An Examination of the Specificity of Motivation and Executive Functioning in ADHD Symptom-Clusters in Adolescence

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Objective Motivation and executive functioning are central to the etiology of attention-deficit/hyperactivity disorder (ADHD). Furthermore, it has been hypothesized that motivation should show specificity of association with ADHD-impulsivity/hyperactivity symptoms, whereas executive functioning should show specificity of association with ADHD-inattention symptoms. This study tests this specificity-hypothesis and extends previous research by conceptualizing motivation to include both reactivity to reward and punishment. Methods Executive functioning was assessed using two different laboratory measures (the Wisconsin-Card-Sort and Stop-Signal Tasks) and motivation was measured using a laboratory measure of sensitivity to reward and punishment (the Point-Scoring-Reaction-Time Task). Results Findings suggested specificity of association between executive functioning and symptoms of inattention, and between motivation and symptoms of impulsivity/hyperactivity. However, support varied across indices of executive functioning. Conclusions Results provide support for multiple component models of ADHD symptoms and extend the literature by providing a theoretically based conceptualization of motivation grounded on developmental neuroscience models of motivated behavior.

Key words ADHD; executive functioning; motivation; punishment; reinforcement sensitivity; reward.

Introduction
Attention-deficit/hyperactivity disorder (ADHD) is a heterogeneous disorder with a complex etiology (Castellanos & Tannock, 2002; Sonuga-Barke & Halperin, 2012). There is evidence suggesting that both motivation and executive functioning are central to the etiology of ADHD, supporting multicomponent models that emphasize both mechanisms (Nigg, Goldsmith, & Sachek, 2004; Sonuga-Barke, 2005). Motivational systems are thought to mediate individual differences in reactivity (e.g., latency, intensity, and duration of response) to incentives, while higher-order executive control processes are thought to be involved in the processing of information to select, execute, and maintain optimal response strategies. It has been hypothesized that motivation and executive functioning should show specificity in their association with ADHD-symptoms clusters, with motivation being related to symptoms of impulsivity/hyperactivity and executive functioning being related to symptoms of inattention (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Nigg, 2010; Sonuga-Barke, Sergeant, Nigg, & Willcut, 2008). There is support for the specificity hypothesis from the few studies that have tested it (Martel, Nigg, & von Eye, 2009; Solanto et al., 2001; Thorell, 2007) though there has also been some evidence to the contrary of specificity (Campbell & von Stauffenberg, 2009). Supporting specificity, Thorell (2007) found that a motivational style characterized by a tendency to prefer small immediate rewards over larger delayed rewards (measured using a delay aversion task) was predictive of impulsivity/hyperactivity symptoms, but not inattention symptoms, and that executive functioning (measured using a composite executive functioning variable composed of performance on the stroop, go/no-go, and digit span tasks) was associated with inattention.
symptoms only. Results such as these suggest that the etiology of ADHD-symptoms involves multiple mechanisms, and that there may be specificity of association between such mechanisms and ADHD-symptom clusters.

Studies testing the specificity hypotheses have two major limitations, one methodological and one conceptual. Methodologically, none of the studies have statistically controlled for one symptom cluster when predicting the other. This is problematic because inattention and impulsivity/hyperactivity symptoms are correlated (Smith, Barkley, & Shapiro, 2007), hence without statistically controlling for one symptom cluster when predicting the other it is difficult to draw firm conclusions about specificity. This methodological limitation may account for inconsistent support for the specificity hypotheses (Campbell & von Stauffenberg, 2009; Martel, Nigg, & von Eye, 2009; Thorell, 2007) because the degree of covariation between symptom clusters is likely to vary from sample to sample. One goal of this study was to address this limitation and test the specificity of motivation and executive functioning by controlling for one symptom cluster when predicting the other.

