Research Square

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Cerebellar Test Performance in Children with ADHD and Autism Spectrum Disorder

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Abstract

Background.

Diagnoses of attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are based on clinical assessments; objectively measured motor performance might provide complementary phenotypic information. **Methods.** We compared 96 typically developing children, 29 children with ADHD, and 37 children with ASD on three cerebellar-related tasks, prism adaptation, finger tapping, and eyeblink conditioning, and a standardised neuromotor examination. **Results.** Both clinical groups differed from controls on measures of prism adaptation, eyeblink conditioning, and neuromotor function. Children with ADHD additionally showed altered motor timing, whereas children with ASD showed lower overall prism-task error. However, the two clinical groups differed on only three of 11 measures. **Limitations.** The sample was modest, particularly for eyeblink conditioning, and group distributions overlapped. The groups differed in some background variables such as age and sex, and medication status were not recorded. Moreover, the cross-sectional design precludes causal inference. **Conclusions.** Motor performance on cerebellar-related tasks appears to reflect differences that cut across neurodevelopmental conditions rather than separating diagnoses, consistent with the substantial phenotypic overlap between ASD and ADHD. These measures should be regarded as candidate markers of atypical neurodevelopment rather than as diagnostic tools.

Background

Attention Deficit Hyperactivity Disorder (ADHD) is characterized by persistent inattention and/or hyperactivity-impulsivity, whereas Autism Spectrum Disorder (ASD) is defined by impaired social communication, impaired reciprocal social interaction, and restricted, repetitive behaviors or interests [1]. The prevalence of ADHD is 5.3–7.1% in children and adolescents worldwide [2, 3], while the prevalence of ASD is 1–3% [4–7]. ADHD and ASD frequently co-occur and share overlapping symptoms. About 12% of people with ADHD also have ASD [8] while 30% of children with ASD also have ADHD [9]. However, prevalence estimates vary by case definition, setting, and ascertainment method. Consequently, estimates should therefore be presented as context rather than as universally applicable values.

Motor difficulties – including Minor Neurological Deficits (MND), fine and gross motor problems, repetitive motor behaviours, and Developmental Coordination Disorder (DCD) – are common in both ADHD and ASD [10–14]. The motor impairments in ASD are widespread spanning: gait [15], manual dexterity [16], vestibular function [17], balance [18], postural control [19], motor learning [20], imitation [21], and interpersonal coordination [22]. It has been suggested that motor impairment could be a clinical specifier for ASD, and that interventions targeting motor function may also improve social communication [10]. ADHD is also linked with motor deficits [14]. Children who have both ADHD and MND, a condition also known as DAMP [23] exhibit poorer adult outcomes than both matched and

general population controls [24]. The link between motor abnormalities and both ASD and ADHD raises the possibility of objective, performance-based diagnostic tools.

Though traditionally considered a structure primarily involved in motor control, the cerebellum is now known to contribute to cognitive functions such as language, executive function, spatial perception, and emotional regulation, and has been implicated in neurodevelopmental disorders including ADHD and ASD [25–27]. Stoodley suggests that different cerebellar networks are involved in ADHD and ASD [27]. Cerebellar volume reductions, particularly in the posterior vermis, have been reported in ADHD and may correlate with symptom severity [28–31]. These differences are more pronounced in medication-naïve individuals [32] and methylphenidate treatment seems to alter cerebellar activation [33] and may normalize posterior vermal volumes [34]. Differences in cerebellar connectivity have also been reported [35]. Likewise, both post-mortem and neuroimaging studies suggest structural and functional alterations in the cerebellum of ASD individuals [36–38]. Strikingly, children who suffer cerebellar injury at birth are 36 times more likely to subsequently be diagnosed with autism compared to controls [39]. In ASD, the total volume of the cerebellum is increased in proportion to the general cerebral enlargement in individuals with ASD [40, 41]. In contrast, the vermis and especially its posterior parts tend to be smaller in comparison to controls [40, 42]. However, the literature is inconsistent regarding hypoplasia of other regions within the vermis [42, 43]. On a histological level, several studies have reported reductions in the number and size of Purkinje cells in individuals with ASD [36–38]. If the morphological differences observed are a result of prenatal factors, genetics, or aberrant development of networks with cerebral cortices remains unclear [40, 42].

Human and animal research has identified several tasks that depend at least partially on the cerebellum. These include eyeblink conditioning, finger tapping, and prism adaptation [44–50]. In eyeblink conditioning, repeated pairing of a tone (CS) with a corneal air puff (US) leads to a conditioned response (CR), an anticipatory blink elicited by the CS alone [51]. In prism adaptation participants point towards a target while wearing glasses that shift the visual field. This requires the participant to adapt to altered sensory input [52]. Finger tapping assesses the ability to maintain a rhythm in the absence of external cues, making it a sensitive measure of fine motor control with particular relevance to cerebellar functioning. [49]. Patients with cerebellar lesions display impairments in eyeblink conditioning, inadequate adaptation during prism adaptation tasks and diminished performance in finger tapping [53, 54]. In line with the aforementioned links to cerebellar abnormalities, individuals with ADHD and ASD also show deficits in these tasks [55–62]. ADHD is associated with higher intraindividual variability in finger tapping [58, 63, 64], impaired prism adaptation [58, 65], and abnormally timed eyeblink responses [56, 66]. Morimoto et al. [59] found increased temporal variability during finger tapping in children with ASD compared to controls. Mostofsky et al. [60] found that children with high-functioning autism had reduced activation of the ipsilateral anterior cerebellum and absent activation of the contralateral anterior cerebellum during finger tapping. In eyeblink conditioning children with ASD exhibited conditioned eye blink responses deviating from typically developing children [61, 62]. Likewise, children with ASD showed earlier onset and peak of conditioned eyeblinks, perhaps due to inadequate cerebellar processing of sensory input [61].

