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A Linear Growth Curve Analysis of Inpatient Treatment Response by Conduct-Disordered, ADHD, and Comorbid Preadolescents

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Conduct disorder (CD) comorbid with attention deficit hyperactivity disorder (ADHD) is widely reputed to be treatment refractory, particularly when accompanied by aggression and early-onset symptoms. Few studies, however, have assessed inpatient treatment response among early onset CD/ADHD children in detail. In the present investigation, behavioral and rating scale data were compared among CD (n = 13), ADHD (n = 20), and CD/ADHD (n = 45) preadolescents during one-month of multimodal inpatient treatment that included methylphenidate administration. As expected, linear growth curve analyses revealed that CD/ADHD children were the most symptomatic of the three groups. However, all groups benefited from hospitalization, with few differences in treatment responsiveness observed. Analyses of residualized symptoms suggested that methylphenidate administration was effective in curbing impulsive but not aggressive behaviors. Aggr. Behav. 29: 440–456, 2003. © 2003 Wiley-Liss, Inc.

Key words: conduct disorder; ADHD; comorbidity; inpatient treatment; methylphenidate

Much has been written in recent years about subtypes of Conduct Disorder (CD), and about heterogeneities in etiology, behavioral expression, and symptom recalcitrance across different CD groups. In large part, these writings emerged following the publication of the third edition of the Diagnostic and Statistical Manual of Mental Disorders [DSM-III; American Psychiatric Association, 1980], which specified an axial format of subtyping CD. According to this system, CD probands were placed into one of four quadrants based on their relative standing on two presumably orthogonal dimensions, socialization and aggression. Thus, a given CD case could be classified as socialized with or without aggression, or as undersocialized with or without aggression. Although a comprehensive review of the literature regarding this classification system will not be presented here, considerable evidence suggests that undersocialized aggressive (UACD) probands represented a diagnostically

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distinct subgroup, characterized by a lack of empathy, frequent use of instrumental aggression [see Quay, 1993, 1986], inability to form meaningful social relationships [Henn et al., 1980], autonomic nervous system dysfunction [see Beauchaine, 2001; Quay, 1993], excessive impulsivity [Daugherty and Quay, 1991; Shapiro et al., 1988], and increased risk for future antisocial behavior and psychopathy [Huesmann et al., 1984]. Moreover, evidence for neuropsychological dysfunction has been reported in children and adolescents selected specifically for aggressive symptoms [Séguin et al., 1995].

In the DSM-III-R [American Psychiatric Association, 1987], a slightly modified system of diagnosing CD emerged in which probands were classified into either solitary aggressive, group, or undifferentiated subtypes. Because the solitary aggressive subtype mapped almost directly onto the former UACD grouping, the new classification system maintained the practice of distinguishing particularly recalcitrant probands from others based on the presence or absence of aggression and socialization deficits. With the arrival of the DSM-IV, however, an alternative approach to subtyping CD emerged in which differences in etiological presentation displaced aggression and socialization as the primary determinant of subtype status [American Psychiatric Association, 1994]. According to this system, CD probands are categorized into childhood onset or adolescent onset groups based on whether criterion behaviors emerge prior to or after age 10.

Although this change represented a shift away from the validated UACD category [see Beauchaine et al., 2001], a reliance on etiological information in diagnosis typically marks progression in the evolution of a nosologic system, and reflects increased understating of the course of a disorder [see Feighner et al., 1972; Kendell, 1989; Robins and Guze 1970]. Thus, etiology-based diagnosis should serve to enhance the validity of a diagnostic entity by more efficiently "carving nature at its joints." In the case of CD, the shift toward age of onset as an indicator of diagnostic subtype followed evidence that differences in the course of symptom expression were of considerable prognostic value [Loeber, 1982; 1988; Moffitt, 1991; 1993; Robins, 1966].

A corpus of literature now supports the childhood onset/adolescent onset distinction. Compared with the adolescent onset subtype, childhood onset symptoms are associated with higher rates of oppositionality and aggression [Lahey et al., 1994; McGee et al., 1992; Moffitt, 1990], higher levels of adult violence and criminality [Henn et al., 1980; Robins, 1966; Stattin and Magnusson, 1989], and increased rates of antisocial behavior in first degree relatives [Lahey et al., 1998]. Childhood onset CD is also characterized by a poor prognosis [see Werry, 1997], and is likely to be accompanied by a multitude of co-occurring symptoms, including ADHD, lack of remorse [Loeber et al., 1998], neuropsychological dysfunction [see Moffitt, 1993; Speltz et al., 1999], and increased risk for future substance dependences [Myers et al.,1995]. These phenotypic/behavioral indicators map closely onto those described above for UACD, which has led some to suggest that the DSM-III and DSM-IV diagnostic systems identify quite similar sets of CD groups [Lahey and Loeber, 1994]. Thus, both systems of classification may be valid diagnostically.

