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Neurocognitive and Behavioral Predictors of Social Problems in ADHD: A Bayesian Framework

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Abstract

Objective—Social problems are a key area of functional impairment for children with ADHD, and converging evidence points to executive dysfunction as a potential mechanism underlying ADHD-related social dysfunction. The evidence is mixed, however, with regard to which neurocognitive abilities account for these relations.

Method—A well-characterized group of 117 children ages 8–13 (*M*=10.45, *SD*=1.53; 43 girls; 69.5% Caucasian/Non-Hispanic) with ADHD (*n*=77) and without ADHD (*n*=40) were administered multiple, counterbalanced tests of neurocognitive functioning and assessed for social skills via multi-informant reports.

Results—Bayesian linear regressions revealed strong support for working memory and cross-informant interfering behaviors (inattention, hyperactivity/impulsivity) as predictors of parent- and teacher-reported social problems. Working memory was also implicated in social skills acquisition deficits, performance deficits, and strengths based on parent and/or teacher report; inattention and/or hyperactivity showed strong correspondence with cross-informant social problems in all models. There was no evidence for, and in most models strong evidence *against*, effects of inhibitory control and processing speed. The ADHD group was impaired relative to the non-ADHD group on social skills (*d*=0.82–0.88), visuospatial working memory (*d*=0.89), and phonological working memory (*d*=0.58). In contrast, the Bayesian ANOVAs indicated that the ADHD and Non-ADHD groups were equivalent on processing speed, IQ, age, gender, and SES. There was no support for or against group differences in inhibition.

Conclusions—These findings confirm that ADHD is associated with impaired social performance, and implicate working memory and core ADHD symptoms in the acquisition and performance of socially-skilled behavior.

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Keywords

ADHD; social skills; executive functions; working memory; Bayesian

Social problems are a key area of functional impairment for a majority of children with ADHD (52%–82%; de Boo & Prins, 2007; Huang-Pollock et al., 2009), and include a broad range of difficulties that include difficulties making and maintaining friendships, peer rejection and neglect, and increased negative peer and caregiver interactions (Hoza et al., 2005). Recent evidence points to executive dysfunction as a potential mechanism underlying ADHD-related social problems (Bunford et al., 2014; Huang-Pollock et al., 2009; Kofler et al., 2011, 2016; Tseng & Gau, 2013). The evidence is mixed, however, with regard to which neurocognitive abilities account for these relations (Rinsky & Hinshaw, 2011). In addition, most studies to date have used mediation-based approaches that have been criticized for producing unreliable estimates of key pathways (Maxwell et al., 2007, 2011), and all have relied on traditional null hypothesis significance testing that disallows strong conclusions regarding the absence of an effect (Rouder & Morey, 2012; Wagenmakers et al., 2016). Finally, no study to date has probed for differential prediction of acquisition-based vs. performance-based social skills deficits despite evidence that these social learning mechanisms can be reliably estimated using behaviorally-anchored parent/teacher reports (Aduen et al., 2017; Frey et al., 2011; Gresham et al., 2010). The current study uses Bayesian linear regression to examine evidence for and against effects of four candidate neurocognitive processes and cross-informant ADHD symptoms on ADHD-related social problems. Primary outcomes include parent- and teacher-reported social skills, with exploratory analyses that probe for differential effects on the acquisition vs. performance of socially skilled behaviors.

Neurocognition and ADHD-related social dysfunction

The current study examines neurocognitive deficits and core ADHD behavioral symptoms that may interfere with the acquisition and/or performance of developmentally-appropriate social behavior (Humphreys et al., 2016; Rapport et al., 2009). Huang-Pollock and colleagues (2009) demonstrated the first link between global neurocognitive dysfunction and social problems in children with ADHD, and five additional studies have demonstrated relations between specific neurocognitive functions – working memory, inhibitory control, and processing speed – and ADHD-related social behavior. *Working memory* refers to the active, top-down manipulation of information held in short-term memory (Baddeley, 2007), and includes interrelated functions of the mid-lateral prefrontal cortex and interconnected networks that involve supervisory attentional control, updating, processing, and reordering (Nee et al., 2013; Wager & Smith, 2003). *Inhibitory control* refers to a set of interrelated cognitive processes that underlie the ability to withhold (action restraint) or stop (action cancellation) an on-going response (Alderson et al., 2007) and are supported by networks involving bilateral frontal, right superior temporal and left inferior occipital gyri, right thalamic, and mid-brain structures (Cortese et al., 2012). *Processing speed* refers to the rate at which individuals encode, evaluate, and act upon external information (Shanahan et al., 2006), is associated with anterior cingulate (Konrad et al., 2006) and fronto-parietal cortical

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regions (Cao et al., 2008) among others, and is often considered an etiologically important mechanism in ADHD (Karalunas & Huang-Pollock, 2013).

Consistent with findings from typically developing samples (McQuade et al., 2013), Kofler and colleagues (2011) found that working memory demonstrated both direct effects on ADHD-related social problems and indirect effects through its influence on ADHD behavioral symptoms. The specificity of these effects may be limited, however, because the study failed to consider working memory's interrelations with additional neurocognitive functions such as inhibitory control and processing speed, as well as potential differential effects of phonological (verbal) versus visuospatial (non-verbal) working memory (Conway et al., 2005).

Four recent studies extended this work, and tested for contributions of both working memory and inhibitory control with mixed results (Bunford et al., 2015a; Hilton et al., 2017; Rinsky & Hinshaw, 2011; Tseng & Gau, 2013). Interestingly, Bunford and colleagues (2015a) found that the relation between neurocognitive dysfunction and social problems was again mediated by ADHD behavioral symptoms (inhibitory control via hyperactivity/impulsivity, working memory via attention problems). Along these lines, Rinsky & Hinshaw (2011) reported a longitudinal association between inhibitory control and social functioning, but associations with a digit span test of short-term memory (Swanson & Kim, 2007) failed to reach significance. Most recently, Kofler and colleagues (2016) classified children with ADHD into subgroups based on the presence/absence of social impairment. Consistent with Tseng & Gau (2013) and Hilton et al. (2017), they failed to replicate a link between inhibitory control and social problems. Instead, they found that socially-impaired children with ADHD demonstrated deficits in phonological working memory and processing speed.

