

Neurocognitive Characteristics of Youth with Noncomorbid and
Comorbid Forms of Conduct Disorder and Attention Deficit
Hyperactivity Disorder

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Deposited 06/08/2021

Citation of published version:

Glenn, A., et al. (2017): Neurocognitive Characteristics of Youth with Noncomorbid and Comorbid Forms of Conduct Disorder and Attention Deficit Hyperactivity Disorder. *Comprehensive Psychiatry*, Volume 77.

This is an accepted manuscript. The fully published version can be found at:

DOI: <https://doi.org/10.1016/j.comppsy.2017.06.005>

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Manuscript Details

Manuscript number **COMPRPSYCHIATRY_2017_53**

Title **Neurocognitive Characteristics of Youth with Noncomorbid and Comorbid Forms of Conduct Disorder and Attention Deficit Hyperactivity Disorder**

Article type **Research Paper**

Abstract

Objective: Studies investigating neurocognitive deficits in youth with Conduct Disorder (CD) and attention deficit hyperactivity disorder (ADHD) are often confounded by the high rates of comorbidity between the two. **Method:** Neurocognitive functioning was examined in three diagnostic groups (ADHD only, CD only, comorbid ADHD and CD) matched by age, sex, IQ, and medication status (n = 28-32 per group). **Results:** No significant differences emerged between the diagnostic groups on measures of risk-taking or response inhibition. Children with CD performed better on a measure of spatial planning than those with comorbid ADHD and CD, and dimensional analyses in the full sample (n = 265) revealed a small association between ADHD symptoms and poorer spatial planning. **Conclusion:** These results suggest that deficits in spatial planning may be more pronounced in individuals with ADHD, but that the neurocognitive functioning of youth with noncomorbid and comorbid CD and ADHD are largely similar.

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Suggested reviewers **Yu Gao, Eva Kimonis, Hedwig Eisenbarth, Jill Portnoy, Inti Brazil, Frances Chen**

Submission Files Included in this PDF

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Supplemental Dimensional Analyses Table.docx [Supporting File]

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January 27, 2017

Dear Dr. Markowitz,

We would like to submit the attached manuscript entitled, “Neurocognitive Characteristics of Youth with Noncomorbid and Comorbid Forms of Conduct Disorder and Attention Deficit Hyperactivity Disorder” for consideration for publication in *Comprehensive Psychiatry*. In this study, we examine the similarities and differences between youth with ADHD only, Conduct Disorder only, and those comorbid for these disorders on three different aspects of neurocognitive functioning – spatial planning, risk-taking, and response inhibition. We found no significant differences between the diagnostic groups on risk-taking or response inhibition. Youth in the comorbid group performed worse on spatial planning than those with Conduct Disorder only. In addition, we conducted dimensional analyses of symptom severity and found that ADHD symptoms were associated with poorer spatial planning abilities, though effects were small. These results suggest that although deficits in spatial planning may be more pronounced in individuals with ADHD, the neurocognitive functioning of youth with noncomorbid and comorbid CD and ADHD are largely similar. These results support the idea that common neurocognitive mechanisms may underlie the development of CD and ADHD and may raise questions about whether these diagnoses should be categorized in separate chapters in DSM-5.

The manuscript, in whole or substantial part, has not been previously published elsewhere, and is not currently being considered by other journals, nor will it be while it is under consideration of *Comprehensive Psychiatry*. There are no papers related to the work presented in this manuscript that have been published or are under consideration elsewhere.

Sincerely,

Andrea L. Glenn, Rheanna J. Remmel, Min Yee Ong, Nikki SJ Lim, Rebecca P. Ang, Nicole Ryerson, Hunter Threadgill, Adrian Raine, Daniel Fung, and Yoon Phaik Ooi

Neurocognitive Characteristics of Youth with Noncomorbid and Comorbid Forms of
Conduct Disorder and Attention Deficit Hyperactivity Disorder

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Abstract

Objective: Studies investigating neurocognitive deficits in youth with Conduct Disorder (CD) and attention deficit hyperactivity disorder (ADHD) are often confounded by the high rates of comorbidity between the two. **Method:** Neurocognitive functioning was examined in three diagnostic groups (ADHD only, CD only, comorbid ADHD and CD) matched by age, sex, IQ, and medication status (n = 28-32 per group). **Results:** No significant differences emerged between the diagnostic groups on measures of risk-taking or response inhibition. Children with CD performed better on a measure of spatial planning than those with comorbid ADHD and CD, and dimensional analyses in the full sample (n = 265) revealed a small association between ADHD symptoms and poorer spatial planning. **Conclusion:** These results suggest that deficits in spatial planning may be more pronounced in individuals with ADHD, but that the neurocognitive functioning of youth with noncomorbid and comorbid CD and ADHD are largely similar.

Keywords: neurocognitive, ADHD, conduct disorder, oppositional defiant disorder, spatial planning

1. Introduction

High rates of comorbidity between childhood conduct disorder (CD), oppositional defiant disorder (ODD), and attention deficit hyperactivity disorder (ADHD) suggest a shared etiology among these disorders (Burt, Krueger, McGue, & Iacono, 2001; Thapar, Harrington, & McGuffin, 2001). There has been recent interest in examining underlying mechanisms that may account for the high rates of comorbidity between these disorders. A number of studies have examined neurocognitive functioning in youth with these diagnoses, but studies of ODD and CD have often been confounded by ADHD comorbidity and vice versa (Hobson, Scott, & Rubia, 2011; Lilienfeld & Waldman, 1990; Matthys, Vanderschuren, & Schutter, 2013). It remains unclear whether youth with noncomorbid forms of ODD/CD have the same neurocognitive deficits as those with ADHD only, and how deficits may differ in youth comorbid for these disorders.

Interestingly, although ADHD and ODD/CD were previously included in the same chapter of DSM-IV (APA, 1994), which included all diagnoses usually first made in infancy, childhood, or adolescence, in the update to the DSM (DSM-5; APA, 2013), ADHD was placed in the neurodevelopmental group of disorders, whereas ODD and CD were placed in the chapter on disruptive, impulse-control and conduct disorders. The categorization of ADHD as a neurodevelopmental disorder was made “to reflect brain developmental correlates with ADHD” (APA, 2013). Although this implies that there are neurocognitive factors specific to ADHD that are not found in ODD/CD, it is unclear from current research whether this is the case. Improving our understanding of the similarities and differences between ADHD and ODD/CD at the neurocognitive level may aid in determining whether the brain developmental correlates are significantly different enough to warrant classification into distinct categories in future iterations of the DSM.

