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ORIGINAL ARTICLE

Differential neuropsychological functioning between adolescents with attention-deficit/hyperactivity disorder with and without conduct disorder

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Abstract *Background/Purpose:* This study aimed to evaluate neuropsychological functioning of attention-deficit/hyperactivity disorder (ADHD) with and without comorbidities of oppositional defiant disorder (ODD) and/or conduct disorder (CD) and the mediation effects of the neuropsychological functions in the relationship between ADHD and ODD/CD symptoms to increase our understanding about these frequently co-occurring disorders.

Methods: Adolescents aged 11–18 years were interviewed by the Kiddie epidemiologic version of the Schedule for Affective Disorders and Schizophrenia to confirm their previous and current ADHD status and other psychiatric diagnoses. The performance of the Cambridge Neuropsychological Testing Automated Battery was compared among four groups: (1) ADHD with CD (ADHD+CD), regardless of ODD; (2) ADHD with ODD (ADHD+ODD) without CD; (3) ADHD without ODD/CD (ADHD-only); and (4) typically developing controls. Mediation effects of neuropsychological functioning were tested.

Results: All three ADHD groups had impaired spatial working memory and short-term memory. Deficits in verbal memory and response inhibition were found in ADHD+ODD, but not in ADHD-only. ADHD+CD did not differ from typically developing controls in verbal working memory, signal detectability, and response inhibition. Spatial working memory partially mediated the association between ADHD and CD symptoms and alerting/signal detectability of arousal partially mediated the association between ADHD and ODD symptoms.

Conclusion: There were both common and distinct neuropsychological deficits between adolescents with ADHD who developed ODD only and who developed CD. ADHD comorbid with

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CD may be a different disease entity and needs different treatment strategies in addition to treating ADHD, while ADHD+ODD may be a severe form of ADHD and warrants intensive treatment for ADHD symptoms.

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Introduction

Oppositional defiant disorder (ODD) and conduct disorder (CD), often together referred to as disruptive behavior disorders (DBDs), are the most common comorbidities in attention-deficit/hyperactivity disorder (ADHD).¹ The comorbid conditions are related to poorer quality of life,² lower educational achievement,³ more antisocial personality disorder,⁴ more psychoactive substance use,⁴ earlier parenthood, and more sexually-transmitted disorders⁵ than pure ADHD. These academic and social difficulties may be associated with neuropsychological deficits intrinsic to these disorders.⁶ Clarifying the common and specific neuropsychological functioning underpinning the comorbid condition would be helpful in understanding the development of DBDs in ADHD.

ADHD is postulated to have multiple and heterogeneous neuropsychological deficits, including executive functions (EF),⁷ with the strongest evidence in response inhibition, spatial working memory and sustained attention⁸ and non-EF, such as spatial short-term memory,⁹ visual memory¹⁰ and signal detectability.^{11,12} In addition to the hypothesis of deficits in the top-down executive control process subserved by the prefrontal areas,⁷ ADHD is also postulated to be associated with impaired cognitive–energetic regulation, which is subserved by the subcortical areas through bottom-up process, presented as slow motor processing and impaired alerting and signal detectability (signal/noise discrimination) in arousal (often including stages of alerting, phasic responding and signal discrimination),¹³ which in turn, influences the executive performance.^{14,15}

It is inconclusive whether executive deficits found in ODD/CD are due to their frequent comorbidity with ADHD or not: some claim that EF deficits of ODD/CD are weak after controlling for ADHD,^{7,16} and others show that executive deficits in ODD, CD, or disruptive behaviors are independent of ADHD in domains of working memory,¹⁷ response inhibition^{18,19} and visuospatial planning.²⁰ Non-EF deficits, such as verbal memory,²¹ visuospatial domain²² (without controlling for ADHD), and inhibitory response speed²³ (after controlling for ADHD symptoms) are also reported in adolescents with delinquent behaviors/CD. The influences of physiological hypoarousal on CD and ODD have also been demonstrated,²⁴ but whether the hypoarousal during cognitive attentional process found in ADHD is correlated to the development of CD and ODD is rarely studied.

Most studies investigating neuropsychological functions of ADHD and DBDs merge ODD and CD together as a group.²⁴ However, divergence between ODD and CD in youths with ADHD gains evidence from cross-sectional²⁵ and longitudinal^{4,26} clinical studies. ODD and CD might be conceptually

considered as distinct but highly correlated dimensions of psychopathology at the phenotypic level²⁶ and genetic level.²⁷ Only few studies tap CD and ODD separately while testing neuropsychological functions of ADHD and DBDs. One recent well-designed study for the relationship of ODD and ADHD demonstrates that verbal memory difficulty is a unique deficit of ODD independent of ADHD²⁸ and another finds that motivational inhibitory control ability could correctly classify ODD and control.²⁹ In a series of studies comparing functional brain imaging between ADHD and pure CD, Rubia³⁰ concludes that *hot* EF deficits, i.e., emotion/motivation related cognitive control, are more specific to CD while *cool* EF deficits, the executive dysfunctions unrelated to motivation/emotion, are more specific to ADHD. Nevertheless, there is still controversy whether there are *cool* EF deficits in ODD/CD,²³ and whether *cool* EF deficits in ADHD+CD would be different from those in ADHD+ODD and ADHD.

It is still not clear why some individuals with ADHD develop ODD, even CD, later in their life but others do not. We intend to approach this question through the neuropsychological functioning, which is thought as the intermediate phenotype between genes and behavioral phenotypes (endophenotype),³¹ by categorical and dimensional approaches. Since ADHD, ODD, and CD are found to share large proportion of common genetic influences but CD especially have higher unique genetic variance than ODD,²⁷ we hypothesize that there might be similar or more neuropsychological deficits in ADHD+ODD as compared to those of ADHD-only, and specific neuropsychological patterns might exist in ADHD+CD. Based on the above-mentioned genetic findings and on the hypothesis that different DBDs might have common and specific deficits in elements of executive control or energetic level,¹⁴ we also expect that differential neuropsychological deficits might mediate the pathway from ADHD to CD and ODD symptoms.

Methods

Study population and procedures

A total of 482 adolescents with childhood diagnosis of ADHD, aged 11–18 years, and 347 typically developing (TD) adolescents without lifetime ADHD were recruited. Details regarding the sample characteristics and procedures of this study have been described elsewhere.¹² They were all clinically diagnosed with ADHD according to the DSM-IV diagnostic criteria by board-certificated child psychiatrists and received outpatient treatments in the same center for 4–5 years before the study implementation. The TD adolescents were recruited from the same school districts as the adolescents with ADHD

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