

Conduct Disorder Subtype and Comorbidity

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Background. Conduct disorder is considered difficult to treat, but comorbid psychiatric disorders may be a basis for treating some youths with conduct disorder. We sought to identify patterns of comorbid psychiatric diagnoses and psychopathology associated with conduct disorder by reported age-of-onset.

Methods. Referred children and adolescents, aged 4–17 years old, were clinically evaluated. Ages of onset of CD symptoms ($N = 53$) were ascertained and divided according to DSM-IV criteria as childhood onset (< 10 years old) or adolescent onset (≥ 10 years old).

Results. Childhood-onset conduct disorder was associated with higher rates of ADHD and anxiety disorders, male gender, and perceived and total hostility scores than adolescent-onset conduct disorder. Adolescent-onset was associated with higher rates of PTSD, alcohol and substance use disorders, complex comorbidity (i.e., 6+ diagnoses lifetime), and female gender.

Conclusions. Understanding age-of-onset-related patterns of comorbidity may facilitate psychiatric treatment planning in children and adolescents with conduct disorder.

Keywords Conduct disorder, Child, Adolescent, Comorbidity

Conduct disorder (CD) is common in children and adolescents referred for mental health services with rates up to 50% in some clinical settings (1,2), and is associated with a poor prognosis, especially with symptom onset before 10 years of age (3). Persistence rates of the disorder are generally high, with 50% to 80% of boys meeting criteria for conduct disorder at time 1 continuing to retain the diagnosis 3 to 4 years later (4,5). Children with CD have a high risk for the eventual development of antisocial personality disorder in adulthood (5–7). The annual costs to society of conduct disorder and associated service use are extremely high, up to six times the rate for non-conduct disorder youths (8–10). Of concern, recent research documents a rising rate of conduct disorder in the population, especially for females (11,12). For these reasons CD remains an important area of clinical research in child and adolescent psychiatry.

CD may differ in etiology, course, and comorbidity depending upon age of onset (3). Early onset (< 10 years old) CD appears to be associated with a greater prevalence of aggressive behavior (13). In the DSM-IV field trials sample, youths with early onset CD were 8.7 times more likely to exhibit at least one aggressive behavior than were youths with later onset CD (13). Some studies have found that average age of onset of CD is earlier for boys (14,15). Other researchers have failed to find a relationship between age of onset and gender (13).

In contrast, other youths do not develop CD until adolescence. Adolescent-onset youths with CD have a more time-limited and generally benign course of disorder and prognosis, with a higher prevalence of females than childhood-onset conduct disorder (3). These differences are reflected in the current DSM-IV subtypes of childhood-onset and adolescent-onset CD (13,16,17).

Historically, there has been discouragement about whether clinicians would ever be able to effectively treat youngsters with CD (18). More recently, this attitude has changed to one of cautious encouragement as research in psychosocial treatments (1,19–22), and psychopharmacological interventions (1,23–28)

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document some success in the treatment of early onset aggression, antisocial problems, and conduct disorder. However, children with CD remain difficult to treat quickly, efficiently, and effectively in clinical settings.

One issue that may contribute to treatment difficulty in conduct disorder is the problem of recognizing patterns of psychiatric diagnostic comorbidity and symptom heterogeneity in children presenting for clinical treatment with CD. In the clinical setting, children with CD frequently have multiple co-morbid psychiatric diagnoses including ADHD (29,30), depression (31), bipolar disorder (32), anxiety disorders (33), and substance use disorders (SUDs) (34). Unrecognized diagnostic comorbidity in CD may confuse and obfuscate effective treatment planning for antisocial youths in the clinical setting.

