Anastopoulos, Smith, et al. Published in the Journal of Attention Disorders 15(7): 583-592. October 2011.

Self-Regulation of Emotion, Functional Impairment, and Comorbidity Among Children With AD/HD

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Abstract

Objective: This study investigated the role of self-regulation of emotion in relation to functional impairment and comorbidity among children with and without AD/HD. **Method:** A total of 358 probands and their siblings participated in the study, with 74% of the sample participants affected by AD/HD. Parent-rated levels of emotional lability served as a marker for self-regulation of emotion. **Results:** Nearly half of the children affected by AD/HD displayed significantly elevated levels of emotional lability versus 15% of those without this disorder. Children with AD/HD also displayed significantly higher rates of functional impairment, comorbidity, and treatment service utilization. Emotional lability partially mediated the association between AD/HD status and these outcomes. **Conclusion:** Findings lent support to the notion that deficits in the self-regulation of emotion are evident in a substantial number of children with AD/HD and that these deficits play an important role in determining functional impairment and comorbidity outcomes.

Keywords

AD/HD, children, emotion regulation, comorbidity, functional impairment

Children with AD/HD are at increased risk for experiencing serious, lifelong impairments in multiple domains of daily functioning (Barkley, 2006). Such impairments are intensified in the presence of comorbid conditions, which occur in up to 60% of clinic-referred children with AD/HD (August, Realmuto, MacDonald, & Nugent, 1996). Oppositional-defiant disorder (ODD) is a particularly common comorbid condition, which, left unchecked, can lead to more serious behavioral complications, most notably conduct disorder (CD; Angold, Costello, & Erkanli, 1999; Cunningham & Boyle, 2002; Jensen, Martin, & Cantwell, 1997).

In addition to being predisposed to co-occurring externalizing difficulties, children with AD/HD are at increased risk for displaying comorbid internalizing problems. For example, in both epidemiological and clinical studies, children with AD/HD have been shown to be at 20% to 30% increased risk for developing depression (Biederman, Mick, & Faraone, 1998). Similar findings have been reported for anxiety disorders, with up to 25% of the child AD/HD population displaying one or more anxiety conditions (Tannock, 2000). Meta-analytic studies lend further support to these findings, suggesting that the odds of having AD/HD and comorbid depression range from 3.5 to 8.4, with an overall median odds ratio of 5.5 (Angold et al., 1999). Elevated but slightly lower odds ratios have also been reported for AD/HD and anxiety disorders, ranging from 2.1 to 4.3, with a median of 3.0 (Angold et al., 1999).

Although the association between AD/HD and internalizing disorders is well established, the processes or mechanisms by which this association occurs have yet to be identified. One commonly held assumption is that having AD/HD places a child at risk for repeated experiences of failure and frustration across the home, school, and social domains, thereby setting the stage for internalizing disorders to occur (Patterson & Capaldi, 1990). Unfortunately, research addressing this possible developmental pathway from primary AD/HD symptoms—inattention, impulsivity,

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hyperactivity—to secondary internalizing psychopathology has been lacking. Thus, questions remain as to how comorbid depression, anxiety, and other internalizing disorders arise.

Another possible explanation for this clinical phenomenon stems from a consideration of what actually constitutes the core features of AD/HD. Inattention, impulsivity, and hyperactivity have long been recognized as primary symptoms of this disorder. Along with these cognitive and behavioral manifestations, it is possible that difficulties regulating emotions are another central feature of AD/HD and that being emotionally labile confers increased risk for experiencing functional impairment and comorbid internalizing problems. Clinical accounts of children with AD/HD are certainly compatible with this possibility. In particular, parents, teachers, and clinicians commonly describe such children as having higher emotional highs and lower emotional lows. Moreover, such children seem more prone to react emotionally to everyday situations and to have greater difficulty regulating their emotions as they are occurring.

In support of these clinical descriptions are recent theoretical accounts that ascribe a more central role to the self-regulation of emotion in the presentation of AD/HD (Barkley, 2006; Nigg, 2001). In Barkley's (2006) model, for example, self-regulation of affect is defined as the process by which an individual's capacity for inhibition allows them to delay responding to events that elicit emotional responses, especially those of a negative nature (e.g., anger). The greater the capacity for delay, the more likely it is that an individual can gather information necessary for understanding an emotionally charged event. This in turn affords an individual greater opportunity for modifying or moderating an emotional response earlier to its public display.

Although limited in number, studies have found evidence of an association between AD/HD and deficits in the self-regulation of affect or emotion. One of the earliest articles addressing this matter was reported by Douglas (1988), who observed that children with AD/HD became overly aroused and excited in response to rewards and more frustrated when rewards were withdrawn and less available. Subsequent research has also shown that children with AD/HD display higher rates of negative affect (e.g., anger, sadness), greater emotional reactivity, and lower levels of empathy relative to normal controls (Braaten & Rosén, 2000; Cole, Zahn-Waxler, & Smith, 1994; Hinshaw & Melnick, 1995; Jensen & Rosén, 2004; Maedgen & Carlson, 2000; Martel, 2009; Melnick & Hinshaw, 2000; Walcott & Landau, 2004). Together, such findings lend support to the notion that deficits in the self-regulation of emotion exist among children with AD/HD (Skirrow, McLoughlin, Kuntsi, & Asherson, 2009).