It has been hypothesized that ODD/CD may be associated with greater impairments in the domains of motivational control and risk-taking, whereas ADHD may be associated with deficits in executive functions such as planning, set-shifting, and behavioral inhibition (Rubia, 2011), but this hypothesis has not been directly tested. Furthermore, it is unclear how these deficits may manifest in youth with comorbid ODD/CD and ADHD. On one hand, having both disorders may serve as a “double

hit,” resulting in more severe or widespread deficits (Matthys, van Goozen, de Vries, Cohen-Kettenis, & van Engeland, 1998). On the other hand, some studies have shown that comorbid youth perform better on some neurocognitive tasks than individuals with ADHD only (Oosterlaan, Scheres, & Sergeant, 2005; Schachar, Mota, Logan, Tannock, & Klim, 2000).

Four prior studies have compared neurocognitive functioning in all three diagnostic groups: ADHD-only, ODD/CD-only, and ADHD+ODD/CD (Humphreys & Lee, 2011; Oosterlaan et al., 2005; Schachar et al., 2000; Schoemaker et al., 2012). These studies each assessed different aspects of neurocognitive functioning (Humphreys & Lee, 2011: risk-taking; Oosterlaan et al., 2005: verbal fluency, working memory, and planning; Schachar et al., 2000: inhibitory control; Schoemaker et al., 2012: inhibition, working memory). However, these aspects of neurocognitive functioning have not been examined in a single study. The purpose of the present study was to examine neurocognitive functioning in these three diagnostic groups. We focused on the domains of planning, inhibition, and risk-taking in order to capture deficits that have been hypothesized to be specific to each disorder (Rubia, 2011).

1.1. Risk taking

Increased risk-taking is commonly described as a feature of both ODD/CD and ADHD, yet findings on tasks assessing risk-taking in these groups are inconsistent. Groen et al. (2013) reviewed fourteen studies that examined performance on gambling tasks, which are a common way of assessing risk-taking, in youth with ADHD. They found that only half of the studies demonstrated evidence that youth with ADHD take more risks on these tasks compared to normal controls. Two of these studies examined comorbid ODD/CD and both demonstrated that comorbidity increases risky behavior. Matthys et al. (1998) found that risk taking on the Door Opening Task (Daugherty & Quay, 1991) was elevated in boys with CD only compared to normal controls, and even further elevated in boys with comorbid CD and ADHD, but the study did not examine boys with ADHD only. Humphreys and Lee (2011) assessed risk taking in all three diagnostic groups using the Balloon Analog Risk Task. They found that youth with comorbid ADHD+ODD took the most risk, followed by the ODD group, ADHD group, and control group, respectively. This study suggests that ODD may be more associated with risk-taking than ADHD.

On the Iowa Gambling Task, Hobson et al. (2011) found that youth with ODD/CD-only and youth with ADHD \pm ODD/CD sampled more from the risky decks than controls. However, dimensional analyses showed that ODD/CD symptoms, but not ADHD symptoms, were related to increased risky decision-making. This further suggests that risk taking may be more associated with ODD/CD. Other studies suggest that the relationship between risk-taking and ADHD may be more nuanced. Kroyzer, Gross-Tsur, & Pollak (2014) found that on a modified version of the Cambridge Gambling Task (CGT), adolescents with ADHD chose unfavorable outcomes more frequently than typically developing controls, but also made smaller bets (i.e., risked less). Further, they did not show deficits in decision speed or risk adjustment, meaning they decreased the amount bet as they chose less likely outcomes. Overall, the ADHD group did perform more poorly on the task than the control group, but this was not due to impulsivity or insensitivity to the concept of probability. However, it should be noted that 41% of the ADHD group also had ODD or CD, although the authors state that the presence of behavior disorders had no significant effect on any of the dependent measures of the CGT. In sum, it is unclear the degree to which risk-taking may differ in youth with noncomorbid versus comorbid forms of ODD/CD and ADHD.

1.2. Response inhibition

Deficits in inhibition have been described as features of both ADHD and CD. Inhibition has been described as a primary deficit in ADHD, and is also described as a feature of ODD and CD. One of the most widely used tasks to assess response inhibition is the Stop Signal Task (SST), which is used in the present study. The SST measures the ability to cancel an ongoing speeded motor response. An early meta-analysis found that deficits in response inhibition as measured by the SST were present in youth with ADHD and also in youth with disruptive behavior disorders without comorbid ADHD (Oosterlaan, Logan, & Sergeant, 1998). However, studies conducted since then have suggested that deficits in response inhibition may be specific to ADHD. Schachar et al. (2000) directly compared youth diagnosed with ADHD only, CD only, or comorbid ADHD/CD with normal control children on the stop signal task. They found that the ADHD-only group had significantly impaired performance on the task compared to the other three groups. Similarly, in a non-referred sample of school-aged boys, Avila et al. (2004) found performance on the SST and other measures of inhibitory control to correlate with ADHD but not ODD

symptoms. A more recent meta-analysis of studies using SST found that across over 9,000 study participants, participants who had ADHD demonstrated medium deficits on stop signal reaction time, but participants who had ODD/CD without comorbid ADHD showed only small deficits in reaction time (Lipszyc & Schachar, 2010). The deficits of the comorbid group were in between the two. The authors speculate that ODD/CD may phenotypically resemble ADHD, but that these individuals may not have the cognitive deficits that are associated with ADHD.

However, other studies have identified deficits on the SST in youth with ODD/CD without ADHD. Hobson et al. (2011) examined youth with only ODD/CD and youth with ADHD with or without ODD/CD (combined into one group) compared to normal controls. They found that both diagnostic groups demonstrated deficits in performance on the SST compared to controls. Dimensional analyses revealed that ODD/CD symptoms were related to slower inhibitory processes on the SST independently of ADHD. Similarly, in a community sample of adolescents with conduct problems, Herba et al. (2006) found deficits in performance on the SST. These differences in performance were observed when individuals with comorbid hyperactivity were excluded from analyses, suggesting that the deficits are not attributable to comorbid ADHD. Finally, Schoemaker et al. (2012) found deficits in inhibition in preschoolers with ODD/CD, but differences were carried mostly by a task in which tangible rewards were used, and deficits were not as pronounced as in the group with comorbid ADHD and ODD/CD.

In contrast to these findings, a recent study found deficits on the SST in adolescents with comorbid ADHD + ODD, but no deficits in adolescents with ADHD only (Noordermeer et al., 2015). Thus, findings regarding the specificity of response inhibition deficits to either ADHD or ODD/CD are mixed.

