Updated Table 1. Six-week stability (ICC) from Time 1 to Time 2 for primary and secondary pre-specified biomarkers.

Values provided are for the TD and ASD included sample and then for ASD subgroups by age and IQ. Updated after publication (2022-12-01) to include 95% confidence intervals in [lower bound-upper bound] and included subject numbers "*n*".

	TD	ASD	ASD <8.5y	ASD ≥8.5y	ASD IQ≤75	ASD IQ>75
Resting						
Slope	.539	.594	.575	.606	.622	.588
95% CI	[.383,.666]	[.501,.674]	[.434,.688]	[.473,.713]	[.306,.816]	[.487,.673]
n	98	217	109	108	24	193
Alpha	.667	.730	.707	.724	.725	.717
95% CI	[.541,.763]	[.662,.787]	[.598,.790]	[.621,.803]	[.458,.871]	[.641,.779]
n	98	217	109	108	24	193
Gamma	.453	.555	.562	.452	.476	.542
95% CI	[.282,.596]	[.455,.641]	[.419,.678]	[.290,.590]	[.099,.733]	[.434,.635]
n	98	217	109	108	24	193
Faces						
FU P100L	.687	.680	.799	.592	.895	.667
95% CI	[.573,.776]	[.595,.751]	[.709,.863]	[.446,.707]	[.649,.973]	[.576,.742]
n	107	186	89	97	10	176
FU N170L	.749	.662	.622	.644	.789	.650
95% CI	[.633,.829]	[.573,.736]	[.476,.734]	[.511,.747]	[.389,.942]	[.556,.727]
n	107	186	89	97	10	176
VEP						
VEP N1A	.697	.732	.640	.801	.403	.751
95% CI	[.576,.789]	[.659,.791]	[.498,.749]	[.719,.861]	[-0.51,.739]	[.678,.809]
n	32	190	87	103	15	175
VEP P100A	.743	.700	.752	.572	.820	.693
95% CI	[.644,.818]	[.624,.763]	[.654,.825]	[.431,.686]	[.587,.928]	[.612,.760]
n	105	212	102	110	18	194
BM						
BMS N200A	.097	.025	.103	.092	*	.026
95% CI	[-.109,.295]	[-.126,.176]	[-.113,.314]	[-.300,.125]		[-.129,.181]
n	92	157	75	82	6	151
BMS P3A	.149	.020	-.109	.211	*	.023
95% CI	[-.052,.340]	[-.137,.176]	[-.322,.116]	[-.006,.409]		[-.137,.182]
n	92	157	75	82	6	151

Table Key: TD=Typical development; ASD=Autism Spectrum Disorder; R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; BM=Biological Motion Experiment; FU=face upright; BMS=biological motion specificity effect; A=amplitude; L=latency.

*ICCs not included due to sample size $n<10$.

Please note, we found an errors in our earlier report of the VEP N1A. These values have been updated in the 12/1/2021 version as well.

SM General Methods

ABC-CT Protocol

The consortium is comprised of five implementation sites ("Sites": Boston Children's Hospital, Duke University, UCLA, University of Washington, and Yale University), who conducted a naturalistic study using clinician, caregiver and lab-based measures of social functioning, and a battery of conceptually related EEG and ET tasks. Participants with Autism Spectrum Disorder (ASD) or Typical Development (TD) were enrolled between 6yr0m and 11yr6m at Time 1 (Day 1), with a 2-day protocol (day 2 occurring from 1 to 14 days following day 1). Time 2 followed the baseline visit by 6 weeks (+/- 2 weeks). The actual temporal distance from T1 to T2 was M=41.49 days (SD 10.46; range 17 to 90) and did not differ by group ($F_{1,379}$=.06, p=.81; M_{ASD}=41.61 SD 10.99; M_{TD}=41.89 SD 9.08). Of note, some of these T2 return dates were "out of range" based on an expected range of 29 to 70 days. We did not remove these participants. Of note, 1 TD participant withdrew before T1 Day 2, and 4 participants with ASD and 5 with TD withdrew before T2 Day 2.

Participant Characteristics

Sample demographic characteristics are provided in ST1 for the total sample and for individuals contributing data to each of the EEG assays. Diagnosis of participants with ASD (n=280) was confirmed using the Autism Diagnostic Observation Schedule-2 (ADOS-2; (1)), the Autism Diagnostic Interview-Revised (ADI-R; (2)), and expert clinical judgment based on the Diagnostic and Statistical Manual, Fifth Edition (DSM-5) criteria for ASD (3). Participants with TD (n=119) were screened for the presence of ASD (via ADOS) or a sibling with ASD and parent report and for the presence of emotional and behavioral disorders via the Child and Adolescent Symptom Inventory, fifth edition (CASI-5; (4)). Age, based on the age at the T1 Day 2 EEG, is days for analysis but reported as years in tables.

Exclusionary criteria for both groups included known genetic or neurological conditions, history of epilepsy (or current use of anti-epileptic medications), clinically significant visual or auditory impairment, sensory-motor difficulties that would limit participation in standardized assessment, prematurity or pre/perinatal birth injury or brain damage, or severe environmental circumstances that would impact neural development. Because many children with ASD are on medications and current medication use is often not an exclusionary criterion for participation in clinical trials, medication use was not deemed exclusionary, although children needed to be on a stable medication protocol for 8 weeks prior to enrollment.

Cognition was assessed using the Differential Ability Scales, Second Edition (DAS-II; (5)). Verbal, nonverbal, and full scale "best IQ" were determined using standard deviation IQ scores when valid; if more than half the T-scores fell below the floor for a particular cluster, the full-scale ratio IQ score (mental age / chronological age) was used. The NEPSY-2 immediate Memory for Faces standard score (6) was acquired as an observational measure of social cognition.

Additional phenotypic characterization of social communication was achieved through parent report using the: Vineland Adaptive Behavior Scale-3 (VABS3) Socialization (Soc) and Communication (Com) Standard Scores (SS) (7); Social Responsiveness Scale (SRS-2) T-Scores for the Social Communication and Interaction (SCI) composite and the Restricted Interests and Repetitive Behaviors (RIRB) subscale (8); Pervasive Developmental Disorder Behavior Inventory (PDDBI) T-Scores for the Social Approach Behaviors domain and the Repetitive, Ritualistic, and Pragmatic Problem composite (REPRIT) (9).

Analytic Plan

Six-week stability. We assessed short-term stability of individual biomarkers in both groups from T1 to T2 using intraclass correlation (ICC) and 95% confidence intervals via two-way mixed models (where people effects are random and measures are fixed) with absolute agreement. In Table 1 and ST5, we provide values for subgroups within the ASD group: split by age (8.5 years) and IQ (full scale IQ of 75). *A priori* acceptability criteria defined ICCs ≥.5 as moderate and ≥.75 as high (34).

EEG Acquisition Standardization

All dependent variables of interest were pre-specified, including the component of interest and its region of interest. This report reflects these primary and secondary variables; a limited set of additional variables necessary to confirm the primary and secondary variables (e.g., peak amplitude was extracted to evaluate a primary peak latency variable; or theta power was abstracted to evaluate slope, which included the theta range). These represent only a fraction of the variables that can be abstracted from EEG data and the topographic pattern of EEG.

Protocol. Centrally, acquisition quality was reviewed and deemed valid if EEG cap placement was acceptable, the participant had completed 50% of the Resting experiment, the EEG equipment functioned according to specification, the recording file contained the expected experiment event markers, and there were no experimenter or participant behaviors that invalidated acquisition.

Resting Assay. Video presentation size was 9.3 cm (width) by 7.0 cm (height), with a visual angle of 8 degrees x 6 degrees. Video stimuli (Shutterstock: 3038821, 3041077, 3191017, 4003732, 4779302, 8398420) consisted of non-social, abstract moving images played forward for 15 seconds and then in reverse for 15 seconds.