Based on the reported differences between childhood onset and adolescent onset CD as well as high rates of overall diagnostic comorbidity in youngsters with CD, we hypothesize that patterns of diagnostic comorbidity will differ between DSM-IV CD subtypes. Childhood-onset CD will be associated with both internalizing and externalizing disorder comorbidity and elevated levels of hostility, while adolescent-onset CD will be associated with a delinquent lifestyle (e.g., substance use disorders) and complex trauma-related comorbidity (e.g., PTSD). Our overall aim is to determine if CD is associated with psychiatric comorbidity, as suggested by prior studies, and to further clarify this relationship by examining differential patterns of comorbidity based on reported age of onset of CD. The specific aims of our study are (1) to identify comorbid psychiatric diagnoses associated with conduct disorder in an independent clinical sample of children and adolescents not previously reported on and compare them across early onset and late onset conduct disorder, and (2) to identify comorbid psychopathology associated with conduct disorder in an independent clinical sample of children and adolescents not previously reported on and compare them across early onset and late onset conduct disorder. The findings may help to clarify foci for psychiatric assessment and treatment planning for youths with CD.

METHODS

Subjects

Patients were accrued by consecutive referral and case ascertainment to a university based child and adolescent psychiatry clinic and were unselected for any characteristic or psychiatric disorder. Patients were assessed between 1996 and 2005. Consecutively referred children and adolescents (referred to as children) ($N = 280$), aged 4–17 years old, were systematically evaluated. The sample for analysis included 53 children meeting DSM-IV criteria for conduct disorder (CD) at the time of clinical referral. The sample included 6 females and 47 males. Ethnicity of the 53 children was 46 Caucasian, 2 African American, 3 Latino, and 2 other. With regard to family characteristics, 40 children were living with their biological

mother and 26 were living with their biological father. Mother's mean number of years of education was 13.7 ± 2.2 years; fathers' mean number of years of education was 13.7 ± 2.1 years. For yearly family income, 10 families earned less than \$20,000, 6 families earned \$30,000–\$39,000, 9 families earned \$40,000–\$49,000, 8 families earned \$50,000–\$75,000, and 10 families earned more than \$75,000. In terms of the child's current family climate, severe parental marital tensions were reported for 22 children. For 14 children, parents were reported to "almost never" agree on child management issues in the home.

When comparing the entire database of 280 children with the subsample of 53 children with CD, no significant differences emerged in age, ethnicity, maternal education, or paternal education. There was a significant difference in gender, with significantly more males in the early onset CD group than the total sample ($P = 0.002$), and a significant difference in family income, with the CD group coming from families with significantly lower income than the sample as a whole ($p = 0.002$).

Procedures

To investigate patterns of comorbidity by DSM-IV CD subtype, at clinical assessment the sample was divided by maternal-report age-of-onset into a childhood-onset type with symptoms beginning < 10 years old and an adolescent-onset type with symptoms starting ≥ 10 years old according to DSM-IV (17). Groups were compared on psychiatric diagnoses, psychopathology, and social variables. Parents and legal guardians provided clinical consent and children assented to all evaluation procedures. The University Institutional Review Board approved the study.

Diagnostic Interview

Children and parents were assessed (about the child) using clinical and structured diagnostic interviewing by five board-eligible or board-certified child and adolescent psychiatrists. Structured interviewing utilized the KSADS-Epidemiological Version 5 for lifetime diagnoses (35). Interrater reliability on a subsample of the entire database ($N = 53$) as assessed by the Kappa statistic (36) was $> .75$ for all psychiatric diagnoses indicating excellent interrater agreement, as previously reported (37). Age-of-onset of CD was established retrospectively by maternal report on KSADS interview.

Aggression and Hostility

The Modified Overt Aggression Scale (MOAS) (38,39) is a 20-item scale that assessed the frequency and severity of overt aggression during the previous month, and is commonly used to rate aggression in clinical settings. Parents completed the MOAS. The MOAS assesses four categories of aggression including Verbal Aggression (threats of harm to others),

Objective Aggression (impulsive property destruction), Self-Aggression (self-injurious behaviors), and Other Aggression (physical assault). The Buss Durkee Hostility Inventory (child version) was used to assess child self-report hostility. This scale yields a total score, an Expressed Hostility factor and an Experienced Hostility factor. The convergent and discriminant validity of this scale is adequate (40,41).

