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Action observation in autism: preserved kinematic-driven sensorimotor resonance, distinct adult neural strategies

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Action observation in autism: preserved kinematic-driven sensorimotor resonance, distinct adult neural strategies

Running title: **Kinematic resonance in Autism**

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Abstract

Background: Difficulties understanding and predicting others' actions are frequently reported in autism spectrum disorder (ASD). Whether they arise from atypicalities in early sensorimotor processing or at higher-order cognitive levels remains unclear.

Methods: We indexed kinematics-driven sensorimotor resonance by assessing cortico-kinematic coupling (CKC) during executed and observed movements in autistic and non-autistic participants. MEG signals were recorded while children (7–13 y.o; 17 autistic, 20 non-autistic) and adults (18–35 y.o; 22 autistic, 23 non-autistic) executed goal-directed hand-grasping movements or observed an actor performing the same task. Subjects' and actor's forefinger movement kinematics were monitored with an accelerometer. CKC was assessed as the coherence between movement acceleration and MEG signals at both sensor and source levels and compared between groups.

Results: Across participants, action execution and observation led to significant CKC within an overlapping visual, parietal and sensorimotor network. Within that network, CKC was stronger in adults than children, with no significant difference between autistic and non-autistic participants. In adults, autistic participants showed enhanced CKC at the right primary motor cortex during action observation, together with trait-dependent scaling of CKC in the somatosensory cortex.

Conclusion: Our results show that kinematics-driven sensorimotor resonance is preserved in autism. Age- and trait-dependent modulations in adulthood suggest a greater reliance on predictable observed action kinematics. These findings challenge accounts positing a primary disruption of embodied kinematic-driven sensorimotor mechanisms in autism. The root of altered action understanding in ASD may thus be located at higher-order cognitive levels.

Limitations: The cross-sectional design limits developmental inferences, while the selected sample restricts generalizability and calls for broader inclusion criteria in future studies. Differences in autistic trait assessment and gender distribution across age groups, together with potential age-related MEG confounds, should also be considered.

Key words:

Autism Spectrum Disorders, Magnetoencephalography, Functional Neuroimaging, Action Observation, Kinematic Coupling, Motor Resonance

Main text

1. Background

Autism Spectrum Disorder (henceforth 'autism') is associated with pervasive and early-emerging motor atypicalities, including delayed motor development, impaired coordination, clumsy or variable movements, atypical movement kinematics, and reduced or atypical imitation (1–7). Autistic individuals also show atypical processing of biological motion (8–10) and minor sensitivity to observed kinematic variations (11), especially when movements are difficult to predict (12,13).

Motor difficulties in autism have been interpreted within several, non-mutually exclusive pathophysiological frameworks, one of which implicates impairments in "motor cognition" rather than isolated motor deficits (14,15). Motor cognition refers to the neural processes by which the human motor system links perception to the planning, prediction, and understanding of one's own and others' actions (15,16). This framework integrates motor processing with social cognition by positing that action understanding emerges from internal sensorimotor models that translate observed movements into sensorimotor representations (17–19). Motor

92 cognition anomalies may lead to reduced access to others' mental states and contribute to core
93 social difficulties in autism (14). Despite these theoretical accounts, the neural bases of motor
94 atypicalities in autism and their developmental trajectory remain poorly understood (20–22).
95 A meta-analytic work indeed highlighted substantial variability across studies, with evidence
96 of more pronounced differences during childhood (22).
97 At the neural level, motor cognition is notably supported by the "action observation network"
98 (AON), a fronto-parieto-temporal neural network engaged during both action execution and
99 observation. Atypical AON dynamics has been widely reported in autism (23,24). However,
100 observed action understanding relies on neural processes within the AON that range from low-
101 level sensory and sensorimotor encoding of observed movements to higher-level associative or
102 inferential representations of action goal or intention (25–28). Still, it remains debated which
103 of these processing levels are altered in autism (29).
104 Cortico-kinematic coupling (CKC) offers a principled means to assess the integrity of the low-
105 level processes in autism. Indeed, CKC measures movement kinematics (e.g., acceleration)
106 representation at the cortical level, and movement kinematics form the lowest level of
107 hierarchical sensorimotor representations within the AON (28). CKC quantifies, using
108 electroencephalography (EEG) or magnetoencephalography (MEG), the frequency-specific
109 coupling between cortical signals and movement kinematics (30). During goal-directed action
110 execution, CKC mainly occurs at movement frequency (*F0*) and its first harmonic (*F1*) (30),
111 and is driven by afferent proprioceptive inputs to some AON nodes, including the primary
112 sensorimotor (SM1) cortex (31,32). During action observation, it involves similar coupling
113 frequencies and nodes, indicating that observed and self-generated movement kinematics
114 engage similar sensorimotor circuits (33). Consequently, the visual kinematic information from
115 others' movements can engage proprioceptive-like cortical coupling, consistent with

kinematics-driven sensorimotor resonance possibly contributing to the understanding of *how* others' actions are performed (33,34).

This MEG study assesses whether atypical action observation in autism is accompanied by alterations in sensorimotor resonance at the kinematic level. To do so, we assessed CKC during executed and observed finger grasping movements in autistic children (*AutisticC,* following identity-first language preferences) (35,36) and adults (*AutisticA*), and age- and sex-matched non-autistic subjects. Given the atypicalities in movement kinematic processing reported in autism (2,3,37), we expected that autistic individuals would exhibit reduced CKC with observed movements compared with non-autistic individuals, with distinct age-related patterns.

2. Methods

2.1 Participants

The final sample comprised 17 autistic children (AutisitcC), 20 age- and sex-matched non-autistic peers (NonautisticC), 22 autistic adults (AutisticA), and 23 age- and sex-matched non-autistic adults (NonautisticA).

Participants' demographics and assessment data are reported in Table 1.

Supplementary Methods presents in details participants' selection procedure.

For autistic participants, eligibility criteria were: (i) age 7–13 years for children and 18–35 years for adults; (ii) autism diagnostic based on DSM-V-TR criteria (38); (iii) a full-scale IQ above 80 on either the Wechsler Intelligence Scale for Children–Fifth Edition (WISC-V)(39), or the Wechsler Adult Intelligence Scale–Fourth Edition (WAIS-IV) (40), and a Verbal Comprehension Index (VCI) above 80 on the WAIS-IV/WISC-V; (iv) right-handedness

according to the Edinburgh Handedness Inventory (41); and (v) good MEG data quality. Matched non-autistic participants had no psychiatric, neurological, or developmental disorders and did not use any psychotropic medication.

The study was approved by the institutional Ethics Committee. All participants and minors' legal representatives provided written informed consent before participation.

2.2 Experimental paradigm

Figure 1 illustrates the experimental paradigm adapted from our previous study (33).

Participants underwent two 5-min conditions (i.e., *Self*, *Other)* in an order that was counterbalanced across participants.

