Common White Matter Microstructure Alterations in Pediatric Motor and Attention Disorders

Lisa Marie Langevin, PhD[1,2,3,4], Frank P. MacMaster, PhD[1,2,3,4,5,6], Susan Crawford, MSc[1,3,4], Catherine Lebel, PhD[2,3,7], and Deborah Dewey, PhD[1,2,3,4,8]

Objective To characterize white matter alterations in children with isolated or concurrent developmental coordination disorder and/or attention-deficit/hyperactivity disorder (ADHD) compared with typically-developing controls, and to determine whether group differences on motor and attention tasks could be explained by differences in diffusion tensor imaging (DTI) measures.

Study design In a cohort of children (n = 85) with developmental coordination disorder, ADHD, or combined developmental coordination disorder+ADHD, we examined 3 major white matter tracts involved in attention and motor processes. Using DTI, the corpus callosum, superior longitudinal fasciculus, and cingulum were analyzed with respect to measures of white matter integrity. Differences in fractional anisotropy (FA), mean diffusivity, radial diffusivity, and axial diffusivity were analyzed using ANOVA. Motor and attentional functioning was assessed using standardized tests, and correlated to DTI measures.

Results FA reductions were noted in the frontal regions of the corpus callosum for children with ADHD ($P = .039$), whereas children with developmental coordination disorder displayed similar reductions in regions of the corpus callosum underlying parietal brain regions ($P = .040$), as well as the left superior longitudinal fasciculus ($P = .026$). White matter integrity was impacted in both frontal and parietal regions for children with comorbid developmental coordination disorder+ADHD ($P = .029$; .046). FA was positively correlated with scores on both motor and attentional assessments in a region-specific manner.

Conclusion Our findings suggest that alterations in the corpus callosum underlie difficulties in motor and attention functioning. These changes are functionally and regionally distinct and could reflect a neurobiological basis for motor and attention disorders in children. *(J Pediatr 2014;164:1157-64)*.

Neurodevelopmental disorders, including attention-deficit hyperactivity disorder (ADHD) and developmental coordination disorder, demonstrate significant overlap with respect to clinical sequelae and prevalence. Disentangling the neurobiological basis of neurodevelopmental disorders is critical to advancing diagnosis and treatment, and maximizing outcomes for affected children. ADHD is a commonly diagnosed neurodevelopmental disorder characterized by inattention, behavioral impulsivity, and hyperactivity.[1] Children with ADHD also display atypical motor behaviors, impairing motor control, motor planning, and coordination.[2-4] In developmental coordination disorder, motor impairment represents the prominent feature, but attention, academics, and social functioning are also impacted.[5] Indeed, ADHD and developmental coordination disorder are found to co-occur in 30%-50% of cases.[4] Developmental coordination disorder and ADHD have also been found to show similar deficits in behavioral correlates of attention and executive function.[6] This high degree of overlap suggests a common neurobiological substrate for ADHD and developmental coordination disorder; however, whether motor and attention problems represent unrelated impairments or are symptomatic of disruption in common localized brain networks has not yet been determined.

Recent studies in white matter circuitry have found decreased white matter integrity in frontal regions of the corpus callosum in individuals with ADHD,[7] whereas others have shown alterations in the isthmus,[8,9] and splenium.[9] In developmental coordination disorder, frontal white matter has not been examined; however, decreases in frontal lobe activity have been reported.[10] Another component of frontostriatal cerebellar circuitry, the superior longitudinal fasciculus

AD	Axial diffusivity	MD	Mean diffusivity
ADHD	Attention deficit-hyperactivity disorder	MRI	Magnetic resonance imaging
ASF	Anterior/superior frontal	NDI	Neurodevelopmental index
DTI	Diffusion tensor imaging	RD	Radial diffusivity
FA	Fractional anisotropy	SLF	Superior longitudinal fasciculus
MAND	McCarron Assessment of Neuromuscular Development	SPP	Superior/posterior parietal

From the [1]Department of Pediatrics, University of Calgary; [2]Hotchkiss Brain Institute; [3]Alberta Children's Hospital Research Institute for Child and Maternal Health, University of Calgary; [4]Behavioral Research Unit, Alberta Children's Hospital; [5]Department of Psychiatry, University of Calgary; [6]The Mathison Center for Mental Health Research and Education; and Departments of [7]Radiology and [8]Community Health Sciences, University of Calgary, Calgary, Alberta, Canada