We note that the "resting" conditions vary across studies and may include eyes open directed toward a fixation point, eyes open viewing videos and eyes closed, all of which may have different implications for brain functioning. We conceptualizing this assay as "resting" while engaging in calm viewing as in Neuhaus et al. (10), as calm viewing has been proposed to be evoke more reliable activation (e.g., (11)).

The analysis pipeline used the Batch EEG Automated Processing Platform (BEAPP (12)) consisting of: (1) format the MFF file for Matlab; (2) filter 1-100Hz; (3) down sampling from 1000Hz to 250Hz (to improve performance of independent components analysis/ICA); (4) implementation of the HAPPE module for artifact detection and rejection (13) including a reduction of the array to 18 channels (electrodes 9, 11, 22, 24, 33, 36, 45, 52, 58, 62, 70, 83, 92, 96, 104, 108, 122, 124; based on the 10-20 system and excluding Cz) to avoid overlearning during ICA, removal of 60 Hz line noise, rejection of bad channels, wavelet enhanced thresholding, ICA with automated component rejection, bad channel interpolation, and re-referencing to average; (5) segmentation into 1 second segments; (6) rejection of files based on HAPPE (12) quality metrics (set at 3 SD): >80% good channels remaining, <30%mean retained artifact probability, <35% median retained artifact probability, <84%percent of independent components rejected as artifact, and >32% percent of EEG signal variance retained after artifact removal. On the remaining valid participant files: (7) discard un-attended segments; (8) calculate the PSD using Hanning window on clean segments without high-amplitude artifact. Valid signal was defined as ≥20 seconds of usable data.

Faces Assay. Three stimuli conditions (72 trials per condition) were included: Female neutral faces from the NimStim Face Stimulus Set (07F, 13F, and 17F; (14)) presented upright and inverted and upright houses (Shutterstock ID 252868810, 150435080, 58015144, scaled to the dimension of the faces). A fixation crosshair was presented at 4.2 cm x 4.2 cm (3.8 degrees); the Faces and Houses were 11.3 cm (width; 12.3 degrees) by 14.3 cm (height; 9.3 degrees). The experiment was aligned with the EU-Aims LEAP faces experiment (15); an important difference is that Faces uses a fixation crosshair while the EU-Aim LEAP uses fixation icons.

The pipeline consists of: (1) PREP (16) to remove line-noise, re-reference to a robust average reference, and detection and interpolation of bad channels relative to this reference. (2) Bandpass filter at .1 to 30hz. (3) Segmentation 200 msec before and 500 msec after stimulus onset; removal of unattended trials. (4) Baseline correction using the 200 msec pre-stimulus interval. (5) Artifact detection using the EEGLAB (17) function *pop_artextval* with a threshold of 150 mV and a time window of -200 msec to 500 msec. A participant's data were included if they had ≥21 artifact free and attended face upright trials (out of 72 or ~30%).

Based on prior work by Neuhaus et al. (19) and Hileman et al. (20), we analyze the P100 and the N170 at lateral posterior temporal leads (and consistent with 21,22,23). Alternative regions of interest, including the left posterior temporal hemisphere and central occipital regions may result in different results.

Peak verification was done by visual inspection. The most common "failure" of the automatic peak identification program was to misidentify the component in cases wherein large oscillations obscured the components, when the component represented a wide or shallow peak, or when there were double peaks present. Further, because the automated algorithm identifies the P100 first, if there is a failure to identify the P100, the N170 would not be identified. Team members were trained (by SJW) on correct peak identification and qualitative descriptors of the ERP waveform. Twenty files from confirmation study were identified and consensus coded for peaks and a set of definitions were developed; 50 cases were coded by the team for training; ~30 visual examples with coding were included in the manual for training and reference. Two team members coded each waveform; reliability was compared and any discrepancies were resolved by JB or SJW.

For Faces, the peak of the P100 or N170 was confirmed when: (1) The maximal amplitude point within the pre-specified window has a slope = 0; (2) and an amplitude change of ≥2μV occurs within the window on either side of the peak; (3) the peak is the largest amplitude peak within the temporal window; (3) morphology of the ERP waveform is defined as Valid (baseline activity min / max amplitude range is <20μV from -100 to 0 msec; no oscillations are present and larger in amplitude than event-related activity; peaks are ≥2μV in relation to baseline and background activity). Individual exemplar waveforms are included in the derived results manuals. See Supplemental Materials References-Manuals.

VEP Assay. The VEP stimuli consist of 2 black and white checkerboards (20x20) displayed at 26cm x 26cm that reversed their phase (i.e., black to white and white to black) every 500 msec. A total of 100 trials (or phase reversals) was presented in 4 blocks of 50 trials for a total of 200 trials. The checkerboards had a mean luminance of 80cd/m2 and a contrast of 99%. A red circle with the same diameter as the length of the square check and was presented at the center of the checkerboards as a fixation point.

The post-acquisition processing of the VEP data used a similar pipeline to Faces except: Segmentation was -100 msec (baseline) to 300 msec (post stimulus), data were defined as usable if there were ≥60 artifact free trials (out of 200 or ~30%).

For VEP, the peak of the N1 and P100 was confirmed when: (1) The maximal amplitude point within the pre-specified window has a slope = 0; (2) and an amplitude change of ≥1μV (N1) or ≥2μV (P100) occured within the window on either side of the peak; (3) the peak is the largest amplitude peak within the temporal window; (3) morphology of the ERP waveform is defined as Valid (baseline activity min / max amplitude range is <20μV from -100 to 0 msec; no oscillations are present and larger in amplitude than event-related activity; peaks are ≥2μV in relation to baseline and background activity). Individual exemplar waveforms are included in the derived results manuals. See Supplemental Materials References-Manuals.

BM Assay. The stimuli were point light displays (black background with white dots) created from live motion capture data of coherent biological motion featuring an adult male walker (CMU MOcap database, http://mocap.cs.cmu.edu/). Scrambled motion animations were created by randomly selecting points from the biological motion displays and plotting trajectories. For each stimulus, 60 static frames were presented each for 17 msec to create the perception of a dynamic image. Images were displayed at 4 cm (width) x 7 cm (height), for a visual angle of 4.26 degrees x 6.18 degrees. For each stimulus, 60 static frames were presented each for 17 msec to create the perception of a dynamic image. Images were displayed at 4 cm (width) x 7 cm (height), for a visual angle of 4.26 degrees x 6.18 degrees.

The post-acquisition processing used the pipeline for Faces. A participant's data were included if they had ≥17 (out of 56 or ~30%) artifact free and attended segments per BIO and SCR condition.

For BM, the peak of the P100 and N200 was confirmed when: (1) The maximal amplitude point within the pre-specified window has a slope = 0; (2) and an amplitude change of ≥2μV (P100/N200) occurred within the window on either

side of the peak; (3) the peak is the largest amplitude peak within the temporal window; (3) morphology of the ERP waveform is defined as Valid (baseline activity min / max amplitude range is <20μV from -100 to 0 msec; no oscillations are present and larger in amplitude than event-related activity; peaks are ≥2μV in relation to baseline and background activity). As the P300 was based on the averaged response within the window of interest, no peak verification was completed. The P300 was only excluded if the overall waveform was invalid. Individual exemplar waveforms are included in the derived results manuals. See Supplemental Materials References-Manuals.

Analytic Plan

We conducted 5 sets of analyses for each assay as presented in the main manuscript. Analytic interpretations of the biomarker results were pre-specified in (18) and predictions are included in the below for construct performance and group discrimination.

Resting Assay. For group discrimination, we predicted that the slope would be more negative in the TD compared to the ASD group. For secondary variables, we predicted lower Alpha power and greater Gamma power in the ASD compared to the TD group.

Faces Assay. Construct performance was defined as a more negative and faster N170 amplitude response to upright faces compared to upright houses in the TD group, reflecting the "face specificity effect". For group discrimination, we predicted that the N170 latency to upright faces (primary) and the P100 latency to upright faces (secondary) would be faster in the TD than ASD group.