Symptom Severity and Impairment in Functioning

Child psychiatrists rated symptom severity using the Clinical Global Impressions Severity Scale (CGI-S) (42), and daily functioning using the Clinical Global Assessment Scale (CGAS), a reliable measure of daily functioning and impairment (43,44). Agreement on this scale was measured with the intraclass correlation coefficient (45) between child psychiatrists for 50 children. Agreement on this measure was good to excellent (ICC = 0.74).

Social Variables

Parents reported on parental education (highest grade achieved for both mother and father), family income, parent-child management agreement between spouses, the presence of marital tension in the household, and family structure (presence/absence of a father in the household). Family income was coded categorically as (1) ≤ \$19,000, (2) \$20,000 to \$29,000, (3) \$30,000 to \$39,000, (4) \$40,000 to \$49,000, (5) \$50,000 to \$75,000, and (6) > \$75,000. Parents rated household marital tension as present/absent. Parents rated their level of agreement on child management on a 3-point scale (almost never agree, sometimes agree, often agree).

Parental Psychopathology

Parents (generally mothers) were queried about psychopathology in themselves and in their spouses using an unstructured

clinical interview. Questions included type and severity of psychiatric symptoms, dates of illness onset and offset, recurrence of illness, impairment, treatment, past medication and psychosocial therapies, and history of previous diagnoses. All parental diagnoses met DSM-IV criteria for disorder (lifetime).

Statistical Analysis

Statistical analysis was by chi-square for categorical data and by *t*-test for continuous variables comparing the two CD groups. For comparison with non-CD children from the entire clinic database with the two CD groups ANOVA with post-hoc Neuman-Kuels tests were used. Significance was set a *p* ≤ .05 (two-sided).

RESULTS

Conduct disorder was present in 53 of 280 (18.9%) consecutive clinic referrals. All children meeting diagnostic criteria for CD also met diagnostic criteria for at least one other psychiatric disorder. High rates of comorbid depression, bipolar disorder, anxiety disorders, ADHD, and substance use disorders (SUDs) were found in the sample (see Table 1).

For the purposes of comparison, the sample was stratified by maternal-report early-age-of-onset CD (mean age onset 5.3 ± 2.6 years; *N* = 40) and adolescent-onset CD (mean age onset 12.0 ± 0.9 years; *N* = 13). Cases were stratified as early onset or adolescent-onset regardless of the age patients presented to the clinic.

Significant differences in diagnostic comorbidity emerged when comparing the two groups (see Table 1). Patients with childhood-onset CD had significantly higher rates of ADHD ($\chi^2_{[1]} 12.6, p = .002$) and non-PTSD anxiety disorders ($\chi^2_{[1]} 8.0, p = .012$) than patients with adolescent-onset CD. Patients with adolescent-onset CD had significantly higher rates of PTSD ($\chi^2_{[1]} 6.5, p = .03$), alcohol use disorders ($\chi^2_{[1]} 9.2, p = .01$), and drug use disorders ($\chi^2_{[1]} 13.8, p = .0001$). The two children with PTSD had experienced chronic physical abuse and witnessing

Table 1 Conduct Disorder by Maternal Report Age-of-Onset and Diagnostic Comorbidity in Referred Children and Adolescents Assessed at the Time of Clinic Referral (N = 53)