1.3. Spatial planning

Spatial planning is an executive function that is thought to rely heavily on frontal lobe functioning (Baker et al., 1996; Cazalis et al., 2003). Deficits in spatial planning have been associated with greater severity of ADHD symptoms (Chiang, Huang, Gau, & Shang, 2013). Poorer spatial planning abilities in youth with ADHD have been associated with more negative peer relationships (Chiang & Gau, 2014) and have been found to mediate the relationship between ADHD and social problems (Tseng & Gau, 2013). Several studies have explored spatial planning in youth with ODD/CD and ADHD but

findings are mixed. Dolan & Lennox (2013) found that on a measure of spatial planning (Stockings of Cambridge; Sahakian & Owen, 1992), youth with comorbid CD+ADHD and youth with CD only performed significantly worse on the measure than healthy controls, but deficits were more pronounced in the comorbid group. They suggest that deficits in planning ability may be limited to youth with ADHD. However, the study did not include a group of participants diagnosed with ADHD only, so they were not able to test this hypothesis directly. Using the same task, Gau and Shang (2010) found that youth with ADHD solved fewer problems in the minimum number of moves and took less time to think before making a move. However, 56% of the sample had comorbid ODD and 19% of the sample had comorbid CD, so it is unclear whether these deficits are attributable to ODD/CD. Barnett, Maruff, and Vance (2009) found that youth with ADHD only and comorbid ADHD +ODD/CD, performed worse than controls on the Tower of London task, another measure of spatial planning. The study did not include a group with ODD/CD only. Similarly, Oosterlaan, Scheres, and Sergeant (2005) found that ADHD was associated with deficits in planning independently of ODD/CD. They suggest that the presence of ADHD accounts for the planning deficits in children with comorbid ADHD and ODD/CD. In comparisons of diagnostic groups, they found that youth with ADHD only performed worse than children with ODD/CD or comorbid ODD/CD and ADHD on the Tower of London task. Neither of the latter groups differed significantly from controls. This is potentially consistent with the study by Dolan & Lennox (2013) which found that those with CD only performed similarly to controls on some aspects of the spatial planning task, whereas those comorbid for ADHD and CD performed worse. Together these studies suggest that deficits in spatial planning may be specific to the diagnosis of ADHD. However, only the study by Oosterlaan, Scheres, and Sergeant (2005) has compared all three diagnostic groups, and sample sizes for each group ranged from 18-22 participants. In the present study, we examine spatial planning abilities in the three diagnostic groups in a larger sample.

1.4. The current study

One challenge in conducting research on these diagnostic groups is that the diagnosis of ODD/CD without comorbid ADHD is much less common than comorbid diagnoses. In the present study, we recruited a larger sample of clinically referred youth in order to be able to conduct comparisons in youth

with ODD/CD only. Although the final sample of this diagnostic group is modest ($n=33$), it is larger than in most of the studies reviewed above. In order to provide appropriate control for potentially confounding variables, we created three matched diagnostic groups that were equivalent on age, gender, IQ, and medication status. Based on the literature reviewed above, we hypothesized that (1) deficits in spatial planning would be greater in youth with ADHD or combined ADHD+ODD/CD than in youth with ODD/CD alone, (2) deficits in response inhibition would be greatest in youth with ADHD and that the comorbid group would more closely resemble the ODD/CD group, (3) risk taking would be most impaired in the comorbid group, followed by the group with ODD/CD-only and then the ADHD-only group. In addition, we also conducted dimensional analyses to examine relationships between neurocognitive functioning and symptoms across the larger sample ($n = 265$).

2. Material and methods

2.1. Participants

Data analyzed for the present study were baseline scores derived from a randomized, placebo-controlled trial (RCT) of an intervention among children with ADHD and/or ODD/CD in Singapore. These children were included in the study if they fulfilled the following inclusion criteria: (a) 7–16 years old; (b) had a clinical diagnosis of ADHD or ODD/CD by the DSM-IV criteria (APA, 1994) as well as met the criteria of ADHD, ODD/CD or both based on the Diagnostic Interview Schedule for Children (C-DISC) (Schaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000); and (c) IQ of 70 and above. Those with serious brain pathology such as epilepsy were excluded. The RCT study was approved by both institutional and hospital group ethics review boards (DSRB A/08/410; CRC 240/2008). All participants and their parents provided informed consent. In this current study, participants included 275 youth (88% male, mean age = 10.61, SD = 1.908, range 7-16 years, 69.8% Chinese). Neurocognitive measures were conducted prior to the beginning of the intervention and therefore represent baseline measures. Ten participants were excluded because they were taking medication other than stimulants (e.g., antidepressant, anti-psychotic). Of the remaining sample, 60.3% were taking stimulant medication.

Of the final sample, 99 participants were diagnosed with ADHD only, 33 participants were diagnosed with CD/ODD only, and 133 participants were diagnosed with comorbid ADHD and CD/ODD (Table 1).

2.2. Measures

2.2.1. Wechsler Intelligence Scale for Children – Fourth Edition

IQ was measured by the Wechsler Intelligence Scale for Children – Fourth Edition (WISC-IV; Wechsler, 2003). For the current study, full-scale IQ scores were calculated for each participant.

2.2.2. Child Behavior Checklist

Conduct and attention problems were measured using the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001) which was completed by parents. The measure includes 118 items that assess the child's behavioral, emotional, and social problems over the past 6 months. Parents rated each item on a 3-point scale (0 = Not True, 1 = Somewhat or Sometimes True, 2 = Very True or Often True). Items on the CBCL can be summed to provide eight 'narrow-band' syndromes (Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule-breaking Behavior, and Aggressive Behavior), two 'broad-band' syndromes (internalizing and externalizing problems), and a total problems scale. For dimensional analyses in the present study, we limited the number of tests performed by selecting the narrow-band Attention Problems syndrome and the broad-band Externalizing Problems syndrome as our primary outcome variables.

2.2.3. Conners' Parent Rating Scale-Revised: Short Form

The 27-item Conners' Parent Rating Scale-Revised: Short Form (CPRS-R:S; Conners, 1997) was used to assess the 12 criteria listed in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994) for ADHD, and also behaviors related to oppositional defiant disorder. Parents rated how much each of the 27 symptoms has been a problem for his or her child during the last month using a 4-point rating scale ranging from 0 (not true at all) to 3 (very much true). Ratings are summed to yield three 6-item subscales: Oppositional, Cognitive Problems/Inattention, and Hyperactivity. The scale also produces a 12-item ADHD index which has been found to discriminate between youth diagnosed with ADHD and controls (Conners, 1997). To limit the number of tests performed for dimensional analyses, we selected

the Oppositional subscale and the ADHD index as primary outcome variables. These scales have been found to be distinct in factor analyses and to have good reliability (Kumar & Steer, 2003).

2.2.4. Cambridge Gambling Task.

The Cambridge Gambling Task (CGT), drawn from the Cambridge Neuropsychological Test Automated Battery (CANTAB; Sahakian & Owen, 1992), assesses decision-making and risk-taking behavior outside of a learning context (e.g., probabilities are explicit). Unlike the Iowa Gambling Task, information retrieval over consecutive trials (i.e., distinguishing a “good” deck from a “bad” deck) is not necessary. On each trial, participants are presented with a row of ten boxes with a mix of red and blue boxes. The number of red or blue boxes differs on each trial (with ratios of 9:1, 8:2, 7:3 and 6:4). The participant must guess whether a token is hidden under a red or blue box. In the gambling stages, participants begin with a number of points and can select a proportion of those points to bet. The correct color choice is rewarded with the number of points bet, whereas the wrong color choice is penalized with the same number of points bet. Five point amounts are displayed in either rising or falling order, and so participants must wait until the amount they want to bet is displayed.

