

Neural Specialization for Faces and Letters in Autism

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Background

Face perception is a vital social ability supported by specialized brain regions

- fMRI studies indicate face-selective activity in the fusiform gyrus
- The N170, an event-related potential (ERP) over occipitotemporal scalp, displays enhanced amplitude and decreased latency to faces¹
- This specialization is honed through extensive developmental exposure, resulting in *perceptual expertise* for faces

Similar neural specialization occurs for other visual stimuli with which people have extensive experience and perceptual expertise

- As one learns to read, one develops expertise for the letters of the alphabet
- Letters in familiar alphabets elicit an enhanced N170², providing an example of non-social expertise
- Specialization requires coordinated function of distributed brain regions in posterior and medial-frontal cortex

Autism spectrum disorder (ASD) is characterized by developmental difficulties with face perception

- Retrospective studies indicate reduced attention to faces by 6 months and enduring anomalies in recognition and visual attention
- N170 response to faces is delayed and insensitive to disruption in configural information³
- The social motivation hypothesis posits that face processing deficits derive from reduced social attention during development and a consequent failure to develop perceptual expertise for faces⁴

The current study investigated neural and behavioral correlates of social (faces) and non-social (letters) expertise in ASD. It was predicted that:

- 1. Individuals with ASD will exhibit atypical patterns of brain activity and behavioral impairment for faces
- 2. Individuals with ASD will exhibit typical brain activity and behavior for letters of the alphabet

Method

- **Participants:** • 36 children with ASD based on ADOS and DSM-IV-TR clinical diagnosis
- 18 typical controls matched for sex, age, Full Scale IQ, and handedness

	Typical (N=18)	ASD (N=36)
Number male (Percent)	15 (83.3)	32 (88.9)
Number White (Percent)	15 (83.3)	34 (94.4)
Number right handed (Percent)	16 (88.9)	31 (86.1)
Mean age (SD)	12.6 (2.4)	11.2 (3.4)
Mean Full Scale IQ (SD)	112.9 (13.4)	105.2 (15.0)

Behavioral measures:

- Benton Facial Recognition Test (Raw score range = 0 54)
- Woodcock-Johnson III: Letter-Word Identification (single word reading) Word Attack (novel word decoding)

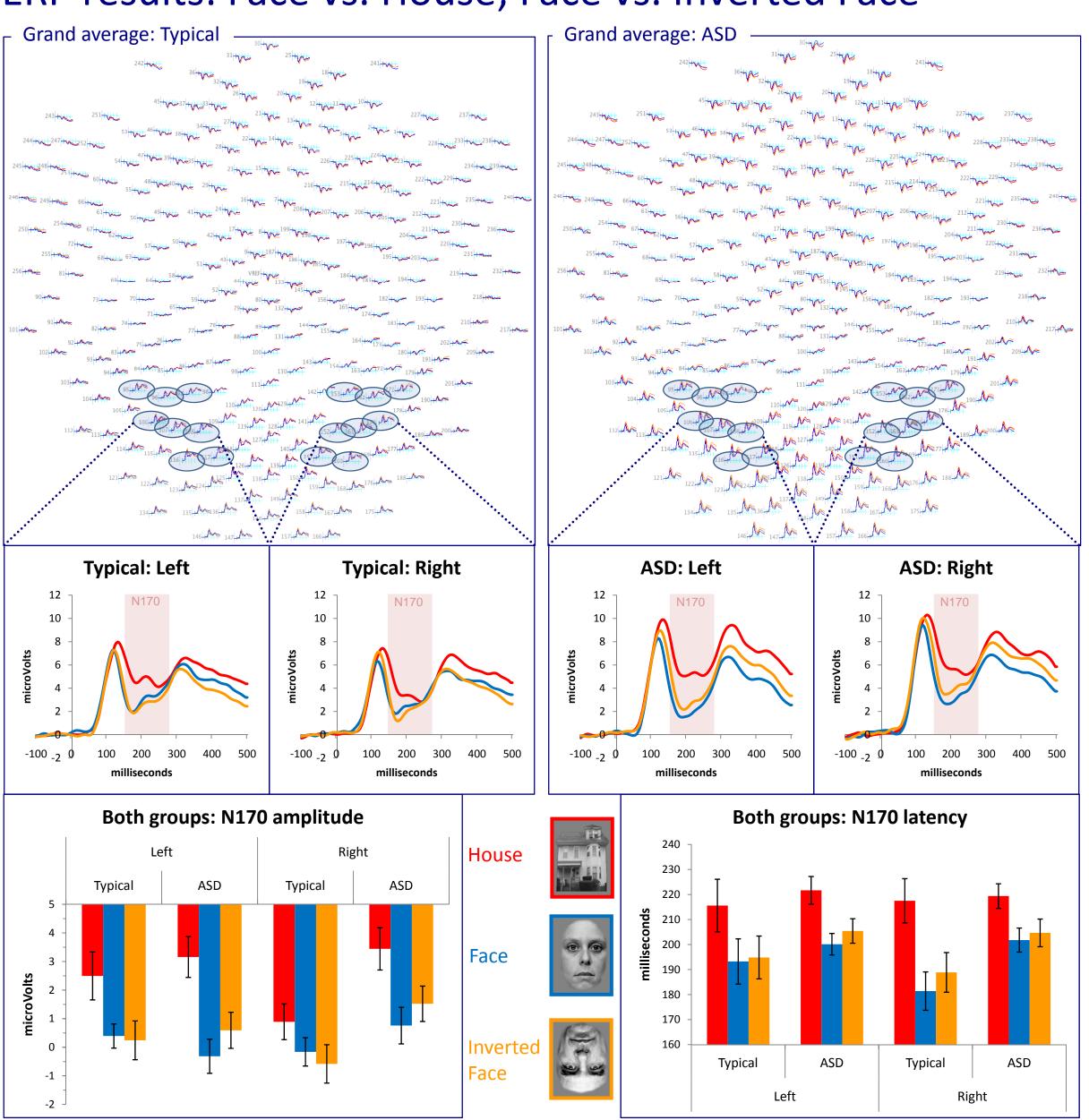
ERP procedure:

- 92 stimuli from each category in pseudorandom sequence in two blocks
- Upright/inverted faces and houses
- Letters of the Roman alphabet and pseudoletters (confabulated alphabet)
- Trial: Crosshair (250–750 ms) → Stimulus (500 ms) → Blank screen (500 ms)
- One-back task with attention monitored by closed-circuit video

ERP data acquisition and processing:

- ERP recorded continuously at 250 Hz using EGI 256-channel sensor net
- N170 peak amplitude and latency extracted from averaged data across 8 electrodes in each hemisphere

ERP results: Face vs. House; Face vs. Inverted Face —

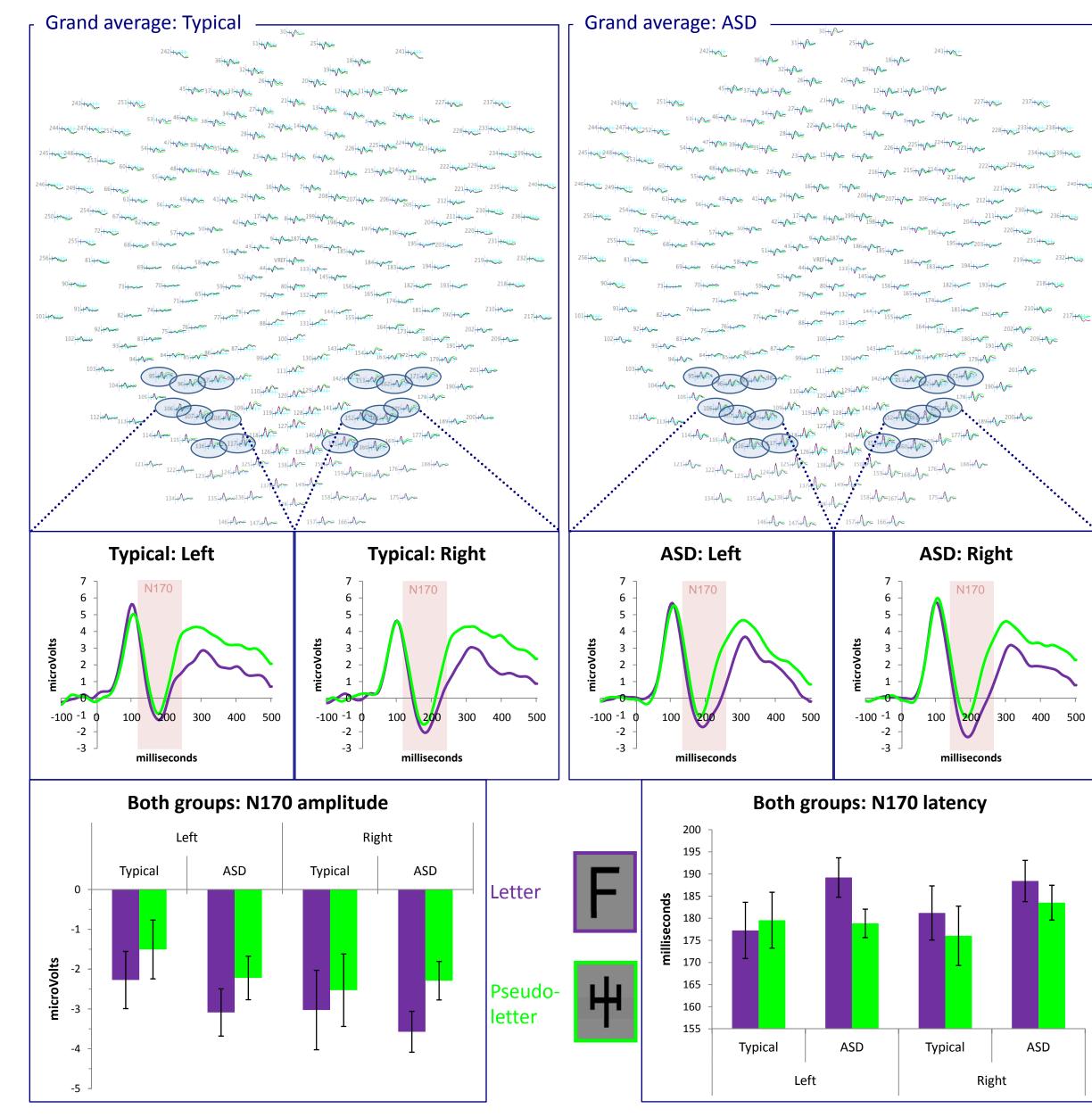


Face vs. house: Faces elicited N170s with shorter latencies and larger amplitudes than houses in both groups and across hemisphere (main effect of Condition; $p \le .01$). Across faces and houses, typically developing individuals displayed enhanced amplitude in the right hemisphere, while those with ASD displayed equivalent amplitude in both hemispheres (Hemisphere by Group interaction; $p \le .01$). Across hemisphere, individuals with ASD exhibited reduced N170 amplitude to houses (Condition by Group interaction; p \leq .05). N170 amplitude to houses was reduced in the left hemisphere across group (Hemisphere by Condition