VEP Assay. For group discrimination, we predicted that the P100 amplitude (primary) would be more positive in amplitude and the N1 amplitude (secondary) would be more negative in amplitude in the TD than ASD group.

BM Assay. Construct performance was defined as a more negative N200 amplitude and a more positive P3 amplitude to biological motion compared to scrambled motion in the TD group. For group discrimination, we predicted that the TD group would show a negative N200 amplitude BMS and a positive P3 amplitude BMS (Biological Motion Specificity Effect; a greater, more negative N200A and more positive P3A to biological motion compared to scrambled motion), while the ASD group would show no differential amplitude between biological and scrambled motion.

SM Expanded Results Time 1

Resting Assay at T1

Acquisition at T1. Children with TD who were older provided more trials (r=.199, p=.037). In the TD group, slope was not related to age (r=-.048, p=.62) nor number of trials (r=-.020, p=.84); however Alpha power correlated with both age (r=.-352, p<.001) and number of trials (r=-.218, p=.02), and Gamma power correlated with age (r=-.251, p=.008), but not number of trials (r=-.126, p=.19).

Relation between age and number of trials for the ASD group are presented in Main Manuscript Table-5.

Group Discrimination at T1. While not included as primary or secondary outcomes, we include the other frequency bands that were included in our Slope calculation. When including covariates, neither Delta, Theta, nor Beta power differed by group (ST7).

Faces Assay at T1

Acquisition at T1. Children with TD who were older provided more trials (r=.301, p<.01). In the TD group, faster responses to faces than houses were related to being older (r=-.243, p<.01; but not amplitude r=.031, p=.75); neither were related to number of valid trials (amplitude r=-.043, p=.65; latency r=.121, p=.20).

To follow up on the TD relation for the face specificity effect N170, we provide the relationship for each stimulus independently: Neither the N170 amplitudes to upright faces nor upright houses were related to age (r=.125, p=.18; r=.119, p=.21, respectively); the number of valid trials was related to face N170 amplitude (r=-.187, p=.045) but not house N170 amplitude (r=.119, p=.21).

Construct Performance at T1. Although analysis of the T1 ASD group was not part of our prespecified analysis for construct performance, the ASD group partially demonstrated the N170 amplitude face specificity effect with a more negative N170 to upright faces than upright houses ($F_{1,206}$=147.13, p<.001), but not a faster response to faces than houses ($F_{1,206}$=3.12, p=.079).

Phenotype Correlations at T1. In exploratory analyses, greater P100 amplitude related to better adaptive skills and fewer autism behaviors (and when covarying for age and trials) (ST6).

VEP at T1

Acquisition at T1. At T1, 2 TD and 4 ASD participants (who had adequate attended artifact free trials) did not have a valid P100 peak per pre-specification and were excluded from the analyses. Not including those who withdrew or did not complete any part of the EEG battery, at T1, rates of acquisition were similar by order (3rd vs 4th position, ASD χ^2=2.6, p=.11; TD χ^2=.48, p=.49).

In the TD group, the number of trials related to age (r=.248, p<.01); all other relations were nonsignificant. Compared to the TD group, the ASD group had fewer data trials at T1 ($F_{1,347}$=29.88, p<.001). Experiment order was not significant ($F_{1,357}$=.013, p=.91) nor was the interaction between group and order ($F_{1,357}$=2.07, p=.15).

In relation to other variables abstracted from the VEP assay**,** in the TD group, neither the P100 amplitude or N1 amplitude were related to age (r=-.037, r=.077, ps>.43) nor number of trials (r=.062, r=-.069, ps>.48).

Group Discrimination at T1. Presented in ST7, the group x order interaction was significant. In post hoc analyses, the order effect was significant within the TD but not the ASD group (presented in Main Manuscript). The group effect was not significant within order 3rd ($F_{1,174}$=2.98, p=.09) nor order 4th ($F_{1,165}$=13.73, p=.39).

In a parallel exploratory analyses to the Faces assay, the P100 latency to checkerboards did not differ by group in analyses including covariates (ST5).

Biomotion at T1.

Acquisition at T1. Within group, sites did not differ in their inclusion rates (ASD χ^2=1.41, p=.84; TD χ^2=6.69, p=.15). Rates of acquisition also did not differ by order (ASD χ^2=2.07, p=.15; TD χ^2=.05, p=.49). ASD children that did provide data (n=188) compared to those who did not (n=92) were older, had higher IQ, and better social and communicative adaptive skills (SM-Table 3).

In examining the numbers of valid trials, the ASD group compared to the TD group had fewer trials at T1 for both BIO and SCR ($F_{1,273}$=8.13, $F_{1,273}$=11.59, respectively, ps<.01). There were no differences based on order ($F_{1,273}$=0.08, $F_{1,273}$=.016, ps≥.78) or site ($F_{4,273}$=1.32, $F_{4,273}$=1.99, ps≥.10). Number of trials for BIO was related to age for the TD group (r=.286, p<.01), social and communicative adaptive skills (rs=.199, .284, ps<.05), and autism symptoms (SRS SCI r=.198, PDDBI REPRIT r=-.331, ps<.05). For the ASD group, number of trials was related to age (r=.249, ps<.01) and IQ (r=.180, p<.05*; r*=.230*, p*<.01).

Construct Performance at T1. In the TD group, neither the N200A or P3A BMS effect was related to age (*r*s=-.055 to .096) or number of trials (*r*s=-.105 to .130).

The BMS effect was not observed in the TD group: No significant stimulus effects were detected at the N200A ($F_{1,103}$=.08, p=.77) and the P3A demonstrated greater amplitude to scrambled than biological motion ($F_{1,103}$=4.79, p=.03, ηp^2=.044; Figure 4A). In exploratory analyses, the P100A did not differ between conditions ($F_{1,98}$=0.22, p=.64), but the response was faster to the scrambled images compared to biological motion at the P100L ($F_{1,99}$=7.00, p=.009) and N200L ($F_{1,103}$=12.87, p=.001).

In exploratory analyses examining the BMS effect, in the ASD group as in the TD group, we also found that the response to BIO vs SCR did not differ at the N200 amplitude ($F_{1,186}$=0.05, p=.83) nor P100A ($F_{1,180}$=0.02, p=.89), but the ASD group did show differentiation at the P100L ($F_{1,180}$=17.32, p<.001) and N200L ($F_{1,186}$=20.4, p<.001). The ASD group did not show differentiation of stimuli at the P3 amplitude ($F_{1,186}$=1.73, p=.19).

Six-Week Stability at T1. The BMS effect (Main Manuscript, Table-3) had very low stability, potentially due to its construct as a difference score. As seen in ST4, the response to BIO itself showed moderate stability and was similar to other ERP components. Data is not provided for the ASD ≤75 IQ group due to the small number of children who provided valid data at both timepoints (n=6).

Group Discrimination at T1. We hypothesized that the BMS effect would be larger in the TD group than the ASD group for the P3 amplitude (primary) and the N200 amplitude (secondary). There were no significant group differences as presented in ST8. Nor as in exploratory analyses for the P100 amplitude, P100 latency, and N200 latency.

When looking at stimulus responses independently, rather than in a difference score, we also found no significant group difference in the response to the P3 amplitude to BIO ($F_{1,289}$=0.01, p=.92), P3 amplitude to SCR ($F_{1,289}$=0.50, p=.48), N200 amplitude to BIO ($F_{1,289}$=0.02, p=.90), nor N200 amplitude to SCR ($F_{1,289}$=0.02, p=.90).

Phenotype Correlations at T1. Presented in ST9, we did not see any significant relations between behavior and the BMS effect (or the response to BIO).

SM Methods Time 2

Analytic Plan

Time 2 analyses are presented in the supplemental materials to check replication of findings related to acquisition and group discrimination.

Acquisition. Acquisition analyses are the same as conducted for T1.