Since prism adaptation also is thought to depend on the cerebellum one could hypothesize that performance on prism adaptation would be impaired in individuals with ASD [55]. Notably, Gidley et al. [57] reported no significant differences between children with ASD and controls on prism adaptation tasks. However, small sample sizes, potential medication effects, and inadequate control matching complicate the interpretation of findings from cerebellar-dependent tasks in children with ASD. In a recent study Gustafsson et al. [58] found that children with ADHD and MND performed worse on finger tapping and prism adaptation than controls, while children with ADHD but without MND performed intermediately. However, it remains unclear whether children with ADHD and ASD exhibit different patterns in finger tapping and prism adaptation.

Here, we compared performance across four motor tests with documented, but non-exclusive, links to cerebellar function in children with ADHD, children with ASD, and typically developing controls. We hypothesised that both clinical groups would differ from controls on several measures and that the pattern of performance would differ between the ADHD and ASD groups.

Methods

A total of 162 children participated in this study (see Table 1). The control group consisted of 96 children (47 boys, 49 girls) with a median age of 11.8 years recruited from a local elementary school in Lund, Sweden. The ADHD group consisted of 29 children diagnosed with ADHD (20 boys, 9 girls) with a median age of 12.6 years recruited from three institutions in southern Sweden: (1) the child and adolescent psychiatry unit in Malmö (BUP), (2) Modigo, a private clinic that assesses and diagnoses children with neurodevelopmental disorders, and (3) Källan Teaching Group, a public teaching group for children and adolescents with special educational needs. The assessments of both groups were performed by specialized medical doctors in child psychiatry and a psychologist. For both groups, the assessments consisted of a comprehensive psychiatric diagnostic interview using the MINI-KID for comorbid disorders, developmental history, neurological assessment, assessment of cognitive functioning with an age-appropriate version of the Wechsler scales, and interviews with the children's teachers.

For the ASD group, the psychologist was specially trained for the assessment of ASD. The ASD-specific assessments included the Autism Diagnostic Interview–Revised (ADI-R) and the Autism Diagnostic Observation Schedule, Second Edition (ADOS-2). For the ADHD group, the assessment additionally included the parent and teacher versions of the Swanson, Nolan, and Pelham Rating Scale (SNAP-IV) and the ADHD Child Evaluation (ACE) with parents. Children with Intellectual Disability were excluded from the study.

The ASD group consisted of 37 children diagnosed with ASD (28 boys, 9 girls) with a median age of 13.3 years (IQR 11.4–14.6), recruited through the child and adolescent psychiatry unit at Lund University hospital. Diagnoses were established clinically according to DSM-5 criteria. The control and ADHD participants were tested between 2017 and 2020 and have been partly described in Gustafsson et. al. [58]. The present study extends previous work by adding an ASD group, whose participants were tested

in 2024, and analysing the full test battery across all three groups. The study was approved by the Swedish Ethical Review Authority in Lund, Sweden (DNR 2017/745) and was conducted in accordance with the Declaration of Helsinki. Written information about the study was sent to families in advance, allowing them to read the material and prepare questions. The same information was given verbally at the session, and both the child and the legal guardian had the opportunity to ask questions before testing began. Informed consent was obtained from legal guardian(s), and from participants themselves. Children were informed that they could end their participation at any point without consequence, and one legal guardian was permitted in the test room if the child or guardian so wished. Participants received a cinema voucher (approximately €10) as a token of appreciation.

Testing procedure and protocol

The test battery comprised four subtests administered in the following order: (1) prism adaptation, (2) finger tapping, (3) eyeblink conditioning, and (4) a standardised neuromotor examination. Sessions lasted approximately 1 h and were conducted in a quiet room at the school or clinic, whichever was most convenient for the participant. Short breaks of a few minutes were given between tests, extended if the child needed more time.

Prism adaptation

Prism adaptation assesses the adjustment of motor output following an imposed change in visual input, and performance on the task has been linked to cerebellar function [67, 68]. Participants stood at arm's length from a measuring tape attached to the wall. On each trial, the participant looked at the tape, identified its midpoint, closed their eyes, and pointed to where they believed the target to be. While still pointing and standing in the same position, the participant opened their eyes to see where they had pointed. This procedure was repeated for 10 baseline trials. For the following 10 trials (trials 11–20), participants wore prism glasses that displaced the visual field laterally by 15 degrees. Wearing the prisms usually produces a pointing error that decreases across trials as the participant adapts to the altered visuomotor relationship. For the final 10 trials (trials 21–30), the glasses were removed, which typically produces a pointing error in the direction opposite to the error observed when the prisms were first introduced. On each trial the experimenter recorded the error in centimetres. Three variables were extracted: the error on the first trial after the prisms were put on (trial 11; prism on), the error on the first trial after the prisms were removed (trial 21; prism off), and the mean absolute pointing error across all trials (prism error). Note that a smaller error on trials 11 and 21 does not indicate better performance, since the adaptive response to an altered visuomotor relationship is to change the motor program.

Finger tapping

Participants listened to a rhythmic auditory sequence and synchronised their taps by pressing the laptop space bar with the dominant hand (synchronisation phase). They then continued tapping at the same pace after the sound stopped (production phase) until a stop signal was presented. Evidence from patients with cerebellar damage [69] and functional magnetic resonance imaging studies [70] indicates

that the cerebellum contributes to this task. The procedure was repeated five times. Each repetition comprised 15 paced intervals followed by approximately 70 self-paced intervals. The target interval was 524 ms, which lies within a range associated with low variability [71, 72]. We derived two synchronisation-phase metrics, the mean inter-tap interval (synch mean) and its standard deviation (synch SD), and three production-phase metrics: mean inter-tap interval (production mean), drift, and local variability [73]. Drift captures systematic change in tapping rate over the production phase; local variability captures trial-to-trial instability after accounting for systematic drift. Intervals deviating by more than 50% from the 524-ms target (< 262 or > 786 ms) were excluded.