A third approach to subtyping CD is to classify cases into groups who do and do not exhibit comorbid ADHD symptoms. In an extensive literature review, Lynam [1996] noted that CD children who exhibit comorbid hyperactivity and impulsivity are at greatest risk for chronic delinquency and recidivism, and suggested that such symptom co-occurrence may be a premorbid indicator of adult psychopathy. In a follow-up study, he demonstrated that adolescents exhibiting comorbid CD and hyperactivity-impulsivity scored higher

on the Psychopathy Checklist – Revised [Hare, 1991] than did those with one set of symptoms or the other [Lynam, 1998]. The comorbid group was also more impulsive, more antisocial, and more impaired based on measures of neuropsychological functioning. Moreover, evidence suggests that CD coupled with ADHD may be characterized by a particularly strong genetic loading [Edelbrock et al., 1995; Faraone et al., 1997]. Thus, considerable support exists for the validity of subtyping CD based on the presence or absence of ADHD symptoms. Once again, however, doing so may be identifying a group that overlaps substantially with the UACD and childhood onset subtypes. Indeed, childhood onset CD symptoms are typically preceded by hyperactivity and impulsivity [Loeber and Keenan, 1994; Loeber et al., 1995; Patterson et al., 2000], with behavioral disinhibition playing a central role in the aggression exhibited by group members [see Walker et al., 1987]. Thus, all three of the discussed CD subtypes are characterized by excessive delinquency, aggression, behavioral disinhibition, neuropsychological dysfunction, and symptom recalcitrance compared with their less severe CD counterparts.

The symptom persistence and severity that marks these groups is especially likely among clinical populations, where comorbid ADHD is observed in the majority of CD cases [Klein et al., 1997; Stewart et al., 1981]. As we have noted elsewhere [Beauchaine et al., 2000], the proportion of treatment refractory cases is likely to be higher still at inpatient settings, as referrals typically follow trials of less restrictive interventions that are of limited utility for severe CD [Offord and Bennet, 1994; Werry, 1997]. Thus, inpatient populations are skewed systematically toward high severity and poor prognosis. Given this, it is somewhat surprising that symptom responsiveness among inpatient CD subtypes has not been assessed directly. The primary purpose of the present investigation was to conduct such an assessment with hospitalized preadolescent males who presented with severe CD, and to contrast their symptom course with those of less severe externalizing subgroups. Thus, symptom profiles during a one month inpatient stay were compared between groups of ADHD, CD, and CD/ADHD probands. Given the literature reviewed above, it was hypothesized that the CD/ADHD group would exhibit greater symptom severity and less symptom improvement during treatment than either the CD or ADHD groups.

A secondary objective was to examine the impact of methylphenidate treatment on symptom improvement among participants with both CD and ADHD. This was of interest due to conflicting reports regarding the efficacy of psychostimulants in treating aggressive behaviors among comorbid children. While it is clear that methylphenidate attenuates symptoms of hyperactivity in such cases [e.g., Gadow et al., 1990; Klein et al., 1997], evidence for attenuation of aggressive symptoms is mixed. Although several authors have reported significant symptom improvement [e.g., Gadow et al., 1990; Kaplan et al., 1990; Klein et al., 1997; Murphy et al., 1992], close scrutiny of these reports reveals that effects were inconsistent across contexts and measures. Thus, while Gadow et al. employed eight observational measures of aggression across three contexts (classroom, lunchroom, recess), only one *F*-statistic was significant. Similarly, although Klein et al. reported significant reductions in conduct problems in general, improvement was not observed on several of the measures of aggression. Moreover, only the Kaplan et al. study was conducted with an inpatient sample, and no medication effects were observed for physical aggression. Similar results were obtained by Kolko et al., [1999], who reported no significant medication

effects on overt aggression in patients enrolled in a partial hospitalization program. Thus, evidence for the efficacy of methylphenidate in reducing aggressive symptoms among the most severe CD populations is lacking. Relations between methylphenidate administration and symptom improvement were therefore also examined among CD/ADHD participants. It was hypothesized that any improvement following stimulant treatment would be stronger for symptoms of impulsivity than for symptoms of aggression.

METHOD

Participants

Following Institutional Review Board approval, charts of children admitted to an inpatient child psychiatry unit between 1987 and 1994 were examined. These children were referred to the inpatient facility by staff members of county clinics and social service agencies, and by private providers. Because few females met the inclusion criteria outlined below ($n = 9$), their data were omitted from all analyses due to the insufficient sample size for examining group effects and group $\times$ sex interactions. Male patients were selected who were age 12 or under at admission (range = 4.1 to 12.0), who remained on the inpatient unit for a minimum of 28 days, and who met DSM-III-R criteria for ADHD, aggressive CD, or both disorders. Age 12 was used as a cutoff because we were interested particularly in early-onset cases. Although the diagnosis of the childhood onset CD subtype requires engagement in criterion behaviors prior to age 10, the symptom severity of children presenting for inpatient admission at age 12 is likely to be associated with an earlier age of onset. Since potential participants were drawn from a child inpatient facility, few were rejected from the study based on this age criterion.

Current diagnoses were rendered within the first week of treatment through consensus of the attending psychiatrist and unit psychologist using the Child Symptom Inventory [Grayson and Carlson, 1991], a validated checklist of DSM-III-R symptoms. Sensitivity and specificity of both the CD and ADHD scales are adequate to excellent [Gadow and Sprafkin, 1997]. In total, 13 males met criteria for CD only, 24 met criteria for ADHD only, and 58 met criteria for both disorders. Sources of information used to assess symptoms included behavioral observation during the first week of stay, interviews with parents, and relevant items from two validated rating scales completed daily by unit nurses and school teachers. These included the Teacher Self Control Rating Scale [TSCRS; Humphrey, 1982], which assesses impulsivity, and the Attention Deficit Disorder-Hyperactivity Comprehensive Teacher Rating Scale [ACTeRS; Ullmann et al., 1984], which assesses attention problems and hyperactivity.

