology revealed in this study strongly suggests that CD is often more than just "misbehavior." Rather, CD is often a complex mental health problem that is intimately linked with several other types of psychopathology concurrently and prospectively. This indicates that children who engage in antisocial behavior must be assessed for comorbid psychopathology to provide the most effective inter-

ventions; it also suggests that theories of antisocial behavior among children and adolescents will need to be embedded within broader theories of the development of psychopathology to be complete.

Several limitations of the present study should be noted. First, the exclusion of girls from the sample means that we cannot shed light on the course of CD in girls. It is somewhat reassuring that Moffitt et al. (2001) recently reported increased levels of depression in both male and female adolescents with a lifetime diagnosis of CD in the Dunedin Longitudinal Study, but their findings suggested higher levels of comorbid depression among boys with CD than girls with CD before adolescence. Thus, the results of the present study may have been different in some ways if the youths had been girls. Second, because ODD, ADHD, and anxiety symptoms appear early in life, our longitudinal study (which first measured these symptoms at 7–12 years of age) may misrepresent the prospective relations between these symptoms and CD. Furthermore, a study that began earlier in life might have found that CD preceded increases in depression that were more consistent with Patterson and Capaldi's (1990) model. Therefore, longitudinal studies beginning at earlier ages are sorely needed to test the generality of the present findings. Third, clinic samples are known to be biased toward higher levels of comorbidity (Goodman et al., 1997). Although CD has consistently been found to co-occur with ODD, ADHD, anxiety, and depression at higher than chance levels among youths in the general population (Lahey et al., 1999), it is possible that the use of a clinic sample exaggerated the degree of temporal covariation of CD with these other types of psychopathology. It is important to study children whose problems are severe enough to lead to clinic referral, but longitudinal studies of representative samples of girls and boys that are initiated in early childhood, and are large enough to contain sufficient numbers of youths with high levels of each type of symptom, may be the most informative.

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