In *Self*, participants sat on the MEG chair with their hands on a table. A plastic bowl was placed near the left hand and a heap of colored foam pieces near the right hand (~55 green items and ~20 red, purple, orange, or yellow items; thickness ~4 mm, surface area ~1 cm^2). Participants were asked to pinch the green pieces at ~1 Hz, one at a time, and place them into the bowl. The movement was repeated until no green piece remained in the heap; the whole process being repeated successively ~5 times during the 5 min. To minimize eye movements (thereby reducing ocular artifacts in the MEG signals), participants were asked to maintain their gaze on the heap of colored pieces while performing movements.

In *Other*, participants observed an actor seated 1.5 m away performing the same task with their right hand. At all times, the actor was concealed behind a screen, leaving only their hands, the plastic pot and the heap of foam pieces visible to the participant. Participants were instructed to remain still, relax, not to move, and gaze at the heap. Experimenters verified that participants were not imitating the actor.

2.3 Data acquisition

Brain activity was recorded (band-pass: 0.1–330 Hz, sampling rate: 1 kHz) with a whole-scalp-covering 306-channel MEG system (Triux, MEGIN, Finland) inside a lightweight magnetically shielded room (Maxshield, MEGIN)(42). Head position was continuously monitored using four head-position indicator (HPI) coils, whose position on the participants' head was digitized with ≥300 scalp and facial points relative to anatomical fiducials (Fastrack, Polhemus, Colchester, VT, USA). Right index-finger movements (participants' in *Self*; actor's in *Other*) were simultaneously recorded with a three-axis accelerometer (ADXL335, Analog Devices, USA).

T1-weighted MRI image was taken from participants' brain using a 3-T scanner (Signa, GE Healthcare) after the completion of the experimental paradigm.

2.4 Data preprocessing

Continuous MEG data were preprocessed offline using temporal signal space separation (tSSS, correlation limit: 0.9, window length: 20 s; MaxFilter, MEGIN, Finland) to subtract external interference and correct for head motion (43,44). Independent component analysis (ICA) was subsequently performed to identify and remove artifacts (e.g., eye blink, eye movement, cardiac and system artifacts). Thirty ICAs were extracted from the MEG signals using the FastICA algorithm (band-pass filter, 0.1-25 Hz; rank reduction, 30; nonlinearity, tanh). Components corresponding to artifacts were visually identified and subtracted from the full-rank data. Between 2-4 components were removed per participant, with no significant difference between autistic and non-autistic participants in any condition (3-way ANOVA, all ps > 0.05), but a significant effect of age (*Self*: $F(1,78)= 54.2$, $p < 0.01$; *Other*: $F(1,78)= 8.75$, $p = 0.004$); more components were removed in children than in adults in both conditions (*Self*, difference= 1.80, $t(78) = 7.36$, $p < 0.001$; *Other*, difference =1.12, $t(78)= 2.96$, $p = 0.004$). To eliminate residual artifacts, segments of MEG signals were marked as bad whenever MEG

amplitude exceeded 5 pT in any magnetometer or 1 pT/cm in any gradiometer channel. Each identified segment was then extended by 1 s before and after the threshold crossing, and these periods were excluded from subsequent analyses.

2.5 Movement kinematics

Movement frequency (*F0*) and movement regularity were derived from accelerometer signals. The three-axis accelerometer signals were first band-pass filtered (0.1–195 Hz) and then reduced to their first principal component using principal component analysis. We estimated the power spectrum of this preprocessed acceleration signal using an FFT applied to the entire data duration at once. Squared Fourier coefficients were further smoothed with a 0.2-Hz FWHM Gaussian kernel. The first spectral peak after 0.5 Hz provided a precise estimate of *F0*. In addition, movement regularity was assessed as the ratio between the spectral power at *F0* and the mean power across frequencies from 0.75 × *F0* to 1.25 × *F0*. Higher values of the movement regularity parameter indicated a sharp spectral peak consistent with low temporal variability in movement kinematics.

2.6 Sensor-space coherence analysis between accelerometer and MEG

Coherence between accelerometer signals and artifact-free MEG data was first computed at the sensor level to determine the frequencies of interest for each participant and condition. Both MEG and the preprocessed accelerometer signals were segmented into 5-s epochs with 4 s overlap (0.2 Hz resolution) (45). Coherence was computed as previously described (46), yielding values that range from 0 (no linear dependency) to 1 (perfect linear dependency) for each frequency bin.

2.7 Source-space coherence analyses

Source-space analyses were conducted on coherence values at *F0* and *F1*. For that, MEG and MRI coordinate systems were first co-registered using three anatomical fiducials and further manual refinement via digitized head-surface points. For participants without MRI (1, AutisticA; 1, NonautisticA), age- and sex-matched surrogate MRIs were warped to fit head points using the Coherent Point Drift (CPD) algorithm (47) (FieldTrip toolbox, Donders Institute for Brain Cognition and Behaviour, Nijmegen, The Netherlands, RRID:SCR_004849). Individual MRIs were then segmented with Freesurfer (Martinos Center for Biomedical Imaging, Boston, MA, USA; RRID:SCR_001847) and normalized to the Montreal Neurological Institute (MNI) template brain using the nonlinear spatial normalization procedure implemented in SPM8 (48). This transformation was used to map a homogeneous 5-mm grid sampling the MNI brain volume onto individual brain volumes. For each subject and grid point, the MEG forward model corresponding to three orthogonal current dipoles was computed using the one-layer Boundary Element Method implemented in the MNE software suite (Martinos Centre for Biomedical Imaging, Boston, MA, RRID:SCR_005972)(49). The forward model was then reduced to its first two principal components. This procedure is justified by the insensitivity of MEG to currents radial to the skull, and hence, this dimension reduction leads to considering only the tangential sources. An inverse solution was then computed using Minimum Norm Estimate approach(50) for gradiometer channels only, assuming a SNR of 1 (51).

The inverse solution was used to generate a CKC source map for each participant, condition (*Self*, *Other*) and frequency of interest (*F0*, *F1*) (31).

2.8 Source-space analyses

Within each group (Autistic, Nonautistic) and age (children, adults), we first generated averaged maps for each condition and frequency of interest. To further characterize CKC

distribution, we extracted coordinates of statistically significant local maxima on the averaged maps. Local maxima were identified as clusters of spatially contiguous voxels exhibiting higher coherence values than their neighbors. This choice to focus only on local maxima and disregard other aspects of coherence distribution is justified by the intrinsic spatial smoothness of MNE-based MEG source estimates (52–54). Since significant local coherence maxima were consistently located in similar cortical regions across groups and age (see Results), we computed grand-average coherence maps, averaging all the individual maps for each condition and frequency of interest. These maps provided the coordinates of local coherence maxima, which were used to extract individual coherence values used for further statistical analyses.

2.9 Statistical analysis

Local maxima and permutation statistics were computed in MATLAB (R2022b, Version 9.13.0). Descriptive analyses, ANOVAs, General Linear Model (GLM) and Linear Mixed Model (LMM) were performed using Jamovi (Version 2.6.26.0).