We were interested in examining the effects of methylphenidate dosage on symptom expression, so potential participants who were administered dextroamphetamine or pemoline were dropped from the ADHD and CD/ADHD groups. This resulted in a loss of four potential ADHD and 13 potential CD/ADHD participants. None of the CD probands received a stimulant, so no participants were lost in this group either. Mean ages were 8.28 ($SD = 1.64$) for the ADHD group, 8.78 ($SD = 2.33$) for the CD group, and 8.99 ($SD = 1.91$) for the CD/ADHD group. Group differences in age were not significant ($F_{(2,75)} = .95$, $p = .39$).

Table I. Mean Methylphenidate Dosages (SDs) Administered Across the Treatment Period by Group[1]

	Week			
Group	1	2	3	4
ADHD ($n = 20$)	.07 (.16)	.27 (.21)	.38 (.25)	.36 (.24)
CD/ADHD ($n = 45$)	.05 (.15)	.14 (.23)	.21 (.25)	.22 (.23)
CD ($n = 13$)	—	—	—	—

[1]No members of the CD group received methylphenidate at any point during treatment. Most changes in dosage were effected in 5mg. increments, which were converted to mg./kg./dose in the presented table.

Stimulant Treatment

Upon admission to the inpatient unit, ongoing stimulant treatment was discontinued for all participants in order to observe native symptom levels, and to assess responsiveness to the behavioral management plan. As noted above, all CD probands remained medication-free throughout the treatment period. Most ADHD (16/20) and CD/ADHD (37/45) participants also remained unmedicated for the first week of admission. Thereafter, all were treated with methylphenidate. In general, dosage increases were prescribed in 5–10mg. increments by the unit psychiatrist following weekly meetings of the interdisciplinary treatment team, where feedback regarding patient progress was provided by unit nurses, teachers, psychologists, residents, and interns. Dosages were administered twice daily, once in the early morning and once at about noon. Records of weekly dosage changes were available for use in predicting behavioral change. Methylphenidate dosages administered across the treatment period are presented in Table I.

Psychosocial Treatment

Each participant received the standard multimodal treatment offered on the inpatient unit, including (a) six hours of school each weekday led by state certified teachers and teacher assistants, at a 5:1:1 ratio (children:teacher:teacher aid); (b) one hour of recreational therapy twice per week led by a unit psychologist; (c) one hour of gym time twice per week including of a variety of team sports; (d) one hour of group social skills training once per week led by unit nurses or clinical psychology interns, including instruction on making friends, initiating conversations, problem solving in conflict situations, recognizing anger cues, and developing effecting coping strategies for anger; (e) one hour of individualized cognitive behavioral therapy led by a unit psychologist; (f) one hour of parent training 1–2 times per week led by unit nurses, focused on altering behavioral contingencies within the home (depending on parent availability); and (g) a one-hour family meeting each week led by a psychiatry (attending or resident) or psychology (Ph.D. or intern) staff member, addressing issues of conflict among family members. Child and parent sessions followed closely the protocol employed by Frankel and colleagues [see Frankel et al., 1995, 1996].

MEASURES

Behavioral measures. Weekly counts of several behaviors were extracted directly from the standardized behavioral management plan (BMP) in effect on the inpatient unit. The

BMP is a response-cost contingency system in which children earn points for prescribed behaviors and lose points for proscribed behaviors. Daily tallies of point gains and losses were used to derive the number of discrete incidents of behavioral transgression within several BMP categories. Included were episodes of (a) disobedience, or refusal to comply with staff-initiated directives; (b) destruction, or inflicting damage to one's own, others', or community property; (c) physical aggression directed toward staff or peers; (d) verbal provocation of staff or peers; and (e) time-outs issued for failure to comply with staff requests following a warning. In addition, participants were subjected to a seclusion procedure for aggressive acts deemed by staff to be so dangerous as to warrant solitary confinement, and for refusal to comply with staff-initiated time-outs. Counts of seclusions thus served as an index of particularly serious aggression and defiance. Finally, compliance with the BMP was assessed via weekly counts of (a) daily living points, which were accumulated by participants for completing required tasks successfully (e.g., getting out of bed in the morning, attending school on time, eating in the cafeteria without disruptive behavior); and (b) daily goal points, which were accumulated by participants for engaging in individualized target behaviors that were identified by the multidisciplinary treatment team as areas needing improvement (e.g., keeping hands to oneself, participating actively in group activities, etc.).

Ratings scales. Several rating scales were completed weekly by staff and were thus available for assessing behavioral change during the treatment period. Impulsivity was assessed by having unit teachers and nurses complete the Teachers Self Control Rating Scale [TSCRS; Humphrey, 1982] at the end of each week. The TSCRS is comprised of 15 statements related to impulsivity (e.g., anticipates the consequences of his/her actions, plans ahead what to do before acting), which are rated on 5-point Likert scales. Because high scores represent poor impulse control, reverse-coding was employed to render the direction of scores consistent with APRS (see below). Although the TSCRS includes both cognitive/personal and behavioral/interpersonal subscales, only full scale scores were available. However, as noted by the scale author, both subscales appear to represent the same domain of impulsive functioning [Humphrey, 1982]. In the validation sample, 2–1/2– to 3–week test-retest reliability of the full scale score was .94, and correlations with the school adjustment, acting-out/aggressive, and frustration tolerance subscales of the Child Behavior Rating Scale (Rochester Social Problem Solving Core Group) were .74, .81, and .78, respectively.