Remaining less clear, however, is the clinical significance of these emotional findings and how specific they are to AD/HD. For example, it has not yet been established whether deficits in the self-regulation of emotion are evident in all children with AD/HD or perhaps only in a subgroup of children with this disorder. Also unclear is whether deficits in the self-regulation of emotion increase the risk for functional impairment or for comorbid conditions, especially those of an emotional nature. Finally, in light of findings suggesting that a small percentage of children with AD/HD may display comorbid bipolar disorder (BD; Biederman et al., 1996), it is also necessary to consider the possibility that comorbid BD, rather than AD/HD, accounts for these emotion regulation difficulties.

The current study examined these issues in the context of a larger-scale investigation of AD/HD among 5- to 12-year-old probands and their siblings. Consistent with the fact that children with AD/HD display different combinations and intensities of inattention, impulsivity, and hyperactivity (e.g., AD/HD subtypes), our expectation was that a substantial number of probands and siblings with AD/HD, but not all, would display evidence of a deficit in the self-regulation of emotion and that these deficits would exist independent of the presence of BD. It was also predicted that, for those children affected by AD/HD, deficits in the self-regulation of emotion would moderate outcomes and be associated with greater functional impairment, as well as with increased levels of comorbid features. Given the heterogeneity of the population, AD/HD subtyping was also expected to play a role, with higher levels of impairment and comorbid features anticipated among those with the combined (C) subtype versus those with either the predominantly inattentive (I) or predominantly hyperactive-impulsive (HI) subtypes. As further evidence of this increased risk for impairment and comorbidity, we expected increased rates of treatment service utilization among those with an impaired capacity for regulating emotions.

Method

Participants

The sample used in this study was drawn from a larger pool of children and their families participating in a longitudinal, multisite investigation of the genetic basis of AD/HD and its comorbid features. To be eligible for initial entry into the study, probands had to be between the age of 5 and 12 years and meet *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., *DSM-IV*) criteria for a diagnosis of AD/HD, any subtype. Probands were initially determined to be affected by AD/HD on the basis of parental responses to structured interview questioning, accompanied by significantly elevated *T*-scores on parent- and teacher-completed rating scale measures of AD/HD symptoms. Final determination of AD/HD status was established by a panel of three senior investigators reviewing each case. The same criteria

and panel-review process were used for determining AD/HD status of all siblings participating in the study. In contrast with probands, siblings could range in age from 5 to 17 years and were not required to meet *DSM-IV* criteria for a diagnosis of AD/HD, although many did.

A total of 216 probands and 142 siblings served as participants. Included among this total were 218 boys and 140 girls, with a mean age of 8.7 years. Approximately 20% of the sample was from ethnically and racially diverse backgrounds, the vast majority of whom (18.3%) were African American families. Almost 74% of the children, including 49 siblings, were affected by AD/HD, with 52.8% of all affected children classified with the C type, 36.6% with the I type, and 10.6% with the HI type. Consistent with previously reported findings (Barkley, 2006), many of the children with AD/HD also met *DSM-IV* criteria for one or more comorbid diagnoses, including ODD (36.9%), CD (8.0%), separation anxiety disorder (11.4%), social phobia (7.6%), generalized anxiety disorder (6.1%), obsessive compulsive disorder (4.2%), major depression/dysthymic disorder (3.8%), tic disorders (4.9%), and elimination disorders (11.8%). Of additional significance, none of the affected children in the sample met criteria for BD as determined by the measures used in this study.

Measures

AD/HD classification and comorbid diagnoses. Structured interview and rating-scale responses were used in combination to establish the presence or absence of an AD/HD diagnosis. The structured interview used for this purpose was the Computerized Diagnostic Interview Schedule for Children, Fourth Edition (C-DISC-IV; National Institutes of Mental Health [NIMH], 1997). Positive parental responses to the AD/HD module of the C-DISC-IV served as the starting point for making an AD/HD diagnosis. Also required were *T*-score elevations on corresponding parent- and teacher-completed Conners' Rating Scales–Revised (CRS-R; Conners, 2001) dimensions. More specifically, parent- and teacher-generated *T*-scores on the CRS-R *DSM-IV* inattention and/or *DSM-IV* hyperactive-impulsive dimensions had to be at or more than 65 and 60, respectively, in order to be of sufficient developmental deviance to warrant consideration of an AD/HD diagnosis. For the AD/HD-C type, there needed to be evidence of significant *T*-score elevations on both AD/HD-symptom dimensions. For an AD/HD-I subtype classification, only *T*-score elevations on the *DSM-IV* inattention score were required. Similarly, elevated *T*-scores on the *DSM-IV* hyperactive-impulsive score were required for establishing an AD/HD-HI subtype classification.