Group discrimination. Similar to T1, we first ran an independent samples ANOVA with group as the between-subject variable and report partial eta squared (ηp^2) for effect size; if variances significantly differed between groups, we report Welch ANOVAs with omega squared (ω^2) for reporting effect size. We also include ANCOVAs with child age, number of trials, sex and full scale IQ as covariates. For BM and VEP, the contrast also included order and group by order. Note, IQ was not acquired at T2 due to the short interval between T1 and T2 (+6 weeks). Thus, the analyses used T1 full scale IQ as a covariate. The time between the T2 EEG and the T1 IQ measure was M=46.3 SD 11.5 (TD M=48.1 SD 10.9; ASD M=45.6 SD 11.7, $F_{1,381}$=3.74, p=.05).

SM Results Time 2

Resting EEG at T2

Acquisition at T2. As shown in ST10, 88% of the ASD sample and 88% of the TD sample provided valid data. The effect of site on the number of trials was significant at T2 in the ASD group ($F_{4,340}$=10.08, p=.02), with a difference of 6 seconds of data between the highest and lowest average sites.

Group Discrimination at T2. As shown in ST11 and as predicted, our primary outcome measure, Slope of the power spectrum, was more negative in the TD than ASD group at T2 as found at T1. When including covariates, the group differences remained significant in T2. Note, the adjusted analyses were not significant at T1.

Our secondary variables were Alpha and Gamma power. Alpha power did not differ by group at T2. Gamma frequency differed at T2 with greater Gamma power in ASD compared to TD as hypothesized (23); although not significant at T1 nor with covariates included at T2. Similar to T1, the other frequency bands (Delta, Theta, Beta) did not differ by group at T2 (ST11).

Faces at T2

Acquisition at T2. A shown in ST10, 77% of the ASD sample and 92% of the TD sample provided valid data. Sites did not differ at T2 in percentage of the sample providing valid usable data for analysis. Compared to the TD group, the ASD group had fewer trials for upright faces (ST11).

Group Discrimination at T2. As hypothesized, the ASD group compared to the TD group demonstrated significantly slower upright face N170 latency and upright face P100 latency (ST11). This pattern was consistent when using covariates and replicates the findings at T1.

VEP at T2

Acquisition at T2. A shown in ST10, 82% of the ASD sample and 91% of the TD sample provided valid data. Rates of acquisition were lower in the ASD group, but did differ by order (3rd vs 4th position, χ^2=5.3, p=.02), with greater loss in the 3rd than 4th position. There was no difference in the TD group by order (χ^2=0.23, p=.63).

Compared to the TD group, the ASD group had fewer data trials at T2 ($F_{1,317}$=28.50, p<.001) but this did not differ by site ($F_{4,317}$=1.07, p=.37) nor experiment order ($F_{1,317}$=0.07, p=.80).

Group Discrimination at T2. In unadjusted analyses, the P100 amplitude showed no group difference (ST11). Unlike T1, the interaction between group and order was not significant (unadjusted and adjusted Fs<1.68, p>.20).

Similar to T1, there were no differences in the N1 amplitude response by group, nor in adjusted analyses. The group x order interaction was not significant (unadjusted and adjusted *Fs≤3.28, ps≥.07)*

The P100 latency to checkerboards did not differ by group (T2 $F_{1,333}$=0.01, p=.93), nor in adjusted analyses (T2 $F_{1,330}$=0.46, p=.50); the interaction between group and order was also not significant (unadjusted and adjusted *Fs*<1.13, *p*s≥.29).

Biomotion at T2.

Acquisition at T2. A shown in ST11, 71% of the ASD sample and 86% of the TD sample provided valid data. Sites did not differ in their inclusion rates (ASD χ^2=2.51, p=.64; TD χ^2=4.44, p=.35). Rates of acquisition decreased when BM was run in the 4th position for the ASD group (ASD χ^2=4.21, p=.04) but not the TD group (TD χ^2=1.55, p=.21). Note: VEP rates also were lower when run 3rd, suggesting that for the ASD group, this order (Resting, Faces, VEP, BM) performed "worse" than the other order (Resting, Faces, BM, VEP).

In examining the numbers of valid trials, the ASD group compared to the TD group had fewer trials for BIO and SCR ($F_{1,280}$=11.10, $F_{1,280}$=12.78, respectively; *p*s≤.001). There were no differences based on order ($F_{1,280}$=012, p=.73). Performance did differ by Site ($F_{1,280}$=3.64, p=.007); the mean difference between the site with the highest vs lowest average number of trials was 5 trials.

Group Discrimination at T2. Our primary variables was the biological motion specificity effect (biological motion minus scrambled motion / BMS) for the P3 amplitude and our secondary variable was the same construct for the N200 amplitude. There were no group differences for BMS N200 amplitude ($F_{1,298}$=0.09, p=.75) but there was for P3 amplitude ($F_{1,298}$=4.074, p=.04, ηp^2=.013).

Presented in ST12 are the results when accounting for group, order, group x order, and covariates of age, number of biological motion trials, and T1 full scale IQ. The BMS was larger in the TD group compared to the ASD group; despite this effect, the ASD group showed a significant discrimination with a larger response to SCR than BIO, and this effect was not significant in the TD group.

Supplemental Discussion

BM Assay.

In reviewing our biological motion assay results, while robust biological motion versus scrambled motion ERP differences in are found in adults (24-30) or when using MEG (31-33), samples with youth are less consistent and prior research is methodologically inconsistent in variable abstraction. First, there are significant age related effects in the components of interest. Hirai et al. (34), who included 50 children aged 7 to 14 years, noted significant developmental effects with greater amplitude P1s in children (≤11 years) compared to 13 year olds and adults, and larger N1 amplitudes (similar to our N200) in the 7 year-olds compared to all other age groups (≥9 years). Biological vs. scrambled motion differentiation was only found in the P1 in the 7 year-old group (n=10) at posterior-temporal lateral hemispheric leads; N1 differentiation was found across the sample at occipital leads; and the N2 was found to be greater (or more negative) to Bio than Scr at posterior-temporal lateral leads with no noted age effects. However, in samples that included ASD (n=12, mean age=14 years range 8 to 22) and TD youth, none of the responses were found to differentiate motion type (35). Kroger et al. (36) found group differences in the P100 amplitude and a trend difference for the P400 amplitude in children with ASD (N=17, mean age=11.9 years) compared to TD children, but no motion differences were identified for any of the components.

Several possible reasons for failing to replicate previous findings exist. First, our sample included a younger age range than the previous reports (6 to 11 year-olds), with no inclusion of adolescents (as in 27, 34, 36). It is possible that the

response is more robust in older samples. Second, sample size is also a consideration when comparing previous results, as our sample (N_{ASD}=188; N_{TD}=105) far exceeds that of previous reports and while heterogeneity is a known concern in ASD, there may be heterogeneity in TD samples as well. Third, the BM assay was run 3rd or 4th and thus children may have been less attentive and more fatigued in comparison to studies in which the BM assay was the *only* experiment collected. Fourth, we defined the N200 differently than in the previous Hirai papers. Hirai et al. (27) identified a N1 and N2 component that overlap in the timing range of our N200; as shown in their Figure 2 grand averages (specifically the responses at T6), the N1/N2 seem to be a part of a large-shallow component. Hirai et al. (24) first identified these as the N200 and N240, although Table 1 in their report suggests little amplitude variation between them (reported N200 M =-2.2μV; N240 M=-2.2μV). In developmental ERP reports, researchers have described a bifid N170, wherein there are two peaks within the same component window, with hypotheses suggesting this may reflect reflecting individual trial jitter, neural system reactivation, or summated scalp-activity from an additional system that becomes active on a different time scale (e.g., 37). However, when we reviewed the components at the individual level, there was not a consistent visible double negative peak that could be abstracted reliably. It is possible that alternative methods that attempt to reduce the waveform into component parts may be informative (e.g., pERPs, 38). Fifth, it is possible that 30-40 trials was "too low" to abstract a reliable averaged signal. Hirai et al. (34) who included children aged 7 to 14 also had a low good trial rate (56%, 50/89) and used a similar minimal number of minimal trials (30) for inclusion; however their average number of trials was 53 (in 7 and 9 year olds), while ours was 40.62 (ST9). Thus, it is possible that even with similar basic trial inclusion criteria, the signal to noise ratio is too low. Overall, the prior literature on ERP responses to biological motion is not consistent and includes: (a) a range of "peak-picking" approaches on both individually averaged waveforms and individually averaged difference waves; (b) use of mean amplitude measures of activity in overlapping, but not identical temporal windows; and; (c) mass-multivariate measures, which obviate the problem of identifying components by comparing activity on a sample-by sample basis. While these approaches demonstrate efficacy in identifying condition level (biological vs scrambled) differences, they highlight the challenge of pre-specifying a marker at the individual level, particularly across period of significant developmental change.

