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Processing Speed Predicts Behavioral Treatment Outcomes in Children with Attention-Deficit/Hyperactivity Disorder Predominantly Inattentive Type

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Abstract

Neuropsychological functioning underlies behavioral symptoms of attention-deficit/hyperactivity disorder (ADHD). Children with all forms of ADHD are vulnerable to working memory deficits and children presenting with the inattentive form of ADHD (ADHD-I) appear particularly vulnerable to processing speed deficits. As ADHD-I is the most common form of ADHD presented by children in community settings, it is important to consider how treatment interventions for children with ADHD-I may be affected by deficits in processing speed and working memory. We utilize data collected from 199 children with ADHD-I, aged 7 to 11 years, who participated in a randomized clinical trial of a psychosocial-behavioral intervention. Our aims are first to determine whether processing speed or working memory predict treatment outcomes in ADHD-I symptom severity, and second whether they moderate treatment effects on ADHD-I symptom severity. Results of linear regression analyses reveal that baseline processing speed significantly predicts posttreatment ADHD-I symptom severity when controlling for baseline ADHD-I symptom severity, such that better processing speed is associated with greater symptom improvement. However, predictive effects of working memory and moderation effects of both working memory and processing speed are not supported in the present study. We discuss study limitations and implications of the relation between processing speed and treatment benefits from psychosocial treatments for children with ADHD-I.

Keywords

ADHD; Inattentiveness; Processing Speed; Clinical Trial; Psychosocial Intervention

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Compliance with Ethical Standards

Conflict of interest: The authors declare that they have no conflict of interest.

Ethical approval: 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. This article does not contain any studies with animals performed by any of the authors.

Informed consent: Informed consent was obtained from all individual participants included in the study.

Children with ADHD-I are particularly vulnerable to processing speed deficits compared to (a) typically developing children (e.g., Arnett, Pennington, Willcutt, DeFries, & Olson, 2015; Rossi et al., 2015), (b) children with the hyperactive-impulsive presentation of ADHD (e.g., Hellwig-Brida, Daseking, Petermann, & Goldbeck, 2010), and (c) children with the combined presentation of ADHD (e.g., Mayes, Calhoun, Chase, Mink, & Stagg, 2009). Indeed, in adults with ADHD, symptoms of hyperactivity-impulsivity have been associated with faster processing speed (Nigg et al., 2005) and symptoms of inattentiveness have been associated with slower processing speed (Hunt, Bienstock, & Qiang, 2012; Nigg et al., 2005). Furthermore, children's processing speed performance has predicted ADHD-I diagnostic status with 69% accuracy (Mayes et al., 2009). There is strong evidence for a largely biological etiology of processing speed impairments in individuals with ADHD-I. White matter abnormalities have been associated with both impaired processing speed (Konrad & Eickhoff, 2010) and ADHD symptoms (van Ewijk, Heslenfeld, Zwiers, Buitelaar, & Oosterlaan, 2012). Furthermore, children with ADHD-I have shown white matter abnormalities in the temporo-occipital circuits (Lei et al., 2014). There may also be a genetic basis for processing speed deficits in individuals with ADHD (Bidwell, Willcutt, DeFries, & Pennington, 2007; Waldman, 2005).

Of the three presentations of ADHD in the DSM-5 (inattentive, hyperactive-impulsive, combined), the inattentive form (ADHD-I) is the most common found in children within community settings (Willcutt, 2012). Symptoms of ADHD-I include failing to give close attention to details, difficulty sustaining attention, appearing not to listen, not following through on instructions, difficulty organizing tasks, avoidance of tasks that require sustained effort, losing things, being easily distracted, and forgetfulness (DSM-5; American Psychiatric Association, 2013). Thus, a diagnosis of ADHD-I, according to the DSM, is based on behavioral symptoms rather than underlying cognitive mechanisms. However, theoretical models of ADHD often implicate underlying neuropsychological processes such as processing speed and working memory as highly relevant to symptom presentation (e.g., Barkley, 1997; Sonuga-Barke, Bitsakou, & Thompson, 2010).

Processing speed is the cognitive capacity to process information and generate an appropriate response within constrained time limits (Weiler, Bernstein, Bellinger, & Waber, 2000). It is often estimated using the Processing Speed Index from the Wechsler intelligence tests (WISC-IV and WISC-V), which comprises two subtests: Coding and Symbol Search. The Coding subtest requires children to match symbols paired with numbers, and the Symbol Search subtest requires children to scan a row of symbols and indicate whether a target symbol appears in the row (Wechsler, 2003a). Both subtest scores reflect accuracy and speed of performance in tests of visual information processing (Weiler et al., 2000).