The C-DISC-IV was also used to determine the presence or absence of *DSM-IV* defined comorbid diagnoses among children affected by AD/HD. This included the routine administration of diagnostic modules addressing: ODD and CD; major depressive disorder, dysthymic disorder, and BD; separation anxiety disorder, generalized anxiety disorder, social phobia, and obsessive compulsive disorder; elimination disorders; tic disorders and Tourettes syndrome; and PTSD. Positive parental responses to any of these C-DISC-IV modules served as the first step for establishing a comorbid diagnosis, with final confirmation of comorbid status determined on the basis of panel review.

Self-regulation of emotion. Emotional lability *T*-scores from the parent-completed CRS-R (Conners, 2001) served as a marker for self-regulation of emotion among probands and siblings.

Functional impairment and comorbid features. *The Behavior Assessment System for Children–Second Edition* (BASC-2; Reynolds & Kamphaus, 2004) is a broadband rating scale that yields information pertinent to both functional impairment and clinical symptom presentation. The *T*-scores for the parent-completed BASC-2 dimensions of social skills, daily living, and overall adaptive functioning served as indices of functional impairment, with lower scores on these dimensions being indicative of increased impairment, and *T*-scores for the parent-completed BASC-2 dimensions of anxiety, depression, internalizing composite, aggression, and conduct problems served as dimensional indices of comorbidity, with higher scores on these dimensions representing greater symptom severity. The decision to use these BASC-2 indices of comorbidity as outcome measures in the statistical analyses, rather than the C-DISC-IV generated comorbid diagnoses, stemmed primarily from a consideration of the fact that comorbid diagnoses were only available for probands and affected siblings, not for unaffected siblings (for whom the full C-DISC-IV was not administered). An additional reason for using these BASC-2 comorbidity indices is that they allowed for more direct comparison with the BASC-2 measures of functional impairment.

Treatment utilization. Seven items (Item 1, Items 3-8) from the Services Use in Children and Adolescents–Parent Interview (Hoagwood et al., 2004; Jensen et al., 2004) were used to assess utilization of stimulant medication therapy and other treatment services (e.g., parent training, individual therapy). Each item was scored as 1 (*present*) or 0 (*absent*). A total score was calculated by summing across these 7 items, with higher scores representing greater use of multiple treatment services.

Procedure

Participating children and their parents were recruited from two separate, university-based AD/HD specialty clinics and from the community. Parental consent and child assent were obtained in accordance with institutional review board guidelines at each university. All participating children and

their parents underwent comprehensive psychological assessments that included structured diagnostic interviewing, semistructured background interviewing, and completion of self- and other-report rating scales. All psychological data were collected by trained staff and graduate-level research assistants working under the supervision of senior project psychologists. At the completion of their participation, all families received US$50 as compensation for their time and effort. Participating families also received written summaries of their psychological testing and rating scale results.

Statistical Analyses

For emotional lability and for the various adverse outcomes, the entire sample was dichotomized into two groups, with one group displaying significantly elevated levels of the feature, whereas the other fell below this level. For emotional lability and the BASC-2 comorbid indices, *T*-scores at or above 65 were used to create groups with significantly elevated features. For the BASC-2 functional impairment indices, *T*-scores at or below 35 were used to create significantly impaired groups. The treatment utilization index was also dichotomized to capture whether or not multiple treatment services (i.e., 2 or more services) were being utilized. Thus, two groups were formed on the basis of receiving 0 to 1 treatment services versus 2 to 7 treatment services.

All analyses were conducted using SAS version 9.1. Correlations were calculated using PROC CORR. Because within-family data are more highly correlated than data collected across families, steps were taken to control for familial correlation between siblings from the same family. More specifically, all logistic regressions were performed using Generalized Estimating Equations (GEE) with PROC GENMOD, which controls for this type of familial correlation. Similarly, to control for the fact that the childhood expression of AD/HD varies as a function of age and gender (Conners, 2001; DuPaul, Power, Anastopoulos, & Reid, 1998), all models included gender and age covariates.

Results

For the entire sample, correlations between the emotional lability subscale and BASC-2 adverse outcomes are presented in Table 1. As expected, higher levels of emotional lability were associated with greater impairment in social skills and daily functioning as well as with respect to overall adaptive functioning. Higher levels of emotional lability were also significantly associated with higher levels of the various comorbid emotional and behavioral indices, with particularly strong correlations evident with respect to comorbid depression and aggression features.