SM Time 1 Tables

ST1: Summary of participant demographics (A) and phenotypic characterization (B) at Time 1.
Mean and standard deviation are presented for phenotypic assessments for the full sample and subsets providing valid data for each assay.

Table 1A Time 1	ASD All	TD All	ASD R	TD R	ASD F	TD F	ASD VEP	TD VEP	ASD BM	TD BM
N total	280	119	242	110	214	116	237	114	188	105
n female	65	36	57	34	51	36	55	35	43	34
% female	23%	30%	24%	31%	24%	31%	23%	31%	23%	32%
Age in years at EEG	8.6 (1.6)	8.5 (1.6)	8.6 (1.6)	8.4 (1.6)	8.8 (1.6)	8.5 (1.6)	8.7 (1.6)	8.5 (1.6)	8.7 (1.6)	8.6 (1.6)
Income <$75000	93 34.1%	23 19.8%	74 31.4%	20 18.7%	69 33.3%	22 19.5%	76 32.9%	22 19.8%	61 33.5%	19 18.6%
Paternal Edu <Bachelors	110 40.4%	14 12.1%	93 39.7%	13 12.2%	81 38.6%	14 12.4%	88 38.4%	14 12.5%	74 40.2%	13 12.8%
Maternal Edu <Bachelors	100 35.7%	15 12.8%	86 35.5%	13 12.0%	70 31.7%	14 12.3%	82 34.6%	14 12.4%	66 35.1%	12 11.7%
Hispanic Ethnicity	52 18.5%	8 6.7%	35 16.4%	6 5.2%	43 17.8%	6 5.5%	38 16.0%	5 4.4%	30 16.0%	6 5.7%
Racial Minority	90 32.1%	21 17.7%	77 31.8%	16 14.6%	55 25.7%	18 15.5%	72 30.4%	19 16.7%	55 29.3%	18 17.1%
African American or Black	22 8%	4 3%	16 7%	2 2%	16 7%	4 3%	17 7%	4 4%	16 9%	4 4%
American Indian & Alaska Native	2 1%	0 0%	2 1%	0 0%	1 <1%	0 0%	2 3%	0 0%	1 1%	0 0%
Asian	15 5%	2 2%	14 6%	2 2%	12 6%	2 2%	13 5%	1 1%	12 6%	2 2%
Multi-racial	45 16%	14 12%	41 17%	11 10%	23 11%	12 10%	36 15%	13 11%	23 12%	12 11%
Other	6 2%	1 1%	4 2%	1 1%	3 1%	0 0%	4 2%	1 1%	3 2%	0 0%

Table 1B Time 1	ASD All	TD All	ASD R	TD R	ASD F	TD F	ASD VEP	TD VEP	ASD BM	TD BM
Full Scale IQ	96.6 (18.1)	115.1 (12.6)	97.7 (18.2)	115.7 (12.3)	99.9 (17.0)	115.4 (12.4)	98.6 (17.9)	115.5 (12.5)	99.7 (17.0)	115.5 (12.3)
Verbal IQ	96.0 (20.6)	116.3 (11.2)	96.7 (20.7)	116.2 (11.0)	99.7 (19.2)	116.4 (11.2)	98.1 (20.3)	116.5 (11.3)	99.7 (18.4)	116.2 (11.2)
Nonverbal IQ	97.5 (16.9)	112.2 (14.1)	98.6 (16.8)	113.0 (13.8)	100.1 (16.3)	112.5 (13.9)	99.0 (16.8)	112.6 (14.0)	99.9 (16.4)	112.7 (14.0)
ADOS CSS	7.7 (1.8)	1.6 (0.9)	7.6 (1.8)	1.6 (0.89)	7.6 (1.8)	1.6 (0.9)	7.6 (1.8)	1.6 (0.9)	7.6 (1.8)	1.6 (0.9)

VABS3 Soc SS	69.9 (16.1)	104.6 (9.2)	70.7 (16.0)	104.7 (9.1)	71.7 (15.4)	104.6 (9.2)	70.7 (16.2)	104.5 (9.3)	71.6 (14.7)	104.1 (9.3)
VABS3 Com SS	76.4 (15.1)	103.4 (9.2)	77.3 (15.4)	103.3 (9.2)	71.7 (15.4)	104.6 (9.2)	77.7 (14.9)	103.4 (9.2)	78.5 (14.0)	103.9 (9.0)
SRS-2 SCI T	72.7 (10.8)	42.5 (5.1)	72.2 (10.9)	42.7 (5.1)	71.5 (10.6)	42.6 (5.1)	72.3 (10.8)	42.7 (5.1)	72.0 (10.8)	42.4 (5.0)
SRS-2 RIRB T	73.7 (12.2)	44.0 (3.7)	73.6 (11.9)	44.0 (3.8)	72.8 (11.8)	44.0 (3.7)	73.3 (12.4)	44.0 (3.8)	73.4 (12.1)	43.9 (3.6)
PDDBI Soc App T	54.2 (9.3)	69.8 (3.0)	55.0 (8.9)	69.9 (3.1)	55.1 (8.8)	69.9 (3.0)	65.8 (8.6)	69.9 (3.1)	55.2 (8.3)	69.8 (3.1)
PDDBI REPRIT T	49.6 (11.5)	28.0 (2.6)	49.3 (11.8)	28.1 (2.7)	48.0 (10.9)	28.1 (2.6)	49.1 (11.5)	28.1 (2.6)	48.7 (11.3)	28.0 (2.4)
Face Memory SS	7.9 (3.7)	10.5 (3.5)	8.0 (3.7)	10.6 (3.5)	8.2 (3.5)	10.6 (3.4)	8.1 (3.7)	10.6 (3.4)	8.1 (3.6)	10.6 (3.5)

Key: TD=Typical development; ASD=Autism Spectrum Disorder; R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; BM=Biological Motion Experiment;
ADOS CSS=Calibrated severity score; VAB3=Vineland Adaptive Behavior Scales 3; Soc=Socialization; Com=Communication; SS=Standard Score; SRS=Social Responsiveness Scale; RIRB=Restricted Interests and Repetitive Behavior subdomain; SCI=Social Communication and Interaction Composite; T=T Score; PDDBI=PDD Behavioral Inventory; SocApp=Social Approach Behaviors Domain; REPRIT=Repetitive, Ritualistic, and Pragmatic Problems Composite.

ST2. Time 1 Summary of Signal Acquisition and Validity.
Loss of data and final included sample for each assay.