A second limitation of the literature involves the conceptualization of motivation. The majority of studies that have tested the specificity hypothesis have conceptualized motivation as individual differences in delay aversion tasks. Delay aversion represents a motivational style characterized by negative reactions to delayed reward and a preference for immediate versus delayed rewards. Individual differences in delay aversion tasks have been proposed to result from hypofunctioning dopamine systems that result in altered reactions to reward (Johansen et al., 2009). Hence, conceptualizing motivation as individual differences in delay aversion tasks fits well with substantial evidence of reward-motivation alterations in ADHD (Plichta et al., 2009; Strohle et al., 2008; Volkow et al., 2009). However, emerging evidence from neuroscience research indicates that motivated behavior is influenced by both reward and punishment systems (Ernst & Fudge, 2009; Spear, 2011). Applications of neuroscience models of motivated behavior to ADHD suggest that it may be fruitful to expand inquiries about the specificity of motivation to include sensitivity to aversive stimuli in addition to reward sensitivity. Indeed, there is some support in the literature for the view that ADHD is characterized not only by alterations in reward processes, but also by alterations in response to punishment (Iaboni, Douglas, & Ditto, 1997; Luman, van Meel, Oosterlann, & Geurts, 2012). Impulsive behavior can be a function of both strong reactivity to reward or weak reactivity to punishment (Avila, 2001; Patterson & Newman, 1993); hence, a second aim of this study is to expand current specificity models of ADHD symptoms by including two facets of motivation, including sensitivity to reward and sensitivity to punishment.

The Present Study

The goal of the current study is to test the specificity hypothesis in a large community sample. We extend prior research in two important ways. First, we provide a more stringent test of the theoretically expected specificity of associations with impulsivity/hyperactivity and inattention symptoms by statistically controlling for one symptom cluster when predicting the other. Second, we invoke a model of motivation with strong theoretical links to emerging developmental neuroscience views of motivated behavior (Ernst & Fudge, 2009; Spear, 2011) to provide a framework to guide our conceptualization of motivational mechanisms germane to ADHD symptom clusters (sensitivity to reward and sensitivity to punishment).

The following hypotheses are proposed: (1) Executive functioning (poor set-shifting and inhibition) will predict high levels of ADHD-inattention symptoms (above and beyond hyperactivity/impulsivity symptoms), and (2) motivational variables (hypersensitivity to reward and hyposensitivity to punishment) will predict high levels of ADHD-impulsivity/hyperactivity symptoms (above and beyond inattention symptoms).

Materials and Methods

Participants

The sample was drawn from the second wave of a longitudinal project examining risk factor for substance use initiation. The sample was recruited via random digit dial phone calls to homes in Erie County, NY. Eligibility criteria required the child to be 11–12 years of age. We used the second wave of data because some of the laboratory tasks of interest in this study were not administered at wave 1. The attrition rate from wave 1 to wave 2 was 4%, and at wave 2 the sample consisted of 373 families (1 caregiver; 1 child). Children’s mean age was 13.13 years (standard deviation = 6.0). The sample compares well with Erie County on a variety of demographic characteristics. Based on the 2006–2008 estimates from the American Community Survey conducted by the U.S. Census Bureau (2010), the sample is 83% Caucasian and 9% African American, compared with 79% and 13%, respectively, for Erie County. Our sample has slightly more females (55% vs. 49%) and more married couple families (76% vs. 65%), but slightly fewer female-headed families (21% vs. 28%) than in Erie County. The median family income in the sample was
In an effort to assess the proportion of the sample that meets symptoms consistent with ADHD, we recoded the data so that scores of 3 or 4 on items would be considered as endorsing an item, while scores of 1 or 2 would be considered nonendorsement of an item. Using this approach, 1.61% of our sample would meet symptoms consistent with ADHD-combined type, 5.36% would meet symptoms consistent with ADHD-predominantly inattentive type, and 0.54% would meet symptoms consistent with ADHD-predominantly hyperactive-impulsive type. These estimated prevalence rates are consistent with epidemiological research suggesting that by adolescence the prevalence of ADHD in community samples ranges from 1% to 5.6% (Smith, Barkley, & Shapiro, 2007). For all analyses, we used the dimensional scoring of ADHD symptoms by summing items within each symptom cluster to represent the full range of ADHD symptomatology.

**Motivation**

Point Scoring Reaction Time Task for Children Revised (PSRT-CR). This measure was adapted by Colder and colleagues (2011) to be used with children and resembles the original task developed with adults (Avila, 2001). Participants were instructed to discriminate between two-digit odd and even numbers presented below a colored circle by pressing the appropriate button on the response box and to work as quickly and accurately as possible. Four experimental blocks were presented in a fixed order: Practice trials, no-reward, reward/prepunishment, reward/punishment, and reward/postpunishment. The rationale for starting the task with a no-reward condition is to obtain a baseline comparison that is not contaminated by the expectation of reward. Children received feedback after each trial in the form of an X (incorrect answer) or O (correct answer). In addition, the child’s points earned per response and total accumulated points for the task were displayed at the bottom of the computer screen at the end of each trial. During the no-reward block (Block 1), participants were asked to respond as quickly as they could and were told that they would not be able to earn points for correct responses but could lose 2 points for incorrect responses. Losing two points for incorrect discrimination remained in effect for all blocks and participants were informed of this. During the reward block (Block 2), participants were told that now they would be able to earn points for correct responses and that the faster they responded the more points they would earn. During the punishment block (Block 3), participants were instructed to inhibit their response if a white circle appeared above the two-digit number and that responses to white circles would cost half of the points they had earned thus far. If the circle was not white instructions repeated those for Block 2.
(able to earn points for correct responses and faster responses earned more points). During the post-punishment block (Block 4) participants were instructed to ignore the colored circles and respond on all trials, even trials with white circles. The rest of the instructions repeated those in Block 2.