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(SLF), regulates somatosensory perception (lateral regions), along with attention, working memory, and motor behavior (medial regions).[11] The SLF in ADHD is correlated with attentional measures; yet examinations of white matter measures have been mixed.[12-15] Therefore, to differentiate regional vs functional alterations, we also examined the cingulum bundle, which runs longitudinally along the dorsal surface of the corpus callosum and connects frontal, parietal, and temporal regions like the SLF. Unlike the SLF, the cingulum has not been implicated in ADHD or motor functioning.[15,16]

The goal of this study was to use diffusion tensor imaging (DTI), an in vivo imaging technique allowing investigation of white matter, to examine the major white matter tracts of children with isolated or concurrent developmental coordination disorder and/or ADHD. DTI measures the movement of water molecules along hydrophobic axonal pathways, and yields quantitative information regarding the orientation, development, and integrity of the tract and related tissues.[17] Specifically, higher fractional anisotropy (FA) values and lower mean diffusivity (MD), axial diffusivity (AD), and radial diffusivity (RD) values, indicate denser and/or more myelinated axons. We also investigated the relationship between microstructural brain alterations and attentional/executive and motor functioning to help understand the contribution of these changes to the manifestations of developmental coordination disorder and ADHD. Identification of shared or distinct white matter alterations in ADHD and developmental coordination disorder could highlight new avenues for research examining the genetic and environmental contributors to these and other concurrent neurodevelopmental disorders.

Methods

A total of 85 children (64 male and 21 female) aged 8 to 17 years with attention and/or motor problems were recruited through local schools and physician referral to our study. Children with a diagnosed metabolic or genetic condition, very low birth weight (<1500 g), gestation <35 weeks, epilepsy or other seizure disorder, IQ below 80, autism, cerebral palsy, or contraindications to magnetic resonance imaging (MRI) scanning (ie, claustrophobia, ferrous implant) were excluded. The study included 9 children with developmental coordination disorder, 23 children with ADHD, 23 children meeting the criteria for both developmental coordination disorder and ADHD, and 26 typically-developing controls. Written consent was obtained from participants' parents under the approval of the Conjoint Health Research Ethics Board of the University of Calgary, and children provided verbal assent to participate in the study.

Demographic information is summarized in **Table I**. Motor skills were evaluated by the *Movement Assessment Battery for Children-2nd edition*,[18] a standardized measure of motor performance. Children were included in the developmental coordination disorder group if their standard score was below 7.0 (<16th percentile) on the *Movement Assessment Battery for Children-2nd edition*. Children classified as developmental coordination disorder also were reported by their parents to have motor difficulties that interfered with daily functioning as shown by lower total scores on the Developmental Coordination Disorder Questionnaire.[19] Children were classified as ADHD if they met the diagnostic criteria for ADHD on the Diagnostic Interview for Children and Adolescents IV,[20] and had a T score above the 95th percentile on the Conner's Parent Rating Scale-Revised.[21] The Diagnostic Interview for Children and Adolescents IV was completed by parents as a computerized interview and evaluates children on 2 criteria (A and B), which assess levels of inattention and hyperactivity, respectively. The Conner's Parent Rating Scale-Revised is a parent-completed questionnaire that includes 2 subscales that measure inattention and hyperactive/impulsive symptoms, respectively. Children meeting only 1 of the previous 2 criteria were classified as having ADHD if a clinician had previously diagnosed them. Children who met both classification criteria for developmental coordination disorder and ADHD were categorized into the developmental coordination disorder+ADHD group. Those participants not meeting either criterion were classified as controls.