Variable	Childhood-Onset CD (N = 40) (Mean, SD or N, %)	Adolescent-Onset CD (N = 13) (Mean, SD or N, %)	Test Value (df)	P Value
Mean Age of Onset (Maternal Report)	5.3 ± 2.6 yrs	12.0 ± 0.9 yrs	<i>t</i> = 13.7 (50,69)	< 0.001
Female Gender	2 (5)	4 (31)	$\chi^2 = 6.5 (1)$	0.03
Depressive Disorder	17 (43)	7 (54)	$\chi^2 = 0.5 (1)$	NS
Bipolar Disorder	9 (23)	4 (31)	$\chi^2 = 0.4 (1)$	NS
Anxiety Disorders	32 (80)	5 (38)	$\chi^2 = 8.0 (1)$	0.012
PTSD	2 (5)	4 (31)	$\chi^2 = 6.5 (1)$	0.03
ADHD	39 (98)	8 (6)	$\chi^2 = 12.6 (1)$	0.002
Alcohol Use Disorders	1 (3)	7 (54)	$\chi^2 = 9.2 (1)$	0.01
Drug Use Disorders	3 (8)	7 (54)	$\chi^2 = 13.8 (1)$	0.001
Cigarette Use	2 (5)	3 (23)	$\chi^2 = 3.8 (1)$	0.09
Total Number of Diagnoses ≥ 6	1 (3)	4 (31)	$\chi^2 = 14.8 (5)$	0.01

of marital violence in the home. The four adolescents with PTSD had all experienced chronic physical and sexual abuse. The diagnoses of PTSD satisfied DSM-IV criteria as assessed by the KSADS structured diagnostic interview. There existed a trend for adolescent-onset CD to be correlated with cigarette use ($\chi^2_{[1]} 3.8, p = .09$). Significantly more adolescent-onset CD patients met criteria for complex psychopathology with 6 or more comorbid psychiatric diagnoses compared with childhood-onset CD ($\chi^2_{[5]} 14.8, p = .01$). A significantly higher percentage of females were found in the adolescent-onset CD group compared with the early onset CD group (5% of 40 patients versus 31% of 13 patients; $\chi^2_{[1]} 6.5, p = .03$).

To compare the average number of comorbid psychiatric diagnoses, the early onset group was compared with the adolescent-onset group, and then with all 228 non-conduct-disordered children in the complete clinic database ($N = 280$). The average number of comorbid diagnoses for the early onset group was 2.63 ± 0.87 , for the adolescent-onset group was 3.23 ± 1.59 , and for the non-CD comparison children was $2.16 \pm .99$. No significant differences emerged comparing the number of comorbid psychiatric disorders between the early and adolescent-onset CD groups. When compared with the non-CD children, a significant difference emerged in the number of comorbid psychiatric diagnoses ($F_{[2,278]} 9.81, p = .000$). Post-hoc analysis using the Newman-Keuls test revealed that adolescent-onset CD patients, but not childhood-onset CD patients, had significantly more comorbid diagnoses than non-CD comparison children.

Table 2 presents comorbid psychopathology across the two groups. As measured by child psychiatrist-assessed CGI-Severity scores at clinic evaluation, high rates of symptom severity were found for both groups (early onset 5.2 ± 0.7 versus adolescent-onset 5.2 ± 0.6) that did not differ by maternal-reported CD age of onset ($p = \text{NS}$). Both groups had marked impairment in functioning as rated by the CGAS (early onset 44.7 ± 5.8 versus adolescent-onset 44.1 ± 3.6) that did not differ across groups ($p = \text{NS}$). High rates of aggression were reported in both groups but scores did not differ by reported age-of-onset (see Table 2). Childhood-onset CD patients reported significantly higher total hostility ($t_{[41]} 2.35, p = .024$) and perceived hostility scores ($t_{[41]} 2.25, p = .03$), but not expressed hostility compared with adolescent-onset CD.

Concerning social variables, there were no significant differences between the early onset and adolescent-onset groups for mother's education ($F_{[1,42]} 2.68, p = \text{NS}$), father's education ($F_{[1,37]} 0.20, p = \text{NS}$), family income ($\chi^2_{[6]} 4.7, p = \text{NS}$), parental marital tension ($\chi^2_{[1]} 0.20, p = \text{NS}$), or parental agreement about child management issues ($\chi^2_{[3]} 1.53, p = \text{NS}$). With regard to male authority figures in the home, 50% of early onset CD children had a father at home and 50% of late onset CD children also had a father at home. Information concerning parental history of psychopathology for the total sample is presented in Table 3. No significant differences emerged across the two groups assessing variables measuring the prevalence of parental psychopathology.

