Psychological Medicine

www.cambridge.org/psm

Developmental milestones and resting-state functional connectivity: Shared and distinct associations with neurodevelopmental traits

Umer Jon Ganai[1,2] and Braj Bhushan[3]

[1]Department of Psychology, Neuroscience & Behaviour, McMaster University, Hamilton, Canada; [2]Jindal School of Psychology & Counselling, O. P. Jindal Global University, Sonipat, India and [3]Department of Humanities and Social Sciences, Indian Institute of Technology Kanpur, India

Original Article

Cite this article: Ganai, U. J., & Bhushan, B. (2026). Developmental milestones and resting-state functional connectivity: Shared and distinct associations with neurodevelopmental traits. *Psychological Medicine*, **56**, e277, 1–13
https://doi.org/10.1017/S003329172610556X

Received: 02 August 2025 UTC
Revised: 13 March 2026 UTC
Accepted: 08 July 2026 UTC

Keywords:
ADHD symptoms; autism traits; early developmental milestones; neurodevelopment; resting-state functional connectivity

Corresponding author:
Umer Jon Ganai;
Email: ganaiu@mcmaster.ca

Abstract

Background. Early motor and language milestones are key indicators of neurodevelopment, yet their associations with later autism traits and attention-deficit/hyperactivity disorder (ADHD) symptoms remain poorly understood. Neurobiological mechanisms linking early developmental delays to these conditions are not well established. Here, we investigate associations between early developmental milestones, resting-state functional connectivity (rsFC), and neurodevelopmental traits in a large adolescent sample.

Methods. Data were drawn from the Adolescent Brain Cognitive Development study (ABCD) and included 6,663 participants (age range = 8.92–11.08 years; mean age = 9.92 years; 48.42% female). Developmental measures included roll over, sit without assistance, walk without assistance, speech development, gross motor development, and coordination. Autism traits were assessed using the Social Responsiveness Scale, and ADHD symptoms were assessed using the Attention Problems subscale of the Child Behavior Checklist. rsFC was examined within and between cortical and subcortical regions. Linear mixed-effects (LME) models and Bayesian mediation analyses assessed associations between developmental measures, rsFC, and neurodevelopmental traits.

Results. LME models revealed shared and condition-specific associations. Poor coordination was associated with higher autism traits and ADHD symptoms, whereas delayed speech development was associated with autism traits, and delayed walking without assistance with ADHD symptoms. We found negligible evidence that rsFC measures mediated associations between early developmental milestones and neurodevelopmental traits. Indirect effects were modest, accounting for less than 2% of associations with autism traits, although somewhat larger effects were observed for ADHD symptoms.

Conclusions. These findings suggest overlapping developmental pathways, with intrinsic connectivity reflecting downstream neural processes rather than primary mediating mechanisms.

Introduction

Autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) are among the most common childhood neurodevelopmental conditions, typically emerging in the first years of life (APA, 2013). ASD is defined by persistent deficits in social communication and interaction, alongside restricted and repetitive patterns of behavior, interests, or activities. In contrast, ADHD is characterized by pervasive patterns of inattention, hyperactivity, and impulsivity that are inconsistent with developmental expectations (APA, 2013). While their diagnostic criteria are distinct, increasing evidence points to substantial overlap in genetic architecture (Rommelse et al., 2010) and shared alterations in brain structure and functional connectivity (Norman et al., 2025). Early motor and language delays are common in both ASD and ADHD (Johnson, Gliga, Jones, & Charman, 2014), supporting a shift toward viewing them as part of a neurodevelopmental continuum, with traits distributed dimensionally across the population (Constantino & Todd, 2003; Lubke et al., 2009).

Early-life motor and language development are a sensitive window into central nervous system maturation, and disruptions in these domains often represent the first observable signs of atypical development (Iverson, 2010). In ASD, delays in the attainment of gross and fine motor milestones, such as walking and hand coordination as well as language impairments, are consistently among the earliest predictors of later diagnosis (Johnson, Gliga, Jones, & Charman, 2014; Jones et al., 2013; Lord, Elsabbagh, Baird, & Veenstra-Vanderweele, 2018; Serdarevic et al., 2019). Longitudinal studies have shown that delays in walking and early expressive language (Kuo et al., 2022), as well as declining fine motor performance over the first three years of life (Landa, Gross, Stuart, & Faherty, 2012), are predictive of ASD. These delays are also linked to the emergence of social communication difficulties (Bhat, Landa, & Galloway, 2011). Maternal

© The Author(s), 2026. Published by Cambridge University Press. This is an Open Access article, distributed under the terms of the Creative Commons Attribution licence (http://creativecommons.org/licenses/by/4.0), which permits unrestricted re-use, distribution and reproduction, provided the original article is properly cited.

reports of fine motor difficulties by six months predict both formal diagnoses and elevated autistic traits in population-based samples (Bolton, Golding, Emond, & Steer, 2012). In addition to milestone delays, specific motor abnormalities, such as atypical gait, altered posture, and poor coordination, are frequently reported in young children with ASD (Ganai, Ratne, Bhushan, & Venkatesh, 2025; Singh et al., 2024). Language development is similarly affected, particularly among infants at familial risk, who often exhibit delays in both expressive and receptive domains (Prelock & Nelson, 2012). Recent evidence has further linked gross motor development and receptive language abilities to variation in autistic traits in the general population (Li et al., 2023), underscoring the relevance of these domains as early behavioral indicators of neurodevelopmental divergence.

In contrast to ASD, the relationship between early motor and language delays and ADHD is less consistent (Havmoeller, Thomsen, & Lemcke, 2018). Although many children with ADHD exhibit motor difficulties, including impairments in both gross and fine motor coordination (Kaiser, Schoemaker, Albaret, & Geuze, 2014), findings related to developmental milestone timing are more heterogeneous. Some studies report delayed walking and motor deficits in infancy (Gurevitz, Geva, Varon, & Leitner, 2012), while others find associations between earlier milestone attainment and later ADHD symptoms (Gui et al., 2025; Lemcke et al., 2016). In a longitudinal community-based cohort, motor, but not language, abilities in the first year of life predicted ADHD symptoms in adolescence (Jaspers et al., 2012). Nevertheless, early expressive and receptive language difficulties have also been implicated in later ADHD traits (Gurevitz, Geva, Varon, & Leitner, 2012), suggesting that developmental delays may contribute to ADHD along more variable and nuanced trajectories than those typically observed in ASD.

Neuroimaging studies provide further insight into the shared neural substrates of early motor and language development. Resting-state functional connectivity (rsFC) analyses in infants and toddlers have shown that these domains develop in parallel, involving overlapping interactions among large-scale brain networks, including the default mode, salience, visual, and frontoparietal networks (Bruchhage et al., 2020). Gross motor abilities in infancy have been linked to rsFC in sensorimotor and cerebellar regions, as well as to connectivity between the motor network and the default mode network (DMN) by 12 months (Marrus et al., 2018). By 24 months, this extends to include the dorsal attention and cingulo-opercular networks, mirroring the growing complexity of motor control and attentional integration. Notably, infants with familial risk for ASD show altered motor-DMN connectivity at 12 months and disrupted integration of attention and control networks by 24 months, potentially underlying both early motor delays and broader developmental divergence. In ADHD, altered rsFC between the primary motor cortex and frontal or subcortical structures, such as the inferior frontal cortex, insula, and basal ganglia, has been associated with motor coordination and planning difficulties (McLeod, Langevin, Goodyear, & Dewey, 2014). These findings implicate disrupted functional network development as a plausible mechanism for early emerging motor and language differences in both conditions.

Genetic evidence supports both shared and distinct developmental mechanisms across ASD and ADHD. Polygenic risk scores (PRSs) for each condition have been linked to variation in early motor and language development, including pragmatic communication and neuromotor coordination (Martin et al., 2014; Serdarevic et al., 2019). A recent large-scale cohort study found that higher ADHD PRSs were associated with earlier attainment of walking milestones, while higher ASD PRSs were associated with delays in walking (Hannigan et al., 2021). These results suggest that the timing of motor milestone attainment reflects underlying genetic susceptibility to neurodevelopmental conditions, even in the general population. Taken together, both rsFC and PRS findings point to biologically rooted, partly overlapping pathways that give rise to behavioral variability in early development.

Given the substantial overlap in neurodevelopmental mechanisms underlying ASD and ADHD, early motor and language milestones may serve as transdiagnostic indicators of neurodevelopmental risk. Yet, most existing studies have examined autism traits and ADHD symptoms in isolation, limiting our understanding of shared early-life predictors and the pathways through which diagnostic specificity emerges. Only a small number of investigations have adopted a dimensional framework that captures trait variability across the population, and fewer still have linked early behavioral development to brain-based measures in non-clinical adolescent cohorts. As a result, the potential of early motor and language milestones to index pluripotent neurodevelopmental risk across diagnostic boundaries remains underexplored.

The present study addresses this gap by leveraging a dimensional, population-based approach to examine how early motor and language milestone attainment relates to later autism traits and ADHD symptoms. We assessed a comprehensive set of early developmental behaviors, including rolling, sitting, walking, speech development, and motor coordination, and their associations with both behavioral outcomes and rsFC. We then tested whether patterns of rsFC mediated the relationship between early milestones and later traits. By jointly evaluating behavioral and neural markers across domains, this study aims to identify early-life pathways of neurodevelopmental divergence, clarify the nature of transdiagnostic risk, and provide insight into mechanisms of developmental differentiation between ASD and ADHD.

Methods

Participants

Participants were drawn from the Adolescent Brain Cognitive Development (ABCD) Study, 'Curated Annual Release 4.0' (https://nda.nih.gov/study.html?id=1299). The ABCD Study is an ongoing, longitudinal, multi-site investigation of brain development and health in youth, with data collected from over 11,000 children representing a diverse demographic background (Casey et al., 2018). For the present study, participants with missing data were excluded. The final analytic sample comprised 6,663 participants (Table 1). Unless otherwise specified, only baseline assessments were used. Written informed consent was obtained from parents or legal guardians after a full explanation of the study procedures, and assent was obtained from all child participants. The ABCD Study was approved by the central Institutional Review Board at the University of California, San Diego, as well as by the institutional review boards of each participating site (Casey et al., 2018).

