

# Impaired Recognition of Social Emotions Following Amygdala Damage

Ralph Adolphs<sup>1</sup>, Simon Baron-Cohen<sup>2</sup>, and Daniel Tranel<sup>1</sup>

## Abstract

■ Lesion, functional imaging, and single-unit studies in human and nonhuman animals have demonstrated a role for the amygdala in processing stimuli with emotional and social significance. We investigated the recognition of a wide variety of facial expressions, including basic emotions (e.g., happiness, anger) and social emotions (e.g., guilt, admiration, flirtatiousness). Prior findings with a standardized set of stimuli indicated that recognition of social emotions can be signaled by the eye region of the face and is disproportionately impaired in autism (Baron-Cohen, Wheelwright, & Jolliffe, 1997). To test the hypothesis that the recognition of social emotions depends on the amygdala, we administered the same stimuli to 30 subjects with unilateral amygdala damage (16 left, 14 right), 2 with

bilateral amygdala damage, 47 brain-damaged controls, and 19 normal controls. Compared with controls, subjects with unilateral or bilateral amygdala damage were impaired when recognizing social emotions; moreover, they were more impaired in recognition of social emotions than in recognition of basic emotions, and, like previously described patients with autism, they were impaired also when asked to recognize social emotions from the eye region of the face alone. The findings suggest that the human amygdala is relatively specialized to process stimuli with complex social significance. The results also provide further support for the idea that some of the impairments in social cognition seen in patients with autism may result from dysfunction of the amygdala. ■

## INTRODUCTION

The amygdala plays an important role in processing social information from the face, as borne out by a large number of studies in both monkeys and humans. Most studies of the human amygdala's role in face recognition have focused on recognition of so-called basic emotional expressions: happiness, surprise, fear, anger, disgust, and sadness, which can be reliably signaled by the face and for which there exist extensively normed and commonly used sets of stimuli (Ekman & Friesen, 1976). The human amygdala is activated when subjects perceive certain basic facial emotions (Blair, Morris, Frith, Perrett, & Dolan, 1999; Phillips et al., 1998; Breiter et al., 1996; Morris et al., 1996), and amygdala damage impairs recognition of basic emotions (Anderson, Spencer, Fulbright, & Phelps, 2000; Broks et al., 1998; Calder et al., 1996; Young, Hellawell, Van de Wal, & Johnson, 1996; Adolphs, Tranel, Damasio, & Damasio, 1994; Adolphs et al., 1999). However, the amygdala's role appears to extend to more complex social judgments as well: Subjects with bilateral amygdala damage are impaired in judging the trustworthiness or approachability of other people from their faces (Adolphs, Tranel, & Damasio, 1998), and amygdala activation in normal subjects correlates

with untrustworthiness judgments (Winston, Strange, O'Doherty, & Dolan, 2002), as well as other social judgments such as aspects of racial stereotyping (Hart et al., 2000; Phelps et al., 2000).

Baron-Cohen, Wheelwright, and Jolliffe (1997) explored the recognition of complex mental and emotional states, including social emotions, from the face. Their findings were threefold: (1) Such complex mental states are recognized disproportionately by information from the region of the eyes in the face (Baron-Cohen et al., 1997; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). (2) When making judgments about such states from images of the eye region of the face, normal subjects activated the amygdala in functional imaging studies (Baron-Cohen et al., 1999). (3) This amygdala activation was not found in subjects diagnosed with autism (Baron-Cohen et al., 1999), who are impaired in their ability to recognize complex mental states from the eyes. These findings, together with many others, have suggested that the severe impairments in everyday social behavior exhibited by people with autism may be attributable in part to dysfunction in circuits including the amygdala (Baron-Cohen et al., 2000).