Taken together, the evidence points to phonological working memory, visuospatial working memory, behavioral inhibition, processing speed, inattentive behavior, and hyperactive/impulsive behavior as important mechanisms underlying global social dysfunction in ADHD. No study to date, however, has concurrently assessed all of these candidate mechanisms – a critical omission given the well-documented 'task impurity' problem associated with neurocognitive measurement (Snyder et al., 2015). That is, no task is process pure (Shipstead et al., 2010): all tasks require multiple executive and non-executive neurocognitive abilities for successful performance, and conclusions regarding effect specificity are limited when these correlated but distinct abilities are not measured and simultaneously controlled (Miyake & Friedman, 2012). In addition, conclusions regarding the nonsignificant effects reported in some previous studies are limited by their exclusive use of frequentist methods (i.e., nonsignificant *p*-values do not provide support for the lack of an effect; Wagenmakers et al., 2016). Finally, mediation methods for modeling causal processes in cross-sectional data have been criticized for producing potentially biased estimates in certain circumstances (Maxwell et al., 2011). To address these concerns, the current study used Bayesian linear regression (van de Schoot et al., 2014) to assess support both for and against effects on ADHD-related social problems of all four candidate neurocognitive processes (i.e., phonological working memory, visuospatial working memory, behavioral inhibition, and processing speed), parent-and teacher-reported ADHD symptoms, and key covariates (IQ, age, gender, SES). Bayesian methods allow stronger conclusions by

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estimating the magnitude of support for both the alternative and null hypotheses (Rouder & Morey, 2012; van de Schoot et al., 2013).

Social behavioral analysis framework

A secondary goal of the current study was to apply the social behavioral analysis framework (Gresham et al., 2010) to determine the extent to which candidate neurocognitive processes and interfering behaviors (core ADHD symptoms of inattention and hyperactivity/impulsivity) differentially contribute to the acquisition vs. performance of social skills. The social behavioral analysis framework defines social skills in terms of *observable*, empirically-identified and developmentally-expected social behaviors (Frey et al., 2011; Gresham et al., 2010), which differentiates it from approaches that focus on declarative social knowledge (Kofler et al., 2015; Leonard et al., 2011) or comprehension (Sibley et al., 2010). This emphasis on procedural skill demonstration is central to the Social Skills Improvement System (SSIS) alternate scoring method (Gresham et al., 2010), which produces estimates of social *acquisition deficits* (social behaviors for which the child lacks the necessary skills repertoire to perform) and *performance deficits* (social behaviors for which the child possesses the requisite procedural knowledge, but does not consistently apply that knowledge at developmentally expected levels).

Previous research supports the reliability and validity of this framework for analyzing parent- and teacher-reported social behaviors in preschool (Frey et al., 2011) and school-aged children (Gresham et al., 2010), both in terms of classifying social behaviors and differentially linking acquisition and performance deficits with specific intervention strategies (Frey et al., 2011). In addition, Aduen and colleagues (2017) recently demonstrated evidence supporting the construct and predictive validities of this method in a mixed sample of children with ADHD, other childhood disorders, and neurotypical children, and produced evidence that social problems in ADHD predominantly reflected social performance deficits.

Current study

The current study used a counterbalanced neurocognitive test battery, parent-and teacher-reports of children's ADHD symptoms and social skills, and Bayesian linear regression to determine the extent to which neurocognitive deficits and core ADHD behavioral symptoms predict ADHD-related social skills deficits. After establishing evidence for and against effects of each candidate mechanism on social skills, we conducted exploratory analyses using the SSIS alternate scoring approach (Gresham et al., 2010) to probe the extent to which these candidate mechanisms differentially predict the acquisition vs. performance of socially skilled behavior. Additionally, analyses of social *strengths* (important social behaviors demonstrated consistently) were included, given our group's broader goal of applying the positive youth development framework (Lerner et al., 2009) to promoting resiliency in children with ADHD.

We hypothesized that deficits in phonological and/or visuospatial working memory would predict overall social problems, as well as poorer social skills acquisition and performance,

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based on the consistency of these findings for global social problems in ADHD. We reasoned that working memory may be important for both skills acquisition and performance due to its role as a gateway between the environment and long-term memory (Baddeley, 2007). Hypotheses regarding processing speed and inhibition are more tentative given conflicting findings in the ADHD literature; however, it seemed reasonable to expect that children who more quickly process information (Shanahan et al., 2006) and are better able to monitor and stop unwanted actions (Alderson et al., 2007) would be better positioned to acquire and perform socially-skilled behavior, respectively.

Method

Bayesian Analyses

The benefits of Bayesian methods over null hypothesis significance testing (NHST) are well documented (Rouder & Morey, 2012; van de Schoot et al., 2013; Wagenmakers et al., 2016). For our purposes, Bayesian analyses were selected because they allow stronger conclusions by estimating the magnitude of support for both the alternative and null hypotheses (Wagenmakers et al., 2016). Bayesian linear regressions with JZS default prior scales (Morey & Rouder, 2015; Rouder et al. 2012) were conducted using JASP 0.8.1 (JASP Team, 2017). Instead of a *p*-value, these analyses provide BF_{10}, which is the Bayes Factor of the alternative hypothesis (H_1) against the null hypothesis (H_0). BF_{10} is an odds ratio, where values above 3.0 are considered moderate evidence supporting the alternative hypothesis (conceptual equivalent of $p < .05$). BF_{10} values above 10.0 are considered strong (≥30 = very strong, ≥100 = decisive/extreme support; Wagenmakers et al., 2016).

Conversely, BF_{01} is the Bayes Factor of the null hypothesis (H_0) against the alternative hypothesis (H_1). BF_{01} is the inverse of BF_{10} (i.e., $BF_{01} = 1/BF_{10}$), and is reported when the evidence indicates a lack of an effect (favors the null hypothesis, i.e., when $BF_{10} < 1$; Rouder & Morey, 2012). BF_{01} values are interpreted identically to BF_{10} (≥3.0 = moderate, ≥ 10.0 = strong, ≥ 100 = decisive/extreme support for the null hypothesis that a predictor does *not* have an effect on an outcome; Rouder & Morey, 2012).