Measures

Autism traits and ADHD symptoms

Autism traits were assessed using the abbreviated version of the Social Responsiveness Scale, Second Edition (Constantino & Gruber, 2011). The Short Social Responsiveness Scale (S-SRC) was administered in year 1, with parents responding to items on a four-point scale ranging from

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

Table 1. Sample characteristics (N = 6,663)

Characteristic	*n* or *M* (SD) or %
Male/female	3,437/3,226
Age in years	9.92 (0.63)
Household income	
≤ $50,000	1,710
≥ $50,000 to < $100,000	1,787
≥ $100,000	2,692
Do not know and refuse to answer	474
Race/Ethnicity	
White	3,667
Black	817
Hispanic	1,385
Asian	121
Other	673
Autism traits	14.18 (3.87)
ADHD symptoms	2.84 (3.41)
Roll over (attained/delayed)	5,599 (84.1) / 1,064 (15.9)
Sit without assistance (attained/delayed)	6336 (95.1) / 327 (4.90)
Walk without assistance (attained/delayed)	6422 (96.38) / 241 (3.62)
Gross motor development	2.60 (0.80)
Speech development	2.70 (0.99)
Coordination	0.13 (0.37)

'Not true' (1) to 'Almost always true' (4). The total score of the S-SRC was calculated by summing all items. ADHD symptoms were measured using the Attention Problems subscale from the Child Behavior Checklist (Achenbach & Ruffle, 2000). The total score on this subscale was used as a continuous measure of ADHD symptom severity.

Developmental assessments

Developmental milestone data were obtained through the Developmental History Questionnaire (DHQ), completed retrospectively by the child's biological mother. The questionnaire assessed the age in months at which the child achieved key milestones, including roll over, sit without assistance, and walk without assistance. Two items assessing gross motor and speech development were rated on a five-point scale ranging from 1 ('much earlier') to 5 ('much later') relative to peers. Milestone ages were binarized based on the Centers for Disease Control and Prevention (CDC) developmental guidelines, which represent the median age (50th percentile) by which children typically acquire these skills: roll over by 4 months, sit without assistance by 9 months, and walk without assistance by 18 months (Zubler et al., 2022). Gross motor and speech development scores were treated as continuous variables. An additional indicator of motor development, the item 'poorly coordinated or clumsy' from the CBCL, was included, with higher scores reflecting greater coordination difficulties (Achenbach & Ruffle, 2000).

Imaging acquisition, preprocessing, and resting-state functional connectivity

Imaging data were acquired as part of the ABCD Study, using harmonized protocols across multiple sites (Casey et al., 2018). Details of image acquisition, preprocessing, and quality control for rsFC are provided in the Supplementary Materials. Briefly, this study utilized preprocessed resting-state fMRI data provided by the ABCD Study (Hagler et al., 2019). Cortical parcellation was based on the Gordon atlas, which delineates functionally distinct cortical networks (Gordon et al., 2014), while subcortical segmentation was performed using FreeSurfer's automated subcortical atlas (Fischl et al., 2002). In total, 55 cortical within- and between-network connectivity measures and 50 cortical–subcortical connectivity measures were derived per participant. These included within- and between-network rsFC of the auditory (AN), cingulo-opercular (CO), default mode (DMN), salience (SAL), fronto-parietal (FPN), ventral attention (VAN), somatomotor hand (SMH), visual (VIS), dorsal attention (DAN), and somatomotor mouth (SMM) networks (Figure 1). Subcortical regions of interest included the averaged left and right cerebellar cortex, thalamus, caudate nucleus, putamen, and nucleus accumbens.

Statistical analysis

All analyses were conducted using *R* (R Core Team, 2025). We first fitted separate linear mixed-effects (LME) models to examine associations between six early developmental measures: roll over, sit without assistance, walk without assistance, gross motor development, speech development, and coordination, and two behavioral outcomes of autism traits and ADHD symptoms. Models were implemented with the *lmerTest* package (Kuznetsova, Brockhoff, & Christensen, 2017). Age, sex, income, and race/ethnicity (as unordered factors) were included as fixed-effect covariates, and study site and family ID were modeled as random effects to account for clustering. When modeling autism traits, ADHD symptoms were included as a covariate; conversely, when modeling ADHD symptoms, autism traits were included as a covariate. All continuous predictors were standardized prior to analysis. Statistical significance was set at $p < 0.05$, and false discovery rate (FDR) correction using the Benjamini–Hochberg procedure (Benjamini & Hochberg, 1995) was applied to adjust for multiple comparisons across developmental predictors.

Subsequently, a series of LME models examined associations between resting-state functional connectivity (rsFC) metrics and the six developmental variables, with each rsFC measure treated as a separate outcome. Models included fixed effects for age, average framewise displacement (motion), sex, income, and race/ethnicity, with site and family ID as random effects. Continuous variables were standardized, and significance was defined at $p < 0.05$ with FDR correction applied to account for multiple comparisons.

Finally, Bayesian multilevel mediation analyses were conducted to test whether specific rsFC measures mediated the relationships between early developmental milestones and behavioral outcomes. Models included the same fixed covariates and site and family ID as random effects. When modeling autism traits, ADHD symptoms were included as a covariate; conversely, when modeling ADHD symptoms, autism traits were included as a covariate. Analyses were performed using the *brms* package for Bayesian hierarchical modeling (Bürkner, 2017), and mediation effects were summarized with *bayestestR* (Makowski, Ben-Shachar, & Lüdecke, 2019). Continuous variables were standardized to improve model convergence and interpretability. Mediation was considered meaningful when the 95% credible interval (CrI) of the indirect effect did not include zero. Indirect effects and proportion mediated were estimated and reported accordingly.

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

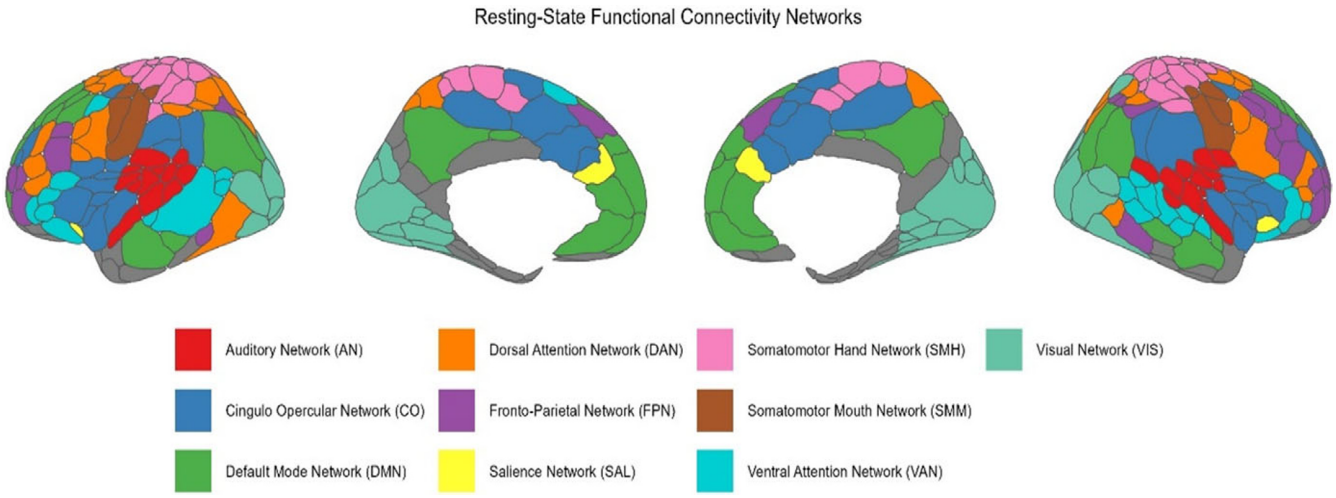


Figure 1. Resting-state functional connectivity (rsFC) networks.

Results

Associations between developmental measures and autism traits and ADHD symptoms

Associations between early developmental measures and later behavioral outcomes are summarized in Table 2. Delayed *speech development* (β = 0.11, 95% CI [0.08, 0.13], FDR-p < 0.0001) and poor *coordination* (β = 0.12, 95% CI [0.09, 0.14], FDR-p < 0.0001) showed significant association with elevated autism traits. In contrast, later attainment of *walk without assistance* (β = 0.14, 95% CI [0.03, 0.26], FDR-p = 0.03), and poor *coordination* (β = 0.26, 95% CI [0.24, 0.28], FDR-p < 0.0001) were associated with higher ADHD symptomatology. More advanced *gross motor development* was inversely associated with ADHD symptoms (β = − 0.04, 95% CI [−0.06, −0.01], FDR-p = 0.006).

Associations between developmental measures and resting-state functional connectivity

Significant associations between early developmental milestones and rsFC are detailed in Supplementary Materials (Supplementary Table S1). The milestone *roll over* was significantly associated with multiple rsFC measures. Positive associations were observed with connectivity between the auditory and somatomotor hand networks (*AN–SMH*; β = 0.11, 95% CI = [0.04, 0.18], FDR-p = 0.009), auditory and somatomotor mouth networks (*AN–SMM*; β = 0.09, 95% CI = [0.02, 0.16], FDR-p = 0.0443), within the auditory network (*AN*; β = 0.08, 95% CI = [0.01, 0.15], FDR-p = 0.0467), somatomotor hand network (*SMH*; β = 0.10, 95% CI = [0.03, 0.17], FDR-p = 0.0108), and between the somatomotor hand and mouth networks (*SMH–SMM*; β = 0.11, 95% CI = [0.04, 0.18], FDR-p = 0.0128). In contrast, *roll over* was negatively associated with connectivity between the cingulo-opercular and visual networks (*CO–VIS*; β = −0.11, 95% CI = [−0.18, −0.04], FDR-p = 0.0155), the fronto-parietal and somatomotor mouth networks (*FPN–SMM*; β = −0.12, 95% CI = [−0.19, −0.05], FDR-p = 0.0070), and between the somatomotor hand and putamen (*SMH–Put*; β = −0.10, 95% CI = [−0.17, −0.04], FDR-p = 0.0179).