Processing speed is distinguished from sluggish cognitive tempo (SCT), with the latter constituting a cluster of symptoms including inconsistent alertness and orientation, evidenced by sluggishness, drowsiness, and daydreaming (McBurnett, Pfiffner, & Frick, 2001; Todd, Rasmussen, Wood, Levy, & Hay, 2004). Processing speed is a specific cognitive ability related to information input and output, whereas SCT is a cluster of behavioral symptoms related to alertness and orientation. Comparing these two constructs is challenging due to both the unknown neuropsychological mechanisms underlying symptoms

of SCT (Barkley, 2014) as well as the unidentified behavioral symptoms resultant of slow processing speed. Both processing speed and SCT are associated with inattention (Barkley, 2014); however, interestingly, processing speed and SCT are not consistently associated with each other (Bauermeister, Barkley, Bauermeister, Martinez, & McBurnett, 2012). Additionally, SCT and ADHD-I have been described as correlated, yet distinct, symptom dimensions (Lee, Burns, Snell, & McBurnett, 2014; Willcutt et al., 2014) with distinct neuropsychological profiles (Araujo Jiménez, Jané Ballabriga, Martin, Arrufat, & Giacobo, 2015; see Barkley, 2014 for a review of SCT and ADHD).

Working memory is an essential executive function, affecting children with ADHD regardless of presentation (Mayes et al., 2009). It constitutes the cognitive capacity to maintain and manipulate information over short periods of time (Takeuchi, Taki, & Kawashima, 2010). Working memory is critical to mathematical skills and reading comprehension and is thus a strong predictor of academic achievement (Buschkuehl, Jaeggi, & Jonides, 2012). Again, whereas processing speed is a cognitive skill that distinguishes between the inattentive versus hyperactive-impulsive or combined presentations of ADHD, children with ADHD-C and ADHD-I typically have similar deficits in working memory performance (Mayes et al., 2009).

Most treatments for ADHD target symptoms and impairment rather than underlying neuropsychological functions. Furthermore, behavioral treatments for ADHD have generally focused on the combined presentation and have targeted the disruptiveness characteristic of this form of ADHD (Fabiano et al., 2009; Pelham & Fabiano, 2008). Interventions targeting disruptiveness and impulsivity may not be as relevant to reducing symptoms of ADHD-I (Pfiffner et al., 2014). Absent from research on behavioral interventions is consideration of how underlying cognitive deficits of ADHD might impact treatment response. Specific to ADHD-I, it is not known whether and how processing speed and working memory weaknesses might impact treatment response. Based on studies linking symptoms and impairments with these cognitive factors (Hunt et al., 2012; Martinussen & Tannock, 2006; Nigg et al., 2005), both processing speed and working memory deficits could be expected to reduce response to behavioral strategies implemented by parents and teachers intended to elicit compliance and completion of routines and academic work.

We conducted the present study with a large sample of children, aged 7 to 11 years, with carefully diagnosed ADHD-I, who participated in a randomized clinical trial of a psychosocial-behavioral intervention. The intervention (Child Life and Attention Skills; CLAS) was developed to specifically address the symptoms and impairment of ADHD-I (Pfiffner et al., 2014). Participants were assigned to either a multimodal treatment (CLAS) which includes parent, child, and teacher components, a parent-focused treatment, or treatment as usual. First, in the primary analyses, data from the active treatment groups (i.e., the multimodal and parent-focused treatments) are combined and contrasted with the treatment as usual group data. Because each active treatment condition received the same parent component, this contrast affords comparison of any study-related treatment components to nonsystematic treatment. Second, we contrast the two active treatment groups directly, without the inclusion of data from the treatment as usual group, pitting multimodal treatment against parent-focused treatment alone.

Our first aim is to determine whether processing speed or working memory independently predict treatment outcomes regardless of intervention type. We hypothesize that children's baseline processing speed and working memory will each significantly predict parent and teacher report of children's posttreatment ADHD-I symptom severity, above and beyond baseline ADHD-I symptom severity, such that better processing speed and working memory will predict greater symptom-based improvement following treatment. In other words, the prediction is that processing speed and working memory will be significantly associated with the inattention-related outcome regardless of intervention type. We also expect that significance will remain for the second contrast of the multimodal versus parent-focused treatment, such that these cognitive factors will predict better inattention-related outcome for the multimodal treatment.

Our second aim is to determine whether processing speed or working memory independently moderate the effects of treatment group on the primary outcome (i.e., whether they differentially predict outcomes across active treatment versus treatment as usual or multimodal treatment versus parent-focused treatment only). Due to the exploratory nature of the moderation analyses, we do not predict directionality of these effects; however, such an investigation could provide insight into potential treatment components most susceptible to influence from neuropsychological functions (e.g., the addition of teacher and child components in the multimodal treatment, which are not in the parent-focused treatment, may protect against effects of neuropsychological functioning on treatment).

Method

Participants

In the present study, we utilize data collected from 199 children participating in a randomized clinical trial held across two sites (Pfiffner et al., 2014). Six participants were excluded from the present analyses due to missing data. Most participants were recruited from schools through mailings to principals, learning specialists, and school mental health providers (65%). Participants were also recruited through mailings to offices of pediatricians, child psychiatrists, and psychologists (18%), postings in online parent networks or professional organizations (11%), or word-of-mouth (6%).