The Wechsler Abbreviated Scale of Intelligence [22] was use to obtain an estimate IQ. Participants receiving pharmaceutical treatment for ADHD were asked to refrain from taking their medication on the day of testing. Attention and executive function were assessed using the Developmental NEuroPSYchological Assessment II, Auditory Attention, Response Set, and Inhibition subtests.[23] Scaled scores were reported, with impairment defined as a scaled score of <8.[23] Motor ability was assessed using the McCarron Assessment of Neuromuscular Development (MAND).[24] The MAND assessment tool uses 10 subtests to determine the level of fine and gross motor performance— (neurodevelopmental index [NDI]). NDI scores below 55 are indicative of severe impairment, 55-70 indicates moderate disability, and scores from 71-85 represent mild disability.[24]

MRI Data Acquisition

MRI was performed on a GE 3T Signa system scanner (GE Healthcare, Waukesha, Wisconsin) using a standard 8-channel phased-array radiofrequency head coil at the Seaman Family MR Research Center, University of Calgary. Single-shot echoplanar diffusion weighted images were acquired, spanning the entire brain. Twenty-six axial-oblique slices of 4 mm thickness with no interslice gaps were obtained, matrix 96 × 96, field of view 22.0 cm, repetition time = 8000 ms, echo time = 30 ms, b0 = 850 s/mm^2, flip angle = 90°. Data were acquired in 11 nonlinear directions with 3 unweighted image repetitions. Head movement was minimized with the use of memory foam pillows. Image quality was examined at the time of acquisition.

MRI Data Analysis

Three-dimensional deterministic tractography of the corpus callosum was completed in DTIStudio (Jiang and Mori,

Table I. Demographic characteristics of each group

	Control (n = 26)	Developmental coordination disorder (n = 9)	ADHD (n = 23)	Developmental coordination disorder +ADHD (n = 23)	*P*
Sex (male/female)	14/12	7/2	24/3	19/4	.019
Age (y)	11.58 ± 3.18	12.22 ± 2.68	11.78 ± 2.99	11.39 ± 2.89	.899
IQ	114.12 ± 13.07	105.56 ± 12.07	107.67 ± 13.07	102.35 ± 14.06	.027
DICA criterion A (yes/no)	2/24	3/6	24/3	21/1	.000*
DICA criterion B (yes/no)	0/26	1/8	12/15	6/16	.001
Conner's inattentive (T score)	51.17 ± 9.40	50.78 ± 4.71	72.16 ± 9.68	73.74 ± 9.62	.0001
Conner's hyperactive (T score)	50.21 ± 7.48	48.22 ± 4.18	70.08 ± 14.75	65.26 ± 13.71	.0001
MABC-2 (standard score)	9.731 ± 1.85	4.89 ± 1.27	9.11 ± 1.53	4.22 ± 1.86	.0001
Developmental coordination disorder questionnaire (total score)	72.870 ± 7.50	48.889 ± 13.18	56.381 ± 11.58	48.050 ± 12.73	.0001

DICA, Diagnostic Interview for Children and Adolescents; *MABC-2*, *Movement Assessment Battery for Children-2nd edition.*
In cases of significant group effect, pairwise testing revealed $P < .05$. Values are mean ± SD. Scores of "1" represent a score indicating significant impairment with respect to attention (DICA A criterion) or hyperactivity (DICA B criterion). On the Conners questionnaires, scores of "0" represent no indication of symptoms. Scores of "0" (not at all) to "3" (very often) are compiled and rated on each subscale.
*$P < .001$.

Baltimore, Maryland), using the fiber assignment by continuous tracking algorithm[25] with an FA initiation/termination threshold of 0.25 and an angle restriction of 70°. One operator (L.L.) blinded to age, sex, diagnosis, and participant identifiers performed the tractography by regions-of-interest drawn on FA-weighed color maps. The corpus callosum, SLF, and cingulum of each individual were traced according to previously described methods[26,27] and then segmented into distinct sections using anatomical landmarks. Within the corpus callosum, the anterior/superior frontal (ASF) region was defined as the region from the front of the head to a coronal slice at the posterior-most border of the genu. The superior/posterior parietal (SPP) region extended from this coronal slice to the boundary of the anterior-most aspect of the splenium. Finally, the temporal/occipital region extended from the SPP region to the back of the head, encompassing the entire posterior and inferior section of the corpus callosum. Spurious fibers not part of the corpus callosum were eliminated based on anatomical knowledge.

The SLF was subdivided at one-half of the distance between the pial surface and dorsolateral aspect of the third ventricle, creating both the medial (II) and lateral (III) SLF.[28] The frontal cingulum was demarcated by the coronal slice located at the inferior border of the underlying callosal genu to the front of the head. The body of the cingulum was delineated using an axial slice halfway through the genu.[27] The parahippocampal region was traced using a region at the most inferior axial slice where the overlying splenium could still be seen to cross the midline. Examples of tract subdivisions drawn can be viewed in Figure 1.