Walk without assistance was associated with reduced connectivity between the somatomotor hand and salience networks (*SMH–SAL*; β = −0.20, 95% CI = [−0.33, −0.06], FDR-p = 0.0128).

Table 2. Associations between developmental measures and autism traits and ADHD symptoms

	Variable	β	95% CI	SE	t	*d.f.*	p	FDR-p
Autism traits	Roll over	0.02	−0.04 to 0.08	0.03	0.64	6547.19	0.52	0.77
	Sit without assistance	0.01	−0.10 to 0.10	0.05	0.00	6515.95	0.99	0.99
	Walk without assistance	0.06	−0.05 to 0.18	0.06	1.06	6506.18	0.29	0.58
	Gross motor development	0.01	−0.02 to 0.03	0.01	0.47	6590.49	0.64	0.77
	Speech development	0.11	0.08 to 0.13	0.01	9.05	6596.82	< 0.0001	< 0.0001
	Coordination	0.12	0.09 to 0.14	0.01	10.37	6634.73	< 0.0001	< 0.0001
ADHD symptoms	Roll over	0.05	−0.01 to 0.11	0.03	1.76	6543.20	0.08	0.08
	Sit without assistance	0.09	−0.01 to 0.19	0.05	1.74	6516.45	0.08	0.08
	Walk without assistance	0.14	0.03 to 0.26	0.06	2.50	6510.80	0.01	0.03
	Gross motor development	−0.04	−0.06 to −0.01	0.01	−3.07	6587.63	0.002	0.006
	Speech development	0.02	0.00 to 0.04	0.01	1.86	6604.82	0.06	0.08
	Coordination	0.26	0.24 to 0.28	0.01	24.81	6636.87	< 0.0001	< 0.0001

Note: SE, standard error; d.f., degrees of freedom; FDR, false discovery rate.

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

Gross motor development showed both positive and negative associations with rsFC. Negative associations were identified for connectivity between the auditory and somatomotor hand (*AN–SMH*; β = −0.04, 95% CI = [−0.07, −0.01], FDR-p = 0.0212), auditory and somatomotor mouth (*AN–SMM*; β = −0.06, 95% CI = [−0.09, −0.03], FDR-p = 0.0002), somatomotor hand and mouth (*SMH–SMM*; β = −0.04, 95% CI = [−0.06, −0.01], FDR-p = 0.0263), within the somatomotor hand network (*SMH*; β = −0.04, 95% CI = [−0.07, −0.02], FDR-p = 0.0091), and auditory network (*AN*; β = −0.03, 95% CI = [−0.06, 0.00], FDR-p = 0.0467). Positive associations emerged with connectivity between the somatomotor hand and salience networks (*SMH–SAL*; β = 0.05, 95% CI = [0.02, 0.08], FDR-p = 0.0042), the somatomotor hand and caudate (*SMH–Caud*; β = 0.05, 95% CI = [0.02, 0.08], FDR-p = 0.0014), and the salience network and cerebellar cortex (*SAL–Cer*; β = 0.04, 95% CI = [0.01, 0.07], FDR-p = 0.0291).

Speech development was negatively associated with connectivity between the auditory network and caudate (*AN–Caud*; β = −0.04, 95% CI = [−0.07, −0.02], FDR-p = 0.0119), between the visual network and putamen (*VIS–Put*; β = −0.04, 95% CI = [−0.07, −0.02], FDR-p = 0.0120), and within-network connectivity of auditory network (*AN*; β = −0.03, 95% CI = [−0.05, 0.00], FDR-p = 0.0467).

Lastly, *coordination* was associated with a distributed pattern of network-level alterations. Negative associations were observed with connectivity between the auditory and somatomotor hand networks (*AN–SMH*; β = −0.03, 95% CI = [−0.05, −0.01], FDR-p = 0.0321), salience network and nucleus accumbens (*SAL–Acc;* β = −0.03, 95% CI = [−0.06, −0.01], FDR-p = 0.0467), the cingulo-opercular network (*CO*; β = −0.04, 95% CI = [−0.06, −0.02], FDR-p = 0.001), the default mode network (*DMN*; β = −0.03, 95% CI = [−0.06, −0.01], FDR-p = 0.0129), the somatomotor hand network (*SMH*; β = −0.03, 95% CI = [−0.05, 0.00], FDR-p = 0.0408), and the auditory network (*AN*; β = −0.03, 95% CI = [−0.05, 0.00], FDR-p = 0.0467). Positive associations were observed between the coordination and connectivity between the default mode and dorsal attention networks (*DMN–DAN*; β = 0.03, 95% CI = [0.01, 0.06], FDR-p = 0.0189), auditory and visual networks (*AN–VIS*; β = 0.03, 95% CI = [0.01, 0.06], FDR-p = 0.0362).

Mediation results

There was negligible evidence that rsFC measures exhibited meaningful indirect effects mediating the associations between early developmental measures and neurodevelopmental traits. Detailed results are provided in the Supplementary Materials (Supplementary Tables S2 and S3 for autism traits and ADHD symptoms, respectively). Key mediation effects are summarized in Figures 2 and 3 for autism traits and ADHD symptoms, respectively, while rsFC networks are visualized in Figure 4 (cortical networks) and Figure 5 (cortical–subcortical connectivity).

For autism traits, connectivity between the *FPN–SMM mediated 1.49% of the association with roll over* (indirect effect: β = 0.001, 95% CrI = [0.00, 0.004]). Connectivity within the *AN* mediated 0.43% of the association between *speech development* and autism traits (indirect effect: β = 0.0005, 95% CrI = [0.000, 0.002]).

For *gross motor development*, connectivity between the *AN–SMM* connectivity *mediated 1.90%* of the association with autism traits (indirect effect: β = 0.001, 95% CrI = [0.00, 0.003]). Connectivity between the *SMH–Caud mediated 1.54%* (indirect effect: β = 0.001, 95% CrI = [0.001, 0.000]), *while connectivity between the SMH–SMM mediated 0.30%* (indirect effect: β = 0.00023, 95% CrI = [0.000, 0.001]). *Within-network connectivity of the SMH* (indirect effect: β = 0.00049, 95% CrI = [0.000, 0.002]) *and AN* (indirect effect: β = 0.00066, 95% CrI = [0.00, 0.002]) mediated 0.63% and 0.86% of the association between *gross motor development* and autism traits, respectively.

For coordination within-network connectivity of *AN* (indirect effect: β = 0.001, 95% CrI = [0.00, 0.004]) and *DMN* (indirect effect: β = 0.00074, 95% CrI = [0.00, 0.003]) mediated 0.32% and 0.23% of the association with autism traits, respectively.

For ADHD symptoms, connectivity between the *AN–Caud mediated 6.70% of the association with speech development* (indirect effect: β = 0.001, 95% CrI = [0.000, 0.003]). *For gross motor development connectivity between the AN–SMH* mediated 10.61% of the association with ADHD symptoms (indirect effect: β = 0.00048, 95% CrI = [0.000, 0.002]). Connectivity between the *SAL–Cer* mediated 28.54% (indirect effect: β = 0.001, 95% CrI = [0.000, 0.003]), connectivity between the *SMH–Caud* mediated 17.65% (indirect effect: β = 0.0009, 95% CrI = [0.000, 0.002]), and connectivity between the *SMH–SAL* mediated 44.32% (indirect effect: β = 0.002, 95% CrI = [0.000, 0.004]). Within-network connectivity of the *SMH* connectivity mediated 11.30% of the association between gross motor development and ADHD symptoms (indirect effect: β = 0.00053, 95% CrI = [0.000, 0.002]).

For *coordination*, connectivity between the *DMN–DAN* mediated 0.32% (indirect effect: β = 0.002, 95% CrI = [0.000, 0.005]) of the association with ADHD symptoms, while within-network connectivity of the *DMN* (indirect effect: β = 0.001, 95% CrI = [0.000, 0.004]) mediated 0.19%.

When examining the path coefficients, several small but credible associations were observed between early developmental predictors, rsFC measures, and neurodevelopmental traits. Inverse associations were observed between *roll over* and connectivity between the *FPN–SMM* (β = −0.08, 95% CrI = [−0.15, −0.02]), *gross motor development* and connectivity between the *AN–SMM* (β = −0.07, 95% CrI = [−0.10, −0.04]), *gross motor development* and within-network connectivity of the *SMH* (β = −0.04, 95% CrI = [−0.07, −0.01]), and *coordination* and connectivity within the *AN* (β = −0.08, 95% CrI = [−0.15, −0.01]).

A small positive association was observed between *gross motor development* and connectivity between the *SMH–Caud* (β = 0.06, 95% CrI = [0.03, 0.08]). In addition, a negative association was observed between connectivity between the *AN–SMM* and autism traits (β = −0.02, 95% CrI = [−0.04, −0.00]).

Similarly, several inverse associations were observed for ADHD symptoms, including between *speech development* and *AN–Caud* connectivity (β = −0.05, 95% CrI = [−0.07, −0.03]), *gross motor development* and *AN–SMH* connectivity (β = −0.03, 95% CrI = [−0.06, −0.01]), and *coordination* and within-network connectivity of the *DMN* (β = −0.07, 95% CrI = [−0.13, −0.01]).