Inclusion criteria included the following (for additional details, see Pfiffner et al., 2014). First, each child must have had a primary DSM-IV diagnosis of ADHD-I confirmed with the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS-PL; Kaufman, Birmaher, Brent, & Rao, 1997). Second, children must have had an IQ greater than 80 confirmed with the Wechsler Intelligence Scale for Children – Fourth Edition (WISC-IV; Wechsler, 2003a). Third, children must have been between 7 and 11 years of age (2^{nd} to 5^{th} grade in school). Fourth, children must have been living with at least one parent for the year prior to the study. Fifth, children must have been attending school full-time in a regular classroom, the school must have been within 45 minutes of the study site, and teachers must have consented to participate in a school-based treatment. Finally, families must have been able to participate in groups on days scheduled. Exclusion criteria comprised receipt of nonstimulant psychoactive medication, intention to initiate or change medication during the study, significant developmental disorders (e.g., autism spectrum disorders or

intellectual disability), and neurological illnesses. Informed written consent was provided by parents, and written assent was provided by children. Study procedures were approved by the Committee on Human Research at the University of California, San Francisco, which also covered participants at the University of California, Berkeley site.

Child demographic characteristics at randomization were as follows: the mean age was 8.6 years (range = 7 to 11 years); 26% were in the 2nd grade, 31% were in the 3rd grade, 27% were in the 4th grade, and 17% were in the 5th grade; 58% of the children were male; 54% were Caucasian, 17% were Latino, 8% were Asian American, 5% were African American, and 17% self-identified as mixed race. At randomization, 4.5% of children were taking stimulant medication to address symptoms related to ADHD.

Parent demographic characteristics at randomization were as follows: total household income was below $50,000 for 14%, $50,000 to $100,000 for 27%, $100,000 to $150,000 for 28%, and above $150,000 for 31% of families; 81% of primary parents reported graduating from college; 13% of participants were living in single-parent homes.

Measures

Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS-PL; Kaufman et al., 1997)—The K-SADS-PL was a primary measure used at baseline to confirm psychiatric diagnostic status in children participating in the study. A licensed clinical psychologist used the K-SADS-PL to ask parents about their children's clinical and developmental histories and administered modules for ADHD, oppositional defiant disorder, conduct disorder, anxiety disorders, major mood disorders, and psychoses. Study inclusion criteria included six or more inattentive symptoms ($M = 7.7$, $SD = 1.1$) and fewer than 6 hyperactive-impulsive symptoms ($M = 1.2$, $SD = 1.2$). The K-SADS-PL has demonstrated good psychometric properties, including adequate test-retest reliability (Kaufman et al., 1997).

Child Symptom Inventory (CSI: Gadow & Sprafkin, 1994)—The CSI is a measure of child symptom severity and was completed by parents and teachers at baseline and posttreatment. Mean symptom severity scores for parent and teacher report on the CSI were used as primary outcome measures and analyzed separately in the present study. Items corresponding to DSM-IV inattentive symptoms were rated on a 4-point scale (ranging from 0 to 3). The Inattention Scale has demonstrated acceptable test-retest reliability and predictive validity for a categorical diagnosis of ADHD (Gadow & Sprafkin, 1994).

Wechsler Intelligence Scale for Children – Fourth Edition (WISC-IV; Wechsler, 2003a)—The WISC-IV is a test of intellectual ability yielding a Full Scale IQ (FSIQ) from four index scores: Verbal Comprehension Index (VCI), Perceptual Reasoning Index (PRI), Working Memory Index (WMI), and Processing Speed Index (PSI). FSIQ was used for study inclusion. The PSI and WMI were used as predictor variables in the present study. PSI includes the core subtests of Coding and Symbol Search; WMI includes the core subtests of Letter-Number Sequencing and Digit Span. In supplemental analyses, selected WISC-IV subtest combinations were included as covariates. The PSI and WMI have demonstrated

good reliability (internal consistency estimate of .88 for the PSI and .92 for the WMI) and validity (Wechsler, 2003b).

Procedure

Participants were randomized across treatment conditions in parallel fashion at one of two treatment sites: the University of California, Berkeley or the University of California, San Francisco. There were no significant site-by-treatment effects on primary outcome measures (Pfiffner et al., 2014). Treatment conditions included Child Life and Attention Skills Treatment (CLAS), Parent-Focused Treatment (PFT), and Treatment as Usual (TAU). Six cohorts of children participated in treatment across four years. A total of 74 children participated in the CLAS group, 74 children participated in the PFT group, and 51 children participated in the TAU group. Cohorts ranged from 24 to 43 children, with a mean of 33 children per cohort. Treatment lasted 10 to 13 weeks. Prior to treatment, parents completed the K-SADS-PL and the CSI, teachers completed the CSI, and children completed the WISC-IV. After treatment, parents and teachers completed the CSI.