Eyeblink conditioning

Eyeblink conditioning is widely used to assess learning with a substantial cerebellar contribution [46, 50, 74]. A conditioned stimulus (CS) is paired with an unconditioned stimulus (US), which elicits an unconditioned response (UR). With repeated pairings, the CS may elicit a conditioned response (CR) before US onset. Participants wore glasses fitted with a flexible plastic tube for air-puff delivery and a magnetic sensor. A 0.5-g neodymium magnet was attached to the eyelid so that eyelid movement changed the magnetic field recorded by the sensor. During testing, children watched a cartoon and were instructed to attend to the film rather than to the stimuli or their own reactions. The CS was a 1-s, 1,000-Hz sinusoidal auditory tone. Before training, we verified that the participant could hear the tone clearly and that it did not itself elicit a blink reflex. The US was an approximately 100-ms air puff to the cornea at 0.5–1.5 bar, individually adjusted to reliably elicit a blink reflex without being perceived as unpleasant. The CS–US interval was 500 ms, and the inter-trial interval varied randomly between 4 and 12 s. Training comprised 100 trials, with 20% randomly interspersed CS-alone probe trials.

Signals from the magnetic sensor were sampled using a Micro 1401 AD converter (Cambridge Electronic Design) connected to a PC running Spike2 v.9 (Cambridge Electronic Design). The raw signal was exported to MATLAB, where custom scripts identified the timing of blink responses relative to CS onset. Trials were classified as invalid if an eyelid movement occurred within 100 ms of CS onset. On valid trials the script determined whether a blink response was present and, based on its onset latency, whether it should be classified as a CR; a CR was defined as a blink response with an onset at least 100 ms after the CS onset that also preceded the air puff. For each participant, ten randomly selected trials were inspected visually to confirm that the algorithm performed correctly across trials. Occasional classification errors were not corrected manually, so as not to introduce subjective bias. Three variables were extracted: the maximum percentage of CRs across a block of ten trials (max CR), the mean CR onset latency relative to CS onset (onset mean), and the within-participant standard deviation of CR onset latency (onset SD).

Neuromotor examination

The final test was a standardised neuromotor examination comprising several short motor tasks [75], including diadochokinesia (rapid alternating pronation and supination), Prechtl’s test [76], standing on one leg, jumping on one leg, heel walking, and Bishop’s test (drawing two squares simultaneously with

both hands). The experimenter scored each task on a 0–2 scale, where 0 indicated no difficulty and 2 indicated severe difficulty, yielding a total score from 0 to 26.

Statistical analysis

Analyses were conducted in MATLAB R2026b (MathWorks, Natick, MA, USA). Given the non-normal distribution of several outcomes, continuous variables are summarised as medians (IQRs), and group comparisons used Kruskal–Wallis tests. Epsilon-squared (ε^2) is reported for continuous outcomes. When an omnibus test was significant, pairwise Mann–Whitney U tests were performed, with Bonferroni adjustment across the three planned group comparisons for that outcome; rank-biserial correlations (r) quantify pairwise effect sizes. Spearman rank correlations were calculated between individual test variables and the total neuromotor score in the pooled sample. These correlations are exploratory; because 11 correlations were examined, their p values are unadjusted. Sample size varies by outcome and is reported in the tables. Exact two-sided p values are reported except where $p < 0.001$.

Results

Participants

Table 1 summarises participant characteristics and task outcomes. The groups differed in sex distribution and age. The groups differed in sex distribution, $\chi^2(2) = 9.41$, $p = .009$, Cramér's V = 0.24, with the imbalance most pronounced in the ASD group. The ASD group was also slightly older than controls. These observed differences should be considered when interpreting unadjusted group comparisons.

Table 1

Descriptive statistics and omnibus group comparisons. Values are medians (IQRs). Group differences were assessed with Kruskal–Wallis tests for continuous variables and a chi-square test for sex distribution. Epsilon-squared (ε^2) is reported for continuous variables; report Cramér's V (rather than ε^2) for sex distribution. Sample sizes vary by measure because not all participants completed every test. Abbreviations: ASD, autism spectrum disorder; ADHD, attention-deficit/hyperactivity disorder; FT, finger tapping; CR, conditioned response; SD, standard deviation; IQR, interquartile range. Units: cm, centimetres; ms, milliseconds; %, percentage.

Variable	n	Control	n	ASD	n	ADHD	V/ε^2	p
Sex (male / female)	96	47/49	37	28/9	29	20/9	**.24**	**.009**
Age (years)	96	11.8 (11.2–12.9)	37	13.3 (11.4–14.6)	29	12.6 (10.9–14.1)	**.049**	**.020**
Prism on (cm)	96	14.0 (10.0–20.0)	36	10.0 (8.0–15.0)	29	10.0 (1.2–13.2)	**.153**	**< .001**
Prism off (cm)	96	-7.0 (-10.0–-3.0)	36	-3.5 (-5.0–-2.0)	29	-4.0 (-7.0–0.0)	**.099**	**< .001**
Prism error (cm)	96	3.5 (2.8–4.6)	36	2.5 (1.8–3.1)	29	3.6 (2.6–4.4)	**.120**	**< .001**
FT Synch mean (ms)	92	519 (512–524)	34	513 (505–520)	29	511 (504–515)	**.094**	**< .001**
FT Synch SD (ms)	92	40 (34–47)	34	44 (35–51)	29	52 (44–62)	**.113**	**< .001**
FT Prodmean (ms)	96	503 (483–519)	34	492 (472–505)	29	474 (424–499)	**.103**	**< .001**
FT Local	96	34 (28–42)	34	33 (28–41)	29	39 (33–58)	**.042**	**.036**
FT Drift	96	12 (7–17)	34	12 (10–19)	29	17 (9–33)	.037	.055
Eyeblink Max CR (%)	38	80 (60–100)	33	56 (42–86)	26	60 (40–80)	**.091**	**.0125**
Onset mean (ms)	38	379 (352–407)	33	377 (353–398)	26	319 (256–351)	**.337**	**< .001**
Onset SD (ms)	38	81 (72–88)	33	92 (80–101)	26	101 (87–124)	**.143**	**.001**
Neuromotor score	94	0 (0–1)	36	2 (0–6)	26	2 (0–5)	**.187**	**< .001**

