Perception of facial expression and facial identity in subjects with social developmental disorders

Rebecca L. Hefter, BSc; Dara S. Manoach, PhD; and Jason J.S. Barton, MD, PhD, FRCPC

Abstract—Background: It has been hypothesized that the social dysfunction in social developmental disorders (SDDs), such as autism, Asperger disorder, and the socioemotional processing disorder, impairs the acquisition of normal face-processing skills. The authors investigated whether this purported perceptual deficit was generalized to both facial expression and facial identity or whether these different types of facial perception were dissociated in SDDs. Methods: They studied 26 adults with a variety of SDD diagnoses, assessing their ability to discriminate famous from anonymous faces, their perception of emotional expression from facial and nonfacial cues, and the relationship between these abilities. They also compared the performance of two defined subgroups of subjects with SDDs on expression analysis: one with normal and one with impaired recognition of facial identity. Results: While perception of facial expression was related to the perception of nonfacial expression, the perception of facial identity was not related to either facial or nonfacial expression. Likewise, subjects with SDDs with impaired facial identity processing perceived facial expression as well as those with normal facial identity processing. Conclusion: The processing of facial identity and that of facial expression are dissociable in social developmental disorders. Deficits in perceiving facial expression may be related to emotional processing more than face processing. Dissociations between the perception of facial identity and facial emotion are consistent with current cognitive models of face processing. The results argue against hypotheses that the social dysfunction in social developmental disorder causes a generalized failure to acquire face-processing skills.

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Autism, Asperger disorder, and the socioemotional processing disorder (SEPD) are social developmental disorders (SDDs) characterized by difficulties with interpersonal interactions. A number of studies suggest that the social difficulties of SDDs are associated with anomalous face processing. Also, other reports note that subjects with developmental prosopagnosia have social disabilities reminiscent of Asperger disorder. These findings imply that social and face-processing abilities are related, perhaps even causally.

Judging interest and intention from the expression of emotion plays a vital role in social reciprocity among individuals. The importance of facial expression in portraying emotional states has led to a focus on the perception of expression in many face-processing studies in SDDs. Individuals with autism or Asperger disorder are less accurate than controls in interpreting emotional expressions and have difficulty judging the emotional states of others. Some functional imaging studies report a lack of activation of the fusiform face area in the right occipitotemporal lobe of these subjects during perception of facial expressions, although others have not replicated this finding.

However, humans garner more than just information about emotional states from faces. Cognitive models have proposed that, after a common early perceptual stage, face-processing streams diverge, in particular for expression and identity, a proposal with support from neurophysiology and functional imaging. Neuropsychological studies have also shown that some subjects with prosopagnosia can perceive facial expression normally, supporting an anatomic dissociation in the perception of facial identity and expression.

It is unclear whether subjects with SDDs are impaired in the processing of facial identity. While one study reported no deficit in recognizing familiar faces, others found impairments. Likewise some studies have shown impaired matching of faces but others have found normal performance. Determining whether facial expression and facial identity are both impaired in subjects with SDDs is important to understanding the nature of their perceptual deficits. A generalized face-processing failure...
might affect judgments about both facial expression and facial identity. General failure could be secondary to a lack of social motivation in subjects with SDDs, impeding the development of normal face expertise in early life.26-28 Alternatively, these subjects may have a primary deficit in face processing,5 implying dysfunction of structures like the fusiform face area.3,4 Whether the perception of facial expression would be impaired with a primary defect of medial occipitotemporal structures remains to be determined. However, face expression analysis may also be affected as part of a general difficulty with processing emotion rather than faces. This would be suggested if facial expression perception correlated more with the processing of expression from other nonfacial cues rather than with the perception of facial identity. This in turn might implicate different structures located at a sensory convergence point for multimodal emotion processing, such as the amygdala.28

We sought to characterize the relationship between face identification, facial expression analysis, and the processing of expression from nonfacial cues in adult subjects with SDDs. Our previous report on facial identity processing in these subjects29 showed a wide range of performance in SDDs, with some subjects performing normally on face identification tasks and others with variable impairment. If failure at a stage of face processing common to identity and facial expression recognition was present in SDDs, we hypothesized that we would find a high correlation between face identification and facial expression analysis. This would be expected if face-processing deficits in SDDs arise as a result of social failure and lack of interest in faces, leading to a possible failure to develop facility with either identity or emotion in faces. On the other hand, if difficulties in the analysis of facial expression in SDD are rooted in a more general failure to process emotional cues, then we would expect the analysis of facial expression to correlate with the analysis of expression from nonfacial cues rather than the analysis of facial identity.