Positive associations were also observed between predictors and rsFC measures for ADHD symptoms, including between *gross motor development* and connectivity between the *SAL–Cer* (β = 0.05, 95% CrI = [0.02, 0.08]) and *SMH–Caud* (β = 0.05, 95% CrI = [0.03, 0.08]), as well as between *coordination* and connectivity between the *DMN–DAN* (β = 0.07, 95% CrI = [0.01, 0.13]).

Lastly, several associations were observed between rsFC measures and ADHD symptoms. Connectivity between the *AN–Caud* was inversely associated with ADHD symptoms for *speech development* (β = −0.03, 95% CrI = [−0.05, −0.01]). Positive associations with ADHD symptoms were observed for connectivity between the *SAL–Cer* (β = 0.03, 95% CrI = [0.01, 0.05]), *SMH–SAL* (β = 0.03, 95%

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

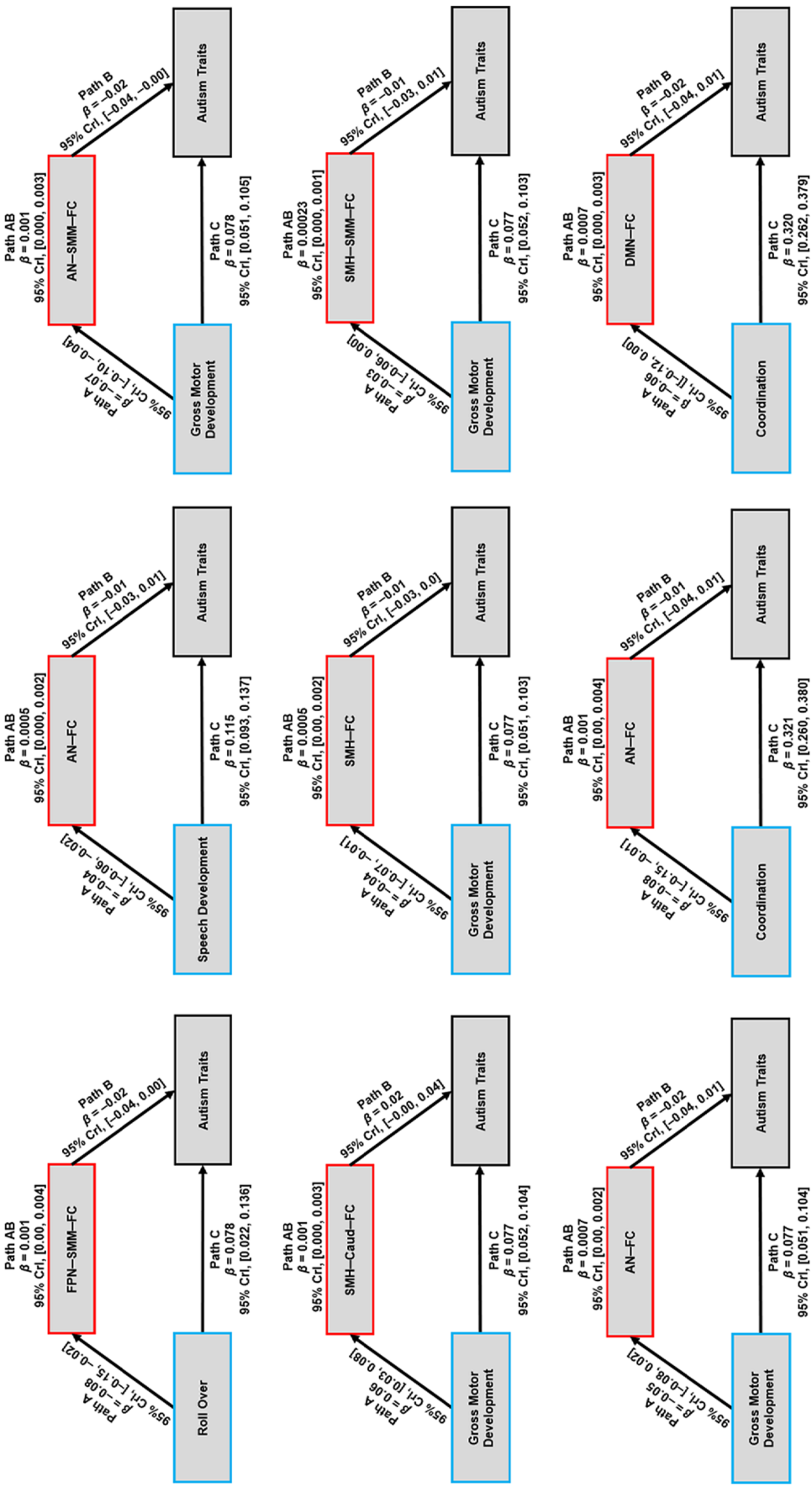


Figure 2. Indirect (mediation) effects of resting-state functional connectivity linking early developmental measures and autism traits. *Note*: AN, auditory network; DMN, default mode network; FPN, fronto-parietal network; SMH, somatomotor hand network; SMM, somatomotor mouth network; Caud, caudate nucleus.

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

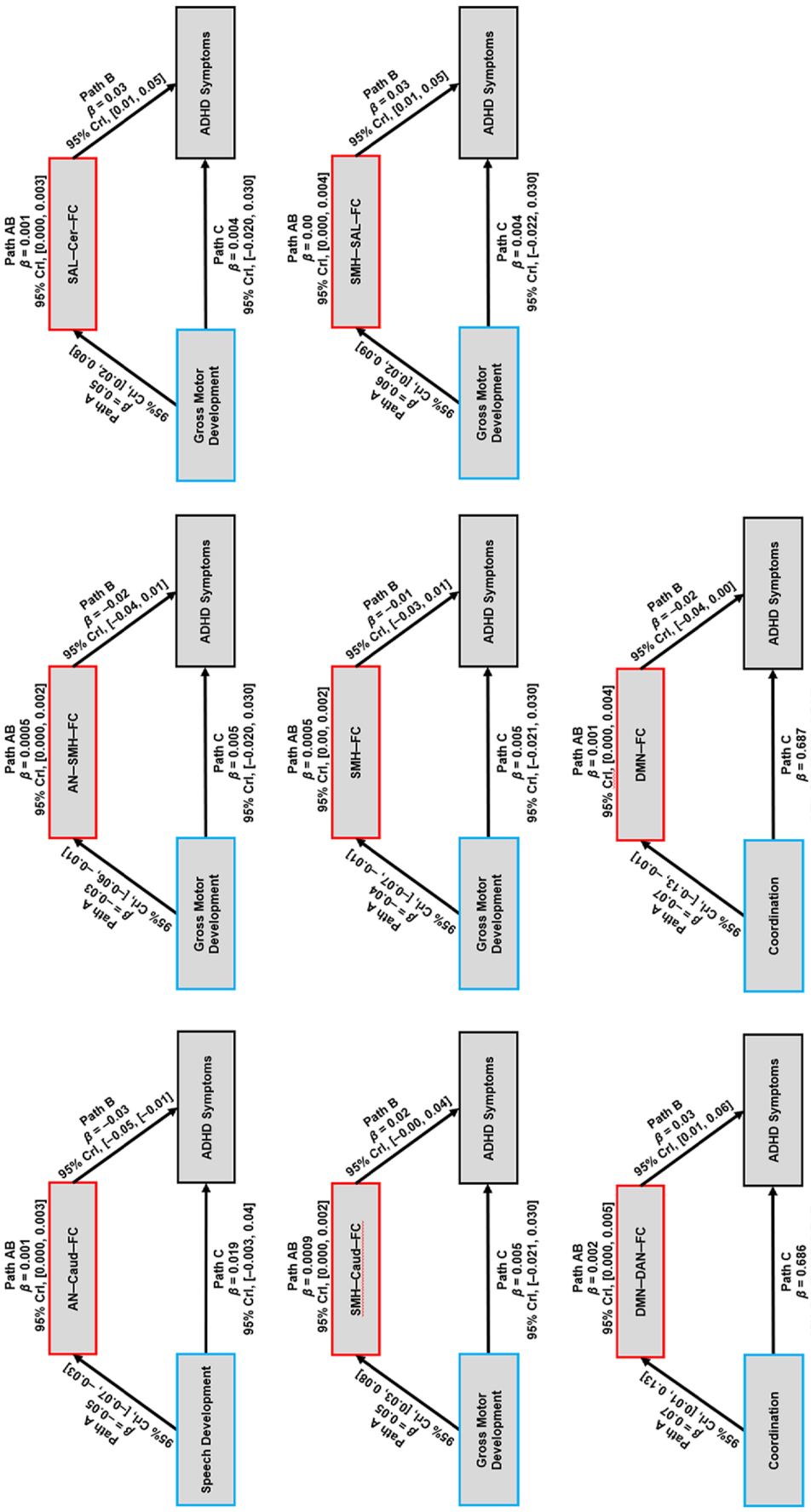


Figure 3. Indirect (mediation) effects of resting-state functional connectivity linking early developmental measures and ADHD symptoms. *Note*: AN, auditory network; DMN, default mode network; SAL, salience network; SMH, somatomotor hand network; DAN, dorsal attention network; Cer, cerebellum cortex; Caud, caudate nucleus.