Diffusion measures were calculated for each region in MatLab using quanTract, an in silico tract segmentation tool (Luis Concha, 2008). Values were calculated for each region by averaging each voxel within the tract; each voxel was counted only once.

Statistical Analyses

Statistical analyses were performed using SPSS for Windows 19.0 (SPSS Inc, Chicago, Illinois). Intra-rater reliability determination was performed on a randomly selected set of 10 datasets with a mean correlation of 0.93. To test for group differences in corpus callosum, SLF, and cingulum subregions, a one-way ANOVA of DTI measures was performed for each tract subdivision. To control for multiple comparison effects on type I error, Tukey honestly significant difference test was used with significance set to $P < .05$. In regions with significant group differences only, relationships between attention/executive function, motor function, and measures of white matter integrity were examined using Pearson correlations, controlling for age. Differences were considered significant at a P value of <.05, following the corrections for between-group comparisons.

Results

Groups did not differ by age, but did show differences in full-scale IQ, with the control group having significantly higher IQs than the 3 other groups (Table I). All groups had average IQ scores within the normal range and no main effect of IQ was found; thus, IQ was not included as a covariate. Children in clinical groups scored significantly lower on standardized tests of motor and attention/executive function performance. Variation in sex distribution was significant; however this bias is consistent with the higher proportion of males diagnosed with attentional and motor problems in clinical pediatric populations.[29,30] No main effect of sex or interaction of group and sex was found, so we did not control for sex in further analyses.

Corpus Callosum

Consistent with our hypothesis, significant group effects were seen in the corpus callosum. Children in the ADHD and developmental coordination disorder+ADHD groups demonstrated reduced FA in the ASF of the corpus callosum with respect to controls (Table II and Figure 1, A). Children in the developmental coordination disorder group demonstrated no change in FA in this region with respect to controls (Table II and Figure 1, A). However, measurements of FA were lower in the SPP subregion for