Time 1	**ASD R**	**TD R**	**ASD F**	**TD F**	**ASD VEP**	**TD VEP**	**ASD BM**	**TD BM**
No EEG	5	2	9	2	18	2	20	2
Acquisition Invalid	15	1	15	0	10	1	8	2
Signal Invalid	18	6	42	1	15	2	64	10
Valid Signal Acquisition rate	242 (86%)	110 (92%)	214 (76%)	116 (97%)	237 (85%)	114 (96%)	188 (67%)	105 (88%)
Difference in Site Acquisition rate	χ^2=.50 p=.97	χ^2=1.4 p=.84	χ^2=10.1 p=.04	χ^2=5.4p =.25	χ^2=1.4 p=.84	χ^2=6.7 p=.15	χ^2=5.6 p=.23	χ^2=4.7 p=.32
Differences in Order Acquisition rate	--	--	--	--	χ^2=2.1 p=.15	χ^2=0.48 p=.49	χ^2=0.2 0 p=.66	χ^2=1.0 p=.32

Key: TD=Typical development; ASD=Autism Spectrum Disorder; R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; BM=Biological Motion Experiment; No EEG=number of participants who were reported as withdrawn, the EEG protocol was not conducted, and/or the specific EEG assay was not conducted;
Acquisition Invalid=number of participants for whom the assay was reported as invalid due to child noncompliance, equipment error, and/or experimenter error;
Signal Invalid=number of participants for whom there were too few valid trials or the waveform component of interest failed morphology metrics.

ST3. At Time 1, comparison of ASD child characteristics for those who were included vs excluded for each experimental assay.
Analyses represent ANOVAs with group as a between subject variable.

Time 1 ASD	R	F	VEP	BM	ASD children included …
Age	$F_{1,275}$=3.51, p=.06	$F_{1,275}$=14.42, p<.001	$F_{1,275}$=9.43, p<.01	$F_{1,275}$=6.98, p<.01	…were older.
Full Scale IQ	$F_{1,278}$=6.81, p=.01	$F_{1,278}$=32.95, p<.001	$F_{1,278}$=20.20, p<.001	$F_{1,278}$=18.08, p<.001	…had higher IQ scores.
Verbal IQ	$F_{1,278}$=2.09, p=.15	$F_{1,278}$=33.86, p<.001	$F_{1,278}$=18.43, p<.001	$F_{1,278}$=19.81, p<.001	…had higher verbal IQ scores.
NV IQ	$F_{1,278}$=7.46, p<.01	$F_{1,278}$=21.99, p<.001	$F_{1,278}$=13.09, p<.001	$F_{1,278}$=11.57, p<.001	…had higher nonverbal IQ scores.
VABS3 Soc SS	$F_{1,277}$=4.50, p=.04	$F_{1,277}$=11.72, p=.001	$F_{1,277}$=3.86, p=.05	$F_{1,277}$=6.59, p=.01	…had better social adaptive skills.
VABS3 Com SS	$F_{1,277}$=5.17, p=.02	$F_{1,277}$=22.64, p<.001	$F_{1,277}$=10.31, p=.001	$F_{1,277}$=11.13, p=.001	…had better communication skills.
SRS SCI T	$F_{1,274}$=3.56, p=.06	$F_{1,274}$=11.23, p=.001	$F_{1,274}$=2.19, p=.14	$F_{1,274}$=1.95, p=.16	…had less autism social communication behaviors.
SRS RIRB T	$F_{1,274}$=.03, p=.60	$F_{1,274}$=5.02, p=.03	$F_{1,274}$=1.84, p=.18	$F_{1,274}$=.42, p=.52	…had less autism restrictive and repetitive behaviors.
PDDBI SocApp T	$F_{1,271}$=3.71 p=.055	$F_{1,268}$=7.49, p<.01	$F_{1,268}$=5.18, p=.03	$F_{1,268}$=6.63, p=.01	…had better social communication skills.
PDDBI REPRIT T	$F_{1,271}$=1.49 p=.22	$F_{1,271}$=17.18, p<.001	$F_{1,271}$=2.61, p=.11	$F_{1,271}$=3.32, p=.07	…had less repetitive, ritualistic, and pragmatic problem behaviors.

Key: R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; BM=Biological Motion Experiment; NV=Nonverbal; VABS3=Vineland Adaptive Behavior Scales-3; Soc=Socialization; Com=Communication; SS=Standard Score; SRS=Social Responsiveness Scale; RIRB=Restricted Interests and Repetitive Behavior subdomain; SCI=Social Communication and Interaction Composite; T=T Score; PDDBI=PDD Behavioral Inventory; SocApp=Social Approach Behaviors Domain; REPRIT=Repetitive, Ritualistic, and Pragmatic Problems Composite.

ST4. Time 1 Summary of the relation between the number of valid trials and ASD child characteristics.
Analyses reported using Pearson correlations.

Time 1 ASD	R Number of Trials	F FU Number of Trials	VEP Number of Trials	BM BIO Number of Trials	More valid trials were provided by children who …
Age	.372**	.331**	.347**	.249**	…were older.
Full Scale IQ	.331**	.223**	.269**	.180*	…had higher IQ scores.
Verbal IQ	.386**	.252**	.264**	.238**	…had higher verbal IQ scores.
NV IQ	.237**	.166*	.224*	.113	…had higher nonverbal IQ scores.
VABS3 Soc SS	.103	.100	.088	.083	
VABS3 Com SS	.166*	.045	.074	-.040	…had better communication skills.

SRS SCI T	-.101	-.042	-.026	.052	
SRS RIRB T	-.120	-.034	-.049	.032	
PDDBI SocApp T	.158*	.100	.060	.001	…had better social communication skills.
PDDBI REPRIT T	-.197**	-.186**	-.152*	-.084	…had less repetitive, ritualistic, and pragmatic problem behaviors.

Key: R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; BM=Biological Motion Experiment; FU=Face Upright; BM=Biological Motion Assay; BIO=Biological Motion Stimuli; NV=Non Verbal; VABS3=Vineland Adaptive Behavior Scales 3; Soc=Socialization; Com=Communication; SS=Standard Score; SRS=Social Responsiveness Scale; RIRB=Restricted Interests and Repetitive Behavior subdomain; SCI=Social Communication and Interaction Composite; T=T Score; PDDBI=PDD Behavioral Inventory; SocApp=Social Approach Behaviors Domain; REPRIT=Repetitive, Ritualistic, and Pragmatic Problems Composite.
*p<.05 or **p<.01

Updated ST5. Six-week stability (ICC) from Time 1 to Time 2 for additional variables derived from the assays and used to evaluate the primary and secondary variables.
See Main Manuscript Table 1 for primary and secondary variables & Supplemental Materials Updated Table 1 for confidence intervals. Updated after publication (2022-10-15) to include 95% confidence intervals in [lower bounds-upper bounds] and included subject numbers "*n*".

		TD	ASD	ASD <8.5y	ASD ≥8.5y	ASD IQ≤75	ASD IQ>75
				Resting			
Delta		.388	.660	.637	.632	.650	.650
	95% CI	[.206,.543]	[.578,.729]	[.511,.737]	[.504,.733]	[.341,.832]	[.560,.724]
	n	98	217	109	108	24	193
Theta		.510	.682	.663	.639	.730	.660
	95% CI	[.349,.643]	[.604,.747]	[.543,.756]	[.512,.739]	[.471,.874]	[.753,.733]
	n	98	217	109	108	24	193
Beta		.682	.753	.727	.776	.751	.728
	95% CI	[.561,.775]	[.689,.805]	[.625,.804]	[.689,.841]	[.508,.884]	[.654,.788]
	n	98	217	109	108	24	193
				Faces			
FU P100A		.703	.723	.750	.668	.816	.718
	95% CI	[.592,.787]	[.646,.785]	[.643,.828]	[.541,.765]	[.045,.960]	[.638,.783]
	n	107	187	85	97	10	176
FU N170A		.710	.739	.783	.679	.534	.752
	95% CI	[.599,.793]	[.665,.799]	[.684,.853]	[.556,.773]	[-.021,.854]	[.679,.810]
	n	107	186	89	97	10	176
				VEP			
VEP P100L		.594	.704	.704	.705	.475	.710
	95% CI	[.455,.705]	[.629,.766]	[.591,.790]	[.596,.788]	[.057,.760]	[.632,.773]
	n	105	212	102	110	18	194
				BM			

BIO P100A		.665	.665	.622	.695	*	.662
	95% CI	[.525,.771]	[.552,.738]	[.450,.749]	[.563,.793]		[.559,.745]
	n	82	149	68	81	6	143
BIO N200A		.766	.688	.704	.665	*	.679
	95% CI	[.667,.839]	[.591,.765]	[.565,.803]	[.524,.770]		[.580,.758]
	n	92	157	75	82	6	151
BIO P3A		.710	.673	.644	.699	*	.671
	95% CI	[.567,.807]	[.573,.753]	[.479,.763]	[569,.794]		[.568,.752]
	n	92	157	75	82	6	151

Key: TD=Typical development; ASD=Autism Spectrum Disorder; R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; BM=Biological Motion Experiment; BIO=biological motion; A=amplitude; L=latency;
*ICC value not calculated due to n<10.