Thus, finding $BF_{10} = 10.0$ would indicate that the observed data are 10 times more likely under the alternative hypothesis model (i.e., strong evidence for an effect), whereas $BF_{01} = 10.0$ would indicate that the observed data are 10 times more likely under the null hypothesis model (i.e., strong evidence of a lack of effect). Regression pathways supportive of an effect are supplemented with β-weights to inform effect magnitude and directionality.

Participants

The sample comprised 117 children aged 8 to 13 years (M=10.45, SD=1.53; 43 girls) from the Southeastern U.S., consecutively recruited by or referred to a university-based Children's Learning Clinic (CLC) through community resources (e.g., pediatricians, community mental health clinics, school system personnel, self-referral) between 2013 and 2017. The CLC is a research-practitioner training clinic known to the surrounding community for conducting developmental and clinical child research and providing *pro bono* comprehensive diagnostic and psychoeducational services. Its client base consists of children with suspected learning,

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behavioral or emotional problems, as well as typically developing children (those without a suspected psychological disorder) whose parents agreed to have them participate in developmental/clinical research studies. Psychoeducational evaluations were provided to caregivers. We obtained IRB approval prior to data collection, and all parents and children gave informed consent/assent. Sample ethnicity was mixed with 82 Caucasian/Non-Hispanic (69.5%), 13 Hispanic/English-speaking (11.0%), 11 African American (9.3%), 4 Asian (3.4%), and 8 multiracial children (6.8%).

Group Assignment

All children and their parents participated in a detailed, semi-structured clinical interview using the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Aged Children (K-SADS; Kaufman et al., 1997). The K-SADS (2013 Update) assesses onset, course, duration, severity, and impairment of current and past episodes of psychopathology in children and adolescents based on DSM-5 criteria (APA, 2013), and was supplemented with parent and teacher ratings from the Behavior Assessment System for Children (BASC-2; Reynolds & Kamphaus, 2004) and Child Symptom Inventory (CSI-IV; Gadow & Sprafkin, 2002).

Children were included in the ADHD group (n=77) based on meeting all of the following: (1) DSM-5 ADHD diagnosis by the directing clinical psychologist based on K-SADS; (2) parent ratings ≥1.5 *SD*s on the BASC-2 Attention Problems and/or Hyperactivity scales, or exceeding criterion scores on the parent CSI-IV ADHD-Inattentive and/or ADHD-Hyperactive/Impulsive subscales; and (3) teacher ratings ≥1.5 *SD*s on the BASC-2 Attention Problems and/or Hyperactivity scales, or exceeding criterion scores on the teacher CSI-IV ADHD-Inattentive and/or ADHD-Hyperactive/Impulsive subscales. All children had current impairment based on the K-SADS interview. Thirty-two children with ADHD (40.3%) were prescribed psychostimulants; medication was withheld for ≥24 hours before administration of neurocognitive tasks.

All ADHD subtypes/presentations were eligible given the instability of ADHD subtypes (Valo & Tannock, 2010). Of the 77 children with ADHD (27 girls), 42 met criteria for Combined, 30 for Inattentive, and 5 for Hyperactive/Impulsive Presentation. To improve generalizability, children with comorbidities were included. Comorbidities reflect clinical consensus best estimates, and included anxiety disorders (17.1%), depressive disorders (10.5%), oppositional defiant disorder (10.5%), and autism spectrum disorder (2.6%).[1]

The Non-ADHD group comprised 40 consecutive case-control referrals (16 girls) who did not meet ADHD criteria, and included both neurotypical children (n=24) and children with psychiatric disorders other than ADHD (n=16). Diagnoses in the non-ADHD group include anxiety (17.5%), autism spectrum (10.0%), depressive (7.5%), oppositional defiant (2.5%), and obsessive-compulsive disorders (2.5%).[1] Neurotypical children (60%) had normal developmental histories and nonclinical parent/teacher ratings, were recruited through community resources, and completed the same evaluation as clinically-referred cases. Non-

[1]The pattern and interpretation of results was unchanged when excluding children with autism spectrum disorder. As recommended in the K-SADS, oppositional defiant disorder was diagnosed clinically only with evidence of multi-informant/multi-setting symptoms.

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ADHD disorders were included to control for comorbidities in the ADHD group. Importantly, the ADHD and Non-ADHD groups were equivalent in the proportion of children diagnosed with a clinical disorder other than ADHD (overall: BF_{01} = 4.11; separated by disorder category: BF_{01} = 10.85).

Exclusion criteria included gross neurological, sensory, or motor impairment; history of seizure disorder, psychosis, or intellectual disability; or non-stimulant medications that could not be withheld for testing. No assessed cases were excluded as a result of these criteria.

Procedures

Neurocognitive testing occurred as part of a larger battery that involved two sessions of approximately three hours each. All tasks were counterbalanced across sessions to minimize order effects. Children received brief breaks after each task, and preset longer breaks every 2–3 tasks to minimize fatigue.

Neurocognitive Performance

Phonological and visuospatial working memory—The Rapport et al. (2009) computerized working memory tasks correctly classify children with vs. without ADHD at similar rates as parent and teacher ADHD rating scales (Tarle et al., 2017), and predict hyperactivity (Rapport et al., 2009), attention (Kofler et al., 2010), impulsivity (Raiker et al., 2012), and ADHD-related functional impairments (Friedman et al., 2016; Kofler et al., 2011, 2016). Reliability and validity evidence includes internal consistency (α=.82–.97), 1- to 3-week test-retest reliability (.76-.90; Sarver et al., 2015), and expected magnitude relations with criterion working memory complex span (*r*=.69) and updating tasks (*r*=.61) (Wells et al., 2015). Internal consistency in the current sample was α=.89 (visuospatial) and α=.81 (phonological).

Six trials were administered at each set size for each task (3–6 stimuli/trial; 1 stimuli/second). The 24 total trials per task were randomized, then grouped into 2 blocks of 12 trials each (Kofler et al., 2016). Five practice trials were administered before each task (80% correct required). Task duration was approximately 5 (visuospatial) to 7 (phonological) minutes.