Prism adaptation

Group differences were found for all three prism adaptation measures (Table 1). Children with ADHD and ASD both showed a markedly smaller pointing error than controls on the first trial after the prisms were

introduced and on the first trial after they were removed (Table 2, Fig. 1B–C). Though this intuitively may seem like patients performing better than controls, a smaller error also means less adaptation. The ASD and ADHD groups did not differ from one another on either measure. The mean pointing error across all trials did however distinguish the two clinical groups. Children with ASD produced smaller errors than both controls and children with ADHD, whereas the ADHD group did not differ from controls (Table 2, Fig. 1D). This was the only prism measure on which the two clinical groups differed.

Table 2

Pairwise comparisons between groups. Bonferroni-adjusted p values from Mann–Whitney U tests are shown for control versus ASD, control versus ADHD, and ASD versus ADHD contrasts. Effect sizes are rank-biserial correlations (r); define the sign convention in a footnote. Significance is indicated by colour and asterisks: dark orange, p < 0.001 (***); mid orange, p < 0.01 (**); light orange, p < 0.05 (*); uncoloured, not significant. Abbreviations: ASD, autism spectrum disorder; ADHD, attention-deficit/hyperactivity disorder; FT, finger tapping; CR, conditioned response; SD, standard deviation. Units: cm, centimetres; ms, milliseconds; %, percentage.

Variable	Control vs Autism	Control vs ADHD	Autism vs ADHD
Prism on (cm)	p = .001***, r = − .40	p < .001***, r = − .51	p = .544, r = − .19
Prism off (cm)	p = .001***, r = .40	p = .030*, r = .32	p = 1.00, r = − .07
Prism error (cm)	p < .001***, r = − .50	p = 1.00, r = − .11	p = .022*, r = .39
FT Synch mean (ms)	p = .024*, r = − .31	p = .003**, r = − .41	p = 1.00, r = − .12
FT Synch SD (ms)	p = .570, r = .15	p < .001***, r = .50	p = .019*, r = .40
FT Prodmean (ms)	p = .260, r = − .20	p < .001***, r = − .48	p = .114, r = − .31
FT Local	p = 1.00, r = − .01	p = .032*, r = .31	p = .158, r = .29
Eyeblink Max CR (%)	p = .039*, r = − .34	p = .034*, r = − .37	p = 1.00, r = .00
Onset mean (ms)	p = 1.00, r = − .02	p < .001***, r = − .77	p < .001***, r = − .74
Onset SD (ms)	p = .103, r = .29	p = .002**, r = .51	p = .111, r = .32
Neuromotor score	p < .001***, r = .43	p < .001***, r = .49	p = 1.00, r = .03

Finger tapping

All finger tapping measures except drift showed overall group differences (Table 1). The pattern was driven predominantly by the ADHD group. During the synchronisation phase, children with ADHD were more variable than controls, and also more variable than children with ASD (Table 2, Fig. 2). During the production phase, children with ADHD tapped at a faster rate than controls and showed greater tapping variability, whereas the ASD group did not differ from controls on either measure. The mean inter-tap interval during the synchronisation phase (synch mean) was the exception to this pattern. On this measure both clinical groups tapped slightly faster than controls, with no difference between them. Drift did not differ between groups in the omnibus test and was not examined further.

Eyeblink conditioning

Figure 3. **Eyeblink conditioning performance across groups**. (A–C) Group-averaged eyelid movement across the conditioning session for controls (A), ASD (B), and ADHD (C). Each row represents one trial and each column one time point within the trial; colour indicates eyelid closure expressed as a percentage of the full reflex blink amplitude. The conditioned stimulus (CS) was presented at 1000 ms and the unconditioned stimulus (US) at 1500 ms, giving a CS–US interval of 500 ms (blue vertical lines; shaded region indicates the CS period). Both paired and CS-alone trials are included. In all three groups, eyelid closure emerges progressively during the CS period across trials, reflecting acquisition of the conditioned response (CR). (D) Percentage of trials containing a CR across ten blocks of ten trials. All groups showed CR acquisition across the session, with controls reaching a higher CR percentage than either clinical group. Symbols indicate group means and error bars the standard error of the mean. (E) Maximum CR percentage attained during the session. (F) Mean CR onset latency, measured relative to CS onset. (G) Within-participant standard deviation of CR onset latency, indexing trial-to-trial timing consistency. In panels E–G, boxes show the median and interquartile range, whiskers extend to 1.5 × IQR, crosses denote outliers, individual participants are shown as filled circles, and the shaded distribution to the right of each box is a kernel density estimate. Group sizes: controls n = 38, ASD n = 33, ADHD n = 26.

Neuromotor examination

Both clinical groups scored higher on the neuromotor examination than controls, indicating more minor neurological deficits, and did not differ from one another (Tables 1 and 2). Spearman correlations between each cerebellar test variable and the total neuromotor examination score, computed across all participants, are presented in Table 3. Eight of the eleven variables tested were significantly correlated with the neuromotor score. The strongest associations were with the finger tapping measures: higher neuromotor scores were associated with altered tapping rate and with greater variability in both the synchronisation and production phases. Among the prism measures, a higher neuromotor score was associated with a smaller pointing error when the prisms were introduced and with a smaller mean error overall. Of the eyeblink measures, only the maximum CR percentage was significantly correlated with the neuromotor score, and the two timing measures were not. All correlations were modest in magnitude, and the p-values are uncorrected for the eleven tests performed.