As noted in Table 2, a substantial number of children with AD/HD (46.92%) displayed high levels of emotional lability relative to that observed for unaffected siblings (15.38%). Similar findings were evident with respect to the measures of functional impairment and comorbidity. As compared to unaffected siblings, children with AD/HD were classified at higher rates for every adverse outcome, ranging from 30 to 51.5% for the impairment indices and from 21% to 38.7% for the measures of comorbidity. Also appearing in Table 2 are the results of the multiple logistic regression analyses that were conducted to address the magnitude of these classification differences. Generally speaking, children with AD/HD were at significantly elevated risk for displaying high levels of emotional lability relative to unaffected children (odds ratio [OR] = 5.703, CI = 2.991-10.878, $p < .001$). Children with AD/HD were also at significantly elevated risk for the functional impairment indices, ranging from a 3.009 increase in risk with respect to social skills (CI = 1.584-5.716, $p < .001$) up to a 19.818 increase in risk for daily living (CI = 7.243-54.223, $p < .001$). A similar pattern was evident among the various comorbidity outcomes, such that children with AD/HD were at significantly elevated risk for depression (OR = 7.334, CI = 3.168-16.978, $p < .001$) and anxiety (OR = 3.601, CI = 1.448-8.956, $p = .001$), as well as for aggression (OR = 3.648, CI = 1.545-8.609, $p < .001$) and conduct problems (OR = 4.753, CI = 1.785-12.660, $p < .001$).

A series of multiple logistic regression analyses was conducted subsequently, in order to test the hypothesis that emotional lability would moderate the effect of AD/HD on adverse outcomes. If present, moderation effects would be indicated by significant interactions between AD/HD status and emotional lability predicting adverse outcomes. None of these interactions was significant and, therefore, provided no evidence of moderation effects. However, models including AD/HD status and emotional lability (but not including an interaction term) showed a reduced effect of AD/HD

Table 1. Correlations Between Emotional Lability and Adverse Outcomes

Outcome	Emotional Lability
Functional impairment	
Social skills	–.39
Daily living	–.40
Adaptive skills composite	–.52
Comorbidity	
Anxiety	.29
Depression	.71
Internalizing composite	.56
Aggression	.64
Conduct	.52

Emotional Lability score derived from Conners' Parent Rating Scales–Revised. All outcome indices derived from Behavior Assessment System for Children–Second Edition. All correlations are significant at $p < .001$.

Table 2. Multiple Logistic Regression Analyses of Relationship Between AD/HD and Adverse Outcomes

	AD/HD[a]	Non-AD/HD[b]	OR	95% CI	*p* value
Emotional lability	46.92	15.38	5.703	2.991-10.878	<.001
Functional impairment					
Social skills	30.04	13.64	3.009	1.584-5.716	<.001
Daily living	51.45	6.82	19.818	7.243-54.223	<.001
Adaptive skills composite	38.27	9.09	6.336	2.979-13.470	<.001
Comorbidity					
Anxiety	20.99	9.09	3.601	1.448-8.956	.001
Depression	38.68	9.09	7.334	3.168-16.978	<.001
Internalizing composite	32.51	9.09	5.716	2.372-13.774	<.001
Aggression	30.45	11.36	3.648	1.545-8.609	<.001
Conduct	38.71	12.50	4.753	1.785-12.660	<.001

Emotional Lability score derived from Conners' Parent Rating Scales–Revised. Functional impairment and comorbidity indices derived from Behavior Assessment System for Children–Second Edition. OR = odds ratios comparing AD/HD versus non-AD/HD, controlling for age, sex, and family correlation. CI = confidence interval.

[a]Percentage of children with AD/HD displaying adverse outcome.

[b]Percentage of non-AD/HD children displaying adverse outcome.

Table 3. Multiple Logistic Regression Analyses of Role of Emotional Lability in Mediating Relationship Between AD/HD and Adverse Outcomes

					Sobel's Test for Mediation	
Outcome	Predictor	OR	95% CI	*p* value	Indirect effect %	*p* value
Social skills	Affected status	2.179	1.093-4.346	.021		
	Emotional lability	1.034	1.015-1.054	.003	21.6	.001
Daily living	Affected status	15.760	5.481-45.317	<.001		
	Emotional lability	1.034	1.016-1.052	.001	17.8	.001
Adaptive skills	Affected status	4.692	1.959-11.242	.000		
	Emotional lability	1.054	1.034-1.074	<.001	30.3	<.001
Anxiety	Affected status	2.240	0.854-5.871	.070		
	Emotional lability	1.030	1.009-1.050	.005	19.1	.009
Depression	Affected status	3.091	1.125-8.499	.018		
	Emotional lability	1.110	1.085-1.137	<.001	51.2	<.001
Internalizing composite	Affected status	2.591	1.009-6.651	.031		
	Emotional lability	1.070	1.050-1.091	<.001	38.7	<.001
Aggression	Affected status	1.413	0.504-3.963	.477		
	Emotional lability	1.101	1.073-1.128	<.001	50.7	<.001
Conduct problems	Affected status	2.758	0.972-7.831	.034		
	Emotional lability	1.063	1.043-1.084	<.001	35.6	<.001

OR = odds ratios controlling for age, sex, and family correlation. CI = confidence interval. Outcome measures and AD/HD affection status are dichotomous and the mediator Emotional lability is continuous; thus, ORs are not on same scale.

status on the outcome, whereas emotional lability remained statistically significant in all models. This raised the possibility that the relationship between AD/HD status and adverse outcomes might be mediated by emotional lability.