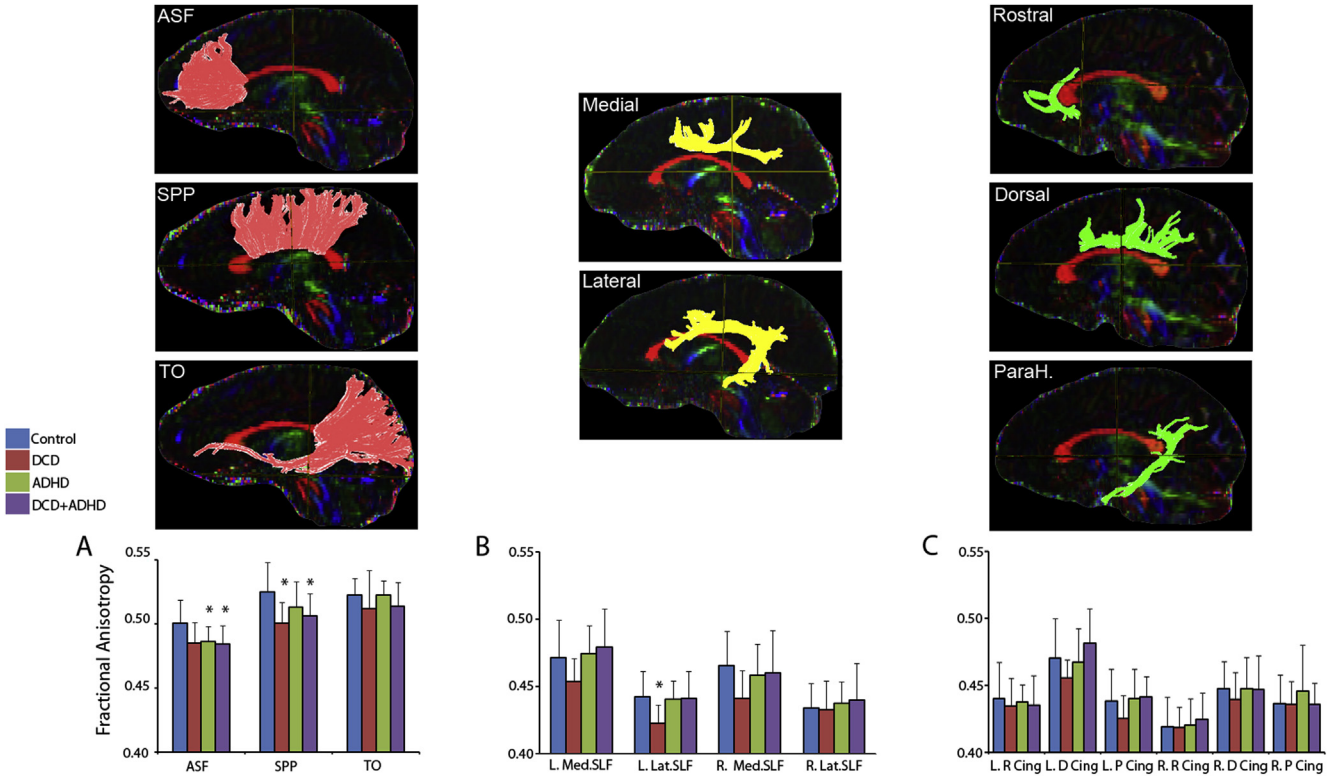

Figure 1. Sagittal colour map regions corresponding to deterministic ROIs and graphs showing average FA differences between clinical groups and controls. *Asterisks* (*) show significant differences between controls and each diagnostic group. No significant differences in the cingulum were observed. *Error bars* indicate SD. *DCD*, developmental coordination disorder; *R cing*, rostral cingulum; *D cing*, dorsal cingulum; *P cing*, parahippocampal cingulum.

the developmental coordination disorder and developmental coordination disorder+ADHD groups (Figure 1, A). No significant group differences were noted in the temporal/occipital subregion of the corpus callosum (Figure 1, A). For the tracts examined, group effects in RD were noted for the SPP corpus callosum (data not shown). Specifically, the developmental coordination disorder+ADHD group had significantly higher RD than controls and the ADHD group.

SLF and Cingulum

Children in the developmental coordination disorder group displayed FA reductions in the left lateral SLF III compared

Table II. Significance values for FA for group comparisons

	Tract Subdivision						
	ASF	SPP	TO	Left SLF II	Left SLF III	Right SLF II	Right SLF III
Developmental coordination disorder							
Mean	0.485	0.500	0.511	0.453	0.422	0.441	0.433
P	.152	.04*	.529	.275	.026*	.089	.998
95% CI	−0.0004, 0.0282	0.007, 0.043	−0.005, 0.027	−0.0017, 0.0373	0.002, 0.030	0.0040, 0.045	−0.015, 0.017
ADHD							
Mean	0.486	0.513	0.523	0.474	0.441	0.458	0.437
P	.039*	.254	.99	.971	.994	.768	.949
95% CI	0.002, 0.023	−0.0009, 0.026	−0.011, 0.012	−0.011, 0.011	−0.0087, 0.011	−0.0077, 0.022	−0.015, 0.009
Developmental coordination disorder+ADHD							
Mean	0.484	0.506	0.514	0.479	0.441	0.460	0.440
P	.029*	.046*	.511	.745	.999	.925	.827
95% CI	0.003, 0.026	0.0037, 0.032	−0.004, 0.021	−0.024, 0.0077	−0.0101, 0.0118	−0.011, 0.022	−0.0185, 0.007

TO, temporal/occipital.
All values are group differences from controls, using Tukey honestly significant difference. SLF II and SLF III denote the medial and lateral subdivisions of the SLF, respectively.
*$P < .05$.

with controls (Table II and Figure 1, B). No group differences were observed with respect to FA measurements of the cingulum (Figure 1, C). No group differences in MD were observed in any tract.

Structural-Functional Relationships

Structure-function relationships were investigated in regions with significant group effects. FA values in the corpus callosum showed positive correlations with scores on the Auditory Attention and Response Set subtests across groups (Figure 2, A and B). Significant positive correlations were also observed between scores on the Inhibition Switching subtest and FA in the ASF of the corpus callosum (Table III and Figure 2, C; Table III available at www.jpeds.com). FA in the SPP of the corpus callosum was correlated with scores on the Inhibition Combined Measure (Table III and Figure 2, D). RD was not correlated with any neurobehavioral scores. Significant positive correlations were found between the FA of the left and right SLF for NDI in the developmental coordination disorder+ADHD group. In the corpus callosum, correlations were significant for NDI scores across groups (Figure 2, E and F). The developmental coordination disorder+ADHD group displayed significant positive correlations between FA and NDI scores within the ASF of the corpus callosum (Figure 2, G). In the SPP portion, significant positive correlations were found between FA and NDI scores for both the developmental coordination disorder and developmental coordination disorder+ADHD groups (Figure 2, H and Table III). RD values in the SPP were not found to correlate with NDI scores in any group.