Table 3

Exploratory Spearman correlations between test variables and neuromotor score. Correlations were calculated in the pooled sample (controls, ASD, and ADHD combined). Significance is indicated by colour and asterisks: red, $p < 0.001$ (***); orange, $p < 0.01$ (**); yellow, $p < 0.05$ (*); uncoloured, not significant. Eleven correlations were tested and p values are unadjusted. Abbreviations: ASD, autism spectrum disorder; ADHD, attention-deficit/hyperactivity disorder; CR, conditioned response; SD, standard deviation. Units: cm, centimetres; ms, milliseconds; %, percentage.

Test variable	n	Spearman ρ	p-value
Prism on (cm)	157	**− .220**	**.006****
Prism off (cm)	157	**.132**	.099
Prism error (cm)	157	**− .173**	**.030***
Synch mean (ms)	151	**− .279**	**< .001*****
Synch SD (ms)	151	**.239**	**.003****
Prod mean (ms)	155	**− .287**	**< .001*****
Local (ms)	155	**.235**	**.003****
Drift (ms)	155	**.233**	**.004****
Max CR (%)	94	**− .232**	**.025***
Onset mean (ms)	94	**− .137**	.189
Onset SD (ms)	94	**.169**	.104

Across these group-level comparisons, the three groups showed only partially distinct patterns. Reduced prism-related after-effects, lower maximum conditioned-response percentages, and higher neuromotor scores were observed in both clinical groups relative to controls. The ASD group had a lower overall prism-task error, whereas the ADHD group showed greater timing variability in finger tapping and shorter, more variable conditioned-response latencies. No individual measure in the present analysis provided evidence sufficient to support diagnostic classification.

Discussion

We compared children with ADHD, children with ASD, and typically developing controls on four motor tasks with documented cerebellar contributions. Both clinical groups differed from controls on several measures, providing partial support for the first hypothesis. The second hypothesis received limited support: the ASD and ADHD groups differed on three of 11 measures. Children with ADHD showed greater differences in motor timing, whereas children with ASD showed a lower overall prism-task error.

However, the two clinical groups were statistically indistinguishable on most measures, and the distributions overlapped substantially.

A largely shared profile

The most consistent feature of the results was how rarely the two clinical groups differed. The ASD and ADHD groups differed on three of 11 measures: mean prism error, synchronisation variability, and mean conditioned-response onset latency. On the remaining outcomes, including the two prism-perturbation measures, maximum conditioned-response percentage, and neuromotor score, the two clinical groups did not differ statistically, although both differed from controls on several measures. This pattern is compatible with shared symptomatology[7, 8], and the ESSENCE framework (Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations), which emphasises co-occurring neurodevelopmental conditions and overlapping difficulties across conventional diagnostic categories [77].

Our results extend this observation from symptom overlap to performance on motor tasks with cerebellar contributions. At a group level, children with ADHD and ASD resembled each other more than typically developing controls on several outcomes, consistent with the recognised co-occurrence of the conditions [8, 9]. These data do not establish that the tests identify neurodevelopmental difficulty broadly or that they have diagnostic utility; validation in independent samples with clinically meaningful classification analyses would be required.

Where the groups diverge

Compared with controls, children with ADHD had greater synchronisation variability, a shorter mean inter-tap interval during production, greater local variability, and shorter and more variable conditioned-response onset latencies. These timing-related differences are consistent with literature describing timing deficits and elevated intra-individual variability in ADHD [63, 64]. The eyeblink-conditioning results are also consistent with altered response timing in ADHD [56]. Children with ADHD blinked earlier relative to the air puff than controls and children with ASD. However, the present design cannot determine whether these differences reflect a specific cerebellar process, a broader timing phenotype, medication effects, attentional factors, or other influences. Children with ASD produced a lower mean pointing error across the prism task than both controls and children with ADHD, the only measure that distinguished the ASD. This finding requires cautious interpretation because a lower error is not necessarily evidence of poorer performance or adaptation. Both clinical groups also showed smaller errors on the first trial with the prisms on and on the first trial after their removal. The smaller after-effect after prism removal is consistent with reduced adaptation during prism exposure. In contrast, the smaller initial error at prism introduction could reflect differences in initial pointing strategy, the effective perturbation, or other non-adaptive processes. Accordingly, the lower overall prism error in the ASD group should be described as a performance difference rather than unequivocal evidence of poorer adaptation. Our results differ from

those of Gidley Larson et al. [57]; differences in study populations, tasks, analytic definitions, and sampling may be relevant, but cannot be resolved directly by the present study.

Convergent validity with the neuromotor examination

Eight of the 11 test variables correlated significantly with the total neuromotor examination score in the pooled sample. These associations suggest that some task measures are related to clinically observable motor difficulty. However, the correlations were modest, the analyses were unadjusted for multiple testing, and pooled associations may partly reflect between-group differences. Some eyeblink measures were unrelated to the neuromotor score. This pattern is consistent with evidence that performance across cerebellar tests correlates only weakly [78] and that the tasks may depend on partially distinct mechanisms rather than a single underlying capacity [27]. The differing profiles in ADHD and ASD could reflect differences in several neural or behavioural processes; the present data cannot localise them to specific cerebellar circuits.

Limitations

Several limitations affect interpretation. First, group differences in central tendency cannot establish clinical impairment; distributions overlap, and individual performance frequently fell within the typically developing range. Second, the tasks have cerebellar contributions but are not selective measures of cerebellar function; therefore, the results should not be interpreted as direct evidence of cerebellar dysfunction. Third, the cross-sectional design cannot establish temporal or causal relationships. The modest and outcome-specific sample sizes, particularly for eyeblink conditioning; unmeasured symptom severity and medication status; group differences in age and sex; and differing recruitment/testing periods and settings may have confounded the observed associations. Replication in larger, contemporaneously recruited cohorts with prespecified covariate adjustment, detailed clinical phenotyping, and transparent handling of missing data is needed.