Therefore, as a follow-up to these planned analyses of moderation effects, formal mediation analyses were conducted to examine whether emotional lability mediated the effect of AD/HD status on adverse outcomes. For all of these analyses, AD/HD status and adverse outcomes were entered as dichotomous variables and emotional lability was entered as a continuous variable. First examined was whether or not AD/HD status predicted outcomes of functional impairment and comorbidity. As may be seen from a summary of these meditational analyses in Table 3, the relationship between AD/HD status and adverse outcomes was significant. The degree to which AD/HD status predicted emotional lability was tested next, and this relationship was also significant ($p < .001$), after controlling for within family correlation, age, and sex. AD/HD-affected individuals had higher emotional lability scores ($M = 64.5$)

Table 4. Multiple Logistic Regression Analyses Examining the Role of AD/HD Subtype and Emotional Lability in Predicting Adverse Outcomes

Outcome	Predictor	Model	OR	95% CI	*p* value
Social skills	AD/HD subtype	Subtype only			.001[a,b]
	AD/HD subtype	Subtype and emotional lability			.006[a,b]
	Emotional lability		1.026	1.004-1.048	.032
Daily living	AD/HD subtype	Subtype only			.002[a,c]
	AD/HD subtype	Subtype and emotional lability			.025[a]
	Emotional lability		1.026	1.007-1.045	.008
Adaptive skills	AD/HD subtype	Subtype only			.000[a,b]
	AD/HD subtype	Subtype and emotional lability			.019[a,b]
	Emotional lability		1.051	1.029-1.073	<.001
Anxiety	AD/HD subtype	Subtype only			.015
	AD/HD subtype	Subtype and emotional lability			.027
	Emotional lability		1.028	1.006-1.050	.016
Depression	AD/HD subtype	Subtype only			<.001[a,c]
	AD/HD subtype	Subtype and emotional lability			.004 [a,c]
	Emotional lability		1.106	1.077-1.136	<.001
Internalizing composite	AD/HD subtype	Subtype only			.016[a]
	AD/HD subtype	Subtype and emotional lability			.488
	Emotional lability		1.071	1.046-1.095	<.001
Aggression	AD/HD subtype	Subtype only			<.001[a,c]
	AD/HD subtype	Subtype and emotional lability			.007[a,c]
	Emotional lability		1.097	1.065-1.129	<.001
Conduct problems	AD/HD subtype	Subtype only			<.001[a,c]
	AD/HD subtype	Subtype and emotional lability			.001[a]
	Emotional lability		1.059	1.035-1.083	<.001

OR = odds ratios controlling for age, sex, and family correlation. CI = confidence interval. Outcome measures and AD/HD affection status are dichotomous and the mediator emotional lability is continuous; thus, ORs are not on same scale.
[a]AD/HD combined subtype > AD/HD predominantly inattentive subtype.
[b]AD/HD predominantly hyperactive-impulsive subtype > AD/HD predominantly inattentive subtype.
[c]AD/HD combined subtype > AD/HD predominantly hyperactive-impulsive subtype.

than did unaffected siblings ($M = 52.0$). Mediation was then tested by including both AD/HD status and emotional lability in the models predicting adverse outcomes, after which estimates of indirect effects were conducted by Sobel's test, using the method outlined for binary outcomes recommended by Jasti, Dudley, and Goldwater (2008). The results indicated that emotional lability partially mediated the association between AD/HD status and all adverse outcomes. The percentage of the total effect mediated by emotional lability ranged from 17.8% (daily living) to 30.3% (adaptive skills composite) for the indices of functional impairment and from 19.1% (anxiety) to 51.2% (depression) for the comorbidity measures, with aggression also being quite high (50.7%).

To examine the relationship between AD/HD subtype, emotional lability, and adverse outcomes among affected children, additional multiple logistic regression analyses were completed. Each regression analysis controlled for within family correlation, age, and sex. A summary of the results of the association between AD/HD subtype, emotional lability, and other adverse outcomes is presented in Table 4. As shown in this table, AD/HD subtype was significantly associated with emotional lability ($p < .001$), such that children with AD/HD-C were at greater risk for emotional lability problems than children with either AD/HD-I (OR = 3.73, CI = 2.09-6.65, $p < .001$) or AD/HD-HI (OR = 5.76, CI = 2.15-15.46, $p < .001$). Children with the AD/HD-C subtype were also more likely to have clinically significant adverse outcomes than those with either AD/HD-HI or AD/HD-I. For social skills and the adaptive skills composite in particular, those with the AD/HD-HI subtype were also more likely to have an adverse outcome as compared to AD/HD-I. When emotional lability was entered into the model, it was associated with every adverse outcome. After accounting for emotional lability, many of the differences between AD/HD subtypes remained significant; however, the odds of having clinically significant outcomes in the subtype comparisons decreased. This suggests that some differences in adverse outcomes between AD/HD subtypes may be partly accounted for by differences in emotional lability.