The CLAS treatment included three manualized components: parent, child, and teacher. The *parent component* was adapted from existing parent training programs (Barkley, 1987; Forehand & McMahon, 1981; Wells et al., 1996) and comprised ten 90-minute parent group meetings and up to six 30-minute family meetings with the parent, child, and therapist. The curriculum focused on strategies for managing ADHD-I symptoms, associated impairments, and executive functioning deficits, strategies to reinforce children's use of skills learned in child group meetings, and strategies for effectively interacting with teachers and reinforcing the classroom intervention. During family meetings, therapists worked with parents and children to individualize treatment content.

The *child component* was adapted from a social skills curriculum for children with ADHD (Pfiffner & McBurnett, 1997) and comprised ten 90-minute child group meetings which occurred at the same time as the parent group meetings. Children learned skills for independence (e.g., academic, study, and organizational skills; self-care and daily living skills), social skills (e.g., good sportsmanship, assertion, conversational skills, dealing with teasing, friendship-making, playdate skills) and cognitive-behavioral techniques to promote attention, time management, and task completion. The curriculum was delivered through didactic instruction, modeling, behavioral rehearsal, corrective feedback, and practice in the context of a reward-based contingency management program.

The *teacher component* included evidence-based classroom management strategies (Fabiano et al., 2010; Pfiffner, 2011; Pfiffner, Barkley, & DuPaul, 2006) and comprised one 30-minute orientation meeting between the teacher and therapist and up to five 30-minute meetings with the teacher, parent, child, and therapist. Teachers were provided with information on ADHD-I and taught strategies to provide in-class support of attention and reinforce the use of skills their student learned in the child group. Specific academic, organizational, and social target behavior goals were selected by the teacher, shaped by the clinician, and discussed with the parent and child.

PFT included the parent component described above, with parent management sessions identical to those in the CLAS parent group; however, PFT did not include the child or teacher components. Families in PFT were not informed of the child component skills taught in the CLAS group and did not receive training in working with teachers. PFT families received the same number of parent group and individual meetings as CLAS families.

TAU did not include treatment components from either study-related treatment (i.e., CLAS or PFT). Families in the TAU group received a written diagnostic report based on the assessment conducted at baseline and a list of community treatment providers. Families did not receive specific treatment recommendations from study staff. During the period between baseline and posttreatment, 53% of children in the TAU group received classroom accommodations, 51% received educational intervention, 33% received psychotherapy, and 14% received medication. At the completion of the study, families in the TAU group had the opportunity to participate in a two-session parenting workshop including strategies taught in the CLAS groups. Note that a completely treatment-free control group in a three-month clinical trial is neither feasible nor ethical; a TAU condition allows families to select community interventions but prohibits them from experiencing the same, evidence-based and intensive interventions experienced by the active treatment conditions. As such, it is a relatively conservative control condition.

Clinicians—Four therapists led parent groups. Three of the therapists were licensed clinical psychologists, and one was a clinical psychology postdoctoral fellow. In the CLAS group, for any given participant, the same therapist provided the parent group sessions, individual sessions, and teacher consultations. In the PFT group, for any given participant, the same therapist provided the parent group sessions and individual sessions. Ten bachelor's or master's level clinicians led the child groups. Two clinicians co-led each child group. Licensed clinical psychologists provided supervision during weekly cross-site conference calls and individually as needed. For the CLAS group, joint supervision was conducted with parent and child group leaders.

Attendance—There was not a significant difference in the number of individual parent meetings attended by participants in the CLAS ($M = 4.2$) and PFT ($M = 3.9$) groups. Participants in the PFT group attended slightly fewer group meetings ($M = 8.8$) than participants in the CLAS group ($M = 9.3$), $p = .02$ ($d = .38$). Teachers of children in the CLAS group attended an average of four meetings.

Data Analytic Plan

In the larger report of the clinical trial from which these data were garnered (Pfiffner et al., 2014), children in the CLAS group showed significantly fewer symptoms of ADHD-I than children in the TAU group at posttreatment, according to parent and teacher report on the CSI. Additionally, children in the CLAS group showed significantly fewer symptoms of ADHD-I than children in the PFT group, according to teacher report on the CSI at posttreatment; however, there was not a significant difference in posttreatment ADHD-I symptom count between children in the CLAS and PFT groups according to parent report.

For the following analyses, we coded treatment group in two ways for two different sets of analyses. First, to compare participants in active treatment to participants in treatment as usual, data from participants in the CLAS and PFT groups were combined to form an active treatment group, contrasted with data from participants from the TAU group. Second, to compare participants in a multimodal treatment versus participants in a parent-focused treatment, the data from participants in the CLAS and PFT groups were analyzed in contrast to each other, with data from TAU participants excluded. The only significant difference in treatment group characteristics at randomization was in report of medication use. Significantly more children in the CLAS group reported ADHD medication use (9.5%) than children in the PFT group (1.4%), but not compared to children in the TAU group (2.0%) (Pfiffner et al., 2014).