ST6. Time 1 Group discrimination for additional biomarker values used to evaluate the primary and secondary variables. Analyses presented using unadjusted ANOVA. Follow up analyses use ANCOVA (in italics), with covariates for age, number of valid trials, sex and full scale IQ. For VEP, the follow up model also includes order and the group by order interaction.

Time 1	**ASD M(SD)**	**TD M(SD)**	**Main effect of group using …unadjusted ANOVA …*ANCOVA***
Resting			
Delta $\mu V^2/Hz$	0.339 (.160)	0.330 (.168)	$F_{1,350}$=0.24, p=.62, ηp^2=.001 *$F_{1,346}$=1.82, p=.18, ηp^2=.005*
Theta $\mu V^2/Hz$	0.320 (.142)	0.309 (.139)	$F_{1,350}$=0.42, p=.52, ηp^2=.001 *$F_{1,346}$=1.44, p=.23, ηp^2=.004*
Beta $\mu V^2/Hz$	0.094 (.041)	0.088 (.034)	$F_{1,350}$=1.46, p=.23, ηp^2=.004 *$F_{1,346}$=1.08, p=.30, ηp^2=.003*
Faces			
FU P100A µV	14.04 (6.3)	13.20 (7.2)	$F_{1,328}$=1.22, p=.27, ηp^2=.004 *$F_{1,324}$=.470, p=.49, ηp^2=.001*
FU N170A µV	1.13 (5.7)	-0.13 (5.8)	*$F_{1,328}$*=3.65, p=.06, ηp^2=.011 *$F_{1,324}$=0.92. p=.34, ηp^2=.003*
VEP			
VEP P100L msec	107.54 (17.3)	105.96 (16.6)	$F_{1,347}$=0.56, p=.44, ηp^2=.002 *$F_{1,3343}$=0.36, p=.57, ηp^2=.001*

Key: TD=Typical development; ASD=Autism Spectrum Disorder; R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; FU: face upright; A: amplitude; L: latency.

ST7. Correlations between Resting State EEG, Faces ERP, and VEP exploratory biomarkers and child behaviors in the ASD group at Time 1.
(See Main Manuscript Table 5.)

Time 1 ASD	**Trials**	**Age**	**Verbal IQ**	**NV IQ**	**Full IQ**	**Face Mem SS**

Resting						
Delta	-.318**	-.326**	-.193**	-.178**	-.212**	-154*
Theta	-.333**	-.356**	-.174**	-.175**	-.204**	-.148*
Beta	-.234**	-.103	-.245**	-.206**	-.251**	-.188**
Faces						
FU P100A	-.230**	-.207**	.018	.070	.047	-.029
FU N170A	-.101	-.021	-.027	-.016	-.023	-.052
VEP						
VEP P100L	-.005	-.023	-.009	.056	.030	-.050

Key: R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; BM=Biological Motion Experiment; NV=Non Verbal; Full=Full scale; VABS3=Vineland Adaptive Behavior Scales 3; Soc=Socialization; Com=Communication; SS=Standard Score; SRS=Social Responsiveness Scale; RIRB=Restricted Interests and Repetitive Behavior subdomain; SCI=Social Communication and Interaction Composite; T=T Score; PDDBI=PDD Behavioral Inventory; SocApp=Social Approach Behaviors Domain; REPRIT=Repetitive, Ritualistic, and Pragmatic Problems Composite.
*p<.05. **p<.01. Underline=significant at p<.05 when covarying for age and valid number of trials in the ASD group.

ST8. Time 1 Means and SD for the ASD and TD VEP N1 and P100 components.
Values are presented by group and by order in the protocol.

Time 1	**All**		**When 3rd**		**When 4th**	
VEP Inclusion	ASD N=237	TD N=114	ASD *n* =119	TD *n* =61	ASD n=118	TD n=53
VEP Trials	152.5 (36.5) 61 to 200	172.8 (21.0) 104 to 199	149.7 (39.0) 61 to 199	175.1 (19.7) 104 to 199	155.4 (33.6) 61 to 200	170.2 (22.3) 111 to 199
VEP N1A Inclusion	ASD *n*=225	TD *n* =108	ASD *n* =111	TD *n* =57	ASD *n* =114	TD *n* =51
VEP N1A µV	-4.4 (3.3)	-4.5 (3.5)	-4.0 (3.1)	-4.6 (3.9)	-4.8 (3.5)	-4.4 (3.1)
VEP P100 Inclusion	ASD *n* =237	TD *n* =114	ASD *n* =119	TD *n* =61	ASD *n* =118	TD *n* =53
VEP P100A µV	8.2 (4.4)	9.0 (4.1)	8.0 (4.3)	9.7 (4.3)	8.5 (4.5)	8.1 (3.7)
VEP P100L msec	107.5 (17.4)	106.0 (16.6)	108.0 (19.2)	105.2 (16.3)	107.1 (15.5)	106.9 (17.2)

Key: TD=Typical development; ASD=Autism Spectrum Disorder; VEP=Visual Evoked Potentials.

ST9. Time 1 Means, SD and Range for the ASD and TD BMS response. We present group effects using unadjusted ANOVA and adjusted analyses that included order and the group by order interaction, and covariates of age, trials, sex, and full scale IQ.

Time 1	**ASD M (SD)**	**TD M (SD)**	**ASD v TD, main effect of group using …unadjusted ANOVA …*ANCOVA***
BIO Trials	37.43 (10.14)	40.62 (8.1)	*$F_{1,257}$=8.69, p=.004, ω^2=.026

BMS P100A μV	0.03 (2.9)	-0.14 (3.0)	$F_{1,280}$=0.09, p=.76, ηp^2=.000 $F_{1,274}$=0.50, p=.48, ηp^2=.002
BMS P100L msec	5.10 (16.5)	5.2 (19.6)	$F_{1,280}$=0.002, p=.96, ηp^2=.000 $F_{1,274}$=0.50, p=.48, ηp^2=.002
BMS N200A μV	0.04 (3.2)	-0.10 (3.2)	$F_{1,291}$=0.12, p=.73, ηp^2=.000 $F_{1,285}$=0.008, p=.93, ηp^2=.000
BMS N200L msec	15.23 (46.2)	15.99 (45.7)	$F_{1,291}$=0.02, p=.89, ηp^2=.000 $F_{1,285}$=0.13, p=.72, ηp^2=.000
BMS P300A μV	-0.34 (3.5)	-0.71 3.4)	$F_{1,291}$=0.80, p=.37, ηp^2=.003 $F_{1,285}$=1.59, p=.21, ηp^2=.0006

BM=Biological Motion Experiment; BIO=Biological motion condition; BMS=Biological motion specificity effect.
*Because Levene's <.05, Welch ANOVA utilized for reporting *F*, df, and *p* and omega squared for reporting effect size.

ST10. Correlations between Biologic Motion biomarkers and child behaviors at Time 1 in the ASD group.