Finally, to examine whether variability in emotional lability predicted treatment utilization among affected participants, a multiple logistic regression analysis was conducted for controlling within-family correlation, age, and sex. For this analysis emotional lability was analyzed continuously, with treatment utilization dichotomized into two groups. Findings showed that increases in emotional lability were associated with multiple-treatment utilization (OR = 1.03, CI = 1.01-1.05, $p = .0048$).

Discussion

For a number of years, the field has recognized that children with AD/HD have difficulties regulating their emotions. In particular, research has shown that children with this disorder display higher rates of negative affect, greater emotional reactivity, and lower levels of empathy relative to normal controls (Braaten & Rosén, 2000; Cole et al., 1994; Hinshaw & Melnick, 1995; Jensen & Rosén, 2004; Maedgen & Carlson, 2000; Martel, 2009; Melnick & Hinshaw, 2000; Walcott & Landau, 2004). Although such findings support the notion that deficits in the self-regulation of emotion exist among children with AD/HD, the diagnostic and prognostic significance of these findings is not yet well understood. Important questions remain with respect to whether or not difficulties regulating emotions are a central feature of AD/HD and/or confer risk for experiencing functional impairment and comorbidity.

The current study examined these issues among affected and unaffected siblings. On the basis of the theoretical and empirical considerations, it was expected that a substantial number of children with AD/HD, but not all, would display significant problems in their self-regulation of emotion, as determined by the their scores on a parent-completed measure of emotional lability. Consistent with this hypothesis, children with AD/HD were found to have a nearly sixfold increased risk for displaying significantly elevated levels of emotional lability, with almost half of the AD/HD-affected children exhibiting this outcome. That this would occur in the absence of any evidence of BD suggests that the deficits in the self-regulation of emotion may indeed be specific to AD/HD and, therefore, serve as a marker for a subgroup of children with AD/HD.

For such a marker to be meaningful, it would need to demonstrate some degree of clinical significance. As a way of addressing this matter, the current study also examined the degree to which emotional lability was associated with functional impairment and comorbidity. Contrary to the study's hypotheses, there was little evidence to suggest that emotional lability moderated the relationship between AD/HD status and these adverse outcomes. However, further inspection of the initial findings raised the possibility that emotional lability might function more as a mediating variable.

To address this possibility, formal meditational analyses were conducted, which showed that emotional lability *partially* mediated the association between AD/HD status and all adverse outcomes. With regard to functional impairment, the percentage of the total effect mediated by emotional lability was found to be as high as 30.3% for a composite measure of adaptive functioning. Among the dimensional measures of comorbidity, the percentage of the total effect mediated by emotional lability was highest for depression (51.2%) and aggression (50.7%), followed by conduct problems (35.6%) and anxiety (19.1%). The fact that both depression and anxiety were partially mediated by emotional lability was in line with study expectations. Less anticipated, however, was the discrepancy in the magnitude of total effect mediated for these two emotional dimensions. At face value, the fact that emotional lability partially mediated the behavioral dimensions of aggression and conduct problems might also seem surprising. However, such results are more easily understood when taking into account that irritability and anger are important components of these two behavioral dimensions.

As expected, AD/HD subtype was significantly associated with emotional lability, such that children with AD/HD-C were at greater risk for emotional lability problems than were those with either the AD/HD-I or AD/HD-HI subtype. AD/HD-C was also associated with higher odds of having clinically significant adverse outcomes relative to both AD/HD-HI and AD/HD-I. When emotional lability was entered into the model, it too was associated with every adverse outcome; however, the odds of having clinically significant outcomes in subtype comparisons decreased, substantially so, in some cases. This suggests that some of the differences in adverse outcomes between AD/HD subtypes may be partly accounted for by differences in the scores for emotional lability.

In line with our final hypothesis, increased rates of treatment service utilization were found among affected children with higher levels of emotional lability. Although the process by which this association arises cannot be determined from the current study, one factor that may contribute to this outcome is the increased risk of functional impairment and comorbidity that was shown to be associated with higher levels of emotional lability.

In sum, the current findings are consistent with the premise that difficulties regulating emotions are a prominent feature of the clinical presentation of AD/HD in children. Among children with this disorder, there would seem to be an increased likelihood that deficits in the self-regulation of emotion will be present, which in turn confer substantially increased risk for functional impairment and comorbid features, especially depression and aggression tendencies. Such results are in line with recently reported empirical findings (Jensen et al., 2004; Martel, 2009; Walcott &

Landau, 2004) and with contemporary conceptualizations of AD/HD (Barkley, 2006; Nigg, 2001).