Phonological working memory (PHWM): Children were presented a series of jumbled numbers and a letter (1 stimuli/second). The letter was never presented first or last to minimize primacy/recency effects, and was counterbalanced to appear equally in the other serial positions. Children reordered and recalled the numbers from least to greatest, and said the letter last (e.g., 4H62 is correctly recalled as 246H). Two trained research assistants, shielded from child view, independently recorded oral responses (interrater reliability=97.33%).

Visuospatial working memory (VSWM): Children were shown nine squares arranged in three offset vertical columns on a computer monitor. A series of 2.5 cm dots were presented sequentially (1 stimuli/second); no two dots appeared in the same square on a given trial. All dots were black except one red dot that never appeared first or last to minimize primacy/

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recency effects. Children reordered the dot locations (black dots in serial order, red dot last) and responded on a modified keyboard.

Dependent variables: Working memory: Partial-credit unit scoring (stimuli correct per trial) at each set size (3–6) was used as recommended (Conway et al., 2005).

Behavioral inhibition and processing speed

Stop-signal: Task and administration instructions were identical to Alderson and colleagues (2008). Psychometric evidence includes high internal consistency, 3-week test-retest reliability (both=.72), and convergent validity with other inhibition tests (Soreni et al., 2009). Internal consistency across the 4 blocks in the current sample was α=.89 (choice reaction time; CRT) and α=.83 (stop-signal delay; SSD).

Go-stimuli were displayed for 1000-ms as uppercase letters X and O positioned in the center of a computer screen (500-ms interstimulus interval; total trial duration=1500-ms). Xs and Os appeared with equal frequency. A 1000-Hz auditory tone (stop-stimulus) was presented randomly on 25% of trials. Stop-signal delay – the latency between go- and stop-stimuli presentation – was initially set at 250-ms, and dynamically adjusted +50-ms contingent on performance. The algorithm was designed to approximate successful inhibition on 50% of stop-trials. In the current study, inhibition success was 60.9%, 58.2%, 57.2%, and 54.6% across the four experimental blocks. Children completed two practice and four consecutive experimental blocks of 32 trials/block (8 stop-trials per block).

Dependent variables: Inhibition: SSD at each of the four blocks is the most direct measure of inhibition in stop-signal tasks that use dynamic stop-signal delays, because SSD changes systematically according to inhibitory success or failure (Alderson et al., 2007; Lijffijt et al., 2005).

Dependent variables: Processing speed: Mean CRT to correct go-trials during each of the four stop-signal blocks served as the primary indices of processing speed. Anticipatory responses (RTs<150-ms) were excluded.

Neurocognitive Dimension Reduction—Statistically, we controlled for task impurity by computing Bartlett weighted averages based on the intercorrelations among task performance scores (DiStefano et al., 2009). Conceptually, this process isolates “common and perfectly reliable variance” (Swanson & Kim, 2007, p.158) associated with each neurocognitive construct by removing task-specific demands associated with non-executive processes, time-on-task effects via inclusion of four blocks per task, and non-construct variance attributable to other measured executive processes. Thus, the 16 neurocognitive performance variables (4 blocks each for PHWM, VSWM, SSD, and CRT; 74.05% of variance explained) were reduced to four principal component estimates (Supplementary Table 1): PHWM (construct-specific loadings=.80-.85), VSWM (.81-.90), CRT (.75–.90), SSD (.66–.76). The ratio of participants (117) to factors (4) was deemed acceptable (Hogarty et al., 2005). By design, the intercorrelations among the varimax-rotated PHWM, VSWM, SSD, and CRT components were r_{all}=.00 (p>.99). Higher scores reflect better working

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memory and inhibition but slower processing speed. These neurocognitive component scores were used in all analyses below.

Global Intellectual Functioning (IQ)—All children were administered the WASI-II (Wechsler, 2011; *n*=32), WISC-IV Short Form (Wechsler, 2003; *n*=2), or WISC-V Short Form (Sattler et al., 2016; Wechsler, 2014; *n*=78) to obtain an overall estimate of intellectual functioning. Following Rapport et al. (2009), a residual IQ score was derived by covarying the neurocognitive variables described above out of Short-Form IQ (SFIQ; $R^2 = .18$, $p < .0005$). This residual IQ score represents intellectual functions that are important for IQ test performance but unrelated to executive functioning, and was computed to improve construct specificity because IQ performance depends heavily on more specific executive functions such as working memory, even for IQ subtests not labeled as 'working memory' measures (Ackerman et al., 2005; Dennis et al., 2009).

Social Skills

The Social Skills Improvement System (SSIS; Gresham et al., 2010) includes 46-item parent and teacher forms that assess social functioning relative to age- and gender-based norms (*N*=4,700; 6- to 8-week test-retest=.82–.86; α=.95-.97). Informants rate the frequency with which children exhibit each social skill on a 4-point scale (*never*, *seldom*, *often*, *almost always*), and the importance of each social skill to the child's development and classroom success on a 3-point scale (*not important*, *important*, *critical*). Internal consistency in the current sample was α= .96 (teacher frequency), α= .99 (teacher importance), α= .96 (parent frequency), and α= .99 (parent importance).

SSIS parent and teacher standard scores served as the primary DVs. Higher scores reflect better developed social skills. Exploratory analyses examined skills acquisition deficits, performance deficits, and strengths using the SSIS alternate scoring approach (Gresham et al., 2010). The SSIS alternate scoring produces estimates of social *acquisition deficits* (operationally defined as important/critical social behaviors that are endorsed as 'never' occurring), *performance deficits* (important/critical social behaviors that 'seldom' occur), and *strengths* (important/critical social behaviors that 'almost always' occur)[2]. Items not meeting the above criteria are considered neither strengths nor weaknesses. The percentage of items falling into each of these categories was computed for each child, separately for parent and teacher ratings. Higher percentages for *acquisition deficits* and *performance deficits* indicate more perceived social problems; higher percentages for *strengths* reflect better social functioning.

Socioeconomic Status (SES)—Hollingshead (1975) SES was estimated based on caregiver(s)' education and occupation.

[2]Notably, Aduen et al. (2017) demonstrated that results were robust to inclusion/exclusion of the SSIS importance ratings when computing acquisition/performance/strengths estimates (i.e., all ADHD/Non-ADHD between-group effect sizes were unchanged within Cohen's $d \pm 0.09$).