Methods. Subjects. The Committee on Clinical Investigations at Beth Israel Deaconess Medical Center approved the study protocol. All subjects gave written informed consent after the experimental procedures had been fully explained according to the Declaration of Helsinki. We tested 26 adults with SDDs who were recruited from adult outpatient clinics offering neuropsychological assessment in the Boston area. We restricted our sample to age 16 years and older due to evidence that face recognition skills may continue to mature during childhood.29 We excluded subjects with history of acquired brain disease or major brain injury. Our total sample included 19 males and seven females (n = 26), with a mean age of 34.4 years (SD 10.1, range 16 to 49).

It should be noted that the diagnosis of SDDs in adults presents several challenges. First, most scales used to measure diagnostic criteria of these disorders are designed for use with children and parents, as the definitions of SDDs are generally based on studies of children. Second, obtaining accurate information about early development is difficult due to its retrospective nature, unless records are available. Finally, the lack of consensus about diagnostic criteria for each of the disorders that fall under the label of SDDs (Asperger disorder, autism, right hemisphere learning disorder, nonverbal learning disorder, and SEPD) makes for variable diagnoses. For example, SEPD, also referred to as right-hemisphere learning disorder21,22 and similar to nonverbal learning disability,23,24 is a disorder mainly defined by the neurologic rather than the psychiatric community. As such, none of these disorders are found in the Diagnostic and Statistical Manual, Fourth Edition (DSM-IV).

We included several diagnoses within the SDD group due to the large overlap of diagnostic criteria common to each of these disorders. Different approaches are used to evaluate and diagnose each (psychiatric, neuropsychological, behavioral), yet they share the central diagnostic criterion of social dysfunction. Given that the aim of the present study was to examine the relationship of social developmental dysfunction to face processing and given the ongoing controversies about current nosologic schemes, we included subjects with a variety of diagnoses. We considered social developmental dysfunction, along with the exclusion of other pervasive developmental disorders and schizophrenia, to be the core criterion for the presence of an SDD.

For the present study, the initial diagnosis of an SDD was made by the referring neuropsychologist and confirmed by a second licensed neuropsychologist (D.S.M.) based on a thorough review of psychological, neuropsychological, and medical evaluations, supplemented by an interview with the subject and a parental informant when possible. We obtained detailed histories with attention to birth-related events, developmental milestones, emotional adjustment, social history, and family history. In addition, behavioral observations from the neuropsychological evaluation (see below) and the interview were recorded. Special attention was given to observations regarding paralinguistic communication ability, including the use of eye contact, facial expression, and gesture. The supplemental interview and behavioral observations addressed the material covered by the Autism Diagnostic Interview-Revised (Short Edition) (ADI-R).25

Diagnosis of SDDs. One diagnostic criterion was common to each of the three SDDs that we examined and was necessary for inclusion in the study (table 1). It is best described by DSM-IV Criterion A for autistic and Asperger disorders as “qualitative impairment in social interaction, manifest in non-verbal social behaviors, peer relationships, spontaneous social engagement and social/emotional reciprocity.”

Table 1 Diagnostic criteria for developmental social processing disorders

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Autism</th>
<th>AD</th>
<th>SEPD</th>
<th>BOTH AD and SEPD</th>
</tr>
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<tbody>
<tr>
<td>Social impairment</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Significant impairment in functioning (i.e., social, occupational, etc.)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Clinically significant general delay in cognitive development</td>
<td>N/A</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Abnormal language acquisition</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Repetitive behavior</td>
<td>Yes</td>
<td>Yes</td>
<td>N/A</td>
<td>Yes</td>
</tr>
<tr>
<td>Right hemisphere dysfunction (VIQ &gt; PIQ by at least 10 points)</td>
<td>N/A</td>
<td>N/A</td>
<td>Yes</td>
<td>Yes</td>
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AD = Asperger disorder; SEPD = socioemotional processing disorder; Yes = criterion required for diagnosis; N/A = not applicable to diagnosis; No = criterion absent for diagnosis; VIQ = Verbal IQ; PIQ = Performance IQ.
The following criteria are specific to one of the three SDDs in our sample. Diagnosis of autism (two subjects, one male, one female). The DSM-IV lists two further criteria for a diagnosis of autism. The first (Criterion B) is "qualitative impairments in communication, manifested by delay in, or total lack of, the development, ... impairment in ... and repetitive use ... of spoken language." Second, subjects demonstrated "restricted repetitive and stereotyped behaviors, interests, and activities." The latter criterion was fulfilled if subjects met the cutoff for autism on the Profile of Nonverbal Sensitivity (PONS). As each of the subjects diagnosed with autism were living independently and scored in the normal range on Full Scale IQ, they were considered high functioning (HFA).