Although promising in nature, the results from this study must be tempered by a consideration of various limitations inherent in this design. First and foremost is the manner in which self-regulation of emotion was defined. In this study, a parent-completed rating of emotional lability served as a marker for emotion regulation difficulties. Within the field there are more direct and precise methods for assessing this construct, which could be incorporated into future studies examining these issues. As noted recently, more detailed information about the type and quality of emotional issues (e.g., type of irritability) may have important implications for clinical assessment and treatment planning (Mick, Spencer, Wozniak, & Biederman, 2005). A related limitation is the manner in which BD was addressed, which was based on C-DISC-IV assessments of the *DSM-IV* criteria for this condition. Although the absence of comorbid BD in the current investigation is consistent with findings from other studies using the same structured-interview approach (e.g., MTA study), some have argued that these *DSM-IV* criteria are not developmentally sensitive enough to capture BD in child populations (Wozniak et al., 2005). Thus, this study cannot definitively rule out the presence of pediatric BD in accordance with these developmentally adjusted diagnostic criteria. Another measurement issue is that all of the outcome measures were derived solely on the basis of parent report. The availability of teacher input in future studies would add an important perspective to this matter, as would direct observations of the child's functioning. The cross-sectional nature of the current investigation represents yet another limitation, precluding any examination of a possible causal relationship between the variables of interest. Observing these same variables in the context of a longitudinal design would allow for a more refined mediational analysis and understanding of the developmental pathways through which deficits in the self-regulation of emotion contribute to the increased risk for functional impairment and comorbidity. Given that rating scales were used to measure various comorbid features, the obtained findings cannot be generalized to children with diagnosable comorbid conditions. Likewise, the study's focus on children and adolescents precludes generalization to populations of adults with AD/HD.

Bearing such limitations in mind, the findings from this study nevertheless have important implications for research and clinical practice. For example, previously reported family and twin research findings have raised the possibility that the associations of AD/HD with depression (Cole, Hall, Radzioch, Olson, & Sameroff, 2009) and with ODD/CD (Faraone, Biederman, Mennin, Russell, & Tsuang, 1998) may represent distinct familial subtypes of AD/HD. Given that the current study found emotional lability to be strongly associated with both aggression and depression, deficits in the self-regulation of emotion may serve as a marker for these comorbid outcomes. Thus, emotion regulation may prove to be useful both as a prognostic indicator and as an intermediate phenotype that underlies AD/HD and its familial association with mood and externalizing disorders (Panksepp, 2006). In terms of clinical-practice issues, it would seem especially prudent for practitioners to conduct evaluations that not only address the diagnostic criteria for AD/HD but also screen for potential signs of deficits in emotion regulation skills. The presence or absence of such clinical markers might then be used to subtype AD/HD, which in turn may inform treatment planning. To the extent that treatment can be tailored in this way, future problems may be prevented or at the very least mitigated.

In conclusion, although much remains to be learned about the role played by the self-regulation of emotions among children with AD/HD, findings from the current study shed new light on this matter. In so doing, it is hoped that future research can build on this foundation in ways that lead to an increased understanding of this clinical phenomenon.

Declaration of Conflicting Interests

The author(s) declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

This research was supported in part through grant # NS049067 awarded by the National Institutes of Health to principal investigator, Allison Ashley-Koch.

References

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry, 40*, 57-87.

August, G. J., Realmuto, G. M., MacDonald, A. W., III., & Nugent, S. M. (1996). Prevalence of AD/HD and comorbid disorders among elementary school children screened for disruptive behavior. *Journal of Abnormal Child Psychology: An Official Publication of the International Society for Research in Child and Adolescent Psychopathology, 24*, 571-595.

Barkley, R. A. (2006). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed.). New York, NY: Guilford.

Biederman, J., Faraone, S., Mick, E., Wozniak, J., Chen, L., Ouellette, C., . . . Lelon, E. (1996). Attention-deficit hyperactivity disorder and juvenile mania: An overlooked comorbidity? *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 997-1008.

Biederman, J., Mick, E., & Faraone, S. V. (1998). Depression in attention deficit hyperactivity disorder AD/HD children: "True" depression or demoralization? *Journal of Affective Disorders, 47*, 113-122.

Braaten, E. B., & Rosén, L. A. (2000). Self-regulation of affect in attention deficit-hyperactivity disorder AD/HD and non-AD/HD boys: Differences in empathic responding. *Journal of Consulting and Clinical Psychology, 68*, 313-321.

Cole, P. M., Hall, S. E., Radzioch, A. M., Olson, S. L., & Sameroff, A. J. (2009). Emotional dysregulation and the development of serious misconduct. In S. L. Olson & A. J. Sameroff (Eds.), *Biopsychosocial regulatory processes in the development of childhood behavioral problems.* (pp. 186-211). New York, NY: Cambridge University Press.

Cole, P. M., Zahn-Waxler, C., & Smith, K. D. (1994). Expressive control during a disappointment: Variations related to preschoolers' behavior problems. *Developmental Psychology, 30*, 835-846.

Conners, C. K. (2001). *Conners' Rating Scales–Revised*. New York, NY: Multi-Health Systems.