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

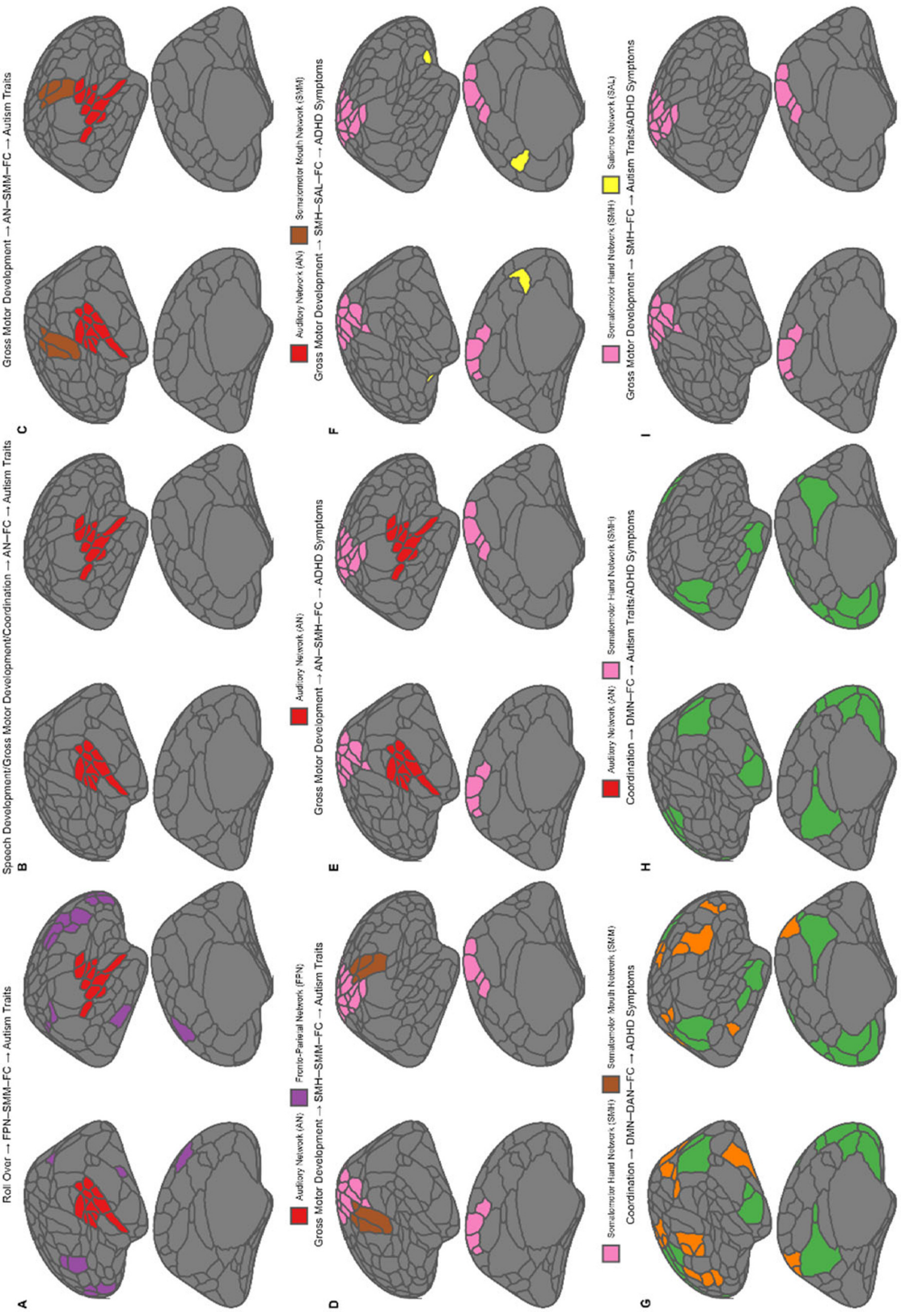


Figure 4. Cortical resting-state functional connectivity mediation networks linking developmental milestones to neurodevelopmental traits. *Note:* AN, auditory network; DMN, default mode network; DAN, dorsal attention network; FPN, fronto-parietal network; SMH, somatomotor hand network; SMM, somatomotor mouth network; SAL, salience network.

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

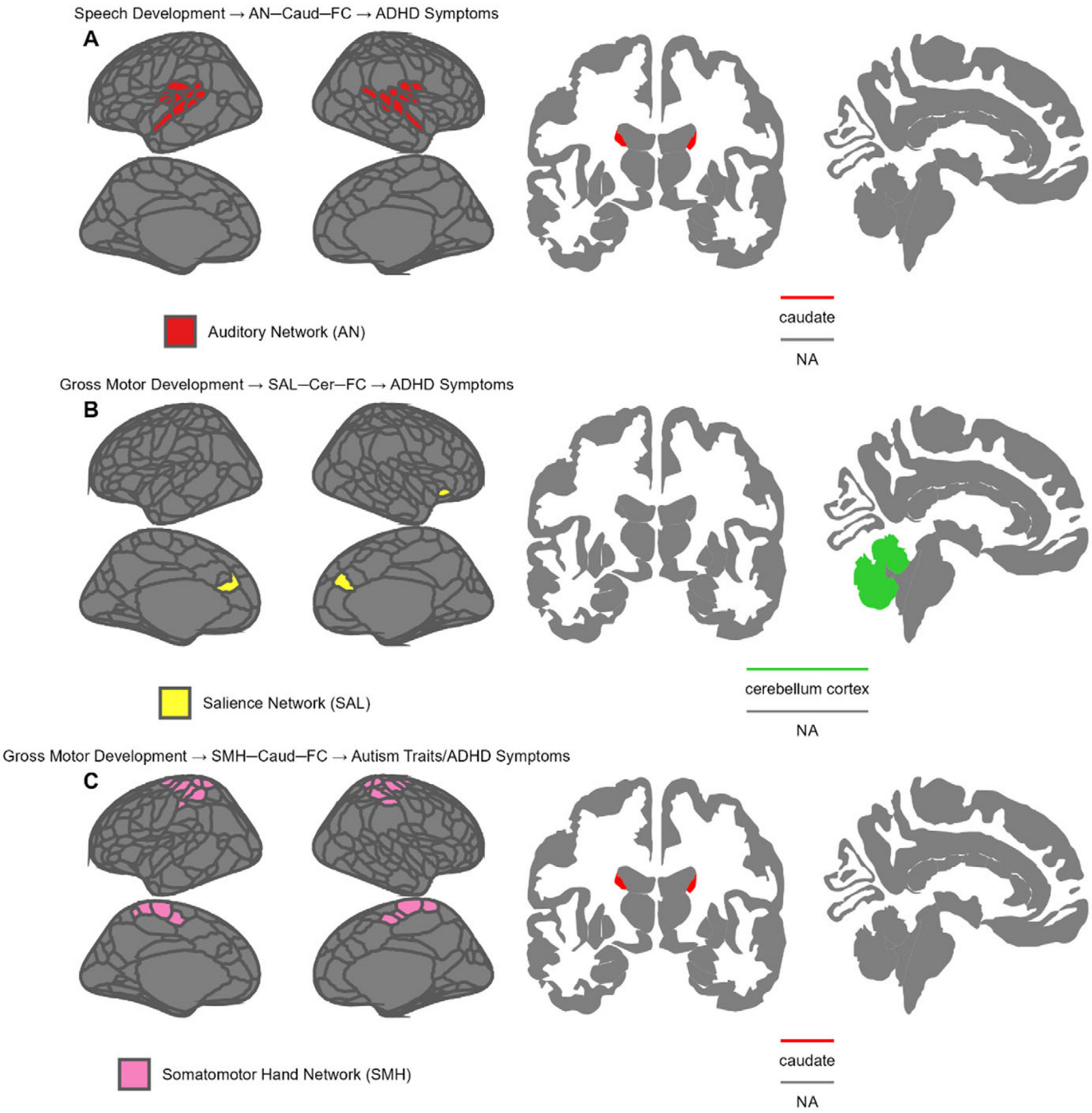


Figure 5. Cortical–subcortical resting-state functional connectivity (rsFC) mediation networks linking developmental milestones to neurodevelopmental traits. *Note*: AN, auditory network; SMH, somatomotor hand network; SAL, salience network; Cer, cerebellum cortex; Caud, caudate nucleus.

CrI = [0.01, 0.05]) for *gross motor development*, and *DMN–DAN* for *coordination* (β = 0.03, 95% CrI = [0.01, 0.06]).

Discussion

In this study, we examined how early motor and language development relate to autism traits and ADHD symptoms, and whether intrinsic functional connectivity mediates these associations. We observed both shared and condition-specific patterns, highlighting early developmental markers as indicators of neurodevelopmental vulnerability.

Delayed speech and poor motor coordination were associated with elevated autism traits, whereas delayed walking without assistance and gross motor delay were more specifically linked to higher ADHD symptoms. Poor motor coordination predicted both autism traits and ADHD symptoms, suggesting a shared developmental liability. In contrast, speech delays were more strongly related to autism traits, underscoring early language acquisition as a salient precursor, while delays in walking indexed attentional and regulatory difficulties characteristic of ADHD. These patterns align with evidence that motor coordination deficits span both conditions, reflecting shared disruptions in cerebellar and cortico-striatal

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

systems implicated in motor control, temporal processing, and executive regulation (Fliers et al., 2008; Fournier et al., 2010; Lemcke et al., 2016; Posar & Visconti, 2022). At the same time, the partially distinct milestone associations support a model in which early deviations confer transdiagnostic vulnerability that differentiates over development.

Previous studies using the ABCD dataset have shown that motor abnormalities are associated with depressive symptoms in adolescents. For instance, developmental motor delays and dyscoordination were significantly related to higher depression symptoms but not to psychotic-like experiences (PLEs). In addition, rsFC between motor networks and the striatum and cerebellum was associated with PLEs, whereas motor network-striatal connectivity was linked to depression symptoms (Damme et al., 2022). Similarly, developmental motor delays and dyscoordination have been shown to predict the number of depression symptoms endorsed, suggesting that motor dysfunction may serve as an early marker of depression risk (Damme et al., 2021).