2.9.1 Effect of age and group on F0 and movement regularity

Two-way ANOVAs with group (Autistic, Nonautistic) and age (children, adults) as independent variables were conducted separately for *Self* and *Other* on *F0* and movement regularity values.

2.9.2 Significance of coherence at sensor level

Statistical significance of coherence was assessed using a surrogate-data-based approach (34,55). For each participant and condition, 1000 surrogate coherence spectra were obtained by computing coherence between real MEG signals and Fourier-transform surrogate acceleration signals; the Fourier-transform surrogate imposes power spectrum to remain the

same as in the original signal, but it replaces the phase of Fourier coefficients by random numbers in the range [-π, π] (56). For each surrogate spectrum, the maximum coherence value across all gradiometers and across 0.4-2 Hz was extracted, yielding a null distribution of maxima. The 95th percentile of this distribution defined the significance threshold ($p < 0.05$, corrected for multiple comparisons).

2.9.3 Significance of coherence at the source level

Subject- and averaged null CKC maps were produced for each condition and frequency of interest using subject-level permutation data in which the accelerometer signal was circularly shifted by approximately half of the recording duration relative to the MEG signals, disrupting their temporal alignment while preserving the spectral content. Difference maps were then calculated by subtracting these surrogate CKC maps from the genuine CKC maps. Under the null hypothesis that CKC maps do not depend on the correct accelerometer–MEG alignment, the labels "genuine" and "surrogate" are exchangeable at the subject level before computing average difference maps. A permutation distribution of the maximum absolute value across all sources was obtained from 1,000 random permutations, and the 95th percentile defined the significance threshold ($p < 0.05$, corrected for multiple comparisons)(55). Local maxima exceeding this threshold were considered as statistically significant CKC sources.

2.9.4 Common cortical sources across Self and Other

To identify cortical regions consistently engaged during action execution and observation, we built, for each age, group, and frequency of interest (*F0* and *F1*), a map that takes value 1 where both conditions display significant CKC, and 0 otherwise. All 8 binary maps were averaged to yield a map of the proportion of consistency between *Self* and *Other* across groups and age.

2.9.5 Effects of age, groups, and conditions on CKC levels

To search for significant effects of age (children, adults) and group (autistic, non-autistic) on CKC levels during movement execution and observation, we first performed intra-condition comparisons. In practice, individual values for each significant local maximum of grand-average CKC maps obtained during *Self* and *Other* were extracted from every participant's source map as the maximal value within a 15-mm sphere centered on each grand-averaged local maximum. Then, for each condition separately, coherence values from selected local maxima were entered into a GLM with age and group as fixed factors. Subsequently, age-specific GLMs were run separately to account for relevant covariates: for adults, models included group and Autism-Spectrum Quotient (AQ); for children, models included group and Social Communication Questionnaire (SCQ). All *p*-values were corrected for multiple comparisons using the Benjamini–Hochberg false discovery rate (FDR) procedure.

Finally, to search for differences between action execution and observation, coherence values extracted from sensorimotor regions common to both conditions were analyzed using linear mixed-effects models (LMMs). Models included condition (*Self* vs. *Other*), hemisphere, age, and group as fixed effects, with subject entered as a random intercept to account for repeated measures across hemispheres.

3. Results

Full statistical results are provided in Supplementary Results; only the main findings are reported below.

3.1 Movement frequency and regularity

In *Self*, autistic participants produced movements at a lower frequency than non-autistic participants (difference = −0.120 Hz, t(78) = - 2.85, p = 0.006). AutisticC also had reduced movement regularity compared with NonautisticC. In *Other*, movements were unintentionally executed by the actor at a lower frequency for autistic than non-autistic participants (difference = -0.035 Hz, t(78) = -2.54, p = 0.013).

3.2 Coherence analysis in sensor space

No significant effect of age, group, condition, or interactions was found ($p < 0.05$) on the number of epochs used to compute CKC (mean epoch number: 293).

Accelerometer and sensor-level CKC spectra peaked at F0 and F1 (Figure 2), which were thus considered as frequencies of interest for further analyses.

Maximum coherence occurred at fronto-parietal MEG sensors.

Significant sensor-level coherence was found in all participants during *Self* and nearly all participants during *Other* (Table 2).

3.3 Coherence analysis in source space

In both *Self* and *Other*, coherent sources were mainly found at bilateral motor (M1), primary (S1) and secondary (S2) somatosensory and occipital cortices, across children/adults and autistic/non-autistic participants (Figure 3 for averaged source level maps). See Supplementary Table 1 for MNI coordinates.

Figure 4 shows that bilateral S1 and M1 cortices consistently showed significant CKC during both *Self* and *Other* across ages, groups and frequencies (*F0*, *F1*).

3.4 Effects of age, autism, and condition on CKC levels

In *Self,* no significant effect of age, group or their interaction was found at *F0* across local coherence maxima. At F1, CKC increased from childhood to adulthood across all local maxima, with a significant age-by-group interaction in right M1 reflecting a stronger age-related increase in the autistic group. CKC was also higher in AutisticA compared with NonautisticA. Including AQ (adults) and SCQ (children) in the GLMs did not reveal any significant effect.

In *Other*, CKC was higher in adults compared with children in several brain areas (*F0*, right primary visual (V1) cortex, right superior parietal lobule (SPL); *F1*, bilateral M1 cortices, right S2 cortex). Furthermore, CKC at F1 was higher in the right M1 cortex in AutisticA compared with AutisticC and NonautisticA (no age-related effects within the non-autistic group) (Supplementary Figure 1). A significant AQ × group interaction at F1 in the right S2 cortex showed that the association between AQ and CKC differed across groups, with CKC increasing with AQ in AutisticA and decreasing with AQ in NonautisticA (Supplementary Figure 2). Including SCQ scores in the GLM did not reveal any significant effects in children.

When comparing *Self* and *Other*, higher coherence was found during movement execution compared with movement observation at *F0* and *F1*. This difference was larger in adults compared with children. At F1, AutisticA also showed higher overall coherence than NonautisticA, whereas no group difference was observed in children.

4. Discussion

This MEG study demonstrates that activity of a bilateral sensorimotor network is coupled with the kinematics of executed and observed repetitive goal-directed hand movements in both autistic and non-autistic individuals, with a general age-related strengthening of CKC with observed movement. Crucially, autism-specific modulations emerged in adulthood, marked by

enhanced CKC with observed movement in right M1 cortex and opposite trait-dependent scaling of CKC level in the right S2 cortex between autistic and non-autistic adults. These findings point to a preserved kinematic-driven sensorimotor resonance in autistic individuals, with trait-dependent modulation in adulthood.