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Data Analysis Overview

We initially used Bayesian ANOVAs to examine evidence for between-group differences/equivalence in each neurocognitive ability and social skills estimate. Our primary and exploratory analyses are organized into two tiers. The primary Tier 1 analyses examine neurocognitive, behavioral, and demographic predictors of overall parent- and teacher-reported SSIS social skills. Parent-reported ADHD behavior ratings (BASC-2 Attention Problems, Hyperactivity) were used to predict teacher-reported social skills, and then reversed with teacher-reported behavior predicting parent-reported social skills, to prevent spurious associations attributable to mono-informant bias. Inattention and hyperactivity were included separately based on evidence that they differentially predict relations between neurocognitive abilities and other ADHD-related social problems (Bunford et al., 2015). We selected a hierarchical approach *a priori* in which we evaluated potential covariates for inclusion/exclusion (step 1) prior to evaluating evidence for the neurocognitive predictors (step 2). In each step, the best fitting model was selected (criteria: combination of predictors with highest $BF_{10} \geq 3$), and each additional predictor was tested relative to this best-fitting model (Rouder & Morey, 2012). Demographic and behavioral variables were entered in the first step of each Bayesian linear regression (age, gender, SES, IQ, attention problems, hyperactivity). Step 1 variables with predictor/outcome associations that favored the null (i.e., $BF_{01} \geq 3$) were removed prior to step 2. The four neurocognitive predictors were added in the second step (phonological working memory, visuospatial working memory, inhibitory control, processing speed). Separate models were estimated for parent- and teacher-reported SSIS social skills. These parent and teacher omnibus models were repeated in the exploratory Tier 2 models, which used the SSIS alternative scoring (described above) to parse overall social behavior into estimates of social skills acquisition deficits, performance deficits, and strengths. Finally, we conducted sensitivity analyses (Tier 3) to examine the impact of non-ADHD disorders on obtained results.

Results

Preliminary Analyses

All variables were screened for univariate/multivariate outliers. Acquisition and performance estimates for one ADHD child were winsorized relative to the ADHD group as recommended (Tabachnick & Fidell, 2007). Working memory and stop-signal data were reported for a subset of the ADHD sample in Kofler et al. (2018a and b); SSIS data were reported for a subset of the current sample in Aduen et al. (2018)[3]. Parent and teacher ADHD ratings were higher for the ADHD relative to Non-ADHD group as expected (Table 1). As shown in Table 1, the 4.15), and gender ($BF_{01} = 3.76$).

The ADHD group showed large deficits in VSWM (Cohen's d=0.89, $BF_{10} = 1.18 \times 10^3$) and medium deficits in PHWM (d=0.58, $BF_{10} = 9.50$). The evidence indicated that the ADHD and Non-ADHD groups were equivalent with regard to processing speed ($BF_{01} = 3.43$) and

[3]The neurocognitive data and SSIS data were reported in separate manuscripts to examine conceptually distinct hypotheses. We have not previously reported associations between SSIS social skills and any of these neurocognitive tasks.

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IQ (BF_{01} = 4.08). There was no evidence to support between-group differences in inhibitory control (BF_{01} = 1.53) (Table 2).

There was strong evidence indicating impaired social skills in the ADHD relative to Non-ADHD group based on both parent (*d*=0.82; BF_{10} = 409.08) and teacher report (*d*=0.88; $BF_{10} = 1.19 \times 10^3$). Parents and teachers were consistent in endorsing greater social performance deficits (*d*=0.87 and 0.70, respectively; both $BF_{10} > 50$) and fewer social strengths (*d*=0.99, 0.60; both $BF_{10} > 12$) in the ADHD vs. Non-ADHD group. Social skills acquisition deficits were indicated based on teacher report (*d* = 0.63; BF_{10} = 18.85), but the evidence indicated ADHD/Non-ADHD equivalence based on parent report (*d*=0.16; BF_{01} = 3.63).

Tier 1 Primary Analyses: Mechanisms associated with global social skills

Parent-reported social problems

Potential covariates: Results failed to provide strong support for any of the covariate models. The evidence trended toward support for effects of teacher-reported hyperactivity (BF_{10} = 2.93) and attention problems (BF_{10} = 1.94), but these effects failed to exhibit an adequate level of support. In contrast, there was evidence for lack of effects of age (BF_{01} = 5.01), gender (BF_{01} = 4.71), SES (BF_{01} = 4.43), and IQ (BF_{01} = 3.68). Therefore, only hyperactivity and inattention were retained for step two.

Neurocognitive predictors: Bayesian linear regression with default prior scales provided the strongest support for the model that included VSWM and teacher-reported hyperactivity symptoms (BF_{10} = 3.20). Inspection of the β-weights indicates that parents report better-developed social skills for children with better-developed VSWM (β= .17) and fewer hyperactivity symptoms (β= −.18).

With reference to this model, there was evidence for lack of an effect of inhibition (BF_{01} = 3.22). The evidence trended toward supporting a lack of effect for processing speed (BF_{01} = 1.45), phonological working memory (BF_{01} = 2.53), and teacher-reported attention problems (BF_{01} = 2.18), but there was insufficient evidence to conclusively rule out these effects.

Teacher-reported social problems

Potential covariates: We found the strongest support for the model that included parent-reported attention problems and IQ ($BF_{10} = 9.12 \times 10^3$). With reference to this model, there was evidence for lack of an effect of parent-reported hyperactivity symptoms (BF_{01} = 3.72), age (BF_{01} = 3.41), and gender (BF_{01} = 3.38). The evidence trended toward supporting a lack of effect for SES (BF_{01} = 2.39) but was insufficient to conclusively rule out this effect. Therefore, attention problems, IQ, and SES were retained for step two.

Neurocognitive predictors: We found the strongest support for the model that included VSWM, PHWM, IQ, and parent-reported attention problems ($BF_{10} = 7.92 \times 10^5$). Inspection of the β-weights indicated that teachers reported better-developed social skills for children with better-developed VSWM (β= .19), PHWM (β= .19), overall intelligence (β= .20), and fewer inattentive behaviors (β= −.34).

