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Research report

# Social cognition and neural substrates of face perception: Implications for neurodevelopmental and neuropsychiatric disorders

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## HIGHLIGHTS

- Autism and schizophrenia are typically regarded as dichotomous clinical entities.
- These conditions are associated with social deficits with known neural correlates.
- Social behavior may be regarded as a continuous, normally distributed trait.
- Here, normal variation in social behavior predicted neural structure and function.
- Brain-behavior links observed in atypical populations are preserved in controls.

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## ABSTRACT

*Background:* Social cognition is an important aspect of social behavior in humans. Social cognitive deficits are associated with neurodevelopmental and neuropsychiatric disorders. In this study we examine the neural substrates of social cognition and face processing in a group of healthy young adults to examine the neural substrates of social cognition.
*Methods:* Fifty-seven undergraduates completed a battery of social cognition tasks and were assessed with electroencephalography (EEG) during a face-perception task. A subset ($N = 22$) were administered a face-perception task during functional magnetic resonance imaging.
*Results:* Variance in the N170 EEG was predicted by social attribution performance and by a quantitative measure of empathy. Neurally, face processing was more bilateral in females than in males. Variance in fMRI voxel count in the face-sensitive fusiform gyrus was predicted by quantitative measures of social behavior, including the Social Responsiveness Scale (SRS) and the Empathizing Quotient.
*Conclusions:* When measured as a quantitative trait, social behaviors in typical and pathological populations share common neural pathways. The results highlight the importance of viewing neurodevelopmental and neuropsychiatric disorders as spectrum phenomena that may be informed by studies of the normal distribution of relevant traits in the general population.


## 1. Introduction

Social cognition comprises a set of skills that enable us to understand thoughts and intentions of others and respond appropriately to their social actions [1]. These skills develop under genetic and experiential influences, including environmental and cultural factors, and are vital for adaptive social behavior. Deficits in social cognition are key features in a variety of neurodevelopmental and psychiatric disorders including autism spectrum disorder (ASD) and schizophrenia [2].

Rather than existing as a dichotomy, behaviors associated with neurodevelopmental and neuropsychiatric syndromes often represent the severe end of continuous distributions of core competencies and/or deficiencies that occur in nature [3,4]. For example, autistic symptoms or traits (social/communication deficits and restricted interests and repetitive behaviors) aggregate in close relatives of children with ASD, including those who do not meet the threshold for clinical diagnosis of ASD – known as the "broader autism phenotype" [5–7]. Indeed, many behavioral traits that are symptomatic of neurodevelopmental and neuropsychiatric disorders are represented in the general population, with the normality of this distribution being highly dependent on the sensitivity of the measurement.

Standard diagnostic measures of ASD (such as the Autism Diagnostic Observation Schedule, or ADOS) reflect the traditional

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categorical approach to symptom expression, and as such yield near floor-effects in non-clinical populations. These floor effects obscure important genes-brain-behavior links that may underlie the typical manifestations of the behaviors in question by attenuating statistical variability. Epidemiologic studies have shown that quantifiable traits that make up the core impairments of ASD (such as those measured with the Social Responsiveness Scale (SRS)), are continuously distributed in the general population [8,9] Other features associated with ASD that are present in the general population include repetitive behaviors and restricted interests [10,11] as well as deficits in empathy, and a systemizing cognitive style (the tendency to analyze, understand, predict, control, and construct rule-based systems) [12,13]. Sex-related differences have also been observed in social-cognitive traits [8,14], and these differences may be linked to the uneven sex distribution observed in ASD diagnoses which approaches a 4:1 male–female ratio, as well as to possible sex differences in the lateralization of certain functions (e.g., face perception) that is also prevalent in ASD [15].

Face processing and its neurophysiologic correlates are important markers of social cognition. Normal infants exhibit visual preference for faces in the first few days of life and within the first 6 months develop the ability to distinguish familiar from unfamiliar faces, differentially process inverted versus upright faces, and differentiate facial emotional expressions [16]. Eye-tracking, functional neuroimaging, and electrophysiological studies have demonstrated that children and adults with ASDs process faces and decode facial expressions differently than typically-developing individuals [17–20]. Converging evidence from ERP and fMRI studies indicates specialized activity in regions of the occipital and temporal cortex that are involved in face processing, including the inferior temporal cortex fusiform gyrus and the superior temporal sulcus [21–23].