interaction; $p \le .05$). A marginally significant ($p \le .10$) Group by Hemisphere by Condition interaction suggested that typically developing children displayed shorter right hemisphere latencies for faces than children with ASD; post-hoc analysis confirmed this effect ($p \le .05$).

Face vs. Inverted face: Across hemisphere and group, inverted faces elicited N170s with longer latencies than upright faces (main effect of Condition; $p \le .05$). Across faces and inverted faces, typically developing individuals displayed enhanced amplitude in the right hemisphere, while those with ASD displayed equivalent amplitude in both hemispheres (Hemisphere by Group interaction; $p \le .05$). Across hemisphere, individuals with ASD displayed attenuated N170 amplitudes to inverted faces relative to upright faces; typically developing individuals displayed an inversion effect in the expected direction, with larger amplitude to inverted relative to upright faces (Condition by Group interaction; $p \le .05$).

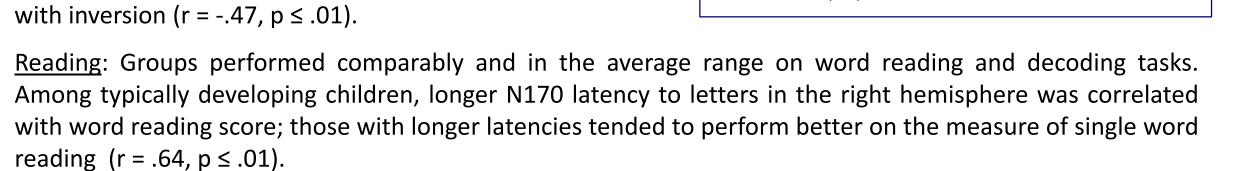
Letter vs. Pseudoletter: Across hemisphere and group, letters elicited N170s with larger amplitudes than pseudoletters (main effect of Condition; $p \le .01$). No between group differences were detected.