Change in reaction time in the reward block (Block 2) compared with the no-reward block (Block 1) indicates motivation for reward. Large declines in Reaction Time (RT) in the reward block indicate strong sensitivity for reward. To obtain a behavioral measure of reward motivation, average RTs from Block 2 (reward) were subtracted from average RTs in Block 1 (no-reward). Thus, high scores represent strong sensitivity to reward.

White circles are established as a punishment cue (losing ½ of accumulated points) in Block 3 (the punishment block). Despite being told to respond to all trials (even white circle trials) in Block 4 (the postpunishment block), slow RTs are expected during trials in which a white circle is presented because of the association between white circles and potential punishment established in the prior block. The degree to which RTs increase on white circle trials compared with nonwhite circle trials in Block 4 (the postpunishment block) represents the strength of punishment sensitivity. Using trials from Block 4 (postpunishment block), average RT of the nonwhite circle trials that immediately preceded each white circle trial was subtracted from the average RT during white circle trials. We used trials that immediately preceded each white circle trial as a comparison condition to control for serial position of trials in the block. High scores on this measure represent strong sensitivity to punishment. Prior work has shown that these task indices are associated with externalizing and internalizing symptoms, and parent reports of reward/punishment sensitivity as expected, supporting the validity of this measure (Colder & O’Connor, 2004; Colder et al. 2011; Rhodes et al., In press).

Executive Functioning

The Wisconsin Card Sorting Test. The computerized version of the Wisconsin Card Sorting Test (WCST, Heaton, Chelune, Talley, Kay, & Curtiss, 2003) a measure of set-shifting, consists of stimulus cards that depict figures that vary in form (crosses, circles, triangles, or stars), color (red, blue, yellow, or green), and number (one, two, three, or four). Participants are instructed to sort the cards without being informed of the correct sorting criteria (i.e., whether they should sort by form, color, or number). Participants were never informed of the correct sorting principle. After 10 consecutive correct responses the sorting principle is changed (without comment or any other indication). In the current study, we used Percent Perseverative Errors as an index of set-shifting. Perseverative errors are when the participant fails to shift strategies in response to changing environmental conditions, or in other words when participants continue to sort cards according to a sorting principle that is no longer correct. Previous work on the latent structure of the WCST using percent perseverative errors as the primary indicator has shown this indicator to be a valid measure of set-shifting (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000).

The Stop Signal Task

The stop signal task (SST; Logan, Schachar, & Tannock, 1997), a measure of behavioral inhibition, assesses the ability to inhibit a prepotent (or dominant) response. This measure involves a “go” task and a “stop” task. The “go” task is a choice reaction time task that requires participants to discriminate between arrows pointing left and arrows pointing right (i.e., pressing a “left” button in response to an arrow pointing left and pressing a “right” button in response to arrows pointing right). The “stop” task involves presentation of a tone (a stop signal) that tells participants to inhibit their response to the “go” signal. This task involved 1 practice “go” block (32 trials) and 1 practice “stop” block (32 trials). After completion of the practice blocks, participants complete three experimental blocks with 64 trials each. The time interval between the “go” and the “stop” signal (the stop-signal delay) is initially set to 250 ms. The stop signal delay is then modified to vary according to the participant’s performance; if the participant is capable of inhibiting the stop signal, delay increases by 50 ms and if the participant is unable to inhibit the stop signal delay decreases by 50 ms. The primary stop task score is the stop signal reaction time (SSRT). The SSRT is computed by subtracting the stop signal delay (the delay between the stop signal and go signal) from the mean reaction time (the average time it took to respond to the go signal). Larger SSRT scores are indicative of poor inhibitory control, while smaller SSRT scores are indicative of good inhibitory control.