The N170 event-related potential (ERP) component exhibits larger amplitudes and shorter latencies to faces relative to other stimuli. ERP face perception studies also report right hemisphere dominance and inversion effects, (larger amplitude and/or longer latency responses to inverted faces relative to upright faces) [24–26]. In high-functioning adolescents and adults with ASD, the N170 response to faces is delayed relative to controls, and the typical right hemisphere lateralized pattern is often absent. Furthermore, unlike controls, the ASD group did not exhibit the inversion effect [27,28], suggesting not only slower processing of faces, but also a qualitatively different processing strategy.

Parents of individuals with autism also fail to show right hemisphere lateralization or a shorter latency N170 to faces compared to objects [29]. A study of adolescent twins provided evidence for substantial heritability of neurophysiologic indicators of face processing; 36–64% of individual variability in the ERP components elicited by changes in facial expression was accounted for by genetic factors [30]. Even in non-clinical populations, quantitative traits of symptoms associated with ASD and obsessive–compulsive disorder are linked to variations in ERPs during the processing of certain stimuli including faces [31,32]. These findings suggest that ERP components sensitive to face processing, including emotional expressions, can potentially serve as endophenotypes for disorders characterized by abnormalities in social cognition and behavior. Similarly, fMRI studies utilizing blood-oxygen-level-dependent (BOLD) contrasts reveal activation of a right lateralized inferior temporal area in the fusiform gyrus (FFG) when subjects look at human faces [33,34]. While right-hemisphere lateralization is relatively robust, the finding is not universally reported [34–36].

Relative expertise in face processing is believed to have developed through evolutionary pressures that place significant import on our ability to recognize and perceive faces [37] which is linked to better social cognition and social behavior, more generally. Individuals with quantitatively low levels of social cognition – specifically those clinically diagnosed with ASD – do not develop face processing expertise to the extent that typically-developing individuals do. Relative to controls, individuals with ASD exhibit hypoactivation in areas related to social cognition and face perception [38–40] such as the orbito-frontal cortex, superior temporal gyrus, amygdala [41] and fusiform gyrus [21,39,42–45].

Few published studies have examined the association between social cognition and fMRI and ERP markers of face processing in non-clinical populations. ASD traits in typically-developing individuals have been shown to predict neural responses to eye gaze [46] and the structure and function in the posterior superior temporal sulcus [47]. Among typically developing children, more negative N170 amplitude (larger ERP) to upright faces is associated with fewer atypical social behaviors [19]. Smaller (less negative) N170 amplitude is thought to reflect less face processing expertise, and this inefficiency in neural processing may result in less fluid and more effortful reciprocal social interactions and therefore more atypical social behavior [19].

The purpose of this study is to explore the associations among quantitative self-report and performance-based measures of social cognition and neurophysiologic correlates of face processing. We hypothesize that measures of social competence will predict variation in (1) face-related N170 amplitude and latency and (2) fusiform gyrus activation on fMRI in response to faces relative to a control condition (houses). Higher social cognitive competence (higher EQ scores, lower SRS scores, higher scores on a social attribution task) is expected to be associated with more activation of the fusiform gyrus in response to faces on fMRI, and with decreased latency and increased amplitude of the N170 on ERP. We also examine sex differences in the lateralization of face processing ability.

## 2. Materials and methods

*Ethics statement.* The research protocol and consent procedures were approved by a university Institutional Review Board (IRB#1112-033, “Social Cognition and Face Perception”). All subjects involved in the study gave written informed consent.

*Statistical analysis.* All analyses were performed using SPSS 20 (IBM) with a significance threshold of $p < 0.05$. Variable distributions were checked for normality, and non-violated assumptions for parametric tests.

*Participants.* Subjects were undergraduate students at a liberal arts university in central Pennsylvania ($N = 57$; 20 males, 37 females). A subset of the participants completed the fMRI portion of the protocol ($N = 24$; 12 males, 12 females). Subjects were recruited through an introductory psychology course, satisfying participation in research. Subjects who completed the entire protocol (including fMRI) received additional monetary compensation.

The average age of the participants was 18.87 years (SD = 0.93). Of the 57 total participants, 50 were self-described as Caucasian (84.7%), three as East Asian or Pacific Islander (5.3%), and two each as African American, South Asian, or of more than one race (3.5% each). Of the 57 participants, 24 participants (42%) volunteered to complete fMRI testing in addition to behavioral and EEG testing. Of these 24 one participant was excluded based on left handedness and another was excluded for taking medication for a psychiatric diagnosis. This left a total of 57 (20 males) participants and a subset of 22 (10 females) with fMRI scans. None of the participants was diagnosed with a psychiatric disorder or reported any known familial history of an ASD diagnosis.

### 2.1. Behavioral and demographic measures

All self-report measures described below (Demographics, Empathizing and Systemizing, and the Social Responsiveness Scale)