4.1 Participants

Groups did not differ in age, sex distribution (within adults and children), or handedness. Full-scale IQ was similar between AutisticA and NonautisticA. AutisticC showed lower and more variable cognitive scores than NonautisticC (107 ± 12.9 vs. 122 ± 7.9) and lower socioeconomic status. However, their IQ remained within the normal range. This pattern may reflect recruitment bias favoring participation of NonautisticC from higher socioeconomic backgrounds in neuroimaging research. Parent-reported motor competence (DCDQ) was lower in AutisticC, consistent with the high prevalence of motor difficulties in autism. Motor atypicalities in autistic participants were reflected in a lower *F0* during *Self* (despite receiving similar instructions), and in less regular movements in AutisticC compared with NonautisticC, which did not affect CKC levels. This is in line with a previous study, which showed that coherence levels at SM1 cortex during movement execution are not influenced by *F0* or movement range in Non-autisticA(57,58). Overall, the included autistic participants spanned a range of traits' severity within the mild-to-moderate spectrum, without intellectual disability.

4.2 Motor resonance

Observing others' hand kinematics entrained brain activity across a broad sensorimotor network that overlapped with that engaged during execution of similar actions. As expected (33), the kinematic-driven coupling was weaker during movement observation than during execution. Action execution–observation difference was larger in adults than in children,

389 suggesting a progressive strengthening of proprioceptive sensorimotor representations with
390 age.
391 This shared coupling involved bilateral sensorimotor (M1, S1, S2) cortex, with additional
392 engagement of frontal and parietal regions. These areas are consistent with the AON, which is
393 implicated in both action execution and observation (34,59–64). The contribution of M1 cortex
394 is in line with the presence of mirror neurons in the M1 cortex (e.g., cortico-spinal tract
395 neurons) in non-human primates (65–67) and the beta-band M1 cortex modulation during
396 movement execution/observation in humans (68,69). These findings replicate prior results in
397 NonautisticA(33) in an independent cohort and, importantly, extend them to NonautisticC,
398 indicating that kinematic mirroring is already present at school-age. More importantly, the
399 preservation of this shared kinematic-driven sensorimotor coupling in autistic participants
400 suggests that the kinematics-driven mapping of observed movement onto the observer's
401 sensorimotor system is preserved in autistic individuals, regardless of age.
402
403 *4.3 Age- and group-related effects on CKC levels*
404 Adults showed stronger CKC than children in *Self* (*F1*) and *Other* (*F0*, *F1*). This may reflect
405 age-related strengthening of proprioceptive sensorimotor representations with age. Still,
406 increased brain-to-sensor distance and lower MEG data quality (higher number of subtracted
407 independent components in children, but similar number of rejected epochs) may have
408 contributed to this difference. Future studies using on-scalp MEG should help clarify this
409 issue(70).
410 Higher CKC level was observed at the right M1 cortex in AutisticA compared with AutisticC
411 and non-autistic participants in both *Self* and *Other*. CKC in the ipsilateral M1 cortex during
412 movement execution has been well documented (57,71) in NonautisticA and is in line with the
413 increased ipsilateral SM1 recruitment described in autistic adolescents/adults (72). In

AutisticA, increased ipsilateral coupling might reflect higher interhemispheric inhibitory processes phase-locked to movement kinematics, neural processing of movement-related proprioceptive inputs (33,71,73), or unintentional muscle activity occurring in contralateral homologous muscles during a movement(33,71,74,75). Noteworthily, although such a motor/mirror overflow is frequently reported in AutisticC, its persistence into adulthood remains unclear (76). During observation of right hand movements in a third person perspective, we previously showed in NonautisticA that CKC is higher in the right than in the left SM1 cortex, opposite to the pattern observed during right-hand movement execution (33). This asymmetry probably reflects a preferential mirror-image mapping of observed actions (33,77).

The increase in CKC level in the right M1 cortex observed in AutisticA during *Other* might reflect increased reliance on a mirror-image perspective. Emerging here under passive observation, this may constitute an implicit sensorimotor bias that can later be recruited in more explicit contexts. In everyday social situations requiring behavioral adaptation, such visually based mapping could indeed facilitate explicit, visually guided imitation and thereby support camouflaging strategies, rather than spontaneous, embodied sensorimotor resonance. Camouflage refers to autistic individuals modifying or compensating for natural social behaviors to adapt to the neurotypical social world (78). It might also be promoted by the —unintentional— slower experimenter's movement in autistic participants (79). Still, some studies reported reduced mirror-image mapping in autism during imitation tasks (77), but evidence in literature remains mixed (80). Further studies are thus needed to clarify the neural bases of this finding.

In *Other*, adults showed a significant group-by-trait interaction at *F1* in the right S2 cortex, with AQ scores positively correlated with CKC levels in AutisticA, but negatively in NonautisticA. This finding may reflect a stronger dependence on kinematic-driven

proprioceptive inputs during visuomotor transformation in AutisticA, whereas the opposite pattern in NonautisticA may indicate a more efficient integration of contextual somatosensory priors (81) . The emergence of this difference only in adults suggests that compensatory processes (e.g., camouflaging) may become more pronounced with age and increasing autistic traits.

5. Limitations

The cross-sectional design precludes direct developmental inferences: age-related effects should therefore be interpreted cautiously. The restriction to right-handed participants, predominance of mild-to-moderate autistic traits without intellectual disability, use of different measures of autistic in children and adults, gender distribution differences across ages, highly predictable movements, and the unintentional lower experimenter's movement *F0* during *Other* when performing the task for autistic participants, limit interpretations and generalizability. Greater head motion (despite similar number of epochs retained for coherence computation) and increased source-to-sensor distance in children may also bias age-related findings. As CKC during *Other* is not driven by imitative movements in NonautisticA, and to facilitate participation of autistic individuals, electromyography was not recorded. Participants were closely monitored to prevent imitative movements, making CKC during *Other* unlikely due to imitative movements.

6. Conclusions

This study argues against a deficit in kinematic-driven sensorimotor resonance in autistic children and adults without intellectual disability. Instead, autism involves atypical neural strategies for processing predictable hand action in adults, potentially reflecting greater

experience-dependent reliance on kinematic cues. These findings suggest that altered action understanding in autism is due to atypicalities at higher-order cognitive levels.

List of abbreviations:

AON: Action observation network

AQ: Autism-Spectrum Quotient

AutisticA: Autistic adults

AutisticC: Autistic children

CKC: Cortico-kinematic coupling

DCDQ : Developmental Coordination Disorder Questionnaire

EEG: Electroencephalography

F0: Fundamental movement frequency

F1: First harmonic of the movement frequency

FDR: False discovery rate

FFT : Fast Fourier transform

FWHM: Full width at half maximum

GLM: General linear model

ICA: Independent component analysis

IQ: Intelligence quotient

LMM: Linear mixed-effects model

M1: Primary motor cortex

MEG: Magnetoencephalography

MNI: Montreal Neurological Institute

NonautisticA: Non-autistic adults

NonautisticC: Non-autistic children

S1: Primary somatosensory cortex

S2: Secondary somatosensory cortex

SCQ : Social Communication Questionnaire

SM1: Primary sensorimotor cortex

tSSS: Temporal signal space separation

VCI: Verbal Comprehension Index

WAIS-IV: Wechsler Adult Intelligence Scale–Fourth Edition

WISC-V: Wechsler Intelligence Scale for Children–Fifth Edition

Declarations

Human Ethics and consent to participate

The study was approved by the Ethical Committees of HUB - Erasme, HUB -Huderf and CHU-Brugmann, in accordance with the Declaration of Helsinki.