Diagnosis of Asperger disorder (four males). The DSM-IV lists three further criteria for a diagnosis of Asperger disorder. The first and second criteria are consistent with those of previous studies at our center and superior verbal vs nonverbal intellect, defined as a Verbal IQ score at least 10 points higher than Performance IQ (a 10-point discrepancy is significant at the p < 0.05 level). Repetitive behaviors do not play a role in the diagnosis of SEPD. These criteria are consistent with those of previous studies at our center and other groups. As with our autistic subjects, our SEPD subject had normal Full Scale IQ. Only one subject had a Performance IQ that fell slightly below the normal range.

Diagnosis of SEPD (10 subjects, seven males). In the present study, beyond the criterion common to all SDDs, a diagnosis of SEPD includes having a good right hemisphere function as our subjects had good Verbal IQ scores, with a mean of 118 (SD 11.4) years. The age and sex of these two groups were not significantly different from our subjects.

Diagnostic Analysis of Non-Verbal Accuracy (DANVA). This computer-based audiovisual test was used to measure ability to discriminate emotional cues from content-standard voices and static faces. This test evaluated the ability of subjects to identify the four basic emotions (happy, sad, angry, fearful) without a stimulus of content. The test was conducted in a dimly lit room. For each stimulus in parts one and two, subjects were able to pause the recordings if they needed more time to answer than allowed on tape (although most were able to keep up with the tapes). The test was administered individually and was scored with a computer program.

This audiovisual test was used to measure accuracy in deciphering the meaning of nonverbal emotional cues expressed by a woman through facial expression, body gesture, and prosody. We used two short forms of the PONS. Part one measured interpretation of prosody via an audiovisual test with 24 stimuli of a woman's voice, content-filtered and randomly spliced to remove semantic content. Part two measured interpretation of facial expression and body gesture via a videotape featuring 40 random black and white dynamic trials with the following features: a woman's face (20) or body (from shoulders to thighs) acting out various emotions. There was no sound and the test was conducted in a dimly lit room. For each stimulus in parts one and two, subjects selected the scenario description most consistent with the affective information conveyed from two choices (e.g., A, reassuring a lost child or B, discussing one's divorce). Neither test was timed, and although repetitions of the stimuli were not permitted, subjects were able to pause the recordings if they needed more time to answer than allowed on tape (although most were able to keep up with the tapes).

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Results. As might be expected, the ability to recognize facial identity and the ability to perceive emotional information in nonfacial cues were not correlated ($r = 0.18$, $p = 0.36$). However, the key finding of our study was that the processing of facial identity was also not correlated with the ability to recognize facial expressions ($r = 0.04$, $p = 0.84$). In contrast, there was a correlation between facial and nonfacial expression analysis ($r = 0.40$, $p < 0.05$). Therefore, the perception of facial emotions was related to the perception of emotions from auditory or body cues more than to the perception of facial identity (figure).

The comparisons between the SDD-1 subgroup (normal facial identity recognition) and the SDD-2 subgroup (impaired facial identity recognition) supported this conclusion (table 2). While by definition these groups differed significantly on $d'$ for face identity, $t$ tests showed no difference for their perception of face expression ($p = 0.27$) and nonfacial expression scores ($p = 0.30$).

We also examined the effect of diagnostic label on performance of facial and nonfacial expression perception using one-way analysis of variance with diagnostic label (Asperger disorder, HFA, SEPD, Asperger disorder and SEPD) as the variable. The results showed that neither facial ($F(3,21) = 1.38$, not significant) nor nonfacial ($F(3,21) = 1.49$, not significant) expression perception were significantly related to diagnostic label. Thus, having a particular clinical diagnosis did not predict the ability to analyze either type of expression cue. This parallels our previous finding that clinical diagnosis did not correlate with the ability to recognize faces.29

Discussion. Our study found that the perception of facial identity in subjects with SDDs is related neither to the perception of facial expression nor to the perception of nonfacial expression. Rather, the ability to process expression from facial cues was correlated with the ability to process expression from nonfacial cues (voices and body), even though some expression items were presented in a different sensory modality (visual or auditory). While both identity and expression information are available in all faces, and our subjects with SDD showed a wide range of abilities for both, performance with one type of facial processing did not correlate with the other. The group analysis confirmed this by showing no significant difference in the processing of facial expressions between the group with normal and the group with impaired recognition of facial identity.