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With reference to this model, there was evidence for lack of an effect of processing speed (BF_{01} = 3.56) and SES (BF_{01} = 3.33). The data supported the null and alternative hypotheses equally with regard to inhibition (BF_{01} = 1.02, BF_{10} = 0.98), indicating no evidence for or against an association between inhibitory control and teacher-reported social skills.

Tier 2 Exploratory Analyses: Mechanisms associated with social skills acquisition vs. performance

We repeated the Tier 1 analyses, separately for social skills acquisition deficits, performance deficits, and strengths. Reporting is condensed for readability.

Social skills acquisition deficits

Parent-reported acquisition deficits: The best fitting final model included PHWM (β= −.20) and teacher-reported hyperactivity symptoms (β= .20) (BF_{10} = 3.65). There was evidence for lack of an effect of inhibition (BF_{01} = 3.06) and IQ (BF_{01} = 3.61) with reference to this model.

Teacher-reported acquisition deficits: The best fitting final model included only IQ (β= −.16) and parent-reported hyperactivity (β= .26) (BF_{10} = 5.40). There was evidence for lack of an effect of inhibition (BF_{01} = 3.05) and SES (BF_{01} = 3.30) with reference to this model.

Social skills performance deficits

Parent-reported performance deficits: The best fitting final model included only teacher-reported hyperactivity (β= .28) (BF_{10} = 12.49). There was evidence for lack of an effect of inhibition (BF_{01} = 3.74), age (BF_{01} = 3.85), gender (BF_{01} = 3.47), and SES (BF_{01} = 3.88) with reference to this model.

Teacher-reported performance deficits: The best fitting final model included PHWM (β= −.20), VSWM (β= −.18), parent-reported attention problems (β= .32), and gender (β= .27) ($BF_{10} = 1.93 \times 10^3$). There was evidence for lack of an effect of inhibition (BF_{01} = 3.09), SES (BF_{01} = 3.18), and parent-reported hyperactivity (BF_{01} = 3.12) with reference to this model.

Social skills strengths

Parent-reported strengths: The best fitting final model included only teacher-reported hyperactivity (β= −.23) (BF_{10} = 4.09). There was evidence for lack of an effect of inhibition (BF_{01} = 3.16), processing speed (BF_{01} = 3.16), PHWM (BF_{01} = 3.04), VSWM (BF_{01} = 3.34), and parent-reported attention problems (BF_{01} = 3.12) with reference to this model.

Teacher-reported strengths: The best fitting final model included PHWM (β= .16), VSWM (β= .14), IQ (β= .17), parent-reported attention problems (β= −.32), and gender (β= −.30) ($BF_{10} = 2.18 \times 10^3$). There was evidence for lack of an effect of processing speed (BF_{01} = 3.04), parent-reported hyperactivity (BF_{01} = 3.14), age (BF_{01} = 3.08), and SES (BF_{01} = 3.51) with reference to this model.

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Tier 3 Sensitivity Analyses: Effects of Comorbidities

Finally, we assessed the impact of our decision to include children with non-ADHD disorders in the ADHD and Non-ADHD groups. Collapsed across ADHD/Non-ADHD status, children with and without non-ADHD disorders were equivalent with regard to PH and VS working memory, inhibition, and teacher-reported social skills (all $BF_{01} > 3.80$) and did not differ significantly on processing speed ($BF_{01} = 2.20$) or parent-reported social skills ($BF_{01} = 0.98$). Non-ADHD Disorder status also failed to predict parent- (BF_{01} range = 0.97–2.23) and teacher-reported (all $BF_{01} > 3.17$) social skills in all Tier 1 and 2 models.

Discussion

The current study used Bayesian modeling, the social behavioral analysis framework, and a counterbalanced test battery to investigate neurocognitive dysfunction and ADHD behavioral symptoms as mechanisms associated with ADHD-related social problems. In doing so, we found strong evidence that both interfering behaviors (i.e., inattention and hyperactivity) and underdeveloped working memory abilities predict parent and teacher perceptions of children's social skills. These findings are consistent with the social behavioral analysis framework (Gresham et al., 2010), as well as studies of global social problems in ADHD (Bunford et al., 2015; Kofler et al., 2010, 2016; Tseng & Gau, 2013). The current study extends these findings by demonstrating that working memory is associated with both the acquisition and performance of prosocial behavior. Effective social interactions require working memory to dynamically decode non-verbal social cues (Phillips et al., 2007), encode and mentally process verbally-presented information in real time, evaluate this information in the context of previous experiences and the immediate context (Baddeley, 2007), and simultaneously organize and plan a socially appropriate response while minimizing interference from both task-unrelated thoughts (Kane et al., 2007) and irrelevant environmental stimuli (Kofler et al., 2011). Our results are consistent also with clinical (Hilton et al., 2017) and developmental studies (Kane et al., 2007; McQuade et al., 2013; Phillips et al., 2007), and indicate that working memory dysfunction may interfere with the acquisition of prosocial skills, in-the-moment performance of previously acquired social skills, and the ability to consistently and skillfully demonstrate acquired skills.

There was strong support for effects of working memory on both parent- and teacher-reported social skills, and working memory was implicated in social skills acquisition, performance, and strengths based on parent and/or teacher report. In contrast, there was no evidence supporting a role of inhibitory control or processing speed in any of the primary or exploratory analyses, and in most models there was strong evidence *against* associations between inhibitory control and social skills. These findings were contrary to our hypotheses, and indicate that inhibitory control likely serves a limited role in the development and practice of children's social behavior (Hilton et al., 2017; Tseng & Gau, 2013) – at least for elementary to early middle-school aged children.

These findings help clarify the mixed findings in the literature (Bunford et al., 2015; Kofler et al., 2016; Rinsky & Hinshaw, 2011; Tseng & Gau, 2013), and provide the first empirical support for the absence of effects (rather than lack of support for effects) of inhibition on ADHD-related social problems. This conclusion is consistent with meta-analytic

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conclusions that inhibitory control is likely intact in ADHD (Alderson et al., 2007; Lijffijt et al., 2005), as well as previous studies showing limited associations between inhibitory control and ADHD symptoms (e.g., Alderson et al., 2008).