Additionally, unit teachers completed the Academic Performance Rating Scale [APRS; DuPaul et al., 1991] at the end of each week. The APRS is a 19–item instrument that includes academic success (e.g., how consistent is the quality of the child's academic work?), academic productivity (e.g., how often is the child able to pay attention without prompts?), and impulse control (e.g., how careless is the child in completing his or her work?) factors. Items are rated on 5–point Likert scales, with high scores representing better functioning. Because individual factor scores were unavailable for 33 participants, only the total score was employed. However, the three factors shared much common variance in the validation sample, with intercorrelations ranging from .59 to .88. Examination of the individual items suggests that all factors assess on-task behaviors and attentional capacity. Coefficients alpha ranged from .94 to .95. Correlations of the total score with national percentile rankings on the Comprehensive Test of Basic Skills [McGraw-Hill, 1982] were .48 for math achievement, .53 for reading achievement, and .53 for language achievement in the validation sample.

Statistical Analyses

Treatment response. Changes in functioning across the treatment period on all measures were assessed via linear growth curve analyses [Rogosa et al., 1982]. This approach avoids the inflation of Type I error associated with repeated measures analysis of variance [Hertzog and Rovine, 1985; Vasey and Thayer, 1987], and is more sensitive in detecting significant change than many alternative approaches [Speer and Greenbaum, 1995]. Least squares regression lines were calculated through scores on each index (e.g., point losses for aggression), at each measurement point (i.e., weeks 1–4), for each patient. Comparisons of intercepts were conducted using Analyses of Variance (ANOVAs), with significant effects suggesting group differences in functioning at intake.[1] In contrast, comparisons of slopes were conducted using Analyses of Covariance (ANCOVAs) in which the impact of intercepts on treatment response were controlled. Significant effects in these analyses suggest group differences in improvement or decline in functioning across the treatment period.

Impact of medication. Assessing the impact of methylphenidate treatment on symptom responsiveness was complicated by two issues. First, because the study was retrospective, no control group was employed. Although CD participants received no medication throughout the treatment period, they were the only group with sub-clinical ADHD symptoms, and thus varied systematically from the ADHD and CD/ADHD groups. Any group contrasts would therefore be confounded with ADHD diagnostic status. This is problematic given the research outlined earlier suggesting that stimulants may be more effective in treating ADHD symptoms than in treating CD symptoms. Second, because methylphenidate dosages across the treatment period were adjusted upward based on symptom recalcitrance, correlations between dosage growth and symptom growth were biased toward positive values for some variables (e.g., episodes of disobedience), and toward negative values for others (e.g., academic performance). Such systematic relations between symptom growth and dosage growth could result in apparent ineffectiveness of methylphenidate treatment in correlational analyses. For these reasons, rather than examining the impact of methylphenidate dose on symptom growth directly, we examined the impact of medication changes on symptom residuals, which were extracted from the growth curve models. Recall that individual growth curves were computed for each participant on each variable across the four weeks of hospitalization. As depicted in Figure 1, observed scores at any week will deviate from the individual regression equation. These deviations, or residuals, may be systematically influenced by changes in methylphenidate dosage. If medication increases are effective in reducing impulsivity, for example, weeks in which dosages are boosted should result in over-prediction by the regression equation vis-à-vis observed scores. Such over-prediction is associated with negative residuals, whereas under-prediction is associated with positive residuals. For any given case, examining symptom change in this fashion indexes improvement or decline in functioning against individual growth rather than against zero growth, thereby reducing the aforementioned biases against finding treatment effects. The impact of methylphenidate dosage changes on symptoms was assessed by conducting χ^2

[1]Interpretation of intercepts depends on the values used in coding a repeated measure. In the present study, weeks 1–4 were represented by the integers 1, 2, 3, and 4. By definition, intercepts are placed at zero and thus served as pre-treatment estimates of functioning on each index. This method of coding was chosen over the commonly used alternative of representing repeated measures with numbers beginning at zero because doing so would have rendered the intercepts as estimates of functioning after week 1 data were collected, thus following a potentially significant dose of multimodal treatment.

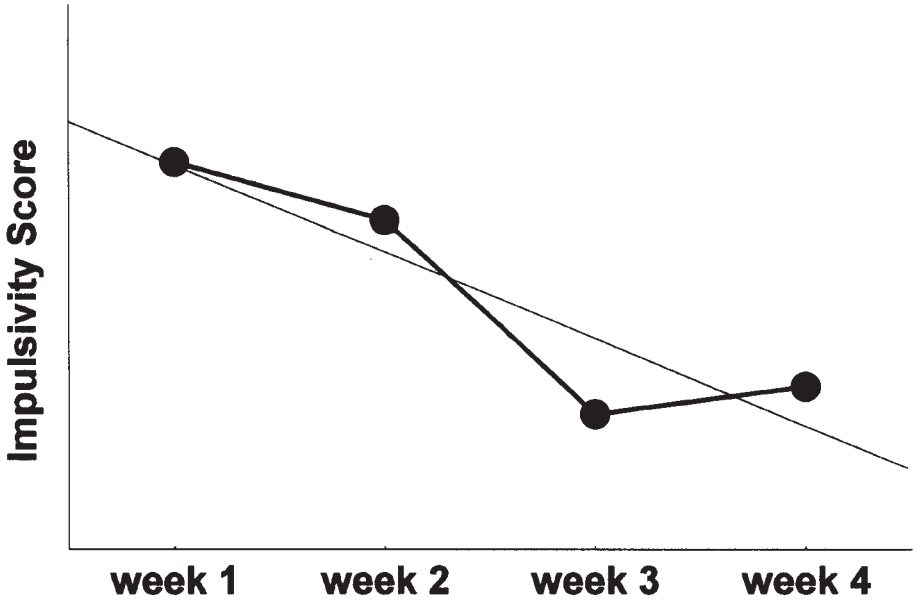

Fig. 1. Hypothetical example of an individual growth curve in which over-prediction by the regression equation occurs at week 3.