The CGT has a number of outcome variables. In this study, the commonly used outcome variables “risk-taking” and “overall proportion bet” were examined, though see Table S1 for analyses involving secondary outcome variables. Risk taking is the mean proportion of current points that the participant chose to bet on trials in which they chose the more likely outcome. Overall proportion bet is a measure of the average proportion of current points that the participant chose to bet on each trial. The duration of this task is 30 minutes.

2.2.5. Stop Signal Task.

The Stop Signal Task (SST), drawn from the CANTAB (Sahakian & Owen, 1992), is a measure of response inhibition and impulse control. It measures the ability to cancel an ongoing speeded motor response. Participants are shown an encircled arrow, which points either to the left or to the right, at the center of the screen. Participants are told to press the left-hand button when a left facing arrow is shown, and the right-hand button when a right facing arrow is shown. After the first block of 16 trials, participants are instructed to continue responding in this manner, but to inhibit their response if they hear

a beep after the arrow is shown. Participants are to complete five blocks of 64 trials with 16 stop trials per block.

The SST also has a number of outcome variables. In this study, we analyzed data from two commonly assessed outcome variables – “proportion of successful stops” and “stop signal reaction time (SSRT).” Proportion of successful stops is the proportion of times the participant correctly stopped a response. SSRT is an estimate of the length of time between the go and stop stimuli at which the participant is able to inhibit their response on 50% of trials. The duration for this task is 20 minutes.

2.2.6. Stockings of Cambridge.

The Stockings of Cambridge (SOC), drawn from the CANTAB (Sahakian & Owen, 1992), is a measure of spatial planning. Participants are shown two displays, each containing three different colored balls. Participants must move the balls in the lower display to match the pattern shown in the upper display. Balls can only be moved one at a time. On some trials, participants are given a minimum number of moves they must make to answer correctly. There are four difficulty levels with different numbers of minimum moves and with different numbers of trials: two moves minimum, the easiest level, has two trials; three moves minimum has two trials; four moves minimum has four trials; and five moves minimum, the highest difficulty level, has four trials.

The SOC outcome variables include problems solved in minimum number of moves, initial thinking time, and subsequent thinking time. Problems solved in minimum number of moves is a count of problems that the participant is able to solve in the minimum required number of moves. Initial thinking time is the time between the problem being presented and the first move made. Mean subsequent thinking time is the time from after the first move is made to when the problem is solved, divided by the number of moves made.

2.3. Procedure

Following referrals by an attending physician, a trained research assistant approached the families to provide explanation of the study and to obtain parental consent and child assent. Parents were asked to complete the C-DISC, CBCL, CPRS-R:S, and also to provide demographic information about their child. Measure of child’s IQ was then assessed by the abbreviated version of the WISC-IV. Participants then

completed the Cambridge Gambling Task, the Stop Signal Task, and the Stockings of Cambridge Task. Assessments were conducted on two different days. At the initial visit, participants completed the CGT. On a subsequent day they completed the SST and SOC. Task order was the same for each participant.

2.4. Data Processing

Demographic characteristics for the overall sample can be found in Table 1. Because of statistical issues related to including age and IQ as covariates in an ANCOVA due to their overlap with the cognitive processes of interest (Chapman, 2002), we decided that the best way to compare the diagnostic groups would be to create groups that were matched on these variables, as well as on gender and medication status. Matched groups were created for the three diagnostic categories (ADHD, ODD/CD, and comorbid) based on age, gender, IQ, whether or not the participant was taking a stimulant medication, and medication status at the time of testing.

[INSERT TABLE 1]

Matched groups were created using a multi-step process. First, 34 participants were missing IQ scores. In order to maintain the largest sample possible for creating matched groups, we investigated whether it was feasible to impute IQ scores (Tabachnick & Fidell, 2013). Results from the “MissMech” package used in R (Jamshidian, Jalal, & Jansen, 2014) revealed that the IQ data was missing at random (i.e., non-significant nonparametric test of homoscedasticity). Therefore, IQ scores were imputed using a regression-based method. We used participants with complete data within each diagnosis to regress age and gender (dummy-coded) on IQ. Then, age and gender were entered into the regression equation to obtain an estimated IQ score for participants missing IQ data.

Next, medication at the time of testing was taken into consideration for creating the matched groups. Because the neurocognitive tests took place across two different days, some participants reported having taken their medication on one day, but not the other. Therefore, two samples from the overall dataset were created in order to control the variation in medication status at time of testing across the battery of tests.¹ Specifically, a matched group was created for the completion of the Cambridge

¹ It is important to note that these matched groups came from the same dataset. Because there were variations in whether an individual participant reported taking their medication on the day of testing, it was important to create

Gambling Task (completed during session 1) and another matched group was created for the completion of the Stop Signal and Stockings of Cambridge tasks (completed during session 2).

Finally, we created matched groups by using propensity score matching via the “MatchIt” package (Ho, Imai, King, & Stuart, 2007) in R. First, we used automated matching to match on the variables age, gender, IQ, whether or not the participant was taking a stimulant medication, and medication status at time of testing. We then reviewed the resulting matched groups, hand-correcting incomplete matches. This was done for both samples, resulting in 28 participants in each diagnostic category at the first testing session (CGT) and 32 participants in each diagnostic category at the second testing session two (SST and SOC).² Across both sets of matched groups, there was no difference in age, gender, IQ, medication type, or medication status at time of testing (see Tables 2 and 3 for testing differences in demographics for matched groups).

[INSERT TABLES 2 AND 3]

2.5. Data Analysis Plan

First, because the current study did not include a control group, we wanted test whether the behavior problems reported in the current sample were, in fact, significantly greater than those of non-referred youth in order to show that the individuals in these diagnostic groups are significantly different from would-be controls. Table 1 shows the means and standard deviations for the CBCL subscales from a sample of non-referred youth from Singapore (Ang et al. 2012). T-test comparisons revealed that all three diagnostic groups had significantly greater attention problems (all $t > 14.7$, all $p < .001$) and externalizing problems (all $t > 12.07$, $p < .001$) than the non-referred youth.

To test for differences between the three diagnostic groups, we conducted one-way ANOVAs comparing performance on the three neuropsychological tasks. Dimensional analyses were also conducted to examine relationships between symptom scores and performance on the task. These analyses included multiple regression models with the full sample ($n = 265$) investigating the two CBCL scales (Attention

two different samples of matched groups. However, there is a high degree of participant overlap between those used for analyses for tasks administered at testing session one and tasks administered at testing session two.