All statistical analyses were conducted using SPSS for Windows, Version 23. Analyses performed were as follows. First, we determined descriptive statistics for the predictor and outcome variables. Next, we conducted a series of linear regression analyses to address the hypotheses from the two aims. For each aim, two series of linear regression analyses were performed based on rater: one including parent report of CSI ADHD-I symptom severity and another based on teacher report of the same measure. Baseline parent and teacher ratings of CSI-ADHD-I symptom severity were not significantly correlated, $r(197) = .11$, $p = .139$. Posttreatment parent and teacher ratings of CSI-ADHD-I symptom severity were significantly yet weakly correlated $r(190) = .24$, $p < .001$.

The hypotheses from the first aim (i.e., to determine whether processing speed or working memory independently predict treatment outcomes regardless of intervention type) were tested by performing eight linear regression analyses: two methods for coding treatment by two reporters (parent or teacher) by two cognitive processes (PSI or WMI). Step 1 included the group variable (active treatment versus TAU or CLAS versus PFT) as well as baseline report of CSI ADHD-I symptom severity (either parent or teacher report). Step 2 included children's baseline neuropsychological performance (either WISC-IV PSI or WMI scores). The hypotheses from the second aim (i.e., to determine whether processing speed or working memory independently moderate the effects of treatment group on treatment outcomes) were tested by adding an interaction term between the group variable and children's baseline neuropsychological performance at Step 3.

Our primary data analytic plan did not include WISC-IV FSIQ as a covariate due to concerns of over-control, particularly due to overlap between processing speed and working memory measures with the IQ measure (i.e., WISC-IV PSI and WMI each contribute to the FSIQ). Indeed, it has been argued that statistical adjustment for IQ in analyses of neuropsychological functioning may produce anomalous findings (Dennis et al., 2009). However, to aid in specificity of findings, we repeated primary analyses with covariation of (a) WISC-IV VCI or (b) all WISC-IV indexes other than the predictor index (i.e., VCI, PRI, and WMI were covaried for PSI analyses and VCI, PRI, and PSI were covaried for WMI analyses).

Results

Descriptive Statistics

Table 1 presents sample characteristics by treatment assignment for CLAS, PFT, active treatment (i.e., CLAS and PFT combined) and TAU. Independent samples *t* tests revealed no significant group differences (i.e., between CLAS and PFT or active treatment and TAU) in pretreatment age, CSI ADHD-I symptom severity, FSIQ, PSI, WMI, or posttreatment CSI ADHD-I symptom severity (with the exception of a comparison of posttreatment parent reported ADHD-I symptom severity between CLAS and PFT groups). Additionally, Chi-Square testing revealed no significant group differences in gender or baseline K-SADS diagnostic criteria status for oppositional defiant disorder (ODD), generalized anxiety disorder (GAD), or depression.

Processing Speed

Linear regression analyses were conducted to determine whether parent or teacher report of children's ADHD-I symptom severity on the CSI at posttreatment could be predicted from children's WISC-IV Processing Speed Index scores at baseline, covarying children's baseline CSI ADHD-I symptom severity as well as treatment group (i.e., active treatment versus TAU). Results indicated that a significant proportion of the total variation in parent report of ADHD-I symptom severity change from pretreatment to posttreatment was predicted by children's baseline processing speed and a small effect size on the overall model (Table 2). According to a standardized Beta coefficient (β), for every one standard deviation increase in baseline processing speed score, posttreatment parent report of ADHD-I symptom severity decreased (i.e., improved) by .13 standard deviations. Results also suggest that a significant proportion of the total variation in teacher report of ADHD-I symptom severity change from pretreatment to posttreatment was predicted by children's baseline processing speed and a small effect size on the overall model (Table 2). According to a standardized Beta coefficient (β), for every one standard deviation increase in baseline processing speed score, posttreatment teacher report of ADHD-I symptom severity decreased (i.e., improved) by .30 standard deviations. In other words, baseline processing speed was a significant predictor of treatment outcomes according to parent and teacher report of ADHD-I symptom severity for children diagnosed with ADHD-I. Additionally, with the inclusion of VCI as a covariate, results reveal that baseline processing speed remained a significant predictor of treatment outcomes according to both parent and teacher report.

For the CLAS versus PFT contrast, baseline processing speed remained a significant predictor of treatment outcomes according to both parent and teacher report (Table 3), over and above variance accounted for by treatment assignment and baseline ADHD-I symptom severity. Additionally, when VCI was included as a covariate in the CLAS versus PFT contrast, baseline processing speed remained a significant predictor of treatment outcomes according to both parent and teacher report.