We previously reported that subjects with SDDs are not uniformly impaired on a range of facial tasks related to the processing of identity, including famous face imagery, perception of the configuration of facial features, face matching, short-term facial memory, and famous face familiarity.29 Indeed, one group of subjects (SDD-1) was consistently normal on all tests of identity. On this basis, we concluded that developmental social dysfunction does not inevitably lead to impaired face processing. One hypothesis about the relationship between defective face processing and defective social function is that impaired social skills limits the interest that subjects with SDDs have in faces and that this limited interest impedes their acquisition of normal perceptual expertise with faces.25-27 Given our data, this hypo-

Table 2  Comparison of scores between SDD-1 and SDD-2 groups

<table>
<thead>
<tr>
<th></th>
<th>SDD-1 mean</th>
<th>SD</th>
<th>SDD-2 mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Face identity ($d'$)</td>
<td>3.3</td>
<td>0.4</td>
<td>1.6</td>
<td>0.5</td>
</tr>
<tr>
<td>Facial expression (/80)</td>
<td>62.3</td>
<td>6.0</td>
<td>59.8</td>
<td>5.0</td>
</tr>
<tr>
<td>Nonfacial expression (/84)</td>
<td>59.5</td>
<td>6.3</td>
<td>56.9</td>
<td>5.8</td>
</tr>
</tbody>
</table>

Only face identity is significant at $p < 0.0001$.
SDD-1 = social developmental disorder, normal face identity recognition; SDD-2 = social developmental disorder, impaired face identity recognition.

Figure. Correlation of face expression, face identity, and nonfacial expression perception in social developmental disorders (SDDs). In each graph, the score for one function is plotted against that of another for each individual patient. Linear regressions are shown for the entire group of subjects with SDDs. (A) The composite score for nonfacial expression is plotted against the identity $d'$ (which is the ratio of hits to false alarms, using normalized values) obtained in judging the familiarity of famous vs anonymous faces. (B) The composite score for facial expression is plotted against identity $d'$. (C) The composite scores for facial and nonfacial expression perception are plotted against each other. The correlation is significant only for the plot in C. Black disks show data for SDD-1 subjects (normal facial identity recognition); gray triangles show data for SDD-2 subjects (impaired facial identity recognition). Unit for $d'$ graphs is a ratio of hits to false alarms.
esis would require modifications to explain why some subjects with SDD can have normal face identification as well as the fact that subjects with SDD with normal and those with abnormal face identification do not differ on indices of the severity of their social dysfunction. Also, thoughts about causality should consider two other potential forms of linkage between face processing and social dysfunction. One is that face perception and social skills in SDD are not causally related but correlated, perhaps due to shared anatomic or pathogenetic susceptibilities to the etiologic agent responsible for the syndrome. The other is that SDD as a syndrome may have multiple causes, and that, in one subgroup, face perception impairments contribute to the failure to develop normal social interactions.

The data in this study add to the debate about the relationship between social deficits and face processing by showing widely varying abilities in facial expression analysis, both in individuals with SDDs with normal face identification and subjects with SDDs with impaired face recognition. This argues against a generalized failure to process faces, particularly in the subgroup with impaired processing of facial identity, and suggests that the perceptual aspects of faces required to correctly identify a familiar face differ from those needed to recognize expression (a conclusion supported by psychophysical data from normal subjects showing that the processing of facial identity and facial expression use different components of visual information from faces, different spatial frequencies, and different regions of the face). A generalized failure for all facial processing might be predicated on the hypothesis that face perception does not develop in those whose social failure leads them to ignore or avoid faces from early in life through adulthood. However, the fact that some subjects with SDDs can perceive facial identity but not facial expression, a finding also reported in one other study, while other subjects with SDDs have the reverse pattern of ability strongly argues against such a generalized failure to achieve expertise with faces. Rather, the findings are consistent with models of face recognition that propose divergent processing streams for the perception of facial expression and facial identity. These models are supported by monkey neurophysiologic work and functional imaging studies that suggest that the superior temporal sulcus plays a greater role in processing social cues from faces, while the inferotemporal cortex in monkeys and the fusiform face area in humans are more involved in discriminating facial identity (for a dissenting view, see Tiberghien et al.). They are also consistent with neuropsychological data from subjects with prosopagnosia that reveal normal perception of expression in some subjects, but not others. The results of this study also suggest that the processing deficits in SDDs can affect these divergent streams independently and variably rather than at an earlier perceptual stage common to both expression and identification of faces.

Our results suggest that deficits in perceiving facial expression in SDD are linked to emotional processing rather than face processing. A multimodal deficit in emotional processing in SDDs would be consistent with previous findings that subjects with autism or Asperger disorder have difficulty recognizing emotions in both visual and auditory modalities. Future work should include functional imaging of individuals with SDDs while performing identity and expression recognition tasks to confirm the presence of divergent streams of identity and expression processing and to identify their anatomic components.

References


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