² Differences in the number of participants for each set of matched groups is due to some participants not reporting whether they had taken their medication that day. Therefore, those participants were dropped from their respective analyses.

Problems, Externalizing Problems) and the two CPRS scales (Oppositional subscale, ADHD index) on the aforementioned outcome variables of the three neurocognitive tasks (CGT, SST, and SOC). Variations in degrees of freedom were due to participants having missing scores for that specific test.

3. Results

3.1. Diagnostic Group Comparisons

3.1.1. Cambridge Gambling Task.

ANOVAs were performed on risk-taking scores and overall proportion bet on the CGT. These variables did not differ between groups ($ps > .68$). Means and standard deviations, as well as ANOVA results, can be found in Table 2.

3.1.2. Stop Signal Task.

ANOVAs were performed on the stop-signal reaction time and the proportion of successful stops in the SST. These variables did not differ between groups ($ps > .48$). Means and standard deviations, as well as ANOVA results, can be found in Table 3.

3.1.3. Stockings of Cambridge.

ANOVAs were performed on the mean initial think time, mean subsequent think time, and problems solved in the minimum number of moves on the SOC. There were no differences between groups for initial thinking time ($p = .35$) or subsequent thinking time ($p = .69$). Problems solved in the minimum number of moves differed significantly between groups, $F(2, 85) = 3.14, p = .05$. Post-hoc analyses revealed that children with CD solved significantly more problems in the minimum number of moves than those with comorbid ADHD/CD, $p = .04, d = .83$. Scores for the ADHD group fell between the CD and comorbid groups, but were not significantly different from either ($ps > .30$) (Table 3).

3.2. Dimensional Analyses

Multiple regression analyses were conducted to assess the relationship between performance on the neurocognitive tasks and specific symptoms measured by the CPRS (Oppositional, ADHD index) and CBCL (Attention problems, Externalizing behavior). Each aforementioned outcome variable from the neurocognitive tasks was analyzed as a dependent variable in two stepwise regression models: one model including the subscales of the CPRS, one model including the subscales of the CBCL. For each stepwise

regression model, control variables (age and IQ) were entered on step 1. Then, the subscales of either the CPRS or the CBCL were entered on step 2. Only significant models are included in the text below (see Table 4 for a full summary of regression analyses). Results from multiple regression models assessing all outcome variables for each neurocognitive task is provided in Table S1.

3.2.1. Cambridge Gambling Task.

There were no significant relationships between the CBCL and CPRS subscales and the outcome variables of the CGT ($ps > .05$).

3.2.2. Stop Signal Task.

There was a significant relationship between proportion of successful stops and the externalizing subscale of the CBCL ($\beta = -.22, p < .01$).

3.2.3. Stockings of Cambridge.

There was a significant relationship between mean subsequent thinking time and the externalizing subscale of the CBCL ($\beta = -.19, p < .01$). There was a significant relationship between total problems solved in the minimum number of moves and the ADHD index of the CPRS ($\beta = -.17, p < .05$).

4. Discussion

In this study, we did not find evidence of significant differences between youth with noncomorbid and comorbid forms of CD and ADHD on measures of risk taking or response inhibition. We did find that children with CD performed better on a measure of spatial planning than those with comorbid CD and ADHD, and that the performance of youth with ADHD fell between these two groups. This finding is consistent with results from Dolan & Lennox (2013) who used the same task in an older sample (mean age = 16.41) and found that adolescents with CD performed better than adolescents with comorbid CD and ADHD, but still performed worse than controls. Although we do not have a control group in the present study, all three diagnostic groups performed significantly worse on the spatial planning task than a control group reported in a study by Gau & Shang (2010), which included 173 youth with a mean age of 12.6 years, slightly older than the present sample. In that sample, the mean problems solved in minimum moves was 8.80 (SD = 1.93), which is significantly more than any of the diagnostic groups in the present study (all $t > 3.3$, all $p < .005$). However, in the only other study to compare all three

diagnostic groups, Oosterlaan, Scheres, and Sergeant (2005) found that youth with noncomorbid ADHD performed worse than other groups on a measure of spatial planning (Tower of London), and that youth with noncomorbid CD and comorbid CD and ADHD performed similarly to controls. Collectively, these results suggest that the deficits in youth with noncomorbid CD are less pronounced than those of individuals with ADHD.

Similar to findings by Oosterlaan, Scheres, & Sergeant (2005) and by Chiang, Huang, Gau & Shang (2013), in dimensional analyses in the larger sample, we found a small association between ADHD symptoms and poorer performance on the spatial planning task. This further supports the idea that spatial planning deficits may be more characteristic of ADHD. Given that poorer spatial planning abilities have been associated with more negative peer relationships (Chiang & Gau, 2014), poorer communication and socialization skills (Clark, Prior, & Kinsella, 2002), and have been found to mediate the relationship between ADHD and social problems (Tseng & Gau, 2013), it may be important to develop interventions that target planning abilities in youth with ADHD.

We did not find differences between the diagnostic groups on response inhibition. However, in dimensional analyses we found that externalizing behavior was associated with a lower proportion of successful stops. We did not observe relationships between ADHD-related symptoms and any of the outcome measures. This finding seems most consistent with the study by Hobson et al. (2011) which found no differences between diagnostic groups, but found that ODD/CD symptoms were related to slower inhibitory processes on the SST independently of ADHD.

The fact that we did not find differences between diagnostic groups on this task does not necessarily mean that the underlying neurobiological functioning does not differ between groups. In order to clarify whether neurobiological deficits are common to ADHD and CD, Rubia et al. (2008) examined brain functioning in boys with ADHD only compared to CD only during a stop signal task. They found that both groups demonstrated reductions in brain functioning relative to controls, but that the regions were not the same for the two groups. Patients with ADHD demonstrated reduced activity in prefrontal regions, whereas patients with CD showed reduced activity in posterior temporal-parietal regions. This study suggests that different neurobiological factors may contribute to deficits in performance in the

different diagnostic groups. In addition, it is also possible that the different diagnostic groups may have difficulties with different aspects of the task. For example, response inhibition has two components. Reactive inhibition occurs when one attempts to cancel an ongoing response. Prospective inhibition occurs when one withholds a response pending a signal to stop. In a sample of 12 adolescents with ADHD (two of whom had comorbid ODD) compared to 12 controls, Bhajiwala, Chevrier, and Schachar (2014) found differences between the groups in brain activity specifically during prospective, but not reactive inhibition on the SST.