Conclusions

Children with ADHD and children with ASD both differed from typically developing controls across a battery of motor tasks linked to cerebellar function. In most respects, the two clinical groups resembled one another. Where they diverged, ADHD was associated with greater timing variability and ASD with a lower overall prism error, though neither pattern was specific enough to identify individual children. These tasks are quick, inexpensive, and objective, and may prove useful as general indicators of atypical neurodevelopment, but establishing clinical value will require studies that account for age, comorbidity, medication, and symptom severity.

Abbreviations

ADHD
Attention-deficit/hyperactivity disorder
ASD

Autism spectrum disorder

CS

Conditioned stimulus

CR

Conditioned response

US

Unconditioned stimulu

UR

Unconditioned response

DAMP

Deficits in attention, motor control and perception

DCD

Developmental coordination disorder

DSM-5

Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition

ESSENCE

Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations

FT

Finger tapping

IQR

Interquartile range

MND

Minor neurological deficits

SD

Standard deviation

Declarations

Ethics approval and consent to participate

All methods were carried out in accordance with relevant guidelines and regulations. Children and legal guardians gave their consent to participate in the study. Upon arrival, the children and their legal guardians received information about the test battery and were given an opportunity to ask questions. The children were informed that they could end their participation at any point without any consequences. The study and associated procedures were approved by the Swedish Ethical Review Authority in Lund, Sweden (DNR. 2017/745), on 2018-04-17, with revisions 2022-10-12 and 2026-06-12.

Consent for publication

All participants were informed that the results would be published but that they would remain anonymous. The manuscript contains no identifiable information.

Competing interests

The authors declare no competing interests.

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Author Contribution

PG, AR, and PT conceived and designed the study. MH and AR conducted the experiments. PG, AR, and MC analysed the data. AR created the figures. AR and ECK acquired funding. AR, PG, and PT supervised the project. AR, and PT wrote the first draft with input from ECK, MC, and MH. All authors critically reviewed the manuscript and approved the final version

Data availability

Data and code will be made available on a GitHub repository upon acceptance of the article. The datasets and code are also available from the corresponding author.