Interestingly, previous evidence implicating inhibitory control (Bunford et al., 2015) and processing speed (Kofler et al., 2016) in ADHD-related social problems is based in part on data from the same stop-signal task used in the current study. A parsimonious explanation for the distinction regarding inhibition may be the use of the stop-signal reaction time (SSRT) metric in Bunford et al. (2015), relative to the stop-signal delay (SSD) metric used in the current study. SSRT reflects the difference between choice reaction time (CRT) and SSD, and has been criticized for confounding inhibition and processing speed in dynamic stop-signal tasks (Alderson et al., 2007). In the current study, we were able to parse variance separately for inhibitory control (SSD) and processing speed (CRT; Kofler et al., 2016). A more likely explanation involves our simultaneous modeling of multiple neurocognitive processes, such that effects of inhibitory control and processing speed may be artifacts of the working memory processes associated with maintaining the rule set required to interpret infrequently-occurring secondary stimuli and efficiently decide to inhibit (Garon et al., 2008).

An additional design feature that may account for these discrepancies involves our modeling of underlying neurocognitive mechanisms and proposed behavioral outcomes of these mechanisms as concurrent predictors. This approach was adopted in response to recent critiques of mediation modeling for cross-sectional data (de los Reyes, 2017), but may obfuscate detection of main effects when underlying mechanisms are entered simultaneously with putative outcomes of those mechanisms. A growing body of evidence indicates that experimentally increasing and decreasing demands on certain executive functions can evoke and rarefy ADHD-related inattentive (Kofler et al., 2010) and hyperactive behavior (Kofler et al., 2015; Rapport et al., 2009), suggesting that overt ADHD symptoms may be most accurately modeled as outcomes rather than concurrent predictors of underlying executive functioning processes (Bunford et al., 2015a). Recent studies of ADHD and social problems have employed mediation modeling to account for this conceptualization, although divergence in the field is indicated by the equal number of these studies that have modeled executive functioning as an outcome of ADHD symptoms (Huang-Pollock et al., 2009; Tseng & Gau, 2013) and vice versa (Bunford et al., 2015a; Kofler et al., 2011). The consistency in findings despite different assumed causal models speaks to the robust association between social problems and both neurocognitive abilities and interfering behaviors (Gresham et al., 2010) – a finding further supported by our primary models that demonstrated unique effects for both ADHD symptoms and working memory.

With regard to ADHD behavioral symptoms, we found strong evidence of associations with social behavior in every model tested despite our control for mono-informant bias. These findings were consistent with previous reports (Andrade et al., 2009; Humphrey et al., 2007), and extend this literature by demonstrating that different interfering behaviors may be implicated in different settings and/or for the acquisition vs. performance of social skills. Thus, our findings were broadly consistent with characterizations of social problems as secondary to the “intrusive … and generally aversive” nature of ADHD symptoms (Landau

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& Moore, 1991, p.235), as well as evidence that the direct link between ADHD symptoms and social problems is conveyed specifically by their shared risk for neurocognitive dysfunction (Bunford et al., 2015a).

Interestingly, there was strong evidence indicating that hyperactive/impulsive behavior likely interferes with the acquisition of social skills based on both parent and teacher report. In contrast, performance deficits and social strengths covaried with inattention based on teacher report but hyperactivity based on parent report. These findings are consistent with social learning models that posit both interfering behaviors (i.e., inattention and hyperactivity) and neurocognitive dysfunction in the acquisition and performance of developmentally-expected social skills (Frey et al., 2011; Gresham et al., 2010), as well as the DSM-5 clinical model conceptualization of social problems as one of three primary functional impairments in ADHD (APA, 2013). The discrepancy between parent and teacher models suggests that these informants may have different behavioral expectations for prosocial peer relationships (Lane et al., 2004), and/or view children's social behaviors in different contexts (e.g., large groups vs. playdates). Alternatively, these findings may be interpreted in light of evidence that teachers may be better reporters of functional impairments than parents (Langberg et al., 2013).

Limitations

The current study supported previous reports that linked ADHD-related social problems with working memory dysfunction and overt ADHD behaviors, and extended this literature by providing the first confirmatory evidence *against* effects of inhibitory control and processing speed. Several caveats merit consideration. Despite our large sample size, there were instances in which the evidence was insufficient to refute associations between social problems and key predictors. These estimates can be considered anecdotal support for the null (conceptually similar to $p>.05$), rather than the strong support that is desirable for conclusively ruling out an effect (Wagenmakers et al., 2016). In addition, the low base rates for social skills acquisition deficits may be related to the age of our sample; replication with younger children is needed to determine whether the nature of social problems in ADHD changes developmentally.

Parents and teachers provide reliable, ecologically valid data on children's social functioning, but generalizability is limited by biases (e.g., negative halo, expectancy). The cross-sectional data preclude conclusions regarding causality; longitudinal and/or experimental studies are needed. Longitudinal studies provide the strongest support for causality for constructs that cannot be manipulated experimentally; however, dual-task methodologies appear well-suited for manipulating specific neurocognitive demands (Baddeley, 2007), and may prove fruitful for understanding ADHD-related social problems when combined with dyadic observation methods (e.g., Stroes et al., 2003; Normand et al., 2011).

Finally, the SSIS alternate scoring used in our exploratory models emphasizes observable behavior and relies on the assumption that social skills can be inferred based on behavioral frequency (Gresham et al., 2010). Although previous studies have supported the construct and predictive validities of this method in ADHD and non-ADHD samples (Aduen et al.,

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2017; Gresham et al., 2010; Frey et al., 2011), it is certainly possible that children are socially skilled in ways that adult informants do not witness, or that they can sometimes perform a skill despite insufficient expertise (e.g., with scaffolding).

Clinical and Research Implications

Taken together, the evidence further emphasizes social problems as a key area of impairment among children with ADHD, and links their underdeveloped social skills with working memory dysfunction and ADHD behavioral symptoms that interfere with both skills acquisition and performance. To our knowledge, working memory has been implicated in every ADHD study to date that has predicted social problems based on specific executive functions (Bunford et al., 2015a; Hilton et al., 2017; Kofler et al., 2011, 2016; Tseng & Gau, 2013), with one exception. Rinsky & Hinshaw (2011) reported a 'marginal' predictive association between a digit span test and social functioning. The discrepancy between this study and those cited above may be related to its longitudinal design, covariation of ADHD status, and/or reliance on a test that shows poor convergence with criterion working memory tests (Bowden et al., 2013; Egeland, 2015; Engle et al., 1999; Swanson & Kim, 2007; Tarle et al., in press; Wells et al., 2015).