DISCUSSION

High rates of diagnostic comorbidity were found in our sample of children with conduct disorder. Considering depression, about 2% of non-referred children and about 6% of non-referred adolescents residing in the community meet criteria for MDD (46–52). In our sample rates of MDD were 43% for reported early onset and 54% for reported adolescent-onset conduct disorder. Considering bipolar disorder, among adolescents in the recently reported Indigenous Hawaiian Behavioral Health study 1.4% met criteria for bipolar disorder (53), while in our clinically referred conduct disorder sample 23% to 31% of children and adolescents met criteria for bipolar disorder. Similarly, a high rate of ADHD comorbidity has repeatedly been demonstrated in children with conduct disorder (30,54–56), and our results are consistent with this association across both maternal-report early onset and adolescent-onset conduct disorder. High rates of non-PTSD anxiety disorders, PTSD, and SUD were also found in our CD sample. Taken together, these data support previous research suggesting that conduct disorder is highly heterogeneous in clinical samples and frequently comorbid with other psychiatric disorders (29,32).

Our sample was compared by stratifying children into an early onset and an adolescent-onset subtype of CD consistent with DSM-IV subtype, assessed by retrospective maternal report at the time of clinical evaluation and investigating diagnostic comorbidity. When stratified in this manner, significant differences emerged in associated comorbid psychiatric

Table 2 Conduct Disorder by Maternal Report Age-of-Onset and Comorbid Psychopathology in Referred Children and Adolescents Assessed at the Time of Clinic Referral ($N = 53$)

Variable	Childhood-Onset CD ($N = 40$) (Mean, SD or N, %)	Adolescent-Onset CD ($N = 13$) (Mean, SD or N, %)	Test Value (<i>df</i>)	<i>P</i> Value
CGI-Severity	5.2 ± 0.7	5.2 ± 0.6	$t = 0.22 (49)$	NS
CGAS	44.7 ± 5.8	44.1 ± 3.6	$t = 0.39 (50)$	NS
Total Hostility	8.26 ± 1.8	6.50 ± 1.4	$t = 2.35 (41)$	0.024
Perceived Hostility	3.29 ± 1.0	2.42 ± 1.4	$t = 2.25 (41)$	0.03
Expressed Hostility	4.97 ± 1.4	4.25 ± 1.8	$t = 1.4 (41)$	NS
Overt Aggression	58.5 ± 36.6	51.0 ± 36.1	$t = 0.59 (43)$	NS

Table 3 Frequency of Positive Parental History of Psychopathology for Children and Adolescents Diagnosed with Conduct Disorder

	Mother	Father
Conduct Disorder	14%	30%
ADHD	14%	40%
Antisocial Personality Disorder	10%	32%
Alcohol Abuse	22%	44%
Substance Abuse	22%	34%
Bipolar Disorder	10%	6%
Major Depressive Disorder	40%	14%

diagnoses. The early onset CD group had significantly higher rates of ADHD and non-PTSD anxiety disorders than the adolescent-onset group regardless of the age at which the child was evaluated in the clinic. The adolescent-onset CD group had significantly higher rates of complex psychopathology, PTSD and SUDs than the early onset CD group. There were no differences in social variables or rates of parental psychopathology found across the two groups that might further account for these differences in patterns of diagnostic comorbidity.

ADHD is the comorbid condition most commonly associated with conduct problems and conduct disorder, especially for children with early onset conduct problems (57). ADHD is thought to precede the development of CD in most cases and many investigators consider the impulsive-hyperactive domain of ADHD to be the motor that drives the onset of CD in children (3,58). The presence of comorbid ADHD and CD usually signals a more severe form of CD with a chronic course and poor outcome (3,58). Importantly, treatment of ADHD, even in the presence of CD, is effective for ADHD symptoms (59), and for aggressive CD symptoms (24,60).