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Figures

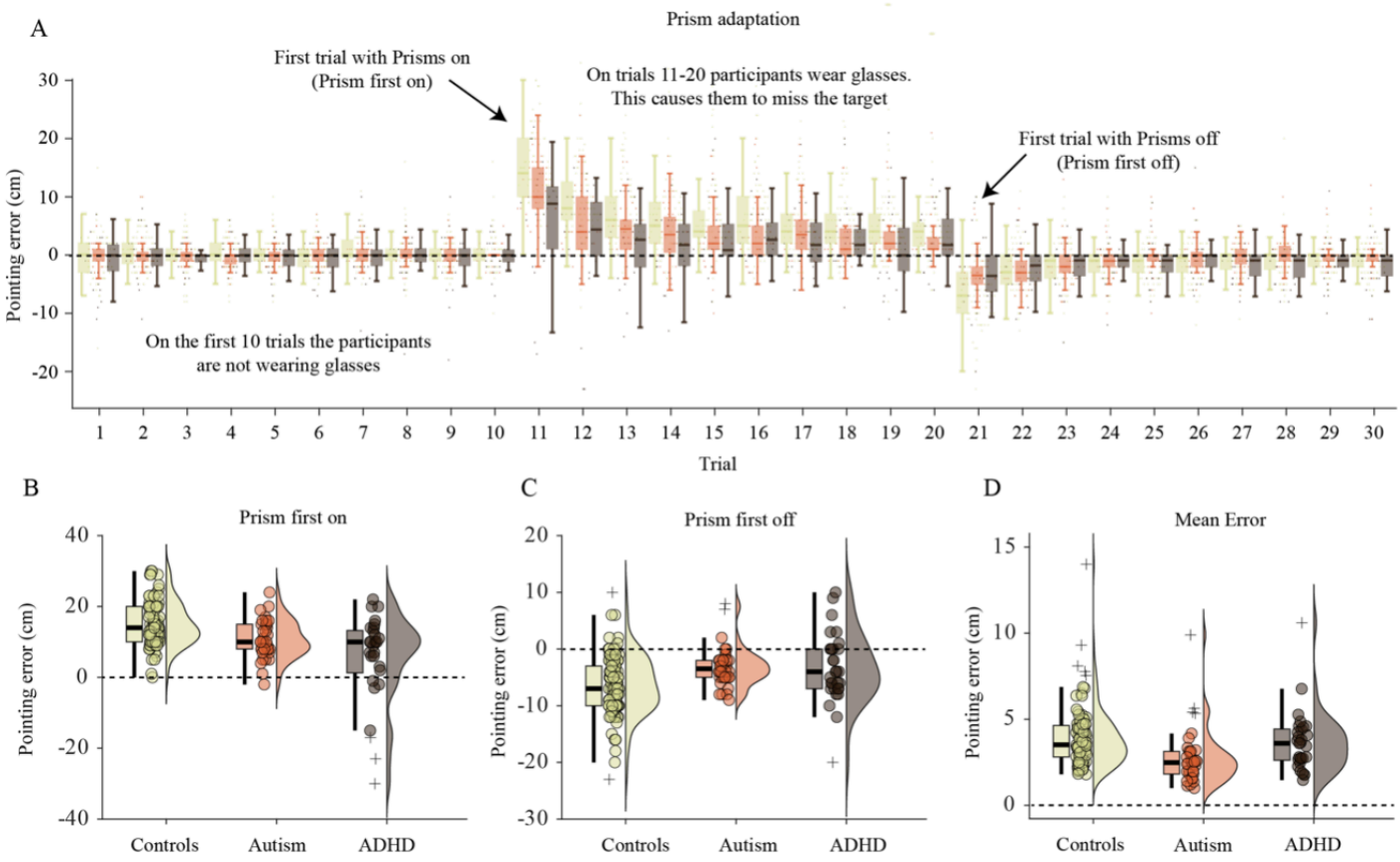


Figure 1

Prism adaptation. (A) Pointing error (cm) across all 30 trials of the prism adaptation task for controls (yellow-green), ASD (orange), and ADHD (dark grey). Participants performed 10 baseline trials without prism glasses (trials 1–10), followed by 10 trials wearing the glasses (trials 11–20), and 10 final trials with the glasses removed (trials 21–30). Introducing the prisms on trial 11 produced a large rightward pointing error that decreased over subsequent trials as participants adapted, while removing the prisms on trial 21 produced an after-effect in the opposite direction that likewise declined across trials. Boxes show the median and interquartile range; whiskers extend to the most extreme values within 1.5 × IQR; individual data points are overlaid with horizontal jitter. The dashed line indicates zero error. (B) Pointing error on the first trial with the prisms on (trial 11), quantifying the initial magnitude of the visuomotor perturbation. (C) Pointing error on the first trial with the prisms removed (trial 21), quantifying the after-effect and thus the degree of adaptation achieved. (D) Mean absolute pointing error across all trials, providing a summary measure of overall task accuracy. In panels B–D, boxes show the median and interquartile range, whiskers extend to 1.5 × IQR, crosses denote outliers, individual data points are

shown as filled circles, and the shaded distribution to the right of each box is a kernel density estimate. Dashed lines indicate zero error. Group sizes: controls n = 96, ASD n = 36, ADHD n = 29.

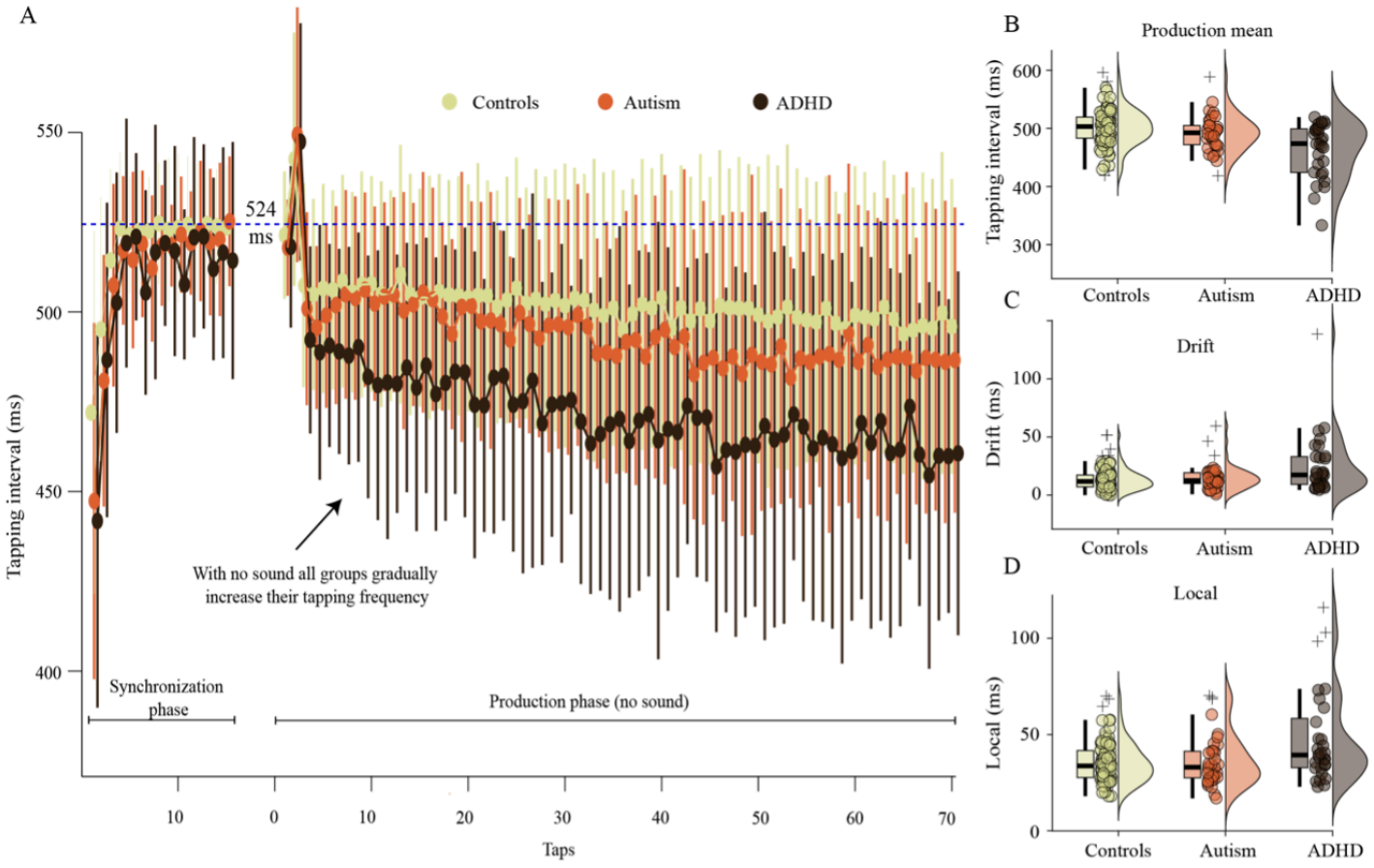


Figure 2

Finger tapping performance across groups. (A) Mean inter-tap interval (ms) in the finger tapping task for controls (yellow-green), ASD (orange), and ADHD (dark grey). Participants first tapped in synchrony with an auditory pacing signal presented at a fixed interval of 524 ms (blue dashed line; synchronisation phase, taps 1–20), after which the signal was withdrawn and participants continued tapping at the same rate from memory (production phase, taps 21–100). All groups converged on the target interval within the first few taps of the synchronisation phase. Once the pacing signal was removed, all groups progressively shortened their inter-tap intervals, with the ADHD group showing the largest and earliest deviation from the target. Filled circles indicate group means and vertical lines indicate ± 1 SD. Group markers are offset horizontally for visibility. Each participant's value at a given tap position is the average across five task repetitions; intervals deviating more than 50% from the 524 ms target were excluded as outliers. (B) Mean inter-tap interval during the production phase. (C) Drift, defined as the systematic change in inter-tap interval across the production phase, indexing the tendency to progressively speed up or slow down. (D) Local variability, defined as the average absolute difference between consecutive inter-tap intervals, indexing tap-to-tap consistency independent of drift. In panels B–D, boxes show the median and interquartile range, whiskers extend to 1.5 × IQR, crosses denote outliers, individual

participants are shown as filled circles, and the shaded distribution to the right of each box is a kernel density estimate. Group sizes: controls n = 96, ASD n = 34, ADHD n = 29.