Written informed consent was obtained from all adult participants and children legal representants prior to inclusion in the study.

Data availability

The dataset supporting the findings of this study is available from corresponding author upon reasonable request. No material from other sources requiring permission was reproduced in this manuscript. This study was not a clinical trial and therefore was not registered.

Fundings

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Conflict of interest

The authors declare that they have no competing interests.

Acknowledgments

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Supplementary Information

Supplementary Methods

Participants

A total of 93 participants were initially recruited for this study, including 45 autistic individuals (23 children, AutisticC; 22 adults, AutisticA) and 48 non-autistic individuals (21 children, NonautisticC; 27 adults, NonautisticA).

Autistic participants were recruited through the Autism Reference Center (CRA) at Hôpital Universitaire des Enfants Reine Fabiola (HUDERF), the Psychiatry Department of CHU Brugmann, and the Autism in Context: Theory and Experiment (ACTE) volunteer database.

Within the autistic group, comorbid attention deficit and hyperactivity disorders (ADHD) and anxio-depressive disorders were not considered as exclusion criteria, given their high prevalence in autism (ADHD, 40–70%: (82,83); depression, 20–40% : (84)). One AutisticC and two AutisticA had comorbid ADHD; the child was treated with risperidone and both adults with methylphenidate. Four AutisticA were on antidepressants (2, Fluoxetine; 1, Venlafaxine; 1, Wellbutrin), and one reported occasional alprazolam use. To limit medication-related effects on MEG: methylphenidate was discontinued ≥24 h before testing, benzodiazepines and risperidone were avoided for 48 h, and antidepressants were maintained for safety despite their possible modest impact on MEG signals (85).

Among autistic participants we also collected scores from the Autism Diagnostic Observation Schedule (ADOS) (86), a standardized semi-structured assessment of social and communicative behavior frequently used in diagnostic settings. Actual autistic traits were measured with the Autism-Spectrum Quotient (AQ) (87) in adults and the parent-reported last-three months Social Communication Questionnaire (SCQ) (88) in children. Anxiety was

assessed using the 6-item State-Trait Anxiety Inventory (STAI-6)(89). Adult participants were further assessed for their empathy using the Empathy Quotient (EQ) (90). Children's motor abilities were evaluated through the parent-completed Developmental Coordination Disorder Questionnaire (DCDQ) in its French-version (91). The questionnaire includes 15 items assessing motor functioning across both gross and fine motor domains, including general coordination, control during movement, and fine motor skills (e.g., handwriting), with age-appropriate items (from 5 to 15 y.o.). In the present study, the DCDQ was administered for descriptive purposes only.

Socioeconomic status (SES) was also assessed using an adapted questionnaire (92). Educational level was rated on a 6-point scale (1 = no primary education, 6 = doctoral degree), and economic status on a 14-point scale (0 = very low, 13 = very high). Total SES was computed as the sum of education and economic ratings in adults, and as the mean parental score (or single score in single-parent families) in children. Following the general assessment, four NonautisticA were excluded: one for IQ < 80, one due to emerging concerns about possible autism (later supported by AQ score), one for poor MEG data quality, and one to maintain age- and sex-matching across groups. Within the child cohort, five AutisticC were excluded due to poor compliance or excessive motion artifacts, and another one for epileptic spikes detected during MEG data preprocessing. In the non-autistic group, one NonautisticC was excluded for a laterality quotient of – 42 (cut-offs: < -40 = left-handed, -40 to +40 = ambidextrous, > +40 = right-handed) (41), despite self-reported ambidexterity.

Supplementary Results

Movement frequency and regularity

In *Self*, a significant main effect of group was observed for *F0* ($F(1,78) = 8.13$, $p = 0.006$), with no significant effects of age ($F(1,78) = 0.93$, $p = 0.337$) or age-by-group interaction ($F(1,78) = 2.48$, $p = 0.119$). Post-hoc comparisons indicated that autistic participants produced movements at a lower frequency than non-autistic participants (difference = −0.120 Hz, t(78) = -2.85, $p = 0.006$; mean *F0* values ± SD, AutisticA: 0.580 ± 0.212 Hz, AutisticC: 0.554 ± 0.185 Hz, NonautisticA: 0.634 ± 0.138 Hz, NonautisticC: 0.740 ± 0.215 Hz). For movement regularity in the *Self* condition, no main effects of age ($F(1,78) = 0.35$, $p = 0.557$) or group ($F(1,78) = 1.44$, $p = 0.234$) were observed. However, a significant age-by-group interaction was found ($F(1,78) = 4.35$, $p = 0.040$), driven by reduced regularity in AutisticC compared to NonautisticC (difference = −0.069, t(78) = −2.21, $p = 0.030$), whereas no group-level differences were observed in adults (difference = 0.018, t(78) = 0.66, $p = 0.511$). In *Other*, regularity did not show any significant main effects of group ($F(1,78) = 2.95$, $p = 0.090$) or age ($F(1, 78) = 0.93$, $p = 0.339$), nor their interaction ($F(1,78) = 1.85$, $p = 0.178$). For *F0*, no effect of age ($F(1,78) = 2.96$, $p = 0.089$) or interaction ($F(1,78) = 0.01$, $p = 0.918$) was observed; however, a main effect of group ($F(1,78) = 6.43$, $p = 0.013$) indicated that movements were unintentionally executed by the actor at a lower frequency for autistic than non-autistic participants (difference = -0.035, t(78)= -2.54, $p < 0.01$).

Effects of age, group and autistic traits on CKC levels in Self

Only significant effects are reported below across the identified local maxima, specifying the seed regions in which they were observed. See Table 3 for coordinates. As mentioned in the main text, all *p*-values reported were corrected for multiple comparisons using the Benjamini–Hochberg FDR procedure.

At *F0* no significant effects of group, age, or their interaction were observed across both sensorimotor regions and IFG (all FDR corrected *p*s >0.05).

At *F1*, a robust main effect of age was consistently observed across all local maxima including bilateral M1 and bilateral IFG, with adults showing higher coherence than children (all *p*s < 0.001).

In addition, a significant age-by-group interaction emerged in right M1 cortex (F(1,78) = 9.29, $p = 0.024$). Post-hoc comparisons revealed that coherence was higher in AutisticA compared to AutisticC (difference = 0.22, t(78) = 5.78, $p < 0.001$), and higher in AutisticA compared to NonautisticA (difference = 0.11, t(78) = 3.10, $p = 0.003$). In contrast, the age-related increase in the non-autistic group was weaker and did not reach significance (difference = 0.061, t(78) = 1.67, $p = 0.098$). No significant difference was observed between AutisticC and NonautisticC (difference = −0.015, t(78) = −0.63, $p = 0.531$).

In both adults and children, GLMs including AQ and SCQ, respectively, did not reveal any significant effects of group, AQ or SCQ, nor their interaction (all *p*s > 0.05).