Discussion

In a cohort of 85 children, we utilized deterministic tractography to examine 3 major white matter tracts that connect frontal and motor areas within the brain (corpus callosum, cingulum, and SLF). We explored associations between attention/executive and motor measures,[14] and white matter microstructure. Concurrent motor and attention problems have not been examined previously, and this approach has allowed us to identify neuroanatomical deficits associated with developmental coordination disorder, ADHD, and comorbid developmental coordination disorder+ADHD. We have, therefore, demonstrated microstructural abnormalities in

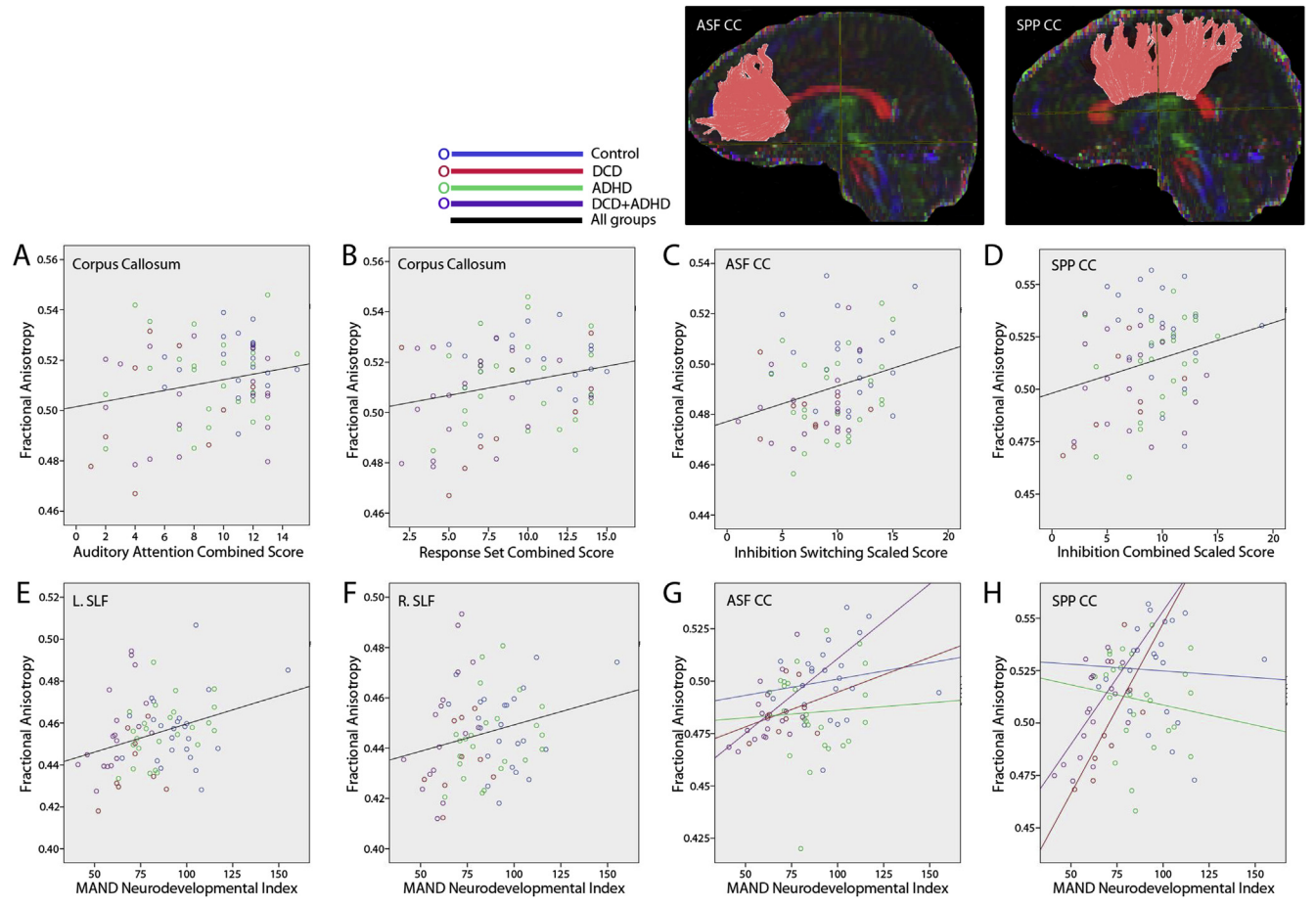

Figure 2. Sagittal colour maps showing callosal regions and significant correlations with measures of executive functioning.

the frontal regions for children with ADHD, as well as abnormalities in white matter connections underlying the primary and somatosensory motor cortices that are unique to developmental coordination disorder. Children with concurrent developmental coordination disorder+ADHD demonstrated alterations in both callosal regions. This suggests that the motor and attention problems in both ADHD and developmental coordination disorder have a common basis in the corpus callosum, but that these alterations are regionally and functionally distinct.

The corpus callosum has been implicated in ADHD in several studies.[7,8] Our study found FA to be significantly decreased in the ADHD and developmental coordination disorder+ADHD groups in the frontal region of the corpus callosum, which connects to prefrontal regions involved in the control of cognition, executive functions, and attention.[31] This finding is in agreement with previous research, which observed decreased FA in frontal white matter in individuals with ADHD.[32,33] The corpus callosum has been implicated in adult motor learning[34]; but this structure has not been previously examined in developmental coordination disorder. We demonstrated that children with developmental coordination disorder alone display abnormalities in the parietal regions of the corpus callosum, comprising connections to both primary and somatosensory motor areas.[35] The detection of significant alterations in motor regions of the corpus callosum is in agreement with core movement deficits seen in developmental coordination disorder. Anatomical disruption in the frontal and parietal regions of the corpus callosum plausibly provides the first neurobiological evidence to explain the high degree of comorbidity between these two disorders.[36]