Finally, we also did not observe differences between diagnostic groups on risk-taking, as measured by the CGT, and did not find any significant associations in dimensional analyses. One reason why differences in risk taking may not have emerged is that unlike the Balloon Analog Risk task or the Iowa Gambling Task, the Cambridge Gambling task is an explicit gambling task. In gambling tasks, participants typically choose between several options that differ in the probability of a reward or penalty. The exact probability distribution of the outcome can be evident for the participant (explicit) or not (implicit). Implicit gambling tasks include the Balloon Analog Risk Task (BART), the Card Playing Task, the Door Opening Task, and the Iowa Gambling Task (IGT). Explicit tasks include the Cambridge Gambling Task, the Game of Dice Task, and the Probabilistic Discounting Task. Implicit gambling tasks are thought to rely on decision-making involving emotional and affective responses as well as on rational and cognitive determinations of risk (Groen et al., 2013). Explicit gambling tasks are more focused on the rational and cognitive aspects of decision-making because the probability distributions are known. However, when examining studies of ADHD in youth, Groen (2013) did not observe differences in effects for studies examining implicit versus explicit gambling tasks.

In sum, results from the present study suggest that neurocognitive deficits do not differ widely simply as a function of diagnosis, though there is some evidence that deficits in spatial planning are more pronounced in individuals with ADHD. These results may raise questions about whether these diagnoses should be categorized in separate chapters in DSM-5. Evidence from other studies suggests that CD and ADHD may share common deficits. Castellanos-Ryan et al. (2014) examined the unique and common variance across CD, ADHD, and substance misuse using behavioral measures and brain imaging. They

found that ADHD and CD symptoms loaded onto the same factor, and that this factor was associated with impulsivity, poor response inhibition, high delay discounting, and reduced activity in the frontal lobes during failed inhibition. The high rates of comorbidity between CD and ADHD have led to the proposal to replace diagnostic categories with a dimensional, neuroscience-based perspective (e.g., Research Domain Criteria; Cuthbert & Insel, 2013). However, identifying the factors that are unique to ADHD and CD is also important. In a brain imaging study comparing youth with noncomorbid forms of ADHD and CD, Stevens & Haney-Caron (2012) found a 13% reduction in grey matter volume in adolescents with conduct disorder, reflecting numerous frontal, temporal, parietal and subcortical deficits, but did not find these same deficits to be present in youth with ADHD.

The primary limitation of this study is that we do not have data from a healthy control group. Because of this, we can only make conclusions about whether diagnostic groups differ from one another and we do not know whether performance levels are significantly reduced compared to healthy controls. In addition, although our sample of youth with CD only is larger than in most previous studies, the sample size was still relatively small. Finally, although this study focuses on more than one domain of neurocognitive functioning, it is still limited in the range of neurocognitive functioning assessed. Future studies including additional measures, such as including an implicit gambling task that taps into the emotional facet of decision-making would be beneficial.

Overall, this study examines differences in neurocognitive functioning between diagnostic groups in a relatively large sample. In the investigation of the neurocognitive factors associated with externalizing problems, it is important that we accurately characterize the factors that are common and unique to symptoms of conduct disorder and ADHD. Doing so may help us to better understand the underlying dimensions reflecting vulnerability to unique or broad psychiatric outcomes.

Conflicts of interest: None

Compliance with Ethical Standards

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

References

- Ang, R. P., Rescorla, L. A., Achenbach, T. M., Ooi, Y. P., Fung, D. S., & Woo, B. (2012). Examining the criterion validity of CBCL and TRF problem scales and items in a large Singapore sample. *Child Psychiatry Hum Dev, 43*(1), 70-86.
- APA. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- APA. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth ed.). Arlington, VA: American Psychiatric Publishing.
- Association, A. P. (2013). Highlights of Changes from DSM-IV-TR to DSM-5.
- Avila, C., Cuenca, I., Felix, V., Parcet, M. A., & Miranda, A. (2004). Measuring impulsivity in school-aged boys and examining its relationship with ADHD and ODD ratings. *J Abnorm Child Psychol, 32*(3), 295-304.
- Barnett, R., Maruff, P., & Vance, A. (2009). Neurocognitive function in attention-deficit-hyperactivity disorder with and without comorbid disruptive behaviour disorders. *Aust N Z J Psychiatry, 43*(8), 722-730.
- Bhajiwal, M., Chevrier, A., & Schachar, R. (2014). Withholding and canceling a response in ADHD adolescents. *Brain and Behavior, 4*(5), 602-614.
- Burt, S. A., Krueger, R. F., McGue, M., & Iacono, W. G. (2001). Sources of covariation among attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder: The importance of shared environment. *J Abnorm Psychol, 110*(4), 516-525.
- Castellanos-Ryan, N., Struve, M., Whelan, R., Banaschewski, T., Barker, G. J., Bokde, A. L., . . . Consortium, I. (2014). Neural and cognitive correlates of the common and specific variance across externalizing problems in young adolescence. *Am J Psychiatry, 171*(12), 1310-1319.
- Chiang, H. L., & Gau, S. S.-F. (2014). Impact of executive functions on school and peer functions in youths with ADHD. *Res Dev Disabil, 35*(5), 963-972.

- Chiang, H. L., Huang, L. W., Gau, S. S., & Shang, C. Y. (2013). Associations of symptoms and subtypes of attention-deficit hyperactivity disorder with visuospatial planning ability in youth. *Res Dev Disabil, 34*(9), 2986-2995.
- Clark, C., Prior, M., & Kinsella, G. (2002). The relationship between executive function abilities, adaptive behaviour, and academic achievement in children with externalising behaviour problems. *Journal of Child Psychology and Psychiatry, 43*(6), 785-796.
- Cuthbert, B. N., & Insel, T. R. (2013). Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Med, 11*, 126.
- Daugherty, T. K., & Quay, H. C. (1991). Response perseveration and delayed responding in childhood behavior disorders. *J Child Psychol Psychiatry, 32*(3), 453-461.
- Dolan, M., & Lennox, C. (2013). Cool and hot executive function in conduct-disordered adolescents with and without co-morbid attention deficit hyperactivity disorder: relationships with externalizing behaviours. *Psychol Med, 43*(11), 2427-2436.
- Gau, S. S., & Shang, C. Y. (2010). Executive functions as endophenotypes in ADHD: evidence from the Cambridge Neuropsychological Test Battery (CANTAB). *J Child Psychol Psychiatry, 51*(7), 838-849.
- Groen, Y., Gaastra, G. F., Lewis-Evans, B., & Tucha, O. (2013). Risky Behavior in Gambling Tasks in Individuals with ADHD – A Systematic Literature Review. *Plos One, 8*(9), e74909.
- Herba, C. M., Tranah, T., Rubia, K., & Yule, W. (2006). Conduct problems in adolescence: Three domains of inhibition and effect of gender. *Developmental Neuropsychology, 30*(2), 659-695.
- Ho, D. E., Imai, K., King, G., & Stuart, E. A. (2007). Matching as Nonparametric Preprocessing for Reducing Model Dependence in Parametric Causal Inference. *Political Analysis, 15*(3), 199-236.
- Hobson, C. W., Scott, S., & Rubia, K. (2011). Investigation of cool and hot executive function in ODD/CD independently of ADHD. *J Child Psychol Psychiatry, 52*(10), 1035-1043.