Regarding the moderator analyses, results indicated that children's baseline processing speed was not a significant moderator of treatment effects on parent report, $\beta = -.38$, $t(192)$

= −0.88, p = .383, or teacher report, β = .38, $t(192)$ = 0.92, p = .359, of ADHD-I symptom severity. In other words, there was not sufficient evidence to suggest that the direction or strength of the relation between treatment group and the primary outcome differed based on children's processing speed. Inclusion of VCI as a covariate did not change the pattern of significance for processing speed moderation analyses. For the CLAS versus PFT contrast, results of moderation analyses for parent and teacher report remained nonsignificant.

Working Memory

For the active treatment versus TAU contrast, children's baseline working memory did not predict a significant proportion of the total variation in parent report or teacher report of ADHD-I symptom severity change from pretreatment to posttreatment (Table 2). Inclusion of VCI as a covariate did not change the nonsignificant findings.

For the CLAS versus PFT contrast, baseline working memory remained a nonsignificant predictor of treatment outcomes according to both parent and teacher report (Table 3). Inclusion of VCI as a covariate in the CLAS versus PFT contrast did not change the nonsignificant findings.

Regarding moderator analyses, results indicated that baseline working memory was not a significant moderator of treatment effects on parent report, β = .07, $t(192)$ = −0.14, p = .892, or teacher report, β = −.67, $t(192)$ = −1.20, p = .232, of ADHD-I symptom severity. Inclusion of VCI as a covariate did not change the pattern of significance for working memory moderation analyses. For the CLAS versus PFT contrast, results of moderation analyses for parent and teacher report remained nonsignificant.

Discussion

Our purpose was to determine whether processing speed and working memory, two neuropsychological functions commonly deficient or relatively weak in children with ADHD-I, would be associated with children's outcomes of a psychosocial-behavioral intervention. The present study is the first to date, to our knowledge, to examine treatment enhancement or interference effects of processing speed in children with ADHD-I. Key findings were that children's baseline processing speed scores predicted both parent and teacher report of posttreatment ADHD-I symptom severity, over and above baseline ADHD-I symptom severity, regardless of treatment group (i.e., contrasting active treatment versus TAU as well as CLAS versus PFT). That is, better baseline processing speed was related to greater treatment-related improvement. However, children's baseline working memory scores did not predict parent or teacher report of posttreatment ADHD-I symptom severity. Additionally, neither baseline processing speed nor working memory scores moderated effects of treatment on parent or teacher report of posttreatment ADHD-I symptom severity. Thus, direct skills training and the teacher component did not appear to mitigate the predictive effects of slow processing speed on ADHD symptom severity after treatment. In short, results reveal that the slower children's processing speeds are when they begin treatment, the less treatment-related improvement with respect to primary symptoms of ADHD-I. Thus, although slower processing speed was still associated with overall decreases in ADHD-I symptom severity from baseline to posttreatment, children with slower

processing speeds may be at risk for reduced treatment-related success compared to children with higher processing speeds.

The current results do not identify why processing slow processing speed predicted less benefit from treatment. It is possible that factors related to the particular intervention (e.g., curriculum, techniques taught) were insufficient for fully addressing these weaknesses or that parents' implementation of the skills learned in treatment (e.g., patience with children, delivery of commands) was adversely affected by children's lower processing speed, resulting in relatively weaker treatment outcomes in terms of symptom severity. In either case, these findings suggest the need for greater attention to processing speed deficits in treatments for ADHD-I. It may be important for clinicians working with families of children with ADHD-I to be aware that processing speed deficits predict poorer outcomes. For example, children with ADHD-I and lower processing speeds may take longer to process and comply with a parent's command. This longer behavioral response time could be related, in part, to processing speed, but parents may interpret children's delayed response as a display of willful noncompliance. Parents of children with ADHD-I may benefit from enhanced training in parenting children with slow processing speed (e.g., targeted techniques for increasing parental patience when waiting for children's compliance, providing rewards for partial completion of requests; and timed practice to encourage quicker responses). Teachers, as well, may benefit from psychoeducation regarding processing speed deficits and learning specific techniques for working with children with ADHD-I and **s**lower processing speeds such as relaxed time constraints, delivery of information at a reduced pace, simplifying multistep tasks, moderating processing demands, and supporting the development of metacognitive strategies (Weiler, Bernstein, Bellinger, & Waber, 2002). Future studies should focus on pinpointing whether including psychoeducation and a skill-building curriculum specific to processing speed deficits yields improved treatment outcomes for children with ADHD-I. Such adjunctive treatment may help children with slower processing speeds reach post-treatment symptom severities similar to children with faster processing speeds.

Non-behavioral interventions targeting processing speed itself also might be useful as adjuncts to behavioral interventions. Some evidence exists for improvement in cognitive abilities through cognitive training interventions including computer-based tasks, video games, and even board games (Burge et al., 2013; Mackey, Hill, Stone, & Bunge, 2011). One study conducted with schoolchildren found that cognitive speed training led to approximately a 30% improvement on the WISC-IV Coding subtest (Mackey et al., 2011). However, there is a lack of evidence supporting the generalization of improvements in trained cognitive tasks to untrained processing speed tasks (Takeuchi & Kawashima, 2012) and everyday tasks (Jak, Seelye, & Jurick, 2013) and other studies, including a meta-analysis of cognitive training studies, question whether cognitive training is an effective approach to reducing ADHD-related impairments (Cortese et al., 2015; Stevens, Gaynor, Bessette, & Pearlson, 2016). Although cognitive training is not an effective frontline treatment for symptoms of ADHD, its role as an adjunctive treatment for improving processing speed is not known.