Time 1 ASD	Trials	Age	Verbal IQ	NV IQ	Full IQ	Face Mem SS
BIO Trials	--	.249 **	.238 **	0.113	.180 *	.200 **
BMS P100A	-.011	.079	-.067	-.033	-.039	.017
BMS P100L	.021	.091	<u>.256</u> **	<u>.194</u> **	<u>.254</u> **	.143
BMS N200A	-.023	-.040	-.048	-.02	-.017	.014
BMS N200L	.052	-.007	-.030	-.036	-.04	-.024
BMS P3A	.029	.006	-.043	-.068	-.045	-.070

<u>Key:</u> BIO=Biological Motion Condition; BMS=Biological Motion Specificity effect; Face Mem=NEPSY immediate Face Memory.
*p<.05; **p<.01; Underlined=significant at p<.05 when covarying for age and number of trials in the ASD group.

ST11 Time 2 Signal Acquisition and Validity. Loss of data and final included sample for each assay.

Time 2	ASD R	TD R	ASD F	TD F	ASD VEP	TD VEP	ASD BM	TD BM
No EEG	9	8	14	8	22	8	23	8
Acquisition Invalid	13	4	10	2	15	2	15	1
Signal Invalid	13	2	41	0	14	1	44	8
Valid Signal Acquisition Rate	245 88%	105 88%	215 77%	109 92%	229 82%	108 91%	198 71%	102 86%
Difference in Site Acquisition Rate	χ^2=2.8 p=.59	χ^2=3.2 p=.53	χ^2=4.8 p=.31	χ^2=3.7 p=.45	χ^2=1.0 p=.90	χ^2=1.2 p=.88	χ^2=2.5 p=.64	χ^2=4.4 p=.35
Difference in Order Acquisition Rate	--	--	--	--	χ^2=5.3 p=.02	χ^2=0.23 p=.63	χ^2=4.2 p=.04	χ^2=1.5 p=.21

Key: TD=Typical development; ASD=Autism Spectrum Disorder; R=Resting; F=Faces; VEP=Visual Evoked Potentials; BM=Biological Motion; No EEG=number of participants who were reported as withdrawn, the EEG protocol was not conducted, and/or the specific EEG assay was not conducted; Acquisition Invalid=number of participants for whom the assay was reported as invalid due to child noncompliance, equipment error, and/or experimenter error;
Signal Invalid=number of participants for whom there were too few valid trials or the waveform component of interest failed morphology metrics.

ST12. T2 Discriminant Validity. Presented first are the analysis results using the unadjusted ANOVA (with group) and then the follow up in italics using an ANCOVA with group covarying for T2 age, T2 number of trials, and T1 full scale IQ. For VEP and BM, order and the group x order interaction are also included in the adjusted analyses. Values are M (SD).

Time 2	**ASD**	**TD**	**Unadjusted ANOVA** ***ANCOVA***
		Resting	
R Trials	144.42 (26.9) 40 to175	160.41 (10.7) 101 to 175	*$F_{1,357}$=63.40, p<.001, ω^2=.151 --
Slope μV^2/Hz	-1.269 (.146)	-1.335 (.140)	$F_{1,348}$=15.48, p<.001, ηp^2=.043 *$F_{1,345}$=7.57, p<.01, ηp^2 =.021*
Delta μV^2/Hz	0.336 (.164)	0.309 (.135)	$F_{1,348}$=2.13, p=.15, ηp^2=.006 *$F_{1,3456}$=0.91, p=.34 ηp^2=.003*
Theta μV^2/Hz	0.314 (.141)	0.290 (.125)	$F_{1,348}$=2.21, p=.14, ηp^2=.006 *$F_{1,345}$=0.89, p=.35, ηp^2=.003*
Alpha μV^2/Hz	0.255 (.115)	0.253 (.106)	$F_{1,348}$=0.02, p=.88, ηp^2=.000 *$F_{1,346}$=1.72 p=.19, ηp^2=.005*
Beta μV^2/Hz	0.093 (.044)	0.085 (.038)	$F_{1,348}$=2.89, p=.09, ηp^2=.008 *$F_{1,345}$=0.42, p=.52, ηp^2=.001*
Gamma μV^2/Hz	0.026 (.015)	0.022 (.011)	$F_{1,348}$=7.50, p=.006, ηp^2=.021 *$F_{1,345}$=139, p=.27, ηp^2=.003*
		Faces	
FU Trials	46.21 (13.05) 21 to 70	52.48 (11.04) 21 to 71	*$F_{1,252}$=20.56, p<.001, ω^2=.057 --
FU P100A µV	14.48 (6.37)	13.30 (5.80)	$F_{1,322}$=2.63, p=.11, ηp^2=.008 *$F_{1,319}$=2.13, p=.15, ηp^2=.007*
FU P100L msec	121.98 (15.89)	118.12 (14.25)	$F_{1,322}$=4.56, p=.03, ηp^2=.014 *$F_{1,319}$=3.68, p=.06, ηp^2=.011*
FU N170A µV	1.71 (5.66)	0.63 (5.64)	$F_{1,322}$=2.63, p=.11, ηp^2=.008 *$F_{1,319}$=1.73, p=.19, ηp^2=.005*
FU N170L msec	206.36 (35.99)	191.31 (26.48)	$F_{1,322}$=14.94, p<.001, ηp^2=.044 *$F_{1,319}$=11.95, p=.001, ηp^2=.036*
		VEP	
VEP Trials	154.2 (35.3) 61 to 200	173.9 (18.7) 114 to 200	*$F_{1,331}$=44.62 p<.001, ω^2=.115
VEP N1A µV	-4.14 (3.35)	-4.20 (3.48)	$F_{1,307}$=0.01, p=.92, ηp^2=.000 *$F_{1,302}$=0.00, p=.99, ηp^2=.000*
VEP P100A µV	8.11 (4.61)	8.78 (4.57)	$F_{1,335}$=1.57, p=.21, ηp^2=.005 *$F_{1,330}$=1.19, p=.28, ηp^2=.004*

VEP P100L msec	107.16 (17.10)	106.90 (15.56)	$F_{1,335}$=0.02, p=.89, ηp^2=.000 *$F_{1,330}$=0.33, p=.57, ηp^2=.003*
		BM	
BIO Trials	36.95 (10.0)	40.80 (8.22)	$F_{1,298}$=11.24, *p*<.001, ηp^2=.036
BMS P100A µV	-0.28 (2.9)	-0.28 (2.4)	$F_{1,272}$=0.00, *p*=.99, ηp^2=.000 $F_{1,267}$=0.01, *p*=.91, ηp^2=.000
BMS P100L msec	1.07 (18.9)	2.60 (18.2)	$F_{1,272}$=0.40, *p*=.53, ηp^2=.001 $F_{1,267}$=0.03, *p*=.87, ηp^2=.000
BMS N200A µV	-0.71 (2.9)	-0.60 (3.1)	$F_{1,298}$=0.94, *p*=.76, ηp^2=.000 $F_{1,293}$=0.26, *p*=.61, ηp^2=.001
BMS N200L msec	11.3 (49.7)	22.75 (49.3)	$F_{1,228}$=3.62, *p*=.06, ηp^2=.012 *$F_{1,223}$=4.36, p=.04, ηp^2=.015*
BMS P300A µV	-0.68 (3.5)	-1.51 (3.2)	$F_{1,298}$=4.07, *p*=042, ηp^2=.013 *$F_{1,293}$=2.42, p=.12, ηp^2=.008*

Key: TD=Typical development; ASD=Autism Spectrum Disorder; R=Resting Experiment; F=Faces Experiment; VEP=Visual Evoked Potential Experiment; BM=Biological Motion Experiment; FU=face upright; BIO=biological motion condition; A=amplitude; L=latency.

*Because Levene's <.05, Welch ANOVA utilized for reporting *F*, df, and *p* and omega squared for reporting effect size.

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Available per request: https://medicine.yale.edu/ycci/researchers/autism/postersandpapers/

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