- Humphreys, K., & Lee, S. (2011). Risk Taking and Sensitivity to Punishment in Children with ADHD, ODD, ADHD+ODD, and Controls. *Journal of Psychopathology and Behavioral Assessment*, 33(3), 299-307.
- Jamshidian, M., Jalal, S., & Jansen, C. (2014). MissMech: An R Package for Testing Homoscedasticity, Multivariate Normality, and Missing Completely at Random (MCAR). *2014*, 56(6), 31.
- Kroyzer, N., Gross-Tsur, V., & Pollak, Y. (2014). Risk taking in adolescents with attention deficit hyperactivity disorder on a probabilistic choice task. *J Nerv Ment Dis*, 202(3), 247-252.
- Kumar, G., & Steer, R. A. (2003). Factorial Validity of the Conners' Parent Rating Scale-Revised: Short Form With Psychiatric Outpatients. *Journal of Personality Assessment*, 80(3), 252-259.
- Lilienfeld, S. O., & Waldman, I. D. (1990). The relation between childhood attention-deficit hyperactivity disorder and adult antisocial behavior reexamined: The problem of heterogeneity. *Clinical Psychology Review*, 10(6), 699-725.
- Lipszyc, J., & Schachar, R. (2010). Inhibitory control and psychopathology: a meta-analysis of studies using the stop signal task. *J Int Neuropsychol Soc*, 16(6), 1064-1076.
- Matthys, W., van Goozen, S. H., de Vries, H., Cohen-Kettenis, P. T., & van Engeland, H. (1998). The dominance of behavioural activation over behavioural inhibition in conduct disordered boys with or without attention deficit hyperactivity disorder. *J Child Psychol Psychiatry*, 39(5), 643-651.
- Matthys, W., Vanderschuren, L. J., & Schutter, D. J. (2013). The neurobiology of oppositional defiant disorder and conduct disorder: altered functioning in three mental domains. *Dev Psychopathol*, 25(1), 193-207.
- Noordermeer, S. D., Luman, M., Buitelaar, J. K., Hartman, C. A., Hoekstra, P. J., Franke, B., . . . Oosterlaan, J. (2015). Neurocognitive Deficits in Attention-Deficit/Hyperactivity Disorder With and Without Comorbid Oppositional Defiant Disorder. *J Atten Disord*.
- Oosterlaan, J., Logan, G. D., & Sergeant, J. A. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD + CD, anxious, and control children: A meta-analysis of studies with the Stop Task. *Journal of Child Psychology & Psychiatry*, 39(3), 411-425.

- Oosterlaan, J., Scheres, A., & Sergeant, J. A. (2005). Which executive functioning deficits are associated with AD/HD, ODD/CD and comorbid AD/HD+ODD/CD? *J Abnorm Child Psychol*, 33(1), 69-85.
- Rubia, K. (2011). "Cool" inferior frontostriatal dysfunction in attention-deficit/hyperactivity disorder versus "hot" ventromedial orbitofrontal-limbic dysfunction in conduct disorder: a review. *Biol Psychiatry*, 69(12), e69-87.
- Sahakian, B. J., & Owen, A. M. (1992). Computerized assessment in neuropsychiatry using CANTAB: discussion paper. *Journal of the Royal Society of Medicine*, 85(7), 399-402.
- Schachar, R., Mota, V. L., Logan, G. D., Tannock, R., & Klim, P. (2000). Confirmation of an inhibitory control deficit in attention-deficit/hyperactivity disorder. *J Abnorm Child Psychol*, 28(3), 227-235.
- Schoemaker, K., Bunte, T., Wiebe, S. A., Espy, K. A., Dekovic, M., & Matthys, W. (2012). Executive function deficits in preschool children with ADHD and DBD. *J Child Psychol Psychiatry*, 53(2), 111-119.
- Stevens, M. C., & Haney-Caron, E. (2012). Comparison of brain volume abnormalities between ADHD and conduct disorder in adolescence. *Journal of Psychiatry & Neuroscience : JPN*, 37(6), 389-398.
- Tabachnick, B. G., & Fidell, L. S. (2013). *Using multivariate statistics (6th ed.)*. Upper Saddle River, NJ: Pearson.
- Thapar, A., Harrington, R., & McGuffin, P. (2001). *Examining the comorbidity of ADHD-related behaviours and conduct problems using a twin study design* (Vol. 179).
- Tseng, W. L., & Gau, S. S. (2013). Executive function as a mediator in the link between attention-deficit/hyperactivity disorder and social problems. *J Child Psychol Psychiatry*, 54(9), 996-1004.

Table 1
Overall Sample Characteristics

Characteristic	ADHD	CD	ADHD + CD	Non-referred youth in a prior study by Ang et al. (2012)
<i>n</i>	99	33	133	840
Age	10.60 (1.78)	11.39 (2.03)	10.44 (1.93)	9.09 (1.76)
Sex (% male)	85.9	87.9	89.5	67.7
IQ	99.98 (14.11)	101.19 (12.71)	102.19 (16.44)	
Medication status (ADHD stimulant %)	65.7	45.5	60.1	
CBCL				
Attention	11.92 (3.22)	10.32 (3.79)	12.44 (3.31)	3.08 (2.73)
Externalizing	14.22 (7.67)	19.93 (12.52)	21.21 (9.29)	6.43 (5.86)
CPRS				
Oppositional	7.30 (4.05)	9.35 (4.99)	11.08 (4.23)	
ADHD	23.06 (6.99)	19.86 (9.38)	25.96 (6.36)	

Note. Standard deviations are in parentheses. Data from non-referred youth are meant for comparison purposes and are from the study by Ang et al. (2012) which assessed behavior problems using the CBCL in a large sample of non-referred children ages 6-12 in Singapore. Scores on Attention and Externalizing subscales were significantly greater in all three diagnostic groups in comparison to scores of non-referred youth.

Table 2
Matched Groups Means and Neurocognitive and Behavioral ANOVAs for Testing Session 1

Characteristic	ADHD	CD	ADHD + CD	<i>F</i>	<i>p</i>
<i>n</i>	28	28	28		
Age	10.96 (1.80)	11.43 (2.10)	11.21 (1.91)	.40	.67
IQ	98.54 (9.67)	101.30 (11.48)	102.99 (11.32)	1.20	.31
Medication type (% on ADHD stimulant)	57.1	39.3	46.4	$\chi^2 = 1.81$.40
Medicated at testing (%)	21.4	25.0	28.6	$\chi^2 = .38$.83
CBCL					
Attention	11.88 (3.79)	10.39 (3.95)	13.04 (3.30)	2.98	.06
Externalizing	13.04 (6.74)	19.91 (13.55)	22.04 (8.79)	5.34	<.01
CPRS					
Oppositional	6.62 (3.71)	9.37 (5.02)	10.17 (4.03)	4.80	.01
ADHD index	22.96 (7.09)	19.71 (9.99)	25.46 (5.35)	3.37	.04
CGT					
Risk-taking	.55 (.12)	.55 (.14)	.53 (.18)	.15	.87
Overall proportion bet	.53 (.10)	.51 (.12)	.50 (.16)	.39	.68

Note. Standard deviations are in parentheses.