Early motor and language milestones showed heterogeneous associations with rsFC across large-scale cortical and subcortical networks. Delayed rollover was linked to stronger connectivity within auditory and somatomotor networks but weaker integration with control and somatomotor-putamen circuits, consistent with altered network specialization (Wheelock et al., 2018). Delayed walking was associated with reduced coupling between somatomotor hand and salience networks, potentially reflecting less efficient coordination between motor execution and attentional monitoring (Menon & Uddin, 2010). Poor gross motor development was related to reduced integration within sensorimotor systems alongside increased coupling with salience, cerebellar, and subcortical networks, suggesting altered recruitment of motor prediction circuits (Buckner, 2013; Doyon et al., 2009). Delayed speech was linked to weaker auditory network connectivity and reduced integration with visual and striatal regions, consistent with disrupted sensory-subcortical coordination relevant to language acquisition (Friederici, 2012; Kotz & Schwartze, 2010). Poor motor coordination was associated with weaker within-network sensorimotor connectivity, reduced segregation among control, default mode, and attention systems, and increased default mode-attention coupling, indicative of less differentiated large-scale organization (Fair et al., 2009). Taken together, these findings are consistent with delayed or altered network specialization, characterized by reduced segregation of higher-order systems alongside persistent coupling of primary sensorimotor circuits (Gao, 2025). However, whether these alterations reflect delayed maturation, atypical specialization, compensatory plasticity, or inefficient synaptic pruning cannot be determined from cross-sectional connectivity data. Thus, these interpretations remain theoretically informed but provisional.

Resting-state connectivity did not meaningfully mediate associations between early motor and language development and later autism traits or ADHD symptoms. Indirect effects were small and accounted for only a minor proportion of total associations. For autism traits, mediation was sparse and inconsistent. Only a weak pathway linked speech delay to reduced auditory-somatomotor connectivity, while other indirect effects across sensory, motor, and subcortical networks were negligible or mixed. This accords with the marked neurodevelopmental heterogeneity of autism, in which diverse and partially independent neural pathways contribute to behavioral outcomes (Happé, Ronald, & Plomin, 2006; Lombardo, Lai, & Baron-Cohen, 2019).

In contrast, ADHD symptoms showed relatively more convergent, though still modest, indirect pathways involving auditory-striatal, salience-cerebellar, sensorimotor-salience, and default mode-dorsal attention interactions. These circuits align with established models implicating cortico-striatal, cerebellar, salience, and attention systems in attentional allocation, motor regulation, and adaptive control (Castellanos & Proal, 2012; Cortese et al., 2021; Menon, 2015). Nevertheless, indirect effects were small, indicating that intrinsic connectivity captures only a partial expression of underlying developmental vulnerability rather than serving as a singular mechanistic conduit.

rsFC such as somatomotor-striatal circuits and default mode-dorsal attention interactions are not specific to autism or ADHD but are core components of large-scale control architectures supporting motor regulation, cognitive control, salience detection, and adaptive task switching (Gao, 2025). Converging evidence indicates that disruptions in these systems extend beyond individual diagnoses and are implicated in transdiagnostic risk dimensions, including executive dysfunction, attentional dysregulation, and general psychopathology (McTeague et al., 2017). Accordingly, the observed mediation effects may reflect relatively general neurodevelopmental vulnerability circuits that influence multiple cognitive and behavioral domains rather than condition-specific pathways. Within this framework, early motor and language delays may perturb foundational integration and control systems during sensitive developmental windows, thereby shaping broad trajectories of cognitive and behavioral functioning, with phenotypic differentiation emerging over time (Karmiloff-Smith, 1998). Notably, the relatively more convergent indirect pathways observed for ADHD symptoms may reflect the central role of control and attention networks in that phenotype (Castellanos & Proal, 2012; Cortese et al., 2021), whereas autism-related outcomes may arise from more distributed and heterogeneous neurodevelopmental perturbations consistent with its marked etiological and neural diversity (Happé, Ronald, & Plomin, 2006; Lombardo, Lai, & Baron-Cohen, 2019).

The overall results suggest that early motor and language delays primarily index upstream neurodevelopmental processes, including atypical circuit formation, synaptic pruning, and experience-dependent plasticity unfolding dynamically across infancy and early childhood (Casey et al., 2018; Johnson, 2011). Because mediation models assume relatively stable intermediary mechanisms, single time-point rsFC measures in middle childhood may underestimate developmental transmission effects, particularly when early perturbations operate through evolving trajectories rather than static network states (Maxwell & Cole, 2007; Selig & Preacher, 2009).

From a developmental cascade perspective, early motor and language milestones scaffold later cognitive, attentional, and social functioning by shaping sensory input, motor exploration, and learning opportunities (Iverson, 2010; Thelen, 2000). Disruptions at these stages likely propagate across distributed and interacting systems rather than through a single intermediate neural phenotype. Accordingly, rsFC measured later in development may reflect downstream or parallel consequences of earlier perturbations, consistent with evidence that large-scale functional networks undergo substantial reorganization across childhood and adolescence (Fair et al., 2009; Grayson & Fair, 2017).

The contrast between autism and ADHD mediation patterns further supports a pluripotent model of neurodevelopmental risk, in which early deviations confer broad vulnerability that differentiates over time (Hartmann et al., 2021; Karmiloff-Smith, 1998). Poor motor coordination, associated with both autism traits and ADHD symptoms, may index a shared upstream liability preceding phenotypic divergence. Autism-related outcomes appear more diffuse and

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

weakly mediated, consistent with greater etiological and neural heterogeneity (Happé, Ronald, & Plomin, 2006; Lombardo, Lai, & Baron-Cohen, 2019). By contrast, ADHD symptoms involve relatively more convergent alterations in large-scale control and attention systems, yielding somewhat more consistent, though still modest, connectivity-linked pathways (Castellanos & Proal, 2012).

Together, these findings suggest that early motor and language delays are best conceptualized as behavioral markers of widespread neurodevelopmental divergence. Later intrinsic connectivity may reflect a downstream or parallel manifestation of dynamic network reorganization rather than a stable intermediary mechanism. Neurodevelopmental risk appears to be expressed through evolving changes in network segregation, integration, and flexibility across childhood, rather than through fixed alterations within isolated circuits (Cui et al., 2020; Fair et al., 2009; Menon, 2013).

Beyond their theoretical implications, these findings carry meaningful clinical relevance. Early motor and language delays may serve as accessible, low-cost behavioral markers of broader neurodevelopmental vulnerability, supporting proactive monitoring and risk stratification even in non-clinical or community samples (Ganai, Ratne, Bhushan, & Venkatesh, 2025). Rather than being viewed as isolated delays, such milestones may signal perturbations in foundational sensorimotor and control systems that scaffold later attentional, social, and executive functioning. Although the observed mediation effects were small, such effect sizes are typical in population-level developmental cascade models, in which multiple modest pathways accumulate over time to shape later behavioral outcomes. Clinically, this suggests that early deviations may not map onto a single neural mechanism but instead reflect diffuse, interacting vulnerabilities that unfold dynamically across development. Accordingly, early intervention efforts targeting motor coordination, language enrichment, and attentional regulation during sensitive developmental periods may confer broad downstream benefits, potentially attenuating transdiagnostic risk trajectories before symptom consolidation. Together, these findings support a developmental systems framework in which early behavioral deviations index upstream neurodevelopmental divergence, with later phenotypic differentiation emerging through the dynamic reorganization of neural circuits across childhood and adolescence.

Several limitations should be acknowledged. First, although the findings provide important insights, longitudinal designs are necessary to clarify how early behavioral markers, brain connectivity, and neurodevelopmental traits unfold and interact over time. rsFC and neurodevelopmental traits were assessed concurrently in adolescence, which precludes firm conclusions regarding the temporal precedence of connectivity over behavioral outcomes. Accordingly, the mediation models tested here should be interpreted as statistically consistent with indirect associations rather than as evidence of causal neurodevelopmental pathways, and it remains plausible that trait-related behaviors also influence brain connectivity across development. Second, motor and language milestones were parent-reported, introducing potential recall bias and measurement variability; future studies should incorporate standardized, observational, or clinician-rated assessments. Third, the non-clinical nature of the sample may limit generalizability to clinical populations, highlighting the need for replication in high-risk or diagnosed cohorts. Finally, although mediation effects were small, future research integrating longitudinal neuroimaging, genetic, and environmental data will be critical for disentangling the directionality and mechanisms underlying early developmental risk.

In conclusion, these findings suggest that early motor and language delays may be best conceptualized as behavioral indicators of broad neurodevelopmental divergence rather than condition-specific precursors. Intrinsic connectivity differences observed later in childhood may represent downstream or parallel manifestations of evolving network reorganization, rather than stable intermediary mechanisms linking early developmental perturbations to later traits. From a developmental systems perspective, neurodevelopmental risk appears to unfold through dynamic changes in network segregation, integration, and flexibility across childhood. Early deviations in motor and language development may therefore signal perturbations in foundational sensorimotor and control systems that shape later cognitive, attentional, and social trajectories, with phenotypic differentiation emerging progressively over development.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1017/S003329172610556X.

Acknowledgments. The first author would like to thank Mitacs and Prof. Nicholas Bock for their support in carrying out this work at McMaster University.

Author contribution. The authors made the following contributions. Conceptualization: U.J.G., B.B.; Data curation: U.J.G., B.B.; Formal Analysis: U.J.G.; Methodology: U.J.G.; Software: U.J.G.; Statical analysis: U.J.G.; Writing – original draft preparation: U.J.G.; Writing – review & editing: U.J.G., B.B.

Funding statement. This research received no specific grant from any funding agency, commercial, or not-for-profit sectors.

Competing interests. The authors declare no conflict of interest regarding this work.