analyses on 2×2 tables in which contingencies between methylphenidate dosage (increased vs. static) and residualized symptoms (positive vs. negative) were examined[2]. Because we were interested specifically in aggressive and hyperactive symptom responsiveness among comorbid participants (following from the above discussion), the pure CD and ADHD groups were omitted from these analyses.[3] Week 1 data were also omitted, since 82% of the CD/ADHD participants received no medication until Week 2.

RESULTS

Results of the growth curve analyses are presented in Table II. For each variable, both intercept and slope parameters are reported. As indicated by the confidence intervals around the intercept values, significant sample-wide symptomatology was observed at intake on almost all measures. Thus, very few of the confidence intervals included zero. These findings are not surprising for an inpatient population. Of greater interest were group differences in

[2]Correlational analyses, which are more powerful than χ^2 analyses, were also conducted toward examining relations between the magnitude of dosage changes and residualized symptoms. Because these analyses did not detect any additional effects, the results of the χ^2 tests are presented here because of the appeal of odds ratios in describing differential dependencies among variables.

[3]Aside from our interest in response to methylphenidate treatment among CD/ADHD participants specifically, it was also important to exclude both the pure CD and ADHD groups from these analyses on conceptual grounds. Because detection of treatment effects depends on changes in dosage, pure CD participants could not be included because they received no medication. In the case of pure ADHD, differences in dosage vis-à-vis the CD/ADHD group would have introduced an interpretive confound into the analyses.

Table II. Results of Linear Growth Curve Analyses by Group[1]

	Parameter Estimate (95% CI)[a]			Diagnostic Status Effect[b]		
Variable	ADHD (n = 20)	CD(n = 13)	CD/ADHD (n = 45)	F	p	η^2
Intercepts						
Disobedience	27.0 (17.1–36.8)	22.1 (10.6–33.5)	3.66 (29.1–44.2)	2.55	.08	.06
Physical aggression	2.26 (.90–3.18)	2.39 (.85–3.93)	3.41 (2.52–4.30)	1.39	.25	.04
Destruction	1.21 (.20–2.21)	.67 (−.19–1.53)	1.70 (.99–2.41)	1.23	.30	.03
Verbal provocation	.76 (.30–1.23)[†]	.88 (−.14–1.91)	1.76 (1.19–2.25)[†]	3.33	.04	.08
Time-outs	9.03 (4.74–13.32)	7.13 (2.71–11.55)	13.03 (10.06–16.01)	2.62	.08	.07
Seclusions	1.47 (.41–2.52)	.34 (−.04–.71)	1.33 (.53–2.14)	1.07	.35	.03
TSCRS-teacher	86.8 (73.6–100.1)	101.8 (88.7–115.0)[†]	72.3 (65.2–79.4)[†]	8.00	$<.001$	.18
TSCRS-nurse	82.4 (74.0–90.8)	98.9 (85.8–112.0)[†‡]	73.8 (67.8–79.9)[†]	8.21	$<.001$	.18
APRS	89.1 (79.9–98.3)	99.5 (90.2–108.8)[†]	80.8 (75.2–86.4)[†]	5.54	$<.01$	.13
Living points	1084 (896–1272)	1175 (954–1396)[†]	870 (755–985)[†]	4.22	$<.02$	.10
Goal points	591 (515–667)	643 (516–769)	605 (526–684)	.20	.82	.02
Methylphenidate dose[c]	.03 (−.08–.15)	0 (0.0–0.0)	.01 (−.06–.09)	.24	.78	.02
Slopes						
Disobedience	−2.18 (−4.60–.24)	−2.61 (−4.95–.27)	−2.90 (−4.88–.92)	3.88	.03	.09
Physical aggression	−.36 (−.70– −.01)	−.34 (−.67– −.01)	−.25 (−.48– −.02)	2.04	.14	.05
Destruction	−.12 (−.41–.16)	−.05 (−.31–.21)	−.25 (−.42– −.07)	.41	.66	.01
Verbal provocation	−.10 (−.23–.03)	.07 (−.29–.44)	−.20 (−.40– −.01)	2.94	.06	.07
Time-outs	−.65 (−1.63–.33)	−1.13 (−2.22– −.04)	−1.34 (−2.13– −.55)	2.86	.06	.07
Seclusions	−.27 (−.51 −−.03)	−.04 (−.17– .09)	−.11 (−.31 −.08)	.78	.46	.02
TSCRS-teacher	5.71 (2.06–9.36)	1.51 (−1.27–4.28)	5.96 (3.96–7.96)	.97	.38	.02
TSCRS-nurse	−.13 (−2.07–1.81)	−1.45 (−3.27–.36)	2.38 (.85–3.91)	1.23	.30	.03
APRS	4.03 (1.47–6.58)	1.82 (.17–3.47)	4.13 (2.62–5.65)	.94	.39	.02
Living points	52.8 (1.2–104.4)	40.2 (−8.0–88.3)	59.5 (29.6–89.4)	3.63	.03	.09
Goal points	15.4 (−8.1–38.9)	−1.14 (−36.8–34.6)	37.0 (13.2–60.8)	5.47	$<.01$	.13
Methylphenidate dose[c]	.10 (.05–.14)[‡]	0 (0.0–0.0)[†‡]	.06 (.03–.08)[†]	11.85	$<.001$	.24

[1]All slope parameters are expressed hi units of change per week. TSCRS, Teachers Self Control Rating Scale; APRS, Academic Performance Rating Scale.