Effects of age, group and autistic traits on CKC in Other

Consistent with the analysis of the *Self* condition, results are reported for significant effects only across the identified local maxima, with p-values corrected. See Table 3 for coordinates.

At *F0*, a significant main effect of age emerged in right V1 cortex ($F(1,78) = 11.6$, difference = 0.090, $p = 0.007$) and right superior parietal lobule (SPL) ($F(1,78) = 6.34$, difference = 0.050, $p = 0.049$), with adults showing higher coherence than children.

At *F1*, age-related increases in coherence were also evident in right S2 ($F(1,78) = 22.9$, difference = 0.087, $p < 0.001$), left M1 cortex (F(1,78) = 20.3, difference = 0.078, $p < 0.001$), and two right M1 cortex maxima ($F(1,78)=24.2$, difference = 0.077 and $F(1,78) = 14.4$, difference = 0.059, both $p < 0.001$). In one right M1 maxima, coherence additionally differed

by diagnostic group ($F(1,78) = 9.51$, $p = 0.012$; Supplementary Figure 1), with a significant age-by-group interaction ($F(1,78) = 16.1$, $p = 0.004$), driven by higher coherence in AutisticA compared with both AutisticC (difference = 0.122, $t(78) = 5.38$, $p < 0.001$) and NonautisticA (difference = 0.110, $t(78) = 5.29$, $p < 0.001$). No age-related effects were observed within the non-autistic group.

In adults, GLMs including AQ only revealed a significant group-by-AQ interaction at the right S2 ($F(1,40) = 7.27$, $p = 0.04$) at *F1*; with increased CKC levels with increasing AQ in AutisticA, but decreased CKC levels with increasing AQ in NonautisticA (Supplementary Figure 2).

In children, GLM models including SCQ scores did not reveal any significant effects (all *p*s > 0.05).

Effects of condition, age, and group on CKC of common sensorimotor regions

To compare CKC between action execution (*Self*) and observation (*Other*), coherence values were extracted from corresponding sensorimotor regions identified in each condition.

For F0, peaks were located within the sensorimotor cortex, encompassing primary motor and somatosensory areas, in both conditions (Self, right [33, −8, 54], left [−41, −15, 48]; Other, right [34, −9, 63], left [−30, −18, 45]). For F1, peaks were similarly identified within bilateral motor cortex (Self, right [44, −11, 52], left [−31, −10, 61]; Other, right [37, −17, 48], left [−32, −14, 37]).

Coherence values were analyzed using LMM with condition, hemisphere, age, and group as fixed effects and subject as a random intercept.

At *F0*, a significant main effect of condition was observed ($F(1, 234) = 66.93$, $p < 0.001$), without significant effect of hemisphere ($F(1, 234) = 0.33$, $p = 0.567$), group ($F(1, 78) = 0.031$, $p = 0.862$), age ($F(1, 78) = 2.75$, $p = 0.102$), nor any of their interactions (all *p*s> 0.05).

Post-hoc analysis revealed higher CKC values during action execution compared to observation (self vs other: difference = 0.10, t(234) = 8.18, $p > 0.01$).

At *F1*, significant effects of condition ($F(1,234) = 72.9$, $p < 0.001$), age ($F(1,78) = 50.0$, $p < 0.001$), age-by-condition ($F(1,234) = 10.8$, $p = 0.001$) and group-by-age interactions ($F(1,78) = 7.50$, $p = 0.008$) were observed. Group didn't show a significant main effect ($F(1, 78) = 1.55$, $p = 0.217$), nor did other interactions (all ps > 0.05). Post-hoc tests again indicated higher CKC during action execution than observation (difference = 0.08, t(234) = 8.54, $p < 0.01$), as well as higher CKC in adults compared to children (difference = 0.11, t(78) = 7.07, $p < 0.01$). The differences between *Self* and *Other* conditions was larger in adults (difference = 0.11, t(234) = 8.81, $p < 0.01$) than in children (difference = 0.05, t(234) = 3.54, $p < 0.001$). AutisticA also showed higher coherence than NonautisticA (difference = 0.06, t(78) = 2.97, $p = 0.004$), while no significant group differences were found in between AutisticC and NonautisticC (difference = 0.02, t(78) = 1.01, $p = 0.318$).

Figures



Figure 1

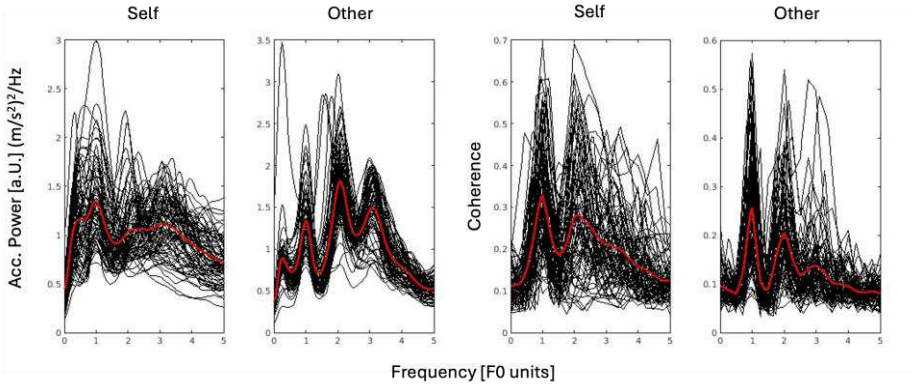
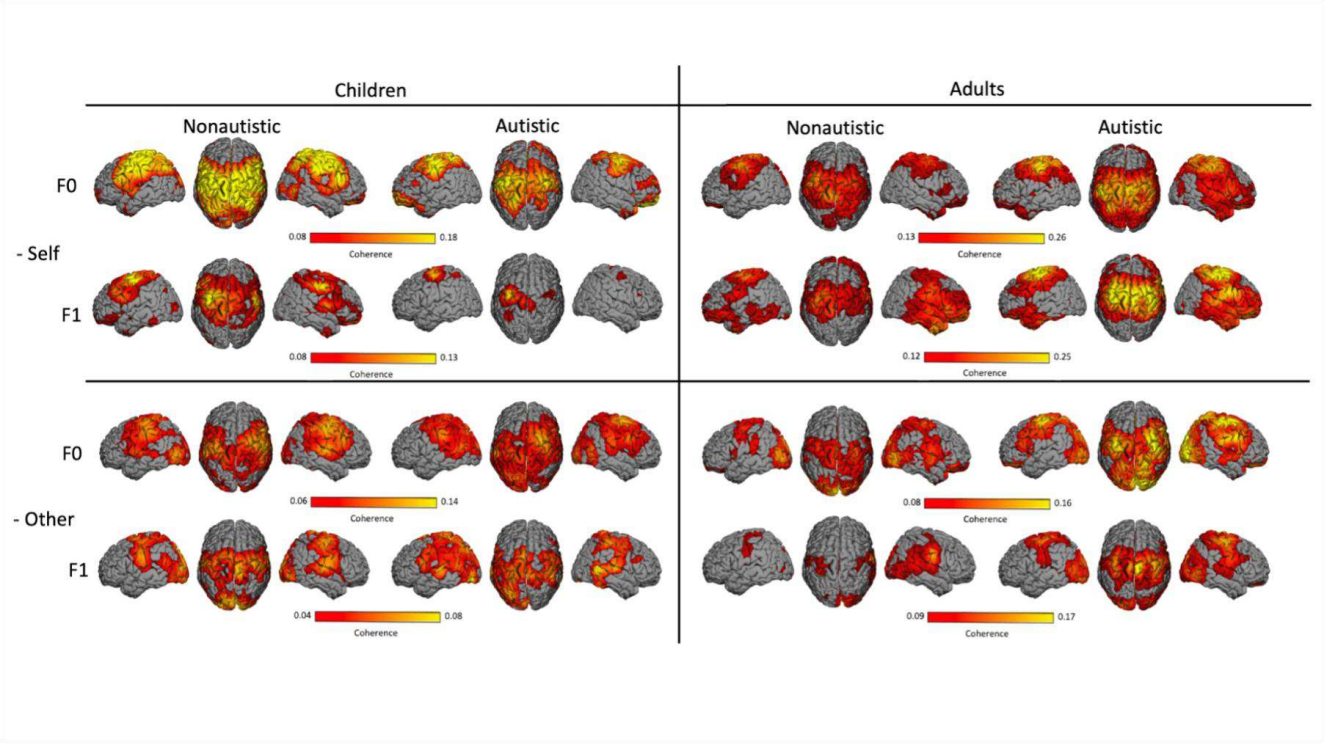