Results

Correlations, means, and standard deviations for all variables are shown in Table I. Executive (set-shifting and inhibition) and motivational (sensitivity to reward and punishment) variables were mostly unrelated, providing some statistical support that our measures were assessing different constructs. Also notable was the lack of association between the two executive functioning variables, which is consistent with the literature and theoretical
accounts that view executive cognitive functioning as “fractionable” (Best & Miller, 2010; Lehto, Juujarvi, Kooistra, & Pulkkinen, 2003; Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). A single sample t-test of the means of the sensitivity to punishment and sensitivity to reward variables suggested that both were significantly different from 0 ($p < .01$). Thus, on average, the introduction of reward activated reward motivation as indicated by faster reaction times in the reward compared with the no-reward condition, and the presence of a punishment cue (a white circle) activated motivation to avoid punishment as indicated by slower reaction times on white circle trials compared with the nonwhite circle trials in the postpunishment block.

To test our hypotheses, we used regression in Mplus using maximum likelihood robust estimation to accommodate the nonnormality of the dependent measures (Finch, West, & MacKinnon, 1997; Ke-Hai & Bentler, 2000; Muthen & Muthen, 2008). Set-shifting, inhibition, sensitivity to reward, and sensitivity to punishment are the independent variables in the models. Age and gender were also included as statistical control variables. Separate models were used to predict ADHD symptoms of inattention and ADHD symptoms of impulsivity/hyperactivity. Parallel analyses were conducted with and without including the other symptom cluster in the model (i.e., predicting inattention symptoms above and beyond the effect of impulsivity/hyperactivity symptoms, and vice versa). We also ran our models with, and without adding our motivational and executive functioning variables as a block to assess the increment in $R^2$.

**Inattention Symptoms**

Regression results are presented in Table II. SSRT was also associated with inattention symptoms, such that poor inhibitory control (high SSRT scores) was associated with high levels of inattention symptoms. Neither sensitivity to punishment nor sensitivity to reward significantly predicted ADHD-inattention symptoms. Including impulsivity/hyperactivity symptoms as a predictor in the model yielded the same pattern of result, with the exception that age became significantly associated with inattention. The effects for SSRT, sensitivity to punishment, and sensitivity to reward remained unchanged, at least with respect to statistical significance. Increment in $R^2$ attributable to motivational and executive functioning variables ranged between .02 and .05, indicating small effect sizes.

**Impulsivity/Hyperactivity**

Regression results are presented in Table III. The laboratory measures of sensitivity to punishment and sensitivity to reward were associated with impulsivity/hyperactivity symptoms, such that high levels of sensitivity to punishment and sensitivity to reward were associated with high levels of impulsivity/hyperactivity symptoms. Executive functioning (neither set-shifting or inhibition) variables were not significantly associated with impulsivity/hyperactivity symptoms. Including inattention symptoms as a covariate in the model resulted in the laboratory measure of the sensitivity to reward dropping to marginal significance ($p = .057$), whereas the effect of sensitivity to punishment remained statistically reliable. Increment in $R^2$ attributable to motivational and executive functioning variables ranged between .03 and .05, indicating small effect sizes.

**Discussion**

There were two goals of this study: (1) Test the specificity of association between motivation and executive functioning in the prediction of ADHD-symptom domains.
(inattention and impulsivity/hyperactivity), and (2) expand theoretical views of motivation by including both reward and punishment motivation. Results offered some support for specificity of associations with ADHD symptom clusters and for the role of both reward and punishment motivation in the etiology of ADHD symptoms.

There was evidence that inhibition (as measured by SSRT in the SST) was associated with inattention symptoms, but not with impulsivity/hyperactivity symptoms and this was true even when statistically controlling for impulsivity/hyperactivity symptoms. Even though bivariate correlations suggested that set-shifting was associated with inattention symptoms, the effect of set-shifting was not significant in our multivariate models. According to Barkley (1997), the executive function of inhibition is a necessary prerequisite for the adequate functioning of other executive functions (e.g., set-shifting) and thus may be the dominant executive function associated involved in ADHD. Our data are consistent with Barkley’s hypothesis and suggest that set-shifting is not associated with inattention symptoms above and beyond inhibition. Overall, these findings suggest that poor executive functioning, response inhibition in particular, may be a core feature of ADHD-inattention symptoms.