[a]Parameters with the same subscript differ significantly at $p < .05$ based on the Tukey test for unequal *ns*.

[b]Group differences in intercepts were tested with 2 numerator and 75 denominator degrees of freedom. Group differences in slopes were tested with 2 numerator and 74 denominator degrees of freedom, as intercepts were employed as covariates.

[c]mg./kg./day.

symptoms at intake, which were observed for verbal provocation, teacher-rated impulsivity (TSCRS), nurse-rated impulsivity (TSCRS), teacher-rated academic performance, and BMP living points earned. In all cases, CD/ADHD participants exhibited higher symptom rates than either the CD group, the ADHD group, or both. The only significant difference in symptoms observed between CD and ADHD participants was in nurse-rated impulsivity, where lower scores (i.e., more impulsivity) were observed in the latter group. Effect sizes (η^2) among these variables ranged from .08 to .18. Although consistent with expectations, these findings should be interpreted with caution given the number of comparisons conducted.

In terms of treatment responsiveness, significant improvement, as indicated by the confidence intervals surrounding slope parameters, was observed on most measures. Once again, few of the confidence intervals included zero. Group differences in slopes were also few, with the ANCOVAs yielding significant F-statistics for episodes of disobedience, BMP living points, and BMP goal points. In each case, group differences were observed on

adjusted but not on unadjusted means. Thus, differences in improvement were evident only when symptom severity at intake was controlled statistically. For episodes of disobedience, adjusted means were −2.51, −3.99, and −1.18 for the ADHD, CD, and CD/ADHD groups, respectively. Follow-up comparisons indicated that the only significant difference was between the CD and CD/ADHD groups, $F_{(1,55)} = 6.67$, $p = .01$, $\eta^2 = .19$. Thus, the CD group exhibited more improvement than the CD/ADHD group, controlling for initial symptoms. In other words, although unadjusted slopes did not differ across groups, the CD group improved more as a function of symptom severity at intake (22.1) than did the CD/ADHD group (36.6). For living points, adjusted means were 61.19, 67.26, and 24.01 for the ADHD, CD, and CD/ADHD groups. The only significant difference was between ADHD and CD/ADHD participants, $F_{(1,62)} = 4.21$, $p = .04$, $\eta^2 = .12$, with the former group exhibiting more improvement, controlling for initial symptoms. Finally, adjusted means for the goal points variable were 9.68, 6.62, and 35.00 for the ADHD, CD, and CD/ADHD groups. Here, the CD/ADHD group exhibited greater improvement than both the ADHD, $F_{(1,62)} = 6.62$, $p = .01$, $\eta^2 = .15$, and the CD, $F_{(1,55)} = 6.17$, $p = .02$, $\eta^2 = .18$, groups, controlling for symptoms at intake. Once again, these findings should be interpreted with caution given the number of comparisons conducted.

Group differences were also observed in methylphenidate dosage, where both the ADHD and the CD/ADHD groups, who did not differ from one another, exhibited larger increases in dosage than the CD only group. This was not surprising given that no members of the CD group received methylphenidate during the treatment period.

Analyses assessing the relations between changes in methylphenidate dosage and symptom residuals for participants with comorbid CD and ADHD are summarized in Table III. For most measures, residualized symptoms were unrelated to medication changes, as indicated by the reported χ^2 statistics and the low odds ratios. Thus, the odds of improving more than predicted by the individual regressions given a dosage increase were roughly equivalent to the odds of such improvement given no dosage increase. The two exceptions to this pattern were found for measures of hyperactivity and impulsivity. Specifically, improvement in both teacher-rated impulsiveness (TSCRS) and on-task academic performance (APRS) were related to increased methylphenidate dosages. For both indices, methylphenidate increases accounted for significant variance in residualized improvement. Patients were 1.43 and 1.33 times more likely to exhibit improvement, as indexed by positive residuals, following a dosage increase than following no dosage increase. No such relation was uncovered for nurse-rated impulsivity.

DISCUSSION

One of the hypotheses outlined in the introduction of this article was that CD/ADHD participants would be more symptomatic than the other two groups. This hypothesis was supported by group differences on five of the 11 criterion measures at intake, as estimated by intercepts in the growth curve analyses. On indices of verbal provocation, impulsivity (TSCRS), on-task behavior (APRS) and compliance with unit rules (living points), CD/ADHD participants performed more poorly than one or both of the other groups. Of note, a greater number of group differences were observed between CD/ADHD and CD participants (four) than between CD/ADHD and ADHD participants (one). This occurred despite a smaller sample of CD participants and therefore less

Table III. Probabilities of Improvement vs. Decline in Symptoms as a Function of Weekly Stasis vs. Participants with Comorbid CD and ADHD[1]