We report high rates of comorbid non-PTSD anxiety disorders, especially in childhood-onset CD, in a sample associated with severe symptoms and marked impairment in daily functioning. Anxiety disorders and problems co-occur in children with CD at rates much higher than chance (58,61). In a majority of cases CD is thought to precede anxiety problems and the development of anxiety disorders. The co-occurrence of anxiety disorders and CD may be especially likely for girls rather than boys (62). The effects of comorbid anxiety on CD severity are presently unclear. Some studies find CD youths with comorbid anxiety are less impaired than those with CD alone (33), and in other studies the presence of anxiety has been reported to increase the severity of conduct problems (63). Other studies find that a low level of comorbid anxiety in some children with CD may be a sign of increased severity of conduct problems (64). In the assessment of anxiety and CD it is important to distinguish between fear, which decreases conduct problems, and anxiety as a negative affect that may be a consequence of the child's behavioral problems and subsequent stress (64).

Although the early onset and adolescent-onset CD groups did not differ in rates of major affective disorder, high rates of depression and bipolar disorder were found in the sample. Our data support previous research suggesting that MDD is often

found in children (31,65), and adolescents (50,66) with CD. Similarly, high rates of bipolar disorder are reported in youths with CD (32,67,68). In general, depression and bipolar disorder are thought to precede the development of CD (31,69). It remains to be seen if CD plus MDD or bipolar disorder represents a distinct subtype of early onset major affective disorder with a distinct family history, prognosis, course of illness, and treatment (70). It is presently unclear if treatment of MDD or bipolar disorder in children with both diagnoses will also alleviate symptoms of aggression and CD. However, the effectiveness of mood stabilizers on symptoms of both early onset bipolar disorder (71), and conduct disorder (25), as well as the effectiveness of SSRIs on symptoms of depression, irritability, and aggression (72,73) in youths with MDD offers hope that effective treatments for youngsters with CD and comorbid depression and/or bipolar disorder will be found.

An association between CD and adolescent alcohol and SUD is well known (74,75), and results from our study are consistent with previous research (76). The relationship between PTSD and CD was significant for adolescent-onset CD in our sample. Correlations between PTSD and CD in children and adolescents have previously been reported (77–79), as well as between CD in childhood and subsequent PTSD in adulthood (80). These associations may be mediated by increased aggression, hostility, and arousal in traumatized individuals which leads them to express conduct disordered behaviors (81), or the fact that a conduct disordered lifestyle increases the chance of being traumatized (80).

Self-reported hostile attributions were significantly associated with early onset CD in our study. Children with childhood onset CD and high rates of comorbid ADHD are frequently in conflict with persons in their environment and possess a poor ability to modulate negative affects (82). As a result, children with early onset conduct problems may develop persistent hostile attributional biases which can result in elevated rates of self-reported hostility and aggression (83–85).

There are several limitations to our study that must be considered. Conduct disorder age-of-onset was established retrospectively by maternal report at the time of clinic evaluation, increasing the possibility of recall bias. Our sample size was small leading to diminished statistical power to detect differences across groups. The study was completed in a university child and adolescent psychiatry clinic, which may have caused a bias for referrals with more severe psychopathology than one would observe in other clinical settings. The sample was mostly Caucasian. Results may be different for different ethnic groups. Despite these limitations we were able to demonstrate several significant differences in comorbidity across maternal-report early onset and adolescent-onset CD groups.

CONCLUSIONS

Clinicians who assess and treat pediatric mental health disorders need to be aware of the heterogeneity inherent in the

diagnosis of conduct disorder in clinically referred children and adolescents. CD is common in clinically referred children and adolescents and presents with high rates of diagnostic comorbidity and psychopathology. In our sample, patterns of comorbidity differ by maternal-report age of onset of CD, with childhood-onset CD presenting with higher rates of ADHD, anxiety disorders, and hostile perceptions, and adolescent-onset CD presenting with higher rates of complex psychopathology, PTSD and SUDs at the time of clinical evaluation. Understanding patterns of comorbidity may facilitate treatment planning in children and adolescents with conduct disorder. Clinicians treating youngsters with CD should routinely and systematically assess for comorbidity.

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