Our findings support conceptualizations of social problems as *in situ* performance deficits that are secondary to working memory 'glitches' (Abikoff et al., 2013). That is, parent and teacher perceptions of children's social skills appear to be associated, to a large extent, with children's ability to efficiently process information in the moment. This finding may explain why emerging interventions that reinforce *in vivo* social skills performance (Mikami et al., 2010) appear to be more effective than traditional approaches that explicitly teach social skills (Evans et al., 2014). That is, *in vivo* behavioral prompts and scaffolding reminders/redirection may inadvertently reduce the dual-processing demands involved in dynamically decoding verbal and nonverbal social cues while concurrently processing this information and organizing a prosocial response (Phillips et al., 2007).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Public Significance Statement

Social problems are prevalent in children with ADHD. Understanding the reasons for these difficulties is important for developing treatments that improve long-term outcomes for these children. The current study found evidence linking their social problems with specific cognitive impairments (working memory) and behavioral symptoms (inattentive/hyperactive behavior). It also refuted links between their social problems and other cognitive impairments (inhibitory control, processing speed) and demographic characteristics (age, gender, socioeconomic status).

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Table 1

Sample and Demographic Variables

Variable	ADHD (N=77)		Non-ADHD (N=40)		Cohen's *d*	BF_{10}	BF_{01}
	M	*SD*	*M*	*SD*			
Gender (Boys/Girls)	(50/27)		(24/16)		–		3.76
Age	10.37	1.48	10.67	1.58	0.20		3.06
SES	48.45	11.12	49.80	12.96	0.12		4.15
SFIQ	105.33	15.64	109.28	11.19	0.28		1.98
BASC-2 Attention Problems (T-score)							
Parent	67.38	10.10	56.53	10.48	−1.06	4.21×10^4	
Teacher	63.71	7.96	52.30	10.04	−1.31	9.91×10^6	
BASC-2 Hyperactivity (T-score)							
Parent	70.38	13.25	55.50	11.66	−1.17	4.37×10^5	
Teacher	61.58	12.37	53.68	12.95	−0.63	18.73	
SSIS Social Skills (Standard Scores)							
Parent	86.27	14.20	99.15	18.18	0.82	409.08	
Teacher	86.97	13.11	98.78	13.93	0.88	1.19×10^3	
SSIS Social Behavioral Subtypes (% of items endorsed)							
Parent							
Skill Acquisition Deficits	0.04	0.07	0.03	0.06	0.16		3.63
Social Performance Deficits	0.33	0.16	0.19	0.17	0.87	562.56	
Social Strengths	0.16	0.14	0.35	0.26	−0.99	4.93×10^3	
Teacher							
Skill Acquisition Deficits	0.06	0.09	0.02	0.03	0.63	18.85	
Social Performance Deficits	0.33	0.18	0.20	0.19	0.70	53.83	
Social Strengths	0.14	0.18	0.26	0.27	−0.60	12.07	

Note. BF_{10} = Bayes Factor for the alternative hypothesis over the null hypothesis (values > 3.0 indicate significant between-group differences). BF_{01} = Bayes Factor for the null hypothesis over the alternative hypothesis (values > 3.0 indicate significant between-group equivalence; $BF_{01} = 1/BF_{10}$); SFIQ = Short Form Intelligence Quotient (Standard Scores); SSIS = Social Skills Improvement System Rating Scales.

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Table 2

Neurocognitive Variables

Variable	ADHD (N=77)		Non-ADHD (N=40)		Cohen's *d*	BF_{10}	BF_{01}
	M	*SD*	*M*	*SD*			
Working Memory Performance Data (Stimuli Correct/Trial)							
PH 3	2.85	0.25	2.84	0.22	−0.04		4.69
PH 4	3.34	0.62	3.68	0.32	0.62	14.28	
PH 5	3.54	1.02	4.28	0.68	0.80	218.89	
PH 6	2.98	1.30	4.28	1.13	1.04	1.80×10^4	
VS 3	2.11	0.62	2.50	0.40	0.71	51.47	
VS 4	2.44	0.93	3.34	0.55	1.10	6.49×10^4	
VS 5	2.48	1.08	3.40	1.02	0.88	775.82	
VS 6	2.12	1.12	3.46	1.20	1.17	2.53×10^5	
Stop Signal Performance Data (milliseconds)							
MRT block 1	603.80	92.10	603.91	89.57	−0.01		4.75
MRT block 2	606.56	98.29	636.61	80.14	0.31		1.47
MRT block 3	604.67	98.42	634.70	85.19	0.33		1.53
MRT block 4	607.20	106.74	642.82	80.22	0.36		1.12
SSD block 1	269.64	76.66	302.96	72.68	0.59	1.85	
SSD block 2	260.99	84.54	303.62	79.53	0.54	3.94	
SSD block 3	265.33	82.09	302.30	73.11	0.44	2.37	
SSD block 4	263.08	90.67	301.15	82.61	0.41	1.68	
Derived Executive Function Component Scores (Z-scores)							
Visuospatial Working Memory	−0.28	1.00	0.54	0.76	0.89	1.18×10^3	
Phonological Working Memory	−0.19	1.09	0.37	0.66	0.58	9.50	
Inhibitory Control	−0.14	1.04	0.27	0.88	0.42	1.53	
Processing Speed	−0.06	1.06	0.11	0.88	0.17		3.43
$SFIQ_{res}$	0.53	13.99	−1.03	11.06	0.12		4.08

Note. BF_{10} = Bayes Factor for the alternative hypothesis over the null hypothesis (values > 3.0 indicate significant between-group differences). BF_{01} = Bayes Factor for the null hypothesis over the alternative hypothesis (values > 3.0 indicate significant between-group equivalence; BF_{01} = $1/BF_{10}$); MRT = Mean reaction time (milliseconds); PH = Phonological Working Memory (Stimuli Correct/Trial); SSD = stop-signal delay (milliseconds); VS = Visuospatial Working Memory (Stimuli Correct/Trial).