	Week of methylphenidate increase		Week of no methylphenidate increase					
Variable	improved	unimproved	improved	unimproved	Odds Ratio[a]	$\chi^2(1)$	p	Φ
Disobedience	21/44 (.48)	23/44 (.52)	45/91 (.49)	46/91 (.51)	.97	.04	.851	.02
Physical aggression	23/44 (.52)	21/44 (.48)	45/91 (.49)	46/91 (.51)	1.06	.09	.759	.08
Destruction	17/44 (.39)	27/44 (.61)	34/91 (.37)	57/91 (.63)	.03	.02	.886	.01
Verbal provocation	10/44 (.23)	34/44 (.77)	32/91 (.35)	59/91 (.65)	.65	2.14	.143	.13
Time-outs	18/44 (.41)	26/44 (.59)	47/91 (.52)	44/91 (.48)	.79	1.37	.242	.10
Seclusions	17/44 (.39)	27/44 (.61)	37/91 (.41)	54/91 (.59)	.95	.05	.822	.02
TSCRS-teacher	29/44 (.66)	15/44 (.34)	42/91 (.46)	49/91 (.54)	1.43	4.64	.031	.19
TSCRS-nurse	22/44 (.50)	22/44 (.50)	38/91 (.42)	53/91 (.58)	1.20	.82	.366	.08
APRS	38/44 (.86)	6/44 (.14)	59/91 (.65)	32/91 (.35)	1.33	6.80	.009	.22
Living points	26/44 (.59)	18/44 (.41)	45/91 (.49)	46/91 (.51)	1.19	1.11	.293	.09
Goal points	19/44 (.43)	25/44 (.57)	44/91 (.48)	47/91 (.52)	.89	.32	.573	.05

[1]Improvement was defined as over-prediction by individual growth curves of variables in which lower numbers represent progress (e.g., aggression) and as under-prediction of variables in which higher numbers represent progress (e.g., academic performance).

[a]Odds of improvement given a methylphenidate increase vs. odds of improvement given no methylphenidate increase. TSCRS, Teachers Self Control Rating Scale; APRS, Academic Performance Rating Scale.

statistical power for comparisons involving this group. Also of note, although CD/ADHD participants were more aggressive and destructive at baseline than their CD and ADHD counterparts, these differences were not significant. One common argument holds that group differences on low base rate behaviors are unlikely to be detected due to low power. However, the significant group effect for verbal provocation, which occurred at a lower base rate than either destruction or physical aggression, argues against this possibility in the present case.

Because comorbid participants suffered from two disorders rather than one, findings of greater symptom severity in the CD/ADHD group are not surprising. In contrast, the equally plausible hypothesis that CD/ADHD participants would exhibit the least improvement over the course of their admission was not supported. After controlling for levels of baseline functioning, few group differences in improvement were observed. Moreover, for group differences that were uncovered, the direction of effects was not consistent. As might be expected, the CD/ADHD group exhibited less improvement than the CD and ADHD groups on measures of disobedience and living points earned. However, the CD/ADHD group exhibited more improvement than both of the other groups on goal points earned. Of the two BMP indices, the living points measure is easier to interpret because it reflects cooperation with required chores and tasks. In contrast, the goal points measure assessed degree of success in meeting individualized treatment objectives.

Taken together, findings of less improvement in episodes of disobedience and living points earned suggest some degree of treatment resistance among CD/ADHD participants. However, on measures of more severe behaviors, including property destruction, physical aggression, and seclusions, no group differences in treatment responsiveness were observed, nor were group differences uncovered on measures of improvement in impulsivity or academic performance. These findings, coupled with findings of sample-wide improvement on most of the outcome measures, suggest that CD/ADHD participants benefited as much or nearly as much from treatment as their non-comorbid counterparts. These benefits included significant reductions in episodes of disobedience or time-out, destruction, verbal provocation, and physical aggression, the latter of which is arguably the behavior of greatest concern exhibited by CD and CD/ADHD children. Additionally, significant improvement in living points earned suggests greater compliance across the treatment period in performing day-to-day tasks. These findings argue against the widely held belief that severe CD is unresponsive to treatment. While this does not speak to long term course, it does indicate that intensive short term multimodal inpatient interventions can be effective in curbing the aversive behaviors associated with severe CD, including physical aggression.

The final hypothesis, that methylphenidate effects would be restricted to indices of impulsivity for CD/ADHD participants, was supported partially. On two of three measures of hyperactivity-impulsivity, significant relations between methylphenidate dosage increases and symptom residuals were found. In comparison, no such relations were uncovered on any of the remaining eight measures. For both on-task behaviors (APRS) during school, and teacher rated impulsivity (TSCRS), the probability of greater than expected improvement was higher during weeks of medication increases than during weeks of no medication increases. This may have implications for academic performance given the validity of the APRS in predicting standardized test scores, and given previous findings of increased reading comprehension in CD/ADHD children following methylphenidate treatment [Forness et al.,

1991]. Curiously, methylphenidate increases were not associated with greater than predicted improvement in nurse report TSCRS data. One possible explanation for the lack of concordance between teacher and nurse reports lies in the time periods during which each informant monitored patient behavior. Because teachers observed behavior during the day, their contact with children was restricted to periods of peak blood concentrations of methylphenidate. Nurses, on the other hand, observed patients primarily after school and in the evenings, as blood concentrations waned. It is therefore possible that the divergence in prediction of teacher versus nurse ratings of impulsivity reflects the efficacy of methylphenidate in curbing impulsive behaviors during periods of peak blood concentration.