We demonstrated higher RD values in the callosal region subserving the primary and supplementary motor areas in the developmental coordination disorder+ADHD group. The increase in RD could reflect delayed or defective myelination, in keeping with Shiverer mutant mouse studies.[37] We observed no changes in participants with ADHD and only subtle decreases in FA for the left SLF III in the developmental coordination disorder group. With respect to MD, RD, or AD, no changes were evident in any of the tracts studied for the single-diagnosis groups. As full tract measurements assume homogeneity, they may lack the discriminatory capacity to detect changes that exist in anatomical subdivisions. This could account for the absence of group differences. Children meeting criteria for both developmental coordination disorder and ADHD had higher FA values in the SPP region of the corpus callosum than for developmental coordination disorder alone. This is an unexpected finding given that lower FA measures are most often associated with clinically relevant disorders.[38] DTI measures are sensitive to myelination and axonal pruning, 2 processes that are active during preadolescence.[39,40] Studies of children and adolescents with ADHD have shown that brain maturation is delayed in prefrontal regions,[41] therefore, the increase in FA could also suggest a delay in the maturational trajectory of white matter in the developmental coordination disorder+ADHD group.[42] Future studies examining white matter in comorbid disorders would benefit from a longitudinal design to discriminate developmental delays vs permanent cortical deficits.

Our finding of anomalous callosal development in motor and attention disorders is validated by correlations between diffusion measurements and participant performance on standardized tests of attention/executive function and motor performance, suggesting that structural changes may impact behavior. Structure-function relationships are shown by the association between lower FA values and poorer scores on the Developmental NEuroPSYchological Assessment II Auditory Attention and Response Set subtests, both of which require sustained attention. White matter effects on executive functioning are demonstrated by the relationship between poorer performance on the Inhibition Switching subtest and reduced FA in the frontal regions of the corpus callosum. Consistent with the topography of the corpus callosum, FA reductions in the middle SPP region of the tract were correlated with lower overall NDI scores on the MAND, a measure of motor function.