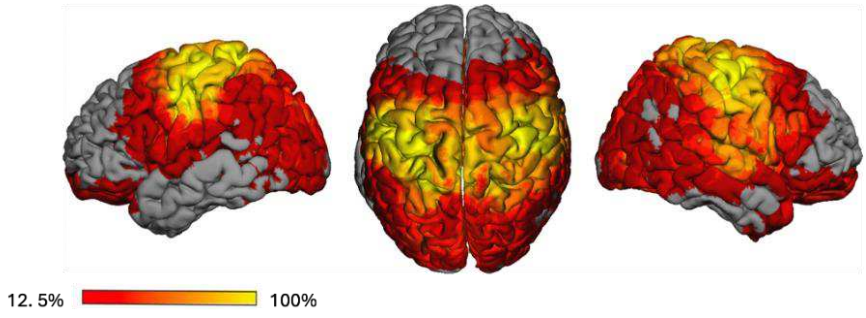
Figure 2



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676 **Figure 3**

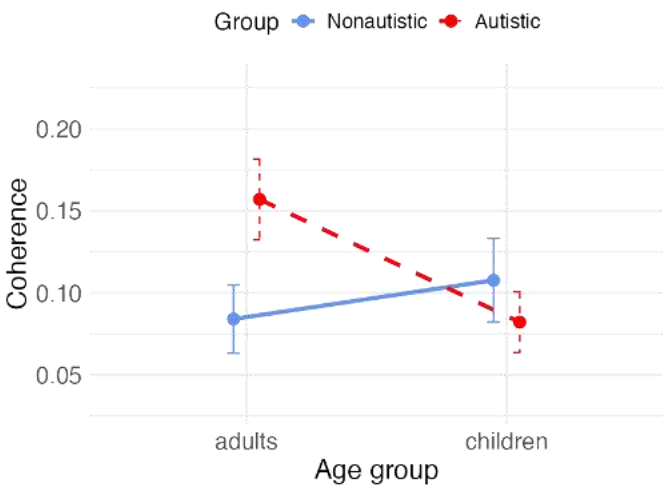
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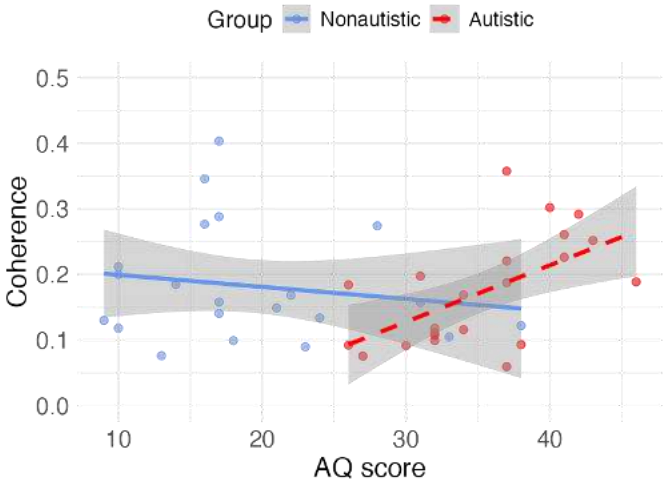
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684 **Supplementary Figure 2**

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Figures' Legends

Figure 1. *Experimental paradigm.*
Top, left. *Self*. Neuromagnetic activity (MEG) was recorded while participants repeatedly pinched green objects with the right thumb and index finger and placed them into a plastic pot positioned on the left. **Top, right.** *Other*. Illustration shown from the participant's viewpoint, in which the experimenter executed the same repetitive hand movements. **Bottom.** A single movement cycle, comprising reaching the green object, pinching, moving towards the plastic pot, and dropping the object. The same movements were repeated during 5 minutes at ~1 Hz (instruction given to the participants) and were similar across *Self* and *Other*. Movement kinematics was measured using a three-axis accelerometer attached to the right index finger of the participant (*Self*) or the experimenter (*Other*).

Figure 2. *Accelerometer power and sensor-level CKC spectra for Self and Other.*
Each plot presents a trace for each participant in black and the group average in red. Frequencies are expressed in *F0* units (i.e., 1 corresponds to the individual movement frequency, 2 to its first harmonic, etc). Note that *F0* (and *F1*) might correspond to different frequency bins in different participants.

Figure 3. *Corticokinematic coherence during Self and Other*.
Age-by-group-level CKC source maps displayed for non-autistic and autistic children (**Left**), as well as non-autistic and autistic adults (**Right**) across conditions (Self, **Top**; Other, **Bottom**) at both fundamental frequency (*F0*) and its first harmonic (*F1*).

Figure 4. *Overlapping cortical regions engaged during Self and Other.*
Spatial overlap of brain areas showing significant coupling between their neural activity and movement kinematics during both Self and Other, across *F0* and *F1*, and across all participants (adults and children, autistic and non-autistic). Each voxel reflects the proportion of maps (out of 8; i.e., Self–Other overlap computed separately for each group and frequency) showing significant CKC in both conditions; thus, the minimum non-zero value corresponds to 12.5% (1/8) and the maximum to 100% (8/8). Color scale (red to yellow) represents increasing overlap (i.e., higher consistency across maps), whereas grey regions indicate absence of overlap (i.e., no significant CKC in either condition).

Supplementary Figure 1. *Significant age-by-group interaction at the right primary motor (M1) cortex at F1 in Other.*
CKC level at right M1 cortex increased from childhood to adulthood in autistic participants (red dashed line),but remained relatively stable across age in non-autistic participants (blue solid line). As a result, autistic adults exhibited higher CKC than both autistic children and non-autistic adults. Error bars represent 95% confidence intervals.