Although findings regarding the differential impact of methylphenidate on specific behaviors are of interest, they should be interpreted with caution for at least two reasons. First, it is unclear how sensitive the method of examining residualized symptoms is, making it possible that medication effects on behavior were undetected. Such effects cannot be assessed unambiguously without a randomized trial. As indicated in the introduction of this article, however, several randomized trials assessing the impact of methylphenidate on aggression among comorbid children have produced results that are consistent with the findings reported here. Thus, it appears that the impact of methylphenidate on hyperactivity and impulsivity is significantly greater than its impact on aggression. While we do not wish to trivialize the importance of reducing less severe externalizing behaviors, it is of some concern that aggression, the behavior of greatest consequence, appears to be the symptom that is impacted the least by stimulant treatment.

Second, the dosages of methylphenidate employed in this study were low to moderate. It is therefore possible that further dosage increases could have produced methylphenidate effects on additional behaviors. However, while reductions in impulsivity have been noted in CD/ADHD samples at lower dosages than were used in the present investigation [e.g., Cunningham et al., 1991], several studies have suggested that higher doses than employed here are not associated with additional improvement on most measures of hyperactivity-impulsivity or aggression [e.g., Barkley et al., 1989; Brown et al., 1991; Gadow et al., 1990]. Thus, moderate methylphenidate doses are an unlikely explanation for the failure to find additional medication effects. This suggests that other components of treatment (e.g., social skills training, behavioral management, etc.) were responsible for the sample-wide reductions in aggression observed. Unfortunately, no dosage estimates of those components were available, so the specific mechanism through which aggression was reduced cannot be identified.

As with all retrospective research, several limitations characterized this study, some of which have been alluded to above. Because it was not a randomized double-blind assessment, staff members were aware of medication dosages and changes, which could have affected their expectations regarding participant improvement. This in turn could affect their ratings of behavior. The likely impact of this would be to boost artificially estimates of methylphenidate effects. Given the pattern of findings, however, this appears not to have occurred systematically. Additionally, outcome measures were limited to the data available within patient's charts. While this was probably an advantage with respect to the BMP, where meticulous records were kept on a host of behavioral categories, rating scale data were limited to broad band scores from the TSCRS and APRS. As noted above, these scores probably did provide valid indices of functioning. However, they did not allow for more specific assessments of teacher or nurse rated changes in behavior. It would have been

informative, for example, to have specific indices of hyperactivity and impulsivity available for analysis.

Results of the growth curve analyses must be interpreted in light of the small sample size, particularly of CD only participants ($n = 13$). This resulted in reduced statistical power for comparisons of the CD only group to the ADHD and CD/ADHD groups. For example, although CD/ADHD participants were more symptomatic at intake than their CD only counterparts on *all* measured indicators, only four of 11 group contrasts were significant statistically. Given more CD participants and increased statistical power, more significant differences would likely have been found. Regarding improvement as indexed by individual growth curves, CD/ADHD participants exhibited faster symptom reduction (i.e., steeper slopes) than CD only participants on 10 of the 11 measures, yet none of these differences were significant. Here again, some of these contrasts probably would have been significant given more power. On the positive side, growth curve analysis is more powerful than several alternative methods of assessing change [Speer and Greenbaum, 1995]. Moreover, the low power associated with the small sample of CD only participants did not affect analyses of residualized symptoms, which did not include the CD group because they received no medication.

Finally, we have noted several times that results should be interpreted with caution given the number of comparisons conducted. Because of the wide range of symptoms assessed, and because of potentially important differences in more vs. less severe symptoms of aggression (e.g., physical aggression vs. disobedience), we chose not to combine indices of aggression and hyperactivity into single constructs. Given this, the pattern of results is probably more meaningful than any single comparison. For example, the finding that methylphenidate affected two of three measures of hyperactivity and zero of eight measures of aggression suggests some specificity of psychostimulants in reducing ADHD symptoms among comorbid children.

Given their early onset symptoms, including aggression and comorbid hyperactivity-impulsivity, the CD/ADHD participants in this study represent the most recalcitrant of all externalizing children. At intake, they were worse off than their counterparts in the CD and/or the ADHD groups on most measures. Nevertheless, they exhibited significant improvement in multiple domains of functioning during hospitalization. This suggests a degree of treatment responsiveness that is not always reflected in the literature. It would be naïve, however, to assume that short term treatment responsiveness necessarily relates to long term course. If Lynam [1996, 1998] is correct in suggesting that comorbid CD and ADHD reflects fledgling psychopathy, then it may useful to draw from the adult literature on relations between short- and long-term treatment responsiveness among psychopaths. This literature suggests that recidivism rates among treated psychopaths are higher than those among untreated psychopaths and non-psychopathic offenders [e.g., Harris et al., 1994; Rice et al., 1992]. Moreover, these findings are paralleled by similar outcomes among treated youth with conduct problems [Dishion et al., 1999]. Nevertheless, adult psychopaths are often able to convince treatment providers that they are progressing in therapy [Seto and Barbaree, 1999]. Thus, short-term outcomes may not predict long-term course, and treatments that place chronic offenders in close proximity of one another may yield iatrogenic effects. It is our hope that continued research will (a) identify the mechanisms of action associated with short-term improvement among children with CD and ADHD, (b) evaluate the long-term outcomes of intensive treatment programs, and (c) develop more effective interventions that reduce core CD behaviors over longer time intervals.

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