When examined in conjunction with scores on neuropsychological testing, children with concurrent developmental coordination disorder+ADHD manifest other neuropsychological deficits that are unique (lower reading, math, and attention/executive functioning scores) than is seen in developmental coordination disorder or ADHD alone.[6] Coupled with the distinct patterns of FA variation, our findings reveal that children meeting the criteria for both developmental coordination disorder and ADHD exhibit specific macro- and microstructural changes that are distinct from those seen in singular diagnoses. This finding demonstrates motor and attention deficits are functionally and neurodevelopmentally distinct, yet share a common neurobiological substrate. The concept of combined motor and attention problems as a distinct disorder has been discussed previously by Gillberg, who introduced a syndrome referred to as Deficits in Attention, Motor control, and Perception as an operational variant of minimal brain dysfunction.[43] The combination of motor and attention problems has also been suggested to comprise a fourth subtype of ADHD.[44] Future investigations including cortical thickness analysis and longitudinal examinations of divergent brain development will be needed to validate this hypothesis. Examinations of the brain basis of developmental coordination disorder alone are few and the contribution of white matter alterations to motor behavior has not been fully investigated. Zwicker et al suggested that structural alterations in the corticospinal tract are correlated with motor impairment scores, and that these changes may contribute to deficits in the motor domain.[45,46] The corticospinal tract would be an important target in future examinations of the shared neurobiological mechanism underlying developmental coordination disorder and ADHD.

Cross-sectional neuroimaging studies do have inherent limitations. White matter development is susceptible to numerous influences, including environmental factors, prenatal infection, and medication use, which may introduce potential confounders.[47] Hand tracing, though highly valid

in avoiding problems with inter-subject registration, increases heterogeneity between studies. Our parcellation of the corpus callosum may introduce some overlap with respect to functional boundaries. Many children were recruited to our study through an assessment for suspected attentional problems and, thus, the number of children with developmental coordination disorder alone is small, and motor impairments in this disorder can be heterogeneous. Future studies could be more aggressive in recruiting this under-represented group. Full-scale IQ did differ among groups; however, all groups fell within the average range. Matching for IQ introduces a relatively narrow criterion for categorization, and group-matching IQ creates an artificial cohort that limits the relevance of the findings to the general population.[48] Future investigations would benefit from longitudinal protocols to better clarify the effects of white matter development on neurobehavioral outcomes.

Identification of the shared and/or separate etiologies for concurrent neurodevelopmental disorders represents a critical step in providing earlier identification and intervention for children and families. Imaging studies can circumvent challenges in clinical diagnostic paradigms by revealing alterations in the brain associated with specific disorders, helping to develop early screening protocols using morphometric indices. Here, we have shown that children with developmental coordination disorder and ADHD display regionally-distinct yet related alterations in the corpus callosum. We have additionally shown that concurrent developmental coordination disorder+ADHD displays aspects of both developmental coordination disorder- and ADHD-specific callosal abnormalities, as well as alterations in other white matter tracts. Our findings support the notion that developmental coordination disorder and ADHD share a common foundation in callosal structure, which may inform other future studies examining related or co-occurring disorders. This evidence may represent the first stage of the creation of an objective framework for empirically defining related neurodevelopmental disorders. ■

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Reprint requests: Lisa Marie Langevin, PhD, Departments of Pediatrics, University of Calgary, Behavioral Research Unit, Alberta Children's Hospital, 2888 Shaganappi Trail NW, Calgary AB T3B 6A8, Canada. E-mail: lisamarie.langevin@albertahealthservices.ca

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Table III. Group correlations of tract FA with executive function measures

Tract	Auditory attention	Response set	Inhibition switching	Inhibition combined	MAND NDI
Corpus callosum	*P* = .049 (all groups) R = 0.230	*P* = .039 (all groups) R = 0.241	ns	ns	ns
Corpus callosum ASF	ns	ns	*P* = .015 (all groups) R = 0.287	ns	*P* = .023 (developmental coordination disorder+ADHD) R = 0.284
Corpus callosum SPP	ns	ns	ns	*P* = .045 (all groups) R = 0.239	*P* = .037 (developmental coordination disorder); 0.001 (developmental coordination disorder+ADHD) R = 0.283 R = 0.238
Corpus callosum TO	ns	ns	ns	ns	ns
Left SLF	ns	ns	ns	ns	*P* = .008 (developmental coordination disorder+ADHD) R = 0.603
Right SLF	ns	ns	ns	ns	*P* = .019 (developmental coordination disorder+ADHD) R = 0.576

ns, not significant.