Cunningham, C. E., & Boyle, M. H. (2002). Preschoolers at risk for attention-deficit hyperactivity disorder and oppositional defiant disorder: Family, parenting, and behavioral correlates. *Journal of Abnormal Child Psychology: An Official Publication of the International Society for Research in Child and Adolescent Psychopathology, 30*, 555-569.

Douglas, V. I. (1988). Cognitive deficits in children with attention deficit disorder with hyperactivity. In L. M. Bloomingdale & J. A. Sergeant (Eds.), *Attention deficit disorder: Criteria, cognition, intervention* (pp. 65-82). London, England: Pergamon.

DuPaul, G. J., Power, T. J., Anastopoulos, A. D., & Reid, R. (1998). *Manual for the AD/HD Rating Scale-IV.* New York, NY: Guilford.

Faraone, S. V., Biederman, J., Mennin, D., Russell, R., & Tsuang, M. T. (1998). Familial subtypes of attention deficit hyperactivity disorder: A 4-year follow-up study of children from antisocial-AD/HD families. *Journal of Child Psychology and Psychiatry, 39*, 1045-1053.

Hinshaw, S. P., & Melnick, S. M. (1995). Peer relationships in boys with attention-deficit hyperactivity disorder with and without comorbid aggression. *Development and Psychopathology, 7*, 627-647.

Hoagwood, K. E., Jensen, P. S., Arnold, L. E., Roper, M., Severe, J., Odbert, C., . . . MTA Cooperative Group. (2004). Reliability of the services for children and adolescents-parent interview. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*, 1345-1354.

Jasti, S., Dudley, W. N., & Goldwater, E. (2008). SAS macros for testing statistical mediation in data with binary mediators or outcomes. *Nursing Research, 57*, 118-122.

Jensen, P. S., Hoagwood, K. E., Roper, M., Arnold, L. E., Odbert, C., Crowe, M., . . . Wells, K. (2004). The services for children and adolescents-parent interview: Development and performance characteristics. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*, 1334-1344.

Jensen, P. S., Martin, D., & Cantwell, D. P. (1997). Comorbidity in AD/HD: Implications for research, practice, and DSM-V. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 1065-1079.

Jensen, S. A., & Rosén, L. A. (2004). Emotional reactivity in children with attention-deficit/hyperactivity disorder. *Journal of Attention Disorders, 8*, 53-61.

Maedgen, J. W., & Carlson, C. L. (2000). Social functioning and emotional regulation in the attention deficit hyperactivity disorder subtypes. *Journal of Clinical Child Psychology, 29*, 30-42.

Martel, M. M. (2009). Research review: A new perspective on attention-deficit hyperactivity disorder: Emotion dysregulation and trait models. *Journal of Child Psychology and Psychiatry, 50*, 1042-1051.

Melnick, S. M., & Hinshaw, S. P. (2000). Emotion regulation and parenting in AD/HD and comparison boys: Linkages with social behaviors and peer preference. *Journal of Abnormal Child Psychology, 28*, 73-86.

Mick, E., Spencer, T., Wozniak, J., & Biederman, J. (2005). Heterogeneity of irritability in attention-deficit/hyperactivity disorder subjects with and without mood disorders. *Biological Psychiatry, 58*, 576-582.

National Institutes of Mental Health (NIMH). (1997). *Diagnostic interview schedule for children–IV*. Bethesda, MD: Author.

Nigg, J. T. (2001). Is AD/HD a disinhibitory disorder? *Psychological Bulletin, 127*, 571-598.

Panksepp, J. (2006). Emotional endophenotypes in evolutionary psychiatry. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 30*, 774-784.

Patterson, G. R., & Capaldi, D. M. (1990). A mediational model for boys' depressed mood. In J. E. Rolf, A. S. Masten, D. Cicchetti, K. H. Nuechterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 141-163). New York, NY: Cambridge University Press.

Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior assessment system for children*. Circle Pines, MN: American Guidance Service.

Skirrow, C., McLoughlin, G., Kuntsi, J., & Asherson, P. (2009). Behavioral, neurocognitive and treatment overlap between attention-deficit/hyperactivity disorder and mood instability. *Expert Review of Neurotherapeutics, 9*, 489-503.

Tannock, R. (2000). Attention-deficit/hyperactivity disorder with anxiety disorders. In T. E. Brown (Ed.), *Attention-deficit disorders and comorbidities in children, adolescents, and adults* (pp. 125-170). Arlington, VA: American Psychiatric Publishing.

Walcott, C. M., & Landau, S. (2004). The relation between disinhibition and emotion regulation in boys with attention deficit hyperactivity disorder. *Journal of Clinical Child and Adolescent Psychology, 33*, 772-782.

Wozniak, J., Biederman, J., Kwon, A., Mick, E., Faraone, S., Orlovsky, K., . . . van Grondelle, A. (2005) How cardinal are cardinal symptoms of pediatric bipolar disorder? An examination of clinical correlates. *Biological Psychiatry 58*, 583-588.

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