References

Achenbach, T. M., & Ruffle, T. M. (2000). The child behavior checklist and related forms for assessing Behavioral/emotional problems and competencies. *Pediatrics in Review*, **21**(8), 265–271. https://doi.org/10.1542/pir.21.8.265

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Publishing. https://doi.org/10.1176/appi.books.9780890425596

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society Series B (Statistical Methodology)*, **57**(1), 289–300. https://doi.org/10.1111/j.2517-6161.1995.tb02031.x

Bhat, A. N., Landa, R. J., & Galloway, J. C. (2011). Current perspectives on motor functioning in infants, children, and adults with autism spectrum disorders. *Physical Therapy*, **91**(7), 1116–1129. https://doi.org/10.2522/ptj.20100294

Bolton, P. F., Golding, J., Emond, A., & Steer, C. D. (2012). Autism Spectrum disorder and autistic traits in the AVON longitudinal study of parents and children: Precursors and early signs. *Journal of the American Academy of Child & Adolescent Psychiatry*, **51**(3), 249–260.e25. https://doi.org/10.1016/j.jaac.2011.12.009

Bruchhage, M. M. K., Ngo, G., Schneider, N., D'Sa, V., & Deoni, S. C. L. (2020). Functional connectivity correlates of infant and early childhood cognitive development. *Brain Structure and Function*, **225**(2), 669–681. https://doi.org/10.1007/s00429-020-02027-4

Buckner, R. L. (2013). The cerebellum and cognitive function: 25 years of insight from anatomy and neuroimaging. *Neuron*, **80**(3), 807–815. https://doi.org/10.1016/j.neuron.2013.10.044

Bürkner, P. (2017). Brms: An R package for Bayesian multilevel models using Stan. *Journal of Statistical Software*, **80**(1). https://doi.org/10.18637/jss.v080.i01

Casey, B., Cannonier, T., Conley, M. I., Cohen, A. O., Barch, D. M., Heitzeg, M. M., Soules, M., Teslovich, T., Dellarco, D. V., Garavan, H., Hanlon, C. A., McQueeny, K., Schubel, T., Castaneda, E., Fontaine, J. J., Wallace, M. C., Snider, K. A., Bhave, S., Flack, J. I., … Dale, A. M. (2018). The adolescent brain cognitive development (ABCD) study: Imaging acquisition across 21 sites. *Developmental Cognitive Neuroscience*, **32**, 43–54. https://doi.org/10.1016/j.dcn.2018.03.001

Castellanos, F. X., & Proal, E. (2012). Large-scale brain systems in ADHD: Beyond the prefrontal–striatal model. *Trends in Cognitive Sciences*, **16**(1), 17–26. https://doi.org/10.1016/j.tics.2011.11.007

Constantino, J. N., & Gruber, C. P. (2011). *Social responsiveness scale (SRS-2)* (2nd ed.). Western Psychological Services.

Constantino, J. N., & Todd, R. D. (2003). Autistic traits in the general population. *Archives of General Psychiatry*, **60**(5), 524. https://doi.org/10.1001/archpsyc.60.5.524

Cortese, S., Aoki, Y. Y., Itahashi, T., Castellanos, F. X., & Eickhoff, S. B. (2021). Systematic review and meta-analysis: Resting-state functional magnetic resonance imaging studies of attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, **60**(1), 61–75. https://doi.org/10.1016/j.jaac.2020.08.014

Cui, Z., Li, H., Xia, C. H., Larsen, B., Adebimpe, A., Baum, G. L., Cieslak, M., Gur, R. E., Gur, R. C., Moore, T. M., Oathes, D. J., Alexander-Bloch, A. F., Raznahan, A., Roalf, D. R., Shinohara, R. T., Wolf, D. H., Davatzikos, C., Bassett, D. S., Fair, D. A., … Satterthwaite, T. D. (2020). Individual variation in functional topography of association networks in youth. *Neuron*, **106**(2), 340–353.e8. https://doi.org/10.1016/j.neuron.2020.01.029

Damme, K. S., Park, J. S., Vargas, T., Walther, S., Shankman, S. A., & Mittal, V. A. (2021). Motor abnormalities, depression risk, and clinical course in adolescence. *Biological Psychiatry Global Open Science*, **2**(1), 61–69. https://doi.org/10.1016/j.bpsgos.2021.06.011

Damme, K. S. F., Park, J. S., Walther, S., Vargas, T., Shankman, S. A., & Mittal, V. A. (2022). Depression and psychosis risk shared vulnerability for motor signs across development, symptom dimensions, and familial risk. *Schizophrenia Bulletin*, **48**(4), 752–762. https://doi.org/10.1093/schbul/sbab133

Doyon, J., Bellec, P., Amsel, R., Penhune, V., Monchi, O., Carrier, J., Lehéricy, S., & Benali, H. (2009). Contributions of the basal ganglia and functionally related brain structures to motor learning. *Behavioural Brain Research*, **199**(1), 61–75. https://doi.org/10.1016/j.bbr.2008.11.012

Fair, D. A., Cohen, A. L., Power, J. D., Dosenbach, N. U. F., Church, J. A., Miezin, F. M., Schlaggar, B. L., & Petersen, S. E. (2009). Functional brain networks develop from a "local to distributed" organization. *PLoS Computational Biology*, **5**(5), e1000381. https://doi.org/10.1371/journal.pcbi.1000381

Fischl, B., Salat, D. H., Busa, E., Albert, M., Dieterich, M., Haselgrove, C., van der Kouwe, A., Killiany, R., Kennedy, D., Klaveness, S., Montillo, A., Makris, N., Rosen, B., & Dale, A. M. (2002). Whole brain segmentation. *Neuron*, **33**(3), 341–355. https://doi.org/10.1016/s0896-6273(02)00569-x

Fliers, E., Rommelse, N., Vermeulen, S. H. H. M., Altink, M., Buschgens, C. J. M., Faraone, S. V., Sergeant, J. A., Franke, B., & Buitelaar, J. K. (2008). Motor coordination problems in children and adolescents with ADHD rated by parents and teachers: Effects of age and gender. *Journal of Neural Transmission*, **115**(2), 211–220. https://doi.org/10.1007/s00702-007-0827-0

Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism Spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, **40**(10), 1227–1240. https://doi.org/10.1007/s10803-010-0981-3

Friederici, A. D. (2012). The cortical language circuit: From auditory perception to sentence comprehension. *Trends in Cognitive Sciences*, **16**(5), 262–268. https://doi.org/10.1016/j.tics.2012.04.001

Ganai, U. J., Ratne, A., Bhushan, B., & Venkatesh, K. S. (2025). Early detection of autism spectrum disorder: Gait deviations and machine learning. *Scientific Reports*, **15**(1), 873. https://doi.org/10.1038/s41598-025-85348-w

Gao, W. (2025). A hierarchical model of early brain functional network development. *Trends in Cognitive Sciences*, **29**(9), 855–868. https://doi.org/10.1016/j.tics.2025.04.001

Gordon, E. M., Laumann, T. O., Adeyemo, B., Huckins, J. F., Kelley, W. M., & Petersen, S. E. (2014). Generation and evaluation of a cortical area parcellation from resting-state correlations. *Cerebral Cortex*, **26**(1), 288–303. https://doi.org/10.1093/cercor/bhu239

Grayson, D. S., & Fair, D. A. (2017). Development of large-scale functional networks from birth to adulthood: A guide to the neuroimaging literature. *NeuroImage*, **160**, 15–31. https://doi.org/10.1016/j.neuroimage.2017.01.079

Gui, A., Hollowell, A., Wigdor, E. M., Morgan, M. J., Hannigan, L. J., Corfield, E. C., Odintsova, V., Hottenga, J. J., Wong, A., Pool, R., Cullen, H., Wilson, S., Warrier, V., Eilertsen, E. M., Andreassen, O. A., Middeldorp, C. M., St Pourcain, B., Bartels, M., Boomsma, D. I., … Ronald, A. (2025). Genome-wide association meta-analysis of age at onset of walking in over 70,000 infants of European ancestry. *Nature Human Behaviour*, **9**(7), 1470–1487. https://doi.org/10.1038/s41562-025-02145-1

Gurevitz, M., Geva, R., Varon, M., & Leitner, Y. (2012). Early markers in infants and toddlers for development of ADHD. *Journal of Attention Disorders*, **18**(1), 14–22. https://doi.org/10.1177/1087054712447858

Hagler, D. J., Hatton, S., Cornejo, M. D., Makowski, C., Fair, D. A., Dick, A. S., … Peltier, S. (2019). Image processing and analysis methods for the adolescent brain cognitive development study. *NeuroImage*, **202**, 116091. https://doi.org/10.1016/j.neuroimage.2019.116091

Hannigan, L. J., Askeland, R. B., Ask, H., Tesli, M., Corfield, E., Ayorech, Z., Magnus, P., Njølstad, P. R., Øyen, A. S., Stoltenberg, C., Andreassen, O. A., Ronald, A., Smith, G. D., Reichborn-Kjennerud, T., & Havdahl, A. (2021). Developmental milestones in early childhood and genetic liability to neurodevelopmental disorders. *Psychological Medicine*, **53**(5), 1750–1758. https://doi.org/10.1017/s0033291721003330

Happé, F., Ronald, A., & Plomin, R. (2006). Time to give up on a single explanation for autism. *Nature Neuroscience*, **9**(10), 1218–1220. https://doi.org/10.1038/nn1770

Hartmann, J. A., McGorry, P. D., Destree, L., Amminger, G. P., Chanen, A. M., Davey, C. G., Ghieh, R., Polari, A., Ratheesh, A., Yuen, H. P., & Nelson, B. (2021). Pluripotential risk and clinical staging: Theoretical considerations and preliminary data from a transdiagnostic risk identification approach. *Frontiers in Psychiatry*, **11**. https://doi.org/10.3389/fpsyt.2020.553578

Havmoeller, S. R., Thomsen, P. H., & Lemcke, S. (2018). The early motor development in children diagnosed with ADHD: A systematic review. *ADHD Attention Deficit and Hyperactivity Disorders*, **11**(3), 233–240. https://doi.org/10.1007/s12402-018-0280-y

Iverson, J. M. (2010). Developing language in a developing body: The relationship between motor development and language development. *Journal of Child Language*, **37**(2), 229–261. https://doi.org/10.1017/s0305000909990432

Jaspers, M., De Winter, A. F., Buitelaar, J. K., Verhulst, F. C., Reijneveld, S. A., & Hartman, C. A. (2012). Early childhood assessments of community pediatric professionals predict autism spectrum and attention deficit hyperactivity problems. *Journal of Abnormal Child Psychology*, **41**(1), 71–80. https://doi.org/10.1007/s10802-012-9653-4