ERP results: Letter vs. Pseudoletter



Behavioral results

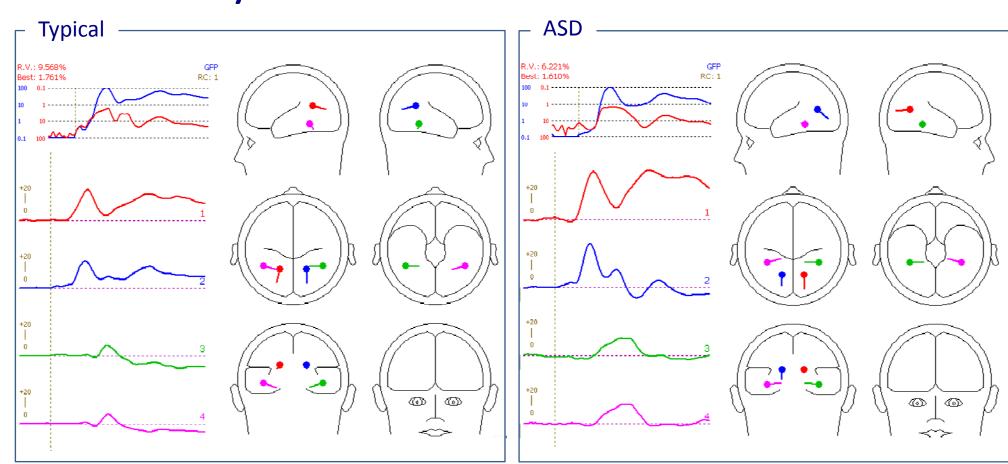
<u>Face recognition</u>: Individuals with ASD obtained significantly lower face recognition scores than typically developing children (p ≤ .01). For both groups, N170 latency to faces in the right hemisphere was correlated with face recognition skill; individuals with faster N170s displayed better face recognition performance (ASD: r = -.39, $p \le .05$; Typical: r = -.39.53, p \leq .05). Among individuals with ASD, N170 amplitude to inverted faces was correlated with face recognition performance; those with better face recognition abilities were more likely to display an enhanced N170 associated



Behavioral performance

Raw Score (+60) Standard Score Standard Score

Preliminary source localization: Faces



Cortical sources of scalp ERPs for face stimuli were estimated using Brain Electrical Source Analysis Software (BESA v5.2.4). A realistic head model with conductivity ratio of 50 was selected to correspond to the age range of study participants.

Grand averaged data for upright face stimuli were analyzed separately for each group. Pairs of symmetric dipoles were fit sequentially to describe the scalp topographies of the P1 and N170 components. Derived Talairach coordinates were assigned Talairach Atlas labels using Talairach Client (v2.4.2).

For typically developing children, the fit interval was determined by the latency of maximal P1 and N170 amplitude in the grand average at specified electrodes. The first pair of dipoles fit between 100 and 132 ms post-stimulus localized to posterior cingulate (Talairach coordinates: +/-19, -54, 18), explaining 87.54% of total variance. A second pair of dipoles were fit at 160 to 180 ms and localized to fusiform gyrus (Talairach coordinates: +/-43, -45, -9). Combined, the two pairs of sources explained 90.45% of total variance.

For the ASD group, fit intervals were extended to correspond to greater individual variation. A first pair of dipoles fit between 72 and 168 ms post-stimulus localized to posterior cingulate (Talairach coordinates: +/-16, -63, 11), explaining 92.42% of total variance. A second pair of dipoles were fit at 144 to 208 ms and localized to fusiform gyrus (Talairach coordinates: +/-38, -38, -10). The sources explained 93.78% of total variance.

Conclusions

Consistent with prior findings, individuals with ASD displayed atypical electrophysiological brain response to faces and correspondingly impaired face recognition ability. In contrast to typically developing children, those with ASD failed to demonstrate a face inversion effect and exhibited slowed face processing in the right hemisphere. Despite these discrepancies, similar brain regions were involved for each group. In the domain of nonsocial perception and behavior, children with autism were comparable to typically developing peers. Both groups displayed neural specialization for letters in terms of enhanced N170 amplitude, and both groups attained average performance on reading tasks. These results concord with theories suggesting specific dysfunction of social brain mechanisms in ASD. Given intact neural specialization for non-social information, a developmental phenomenon requiring distributed brain function, future research should examine connectivity within specific brain systems, rather than nonspecific patterns of dysfunction.

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