Other treatments, such as neurofeedback and psychopharmacological intervention, have also been evaluated for effects on processing speed performance. Effects of neurofeedback on neuropsychological functioning are reportedly mixed (Bink, van Nieuwenhuizen, Popma, Bongers, & van Boxtel, 2014). For example, in a sample of adolescents and young adults with ADHD, participants receiving neurofeedback had improved processing speed at posttreatment, but improvements were not beyond that of the control group not receiving neurofeedback (Bink et al., 2014). It is also worth noting there are mixed results regarding stimulant and nonstimulant medication improving performance on processing speed tasks (Biederman et al., 2008; Finke et al., 2010; Graziano, Geffken, & Lall, 2011; Lajoie et al., 2005; Nielsen & Wiig, 2011). In a sample of children with ADHD, medication status (i.e., medication naïve, pure stimulant, stimulant plus another medication, or nonstimulant) did not have a significant effect on processing speed (Graziano et al., 2011). Additionally, Nielsen and Wiig (2011) found that adults with ADHD taking methylphenidate improved processing speed performance after stabilization of ADHD symptoms; however, it remains unknown whether methylphenidate had a direct impact on processing speed or whether the stabilization of ADHD symptoms influenced improvement in processing speed.

In contrast to processing speed, working memory was not a significant predictor of inattention-related treatment outcomes in the present study. Although working memory has been associated with symptoms of inattention (Martinussen & Tannock, 2006), the WMI was somewhat higher than the PSI in the current sample (see Table 1) indicating somewhat less impaired working memory. Consistent with other studies, slow processing speed may be the more pervasive cognitive deficit of ADHD-I (Thaler, Bello, & Etcoff, 2013). In addition, the intervention in the present study included components for addressing executive function deficits which may have mitigated working memory problems and therefore the potential adverse effect of these problems on inattention symptom reduction.

Limitations

Although this is a preliminary study investigating treatment interference effects of cognitive skills in children with ADHD-I, there are limitations that must be considered. First, the relatively high socioeconomic status of this sample may limit the generalizability of these findings. Second, because the outcome measures of the current study were parent and teacher report of children's ADHD-I symptom severity, it is possible that rater or expectancy bias influenced results. Inclusion of objective measures, such as blinded observations of child behaviors related to ADHD-I, could remedy rater or expectancy bias. Third, CLAS and PFT treatment groups were collapsed into a single active treatment group in primary analyses. The groups were collapsed to compare active treatment to treatment as usual; however, this procedure may have inadvertently affected results as the CLAS group received all three treatment components whereas the PFT group only received the parent component. However, in additional analyses in which CLAS and PFT data were contrasted and TAU data were excluded, the significant effect of processing speed on both parent and teacher report of ADHD-I symptoms remained. Finally, regarding processing speed, we examined only visual information processing and did not have a measure of auditory information processing. Although children with ADHD-I have shown particular vulnerability in visual information processing and not auditory information processing (Weiler et al., 2002), providing a

measure of both processing capacities could provide better understanding of how processing speed influences treatment.

It is also important to consider that, as a group, the current sample of children with ADHD-I is not exceptionally impaired in processing speed, with group means for PSI in the average range, although there were children in the sample with PSI standard scores as low as 62 (see Table 1). Nonetheless, results of the regression analyses revealed that slower processing speed was associated with less treatment-related change in inattention symptoms. These effects may be even greater in a sample with more significant processing speed impairments. To better understand how low PSI must be to detrimentally influence treatment, future studies may include comparisons between samples grouped by PSI ranges (e.g., below average, average, above average).

Another consideration is whether to include other neuropsychological or intellectual measures as covariates in analyses. It has been argued that IQ should not be included as a covariate in analyses in which the variable of interest is a measure of neuropsychological function, as doing so may be statistical over-control and could yield anomalous findings (Dennis et al., 2009). Specifically, it has been noted that IQ comprises multiple abilities that are correlated with each other and with neuropsychological variables of interest. Additionally, deficits in overall cognitive ability are a feature of ADHD, making it difficult to be covaried (Dennis et al., 2009). Still, to aid in specificity of our findings, we included VCI as a covariate in additional analyses and significant prediction effects of processing speed remained. In even more stringent additional analyses, all WISC-IV indexes aside from PSI (i.e., VCI, PRI, and WMI) were included as covariates and significant prediction effects of processing speed remained.