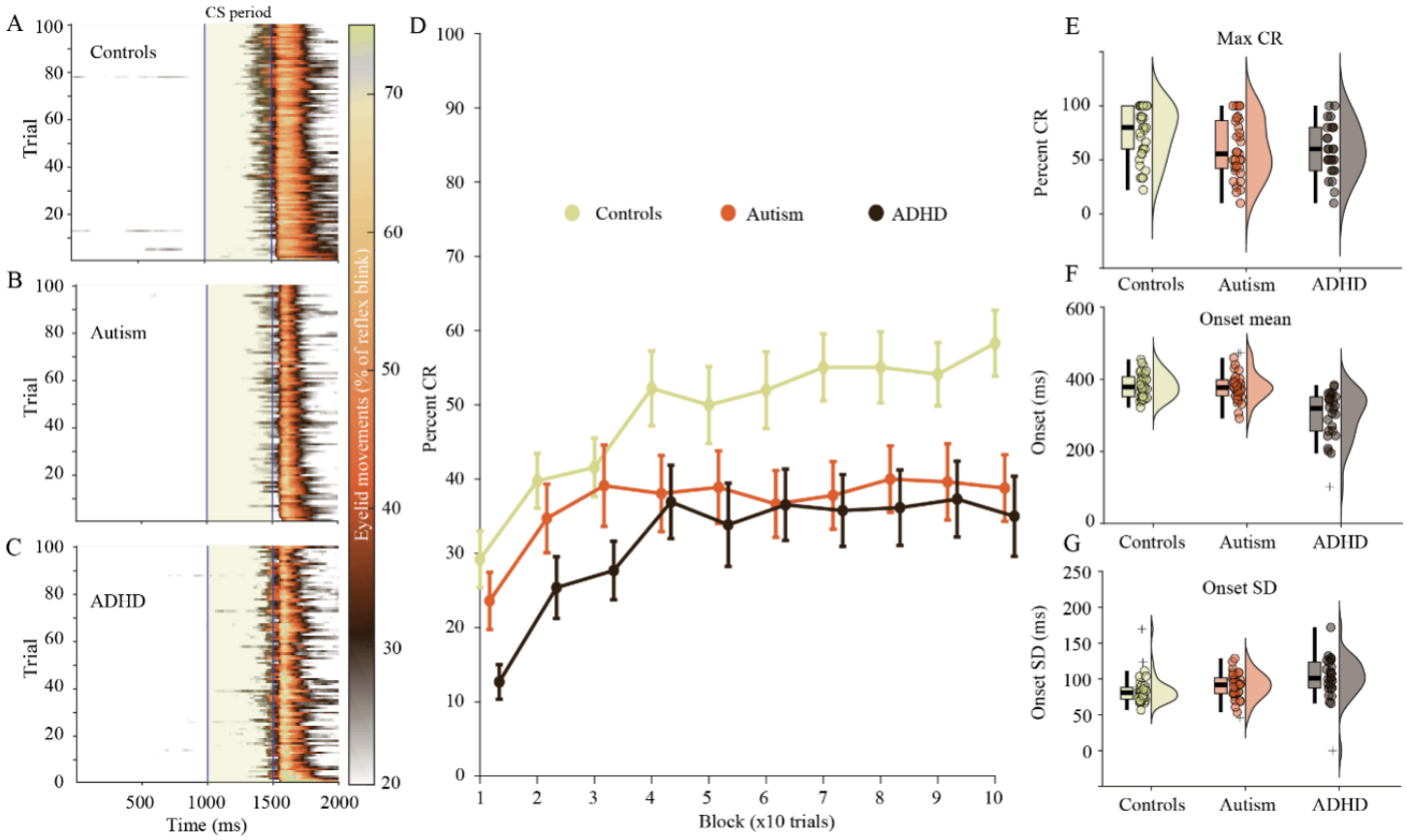


Figure 3

Eyeblink conditioning performance across groups. (A–C) Group-averaged eyelid movement across the conditioning session for controls (A), ASD (B), and ADHD (C). Each row represents one trial and each column one time point within the trial; colour indicates eyelid closure expressed as a percentage of the full reflex blink amplitude. The conditioned stimulus (CS) was presented at 1000 ms and the unconditioned stimulus (US) at 1500 ms, giving a CS–US interval of 500 ms (blue vertical lines; shaded region indicates the CS period). Both paired and CS-alone trials are included. In all three groups, eyelid closure emerges progressively during the CS period across trials, reflecting acquisition of the conditioned response (CR). (D) Percentage of trials containing a CR across ten blocks of ten trials. All groups showed CR acquisition across the session, with controls reaching a higher CR percentage than either clinical group. Symbols indicate group means and error bars the standard error of the mean. (E) Maximum CR percentage attained during the session. (F) Mean CR onset latency, measured relative to CS onset. (G) Within-participant standard deviation of CR onset latency, indexing trial-to-trial timing consistency. In panels E–G, boxes show the median and interquartile range, whiskers extend to 1.5 × IQR, crosses denote outliers, individual participants are shown as filled circles, and the shaded distribution to the right of each box is a kernel density estimate. Group sizes: controls n = 38, ASD n = 33, ADHD n = 26.