Our findings suggested mixed support for the specificity of association between sensitivity to reward and impulsivity/hyperactivity symptoms. Sensitivity to reward was positively associated with ADHD symptoms of impulsivity/hyperactivity. This is consistent with previous research that has found individual differences in sensitivity to reward to correlate with ADHD (Gomez & Corr, 2010; Huntd, Kimbrel, Mitchell, & Nelson-Gray, 2008; Mitchell & Nelson-Gray, 2006). Zero-order correlations suggested that sensitivity to reward was associated with both inattention and hyperactivity/impulsivity symptoms. However, in the context of our multivariate models, sensitivity to reward only predicted impulsivity/hyperactivity symptoms, albeit this effect fell just short of conventional levels of statistical significance ($p = .057$) after controlling for inattention symptoms.

Specificity of motivational processes was also found for individual differences in sensitivity to punishment. Sensitivity to punishment was also positively associated with ADHD symptoms of impulsivity/hyperactivity, even when we included inattention symptoms as statistical control variable in the model. This association was surprising and inconsistent with theoretical expectations that ADHD is a disorder marked by deficient reactions to aversive stimuli (Quay, 1997). However, our results are consistent with a recent study that found that a strong punishment system is associated with ADHD symptoms of impulsivity/hyperactivity (Gomez & Corr, 2010). The emotional consequence of sensitivity to punishment are worry and anxiety (Smillie, Pickering, & Jackson, 2006), and other studies have found temperament/personality traits with a worry/anxiety component to be positively associated with ADHD symptoms (e.g., neuroticism see Martel, Nigg, & von Eye, 2009; Martel, Nikolas, Jernigan, Friderici, & Nigg, 2010; Miller, Miller, Newcorn, & Halperin, 2008; negative emotionality see Cukrowicz, Taylor, Schatschneider, & Iacono, 2006; Gomez & Corr, 2010; Martel, Nigg, & von Eye, 2009). Individual differences in reaction to aversive stimuli can lead to dysregulated impulsive behavior because individuals high in sensitivity to punishment...
have an attentional bias to certain emotional cues (Wallace, Bachorowski, & Newman, 1991), decreasing capacity to process other environmental cues that would facilitate behavioral regulation. This perspective is consistent with the high rates of comorbidity between ADHD and anxiety disorders observed in both clinic-referred (Biederman, Newcorn, & Sprich, 1991; Tannock, 2000) and community samples (Angold, Costello, & Erkanli, 1999). The two conditions may share etiological factors.

**Limitations, Conclusions, and Clinical Implications**

Limitations of the current study include the cross-sectional design, a sample with limited ethnic diversity, small effect sizes, and lack of collateral teacher report of ADHD symptoms. Additionally, research has suggested that there are separable components of executive functioning (Lehto, Juujarvi, Kooistra, & Pullkinen, 2003; Miyake, Friedman, Emerson, Wiszki, & Howarter, 2000) that include set-shifting, inhibition, and updating and monitoring of working memory representations. Future work should investigate this third facet of executive functions, as working memory has become prominent feature in some models of ADHD (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005). Finally, our use of a community sample has both strengths and limitations. Although community samples naturally have low rates of clinical diagnosis, they include the full range of symptomatology, whereas clinical samples typically only include the extreme range of symptoms. Although assessing the full range of symptoms is particularly important in ADHD owing to taxometric evidence indicating that the condition is best represented as a continuum of symptom severity (e.g., Marcus & Barry, 2011), it will be important for future studies to replicate our findings in clinical samples.

In conclusion, the present study provides support for the view that different processes may be involved in the etiology of different symptom clusters of ADHD, and that with respect to motivation, it is important to consider sensitivity to both reward and punishment. Heterogeneity in the etiology of ADHD suggests that it may be important for future studies to use person-centered approaches to advance intervention efforts because youth with different symptom and etiological profiles may respond differently to various intervention strategies.

Heterogeneity of etiology suggests that using functional behavioral analysis techniques (DuPaul & Ervin, 1996) to assess whether some youth respond better to reinforcement or response cost strategies may be a particularly useful treatment approach. Such efforts may be refined by the development of norms for measures of inhibition, sensitivity to reward, and sensitivity to punishment. Our findings suggest that normed measures of these three individual differences may help clinicians focus their behavioral treatment efforts to maximize successful clinical outcomes. For example, when using a token economy for behavior management, a ratio of incentives favoring earning points for appropriate behaviors may be most effective for individuals high in reward sensitivity, whereas a ratio favoring response cost may be important for those high in punishment sensitivity. Similarly, the effects of stimulant medications could potentially vary as either a function of reward sensitivity and/or executive functioning. Although speculative, recently proposed interventions to improve working memory (Diamond, 2012; Klingberg, 2012) may be better targeted at etiological profiles that are predominantly driven by deficits in executive functioning.

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