In conclusion, the present study has provided evidence that children's processing speed is negatively associated with beneficial treatment outcomes in a psychosocial-behavioral intervention. To our knowledge, this is the first study to date to examine treatment-related effects of processing speed in children with ADHD-I. Further investigation into specific ways children's processing speed influences treatment outcomes will extend knowledge from the current study and may help influence the development of strategies clinicians and parents can use to treat children with ADHD-I and lower processing speeds.

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Table 1

Child Characteristics by Treatment Assignment

Measure	Sample range (*n* = 199)	CLAS (*n* = 74)		PFT (*n* = 74)		CLAS-PFT Comparison	AT (*n* = 148)		TAU (*n* = 51)		AT-TAU Comparison
		M or %	*SD*	*M* or %	*SD*	*p*	*M* or %	*SD*	*M* or %	*SD*	*p*
Age	7–11	8.78	1.15	8.69	1.18	.591	8.74	1.16	8.37	1.13	.681
Gender (% male)		51.4%		64.9%		.096[a]	58.1%		58.8%		.929[a]
CSI ADHD-I											
Parent (Pre)	1.11–3.00	1.89	0.47	2.09	0.50	.350	1.99	0.49	2.06	0.42	.108
Teacher (Pre)	.78–3.00	2.04	0.53	1.95	0.55	.779	1.99	0.54	2.03	0.55	.820
Parent (Post)	.44–2.89	1.27	0.40	1.46	0.52	.001	1.37	0.47	1.68	0.56	.086
Teacher (Post)	.22–3.00	1.28	0.64	1.53	0.65	.811	1.41	0.66	1.74	0.71	.643
FSIQ (Pre)	80–135	103.64	11.04	102.67	11.32	.943	103.16	11.15	105.47	11.53	.543
PSI (Pre)	62–128	93.75	12.52	92.44	13.1	.485	93.10	12.79	96.61	14.42	.220
WMI (Pre)	62–135	97.44	12.28	97.59	10.53	.243	97.51	11.4	100.31	11.2	.258
K-SADS ODD		6.76%		6.76%		.627[a]	6.76%		5.88%		.563[a]
K-SADS Anx		5.41%		10.96%		.177[a]	8.16%		5.88%		.429[a]
K-SADS Dep		1.35%		2.7%		.500[a]	2.03%		1.96%		.729[a]

Note. CLAS = Child Life and Attention Skills treatment; PFT = parent-focused treatment; AT = active treatment (i.e., CLAS and PFT combined); TAU = treatment as usual; Pre = pretreatment scores; Post = posttreatment scores; CSI = Child Symptoms Inventories (Gadow & Sprafkin, 1994); ADHD-I = Attention-Deficit/Hyperactivity Disorder Predominantly Inattentive Type; FSIQ = Full Scale IQ; PSI = Processing Speed Index; WMI = Working Memory Index; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children (Kaufman, Birmaher, Brent, & Rao, 1997); ODD = oppositional defiant disorder; Anx = anxiety disorder; Dep = depressive disorder

[a]Significance value for Pearson Chi-Square test

Table 2

Predictive Effects of PSI and WMI in Active Treatment versus Treatment as Usual

		Parent Report of CSI					Teacher Report of CSI				
		β	**Δ*F***	***df***	***p***	**ΔR^2**	***β***	**Δ*F***	***df***	***p***	**ΔR^2**
Step 1	Group	.241	28.280	190	.000	.229	.202	31.507	190	<.001	.249
	CSI ADHD-I (Pre)	.401					.452				
Step 2	PSI	−.132	4.260	189	.040	.017	−.295	23.227	189	<.001	.082
Step 1	Group	.241	28.280	190	<.001	.229	.202	31.507	190	<.001	.249
	CSI ADHD-I (Pre)	.401					.452				
Step 2	WMI	.058	0.828	189	.364	.003	.013	0.039	189	.843	.000

Note. CSI = Child Symptom Inventory; Pre = pretreatment scores; PSI = Processing Speed Index, WMI = Working Memory Index

Table 3

Predictive Effects of PSI and WMI in Multimodal treatment (CLAS) versus Parent-Focused Treatment (PFT)

		Parent Report of CSI					Teacher Report of CSI				
		β	**Δ*F***	***df***	***p***	**ΔR^2**	***β***	**Δ*F***	***df***	***p***	**ΔR^2**
Step 1	Group	.139	15.377	143	<.001	.177	.221	22.605	141	<.001	.243
	CSI ADHD-I (Pre)	.371					.456				
Step 2	PSI	−.173	5.342	142	.022	.030	−.247	11.675	140	.001	.058
Step 1	Group	.139	15.377	143	<.001	.177	.221	22.605	141	<.001	.243
	CSI ADHD-I (Pre)	.371					.456				
Step 2	WMI	.058	0.575	142	.449	.003	−.034	0.213	140	.645	.001

Note. CSI = Child Symptom Inventory; Pre = pretreatment scores; PSI = Processing Speed Index, WMI = Working Memory Index