Johnson, M. H. (2011). Interactive specialization: A domain-general framework for human functional brain development? *Developmental Cognitive Neuroscience*, **1**(1), 7–21. https://doi.org/10.1016/j.dcn.2010.07.003

Johnson, M. H., Gliga, T., Jones, E., & Charman, T. (2014). Annual research review: Infant development, autism, and ADHD – Early pathways to emerging disorders. *Journal of Child Psychology and Psychiatry*, **56**(3), 228–247. https://doi.org/10.1111/jcpp.12328

Jones, E. J., Gliga, T., Bedford, R., Charman, T., & Johnson, M. H. (2013). Developmental pathways to autism: A review of prospective studies of infants at risk. *Neuroscience & Biobehavioral Reviews*, **39**(100), 1–33. https://doi.org/10.1016/j.neubiorev.2013.12.001

Kaiser, M., Schoemaker, M., Albaret, J., & Geuze, R. (2014). What is the evidence of impaired motor skills and motor control among children with attention deficit hyperactivity disorder (ADHD)? Systematic review of the literature. *Research in Developmental Disabilities*, **36**, 338–357. https://doi.org/10.1016/j.ridd.2014.09.023

Karmiloff-Smith, A. (1998). Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences*, **2**(10), 389–398. https://doi.org/10.1016/s1364-6613(98)01230-3

Kotz, S. A., & Schwartze, M. (2010). Cortical speech processing unplugged: A timely subcortico-cortical framework. *Trends in Cognitive Sciences*, **14**(9), 392–399. https://doi.org/10.1016/j.tics.2010.06.005

Kuo, S. S., Van Der Merwe, C., Fu, J. M., Carey, C. E., Talkowski, M. E., Bishop, S. L., & Robinson, E. B. (2022). Developmental variability in autism across 17 000 autistic individuals and 4000 siblings without an autism diagnosis. *JAMA Pediatrics*, **176**(9), 915–923. https://doi.org/10.1001/jamapediatrics.2022.2423

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2017). LMERTest package: Tests in linear mixed effects models. *Journal of Statistical Software*, **82**(13). https://doi.org/10.18637/jss.v082.i13

Landa, R. J., Gross, A. L., Stuart, E. A., & Faherty, A. (2012). Developmental trajectories in children with and without autism spectrum disorders: The first 3 years. *Child Development*, **84**(2), 429–442. https://doi.org/10.1111/j.1467-8624.2012.01870.x

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press

Lemcke, S., Parner, E. T., Bjerrum, M., Thomsen, P. H., & Lauritsen, M. B. (2016). Early development in children that are later diagnosed with disorders of attention and activity: A longitudinal study in the Danish National Birth Cohort. *European Child & Adolescent Psychiatry*, **25**(10), 1055–1066. https://doi.org/10.1007/s00787-016-0825-6

Li, L., Pasco, G., Ali, J. B., Johnson, M. H., Jones, E. J. H., & Charman, T. (2023). Associations between early language, motor abilities, and later autism traits in infants with typical and elevated likelihood of autism. *Autism Research*, **16**(11), 2184–2197. https://doi.org/10.1002/aur.3023

Lombardo, M. V., Lai, M., & Baron-Cohen, S. (2019). Big data approaches to decomposing heterogeneity across the autism spectrum. *Molecular Psychiatry*, **24**(10), 1435–1450. https://doi.org/10.1038/s41380-018-0321-0

Lord, C., Elsabbagh, M., Baird, G., & Veenstra-Vanderweele, J. (2018). Autism spectrum disorder. *The Lancet*, **392**(10146), 508–520. https://doi.org/10.1016/s0140-6736(18)31129-2

Lubke, G. H., Hudziak, J. J., Derks, E. M., Van, T. C., & Boomsma, D. I. (2009). Maternal ratings of attention problems in ADHD: Evidence for the existence of a continuum. *Journal of the American Academy of Child & Adolescent Psychiatry*, **48**(11), 1085–1093. https://doi.org/10.1097/chi.0b013e3181ba3dbb

Makowski, D., Ben-Shachar, M., & Lüdecke, D. (2019). bayestestR: Describing effects and their uncertainty, existence and significance within the Bayesian framework. *The Journal of Open Source Software*, **4**(40), 1541. https://doi.org/10.21105/joss.01541

Marrus, N., Eggebrecht, A. T., Todorov, A., Elison, J. T., Wolff, J. J., Cole, L., Gao, W., Pandey, J., Shen, M. D., Swanson, M. R., Emerson, R. W., Klohr, C. L., Adams, C. M., Estes, A. M., Zwaigenbaum, L., Botteron, K. N., McKinstry, R. C., Constantino, J. N., Evans, A. C., … Pruett, J. R. (2018). Walking, gross motor development, and brain functional connectivity in infants and toddlers. *Cerebral Cortex*, **28**(2), 750–763. https://doi.org/10.1093/cercor/bhx313

Martin, J., Hamshere, M. L., Stergiakouli, E., O'Donovan, M. C., & Thapar, A. (2014). Genetic risk for attention-deficit/hyperactivity disorder contributes to neurodevelopmental traits in the general population. *Biological Psychiatry*, **76**(8), 664–671. https://doi.org/10.1016/j.biopsych.2014.02.013

Maxwell, S. E., & Cole, D. A. (2007). Bias in cross-sectional analyses of longitudinal mediation. *Psychological Methods*, **12**(1), 23–44. https://doi.org/10.1037/1082-989x.12.1.23

McLeod, K. R., Langevin, L. M., Goodyear, B. G., & Dewey, D. (2014). Functional connectivity of neural motor networks is disrupted in children with developmental coordination disorder and attention-deficit/hyperactivity disorder. *NeuroImage Clinical*, **4**, 566–575. https://doi.org/10.1016/j.nicl.2014.03.010

McTeague, L. M., Huemer, J., Carreon, D. M., Jiang, Y., Eickhoff, S. B., & Etkin, A. (2017). Identification of common neural circuit disruptions in cognitive control across psychiatric disorders. *American Journal of Psychiatry*, **174**(7), 676–685. https://doi.org/10.1176/appi.ajp.2017.16040400

Menon, V. (2013). Developmental pathways to functional brain networks: Emerging principles. *Trends in Cognitive Sciences*, **17**(12), 627–640. https://doi.org/10.1016/j.tics.2013.09.015

Menon, V. (2015). Salience network. In A. W. Toga (Ed.), *Brain mapping: An encyclopedic reference* (pp. 597–611). https://doi.org/10.1016/b978-0-12-397025-1.00052-x

Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention and control: A network model of insula function. *Brain Structure and Function*, **214**(5–6), 655–667. https://doi.org/10.1007/s00429-010-0262-0

Norman, L. J., Sudre, G., Bouyssi-Kobar, M., Jiao, M., Gligorovic, S., Jean, J., White, T., & Shaw, P. (2025). Cross-sectional mega-analysis of resting-state alterations associated with autism and attention-deficit/hyperactivity disorder in children and adolescents. *Nature Mental Health*, **3**(6), 709–723. https://doi.org/10.1038/s44220-025-00431-5

Posar, A., & Visconti, P. (2022). Early motor signs in autism spectrum disorder. *Children*, **9**(2), 294. https://doi.org/10.3390/children9020294

Prelock, P. J., & Nelson, N. W. (2012). Language and communication in autism: An integrated view. *Pediatric Clinics of North America*, **59**(1), 129–145. https://doi.org/10.1016/j.pcl.2011.10.008

R Core Team. (2025). *R: A language and environment for statistical computing* (Version 4.5.2). https://www.r-project.org

Rommelse, N. N. J., Franke, B., Geurts, H. M., Hartman, C. A., & Buitelaar, J. K. (2010). Shared heritability of attention-deficit/hyperactivity disorder and autism spectrum disorder. *European Child & Adolescent Psychiatry*, **19**(3), 281–295. https://doi.org/10.1007/s00787-010-0092-x

Selig, J. P., & Preacher, K. J. (2009). Mediation models for longitudinal data in developmental research. *Research in Human Development*, **6**(2–3), 144–164. https://doi.org/10.1080/15427600902911247

Serdarevic, F., Tiemeier, H., Jansen, P. R., Alemany, S., Xerxa, Y., Neumann, A., Robinson, E., Hillegers, M. H. J., Verhulst, F. C., & Ghassabian, A. (2019). Polygenic risk scores for developmental disorders, neuromotor functioning during infancy, and autistic traits in childhood. *Biological Psychiatry*, **87**(2), 132–138. https://doi.org/10.1016/j.biopsych.2019.06.006

Singh, P., Pathak, A., Ganai, U. J., Bhushan, B., & Subramanian, V. K. (2024). Video based computational coding of movement anomalies in ASD children. In *2022 IEEE/CVF Conference on Computer Vision and Pattern Recognition Workshops (CVPRW)*, pp. 400–409. https://doi.org/10.1109/cvprw63382.2024.00045

Thelen, E. (2000). Grounded in the world: Developmental origins of the embodied mind. *Infancy*, **1**(1), 3–28. https://doi.org/10.1207/s15327078in0101_02

Wheelock, M., Austin, N., Bora, S., Eggebrecht, A., Melzer, T., Woodward, L., & Smyser, C. (2018). Altered functional network connectivity relates to motor development in children born very preterm. *NeuroImage*, **183**, 574–583. https://doi.org/10.1016/j.neuroimage.2018.08.051

Zubler, J. M., Wiggins, L. D., Macias, M. M., Whitaker, T. M., Shaw, J. S., Squires, J. K., Pajek, J. A., Wolf, R. B., Slaughter, K. S., Broughton, A. S., Gerndt, K. L., Mlodoch, B. J., & Lipkin, P. H. (2022). Evidence-informed milestones for developmental surveillance tools. *Pediatrics*, **149**(3). https://doi.org/10.1542/peds.2021-052138

https://doi.org/10.1017/S003329172610556X Published online by Cambridge University Press