Supplementary Figure 2. *Significant interaction between Autism Quotient (AQ) and adult groups in the right secondary somatosensory (S2) cortex at F1 in Other*.
Estimated marginal means (EMM) showed opposite AQ-related trends across groups: CKC increased from low to high AQ in autistic adults (EMM : mean−1SD → mean+1SD, 0.011 → 0.195) (red, dashed line) and decreased in non-autistic adults (0.206 → 0.136) (blue, solid line). Shaded areas represent the 95% confidence intervals of the fitted regression lines.

Table 1

	Adults			Children		
	Autistic	Nonautistic	Test (p)	Autistic	Nonautistic	Test (p)
N	22	23		17	20	
Gender	7M,15F	9M,14F	$\chi^2(1) = 0.26$, $p = 0.61$	12M,5F	12M,8F	$\chi^2(1) = 0.45$, $p = 0.50$
Age	27.9(4.6)	25.3(4.7)	$t(43) = 1.89$, $p = 0.07$	10.6(1.5)	10.2(1.6)	$t(35) = 0.87$ $p = 0.39$
Handness	93.6(10.2)	92.3(13.7)	$t(43) = 0.34$, $p = 0.73$	91.4(11.2)	94.7(9.50)	$t(35) = -0.93$, $p = 0.36$
IQ tot	111(15.5)	108(12.7)	$U(42) = 182$, $p = 0.16$	107(12.9)	122(7.9)	$t(35) = -4.41$, $p < 0.001$
VCI	113(14.2)	112(15.3)	$t(42) = 0.71$, $p = 0.48$	104(17.6)	123(9.53)	$t(35) = -4.11$, $p < 0.001$
DCDQ	NaN	NaN	NaN	45.6(11.6)	67.2(12.5)	$U = 35$, $p < 0.001$
ADOS	Module 4 (n=21): 10.3(4.14)	NaN	NaN	Module 1 (n=4):12.3(4.3) Module 2 (n=3): 10(0.8) Module 3 (n=5): 9(1.3) Module 4 (n=1) :14 Module unknow (n=3): 18, 14, 10	NaN	NaN
AQ	35.2(5.6)	19.0(7.9)	$t(42) = 7.88$, $p < 0.001$	NaN	NaN	NaN
EQ	28.9(12.3)	43.4(15.0)	$t(42) = -3.50$, $p = 0.002$	NaN	NaN	NaN
SCQ	NaN	NaN	NaN	11(5.42)	1.9(1.37)	$U = 10.0$, $p < 0.001$
STAI	16.1(6.3)	11.4(4.7)	$U(43) = 138$, $p = 0.009$	12.0(3.6)	9.3(2.6)	$t(30) = 2.48$, $p = 0.02$
SES	6.5(1.9)	7.5(1.5)	$t(40) = -1.93$, $p = 0.06$	11.4(2.6)	14.1(2.15)	$t(34) = -3.42$, $p = 0.002$

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Table 2

Condition	AutisticA (n=22)	NonautisticA (n=23)	AutisticC (n=17)	NonAutisticC (n=20)
Self	22 (100%)	23 (100%)	17 (100%)	20 (100%)
Other	22 (100%)	23 (100%)	15 (88%)	17 (85%)

Supplementary Table 1

SELF

F0			F1		
MNI	Coh	Area	MNI	Coh	Area
[-41, -15, 48]	0.2267	left S1	[-31, -10, 61]	0.1871	left M1
[-9, -30, 63]	0.2250	left M1	[-3, -21, 75]	0.1678	left M1
[33, -8, 54]	0.2140	right M1	[44, -11, 52]	0.1570	right M1
[44, 19, 16]	0.1638	right IFG	[51, 9, 22]	0.1481	right IFG
[-51, 22, 24]	0.1423	left IFG	[56, 1, 21]	0.1479	right M1
			[55, 24, 0]	0.1353	right IFG
			[-54, 8, 28]	0.1282	left IFG
			[7, -92, -4]	0.1023	right V1

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OTHER

F0			F1		
MNI	Coh	Area	MNI	Coh	Area
[5, -83, -1]	0.1425	right V1	[51, -10, 19]	0.1081	right S2
[34, -9, 63]	0.1352	right M1	[-32, -14, 37]	0.1012	left M1
[-30, -18, 45]	0.1278	left M1	[37, -17, 48]	0.0999	right M1
[44, -10, 12]	0.1217	right S2	[4, -23, 77]	0.0960	right M1
[56, 9, 31]	0.1184	right M1			
[18, -48, 73]	0.1078	right SPL			
[-1, -35, 74]	0.1058	left M1			

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Tables' Legends

Table 1. *Descriptive information.*
Data are reported as follow: mean (standard deviation, SD).
Handedness scores were obtained using the Edinburgh Handedness Inventory (41). Values ≥ 40 indicate a right-handed profile; **IQ tot**: intellectual quotient calculated via Wechsler Adult Intelligence Scale, IV Edition (WAIS-IV)(40) for adults and Wechsler Intelligence Scale for Children - Fifth Edition (WISC-V) (39)for children; **VCI**: Verbal Comprehension Index of WAIS-IV; **DCDQ**: Developmental Coordination disorder Questionnaire – French version (91); **ADOS**: Autism Diagnostic Observation Schedule-Generic (86). Scores are the results of communication + social Interaction and are shown per Module. For three participants module was unkown*;* **AQ**: Autism Spectrum Quotient (87); **EQ**: Empathy Quotient(90); **STAI**: short version (6 items) of State-Trait Anxiety Inventory (89); **SES**: Socio-Economic Score assed by an adaptation of the family affluence scale (92).
Data for hearing, cognitive and linguistic evaluation, as well as AQ and EQ is missing for one NonautisticA participant that never reported impairments in hearing performance, and had completed higher education, including university and a master's degree, without any suspicion of autism. ADOS is missing for one AutisticA participant that received medical diagnosis after clinical evaluation and for four AutisticC for which this data was unavailable.

Table 2. *Significant sensor-space CKC.*
Number (and percentage) of autistic adults (AutisticA), non-autistic adults (NonautisticA), autistic children (AutisticC), and non-autistic children (NonautisticC) showing statistically significant CKC (surrogate data-based statistics) at least one sensor during the Self and Other conditions, considering the principal component across both movement frequencies (F0 and F1).

Supplementary Table 1. *Source-space CKC local maxima.*
MNI coordinates and coherence values (Coh) for significant local maxima and respective localization (Area) (according to MNI coordinates) on grand average maps of *Self* and *Other* condition at F0 and F1. Anatomical labels are reported (Area) according to following nomenclature (listed in alphabetical order): inferior frontal gyrus (**IFG**), primary motor cortex (**M1**), primary somatosensory cortex (**S1**), secondary somatosensory cortex (**S2**), primary visual cortex (**V1**), superior parietal lobule (**SPL**).

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