Table 3
Matched Groups Neurocognitive and Behavioral ANOVAs for Testing Session 2

Characteristic	ADHD	CD	ADHD + CD	<i>F</i>	<i>p</i>
<i>N</i>	32	32	32		
Age	11.03 (1.89)	11.44 (2.05)	11.13 (1.79)	.40	.67
IQ	100.08 (10.80)	101.07 (12.89)	101.35 (11.50)	.10	.90
Medication type (% on ADHD stimulant)	56.3	46.9	46.9	.75	.69
Medicated at testing (%)	15.6	12.5	19.4	.56	.76
CBCL					
Attention	11.00 (2.92)	10.22 (3.83)	12.90 (3.05)	4.97	<.01
Externalizing	11.25 (5.16)	19.77 (12.73)	23.07 (9.96)	11.06	<.001
CPRS					
Oppositional	6.10 (3.07)	9.29 (5.08)	11.37 (4.33)	11.53	<.001
ADHD index	21.66 (8.16)	19.64 (9.48)	25.87 (6.41)	4.51	.01
SST					
SSRT (last half)	264.28 (111.35)	244.92 (103.95)	244.97 (85.70)	.75	.47
Proportion of successful stops	.48 (.15)	.42 (.25)	.45 (.18)	.37	.69
SOC					
Initial thinking time (ms)	2067.56 (1147.70)	3166.82 (4561.66)	2691.74 (2211.04)	1.06	.35
Subsequent thinking time (ms)	987.50 (1015.05)	1144.74 (1938.43)	1326.02 (1575.51)	.37	.69
Problems solved in minimum moves	6.91 (1.55)	7.59 (1.65)	6.41 (2.06)	3.14	.05

Note. Standard deviations are in parentheses.

Table 4
Regression Analyses Demonstrating Association between Neurocognitive Variables and CBCL and CPRS Subscales

	CBCL				CPRS			
	Attention		Externalizing		Oppositional		ADHD index	
	β	<i>p</i>	β	<i>p</i>	β	<i>p</i>	β	<i>p</i>
CGT								
Risk taking	-.133	.06	.095	.18	.113	.16	-.108	.18
Overall proportion bet	-.080	.27	.075	.30	.116	.15	-.087	.29
SOC								
Initial thinking time (ms)	.053	.46	-.075	.30	.035	.66	-.014	.86
Subsequent thinking time (ms)	.066	.34	-.189	<.01	-.070	.36	-.048	.53
Problems solved in minimum moves	-.100	.15	-.003	.97	.041	.56	-.172	.03
SST								
SSRT	.098	.15	-.104	.13	-.123	.11	.066	.94
Proportion of successful stops	.121	.09	-.217	<.01	-.133	.10	.155	.06

Note. Regression analyses control for age and full-scale IQ. Values in bold indicate significant associations

Supplemental Analyses

Table S1. Regression Analyses Demonstrating Association between Neurocognitive Variables and CBCL and CPRS Subscales

	CBCL				CPRS			
	Attention		Externalizing		Oppositional		ADHD index	
	β	p	β	p	β	p	β	p
SOC								
Total problems solved in minimum moves	-.100	.151	-.003	.971	.041	.595	-.172	.028
Number solved in 2 moves*	.124	.087	.029	.686	-.007	.935	.012	.882
Number solved in 3 moves*	.078	.267	-.040	.564	.054	.486	.114	.146
Number solved in 4 moves*	-.067	.357	-.030	.680	.037	.645	.020	.806
Number solved in 5 moves*	.113	.111	.038	.591	-.016	.840	.134	.092
Solved in 2 moves*	-.125	.086	-.017	.817	.027	.737	-.031	.704
Solved in 3 moves*	-.061	.387	-.023	.740	-.063	.416	-.129	.101
Solved in 4 moves*	-.017	.810	.081	.260	.086	.288	-.132	.107
Solved in 5 moves*	-.077	.276	-.060	.387	.022	.781	-.077	.335
Mean initial thinking time	.053	.461	-.075	.301	.035	.664	-.014	.862
Initial thinking time 2-move*	.095	.186	.016	.823	.085	.294	-.012	.885
Initial thinking time 3-move*	.026	.722	-.150	.037	-.039	.634	.012	.883
Initial thinking time 4-move*	.024	.742	-.048	.505	.018	.826	-.043	.604
Initial thinking time 5-move*	.053	.456	-.018	.795	.070	.374	.001	.990
Mean subsequent thinking time	.066	.336	-.189	.006	-.070	.360	-.048	.534

Subseq. thinking time 2-move*	.087	.224	-.170	.017	-.120	.135	.035	.667
Subseq. thinking time 3-move*	.074	.303	-.089	.213	.010	.902	.028	.734
Subseq. thinking time 4-move*	.001	.985	-.113	.103	-.002	.976	-.050	.530
Subseq. thinking time 5-move*	.041	.557	-.151	.031	-.105	.178	-.093	.239
SST								
Proportion of successful stops in last half	.121	.087	-.217	.002	-.133	.095	.155	.056
Stop signal reaction time	.098	.149	-.104	.127	-.123	.109	.066	.939
Direction errors go trials*	-.039	.564	-.046	.492	-.101	.183	.068	.377
Direction errors stop trials*	.011	.870	-.091	.187	-.181	.019	.116	.137
Direction errors stop and go trials*	-.025	.705	-.078	.244	-.131	.080	.098	.197
Median correct RT go trials (ms)*	.130	.060	-.144	.037	-.038	.625	.089	.257
Median incorrect RT go trials (ms)*	.102	.151	-.138	.052	-.186	.018	.143	.073
CGT								
Risk taking	-.133	.064	.095	.184	.113	.158	-.108	.182
Overall proportion bet	-.080	.274	.075	.304	.116	.153	-.087	.288
Quality of decision making*	-.130	.062	.165	.019	.106	.178	-.065	.414
Deliberation time*	.148	.037	-.187	.008	-.108	.175	.069	.391
Risk adjustment*	-.073	.284	.082	.230	-.004	.959	-.021	.788
Delay aversion*	-.010	.888	.038	.600	.040	.618	.049	.543

Note. Regression analyses control for age and full-scale IQ. Values in bold indicate significant associations. *Indicates this information is provided in the online supplement only.

Neurocognitive Characteristics of Youth with Noncomorbid and Comorbid Forms of
Conduct Disorder and Attention Deficit Hyperactivity Disorder

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