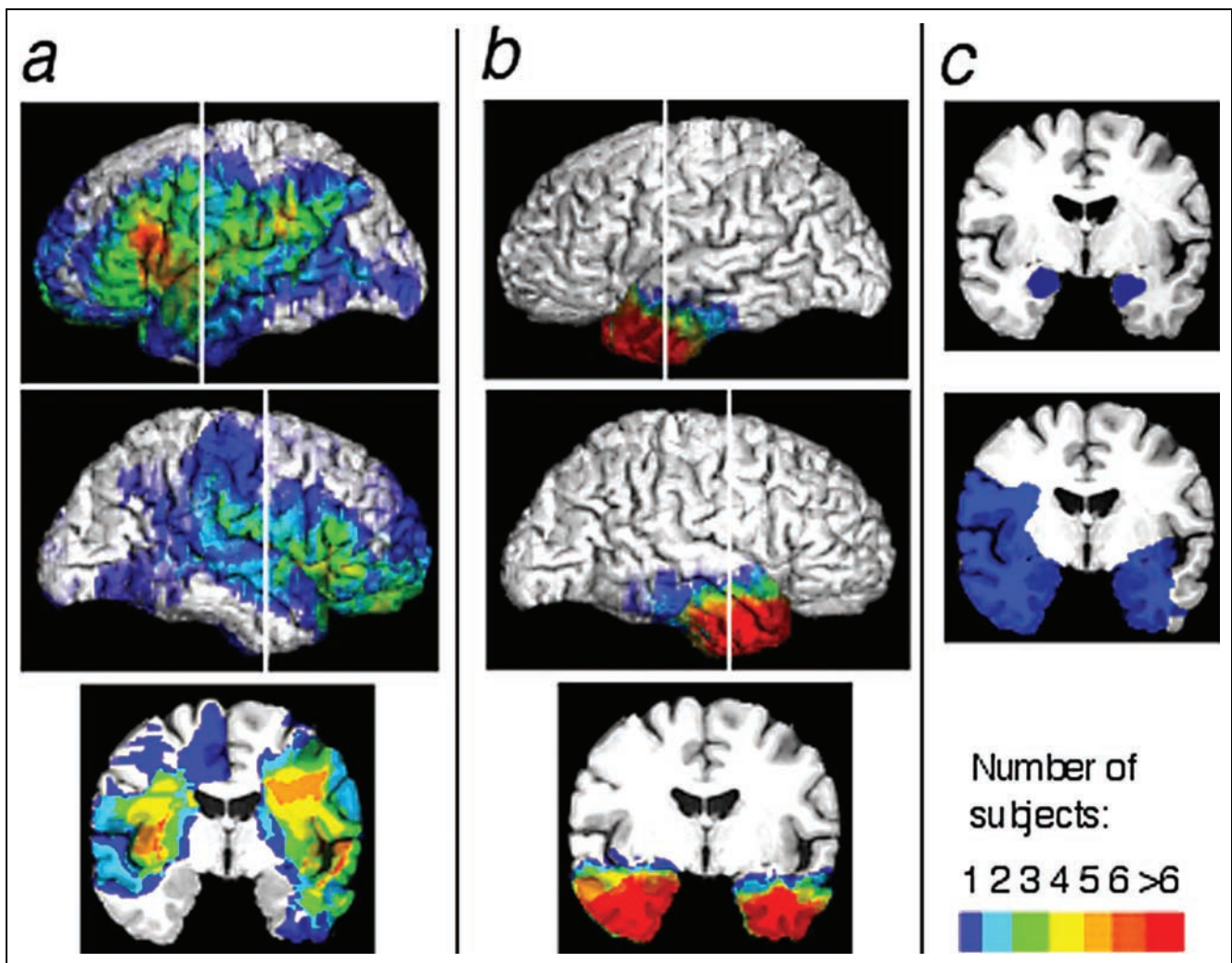
We set out to test the hypothesis that the amygdala is necessary to recognize social emotions from the face. The hypothesis predicts that damage to the amygdala will impair performance on tasks that assess the ability to recognize facial expressions showing social emotions.

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<sup>1</sup>University of Iowa, <sup>2</sup>Cambridge University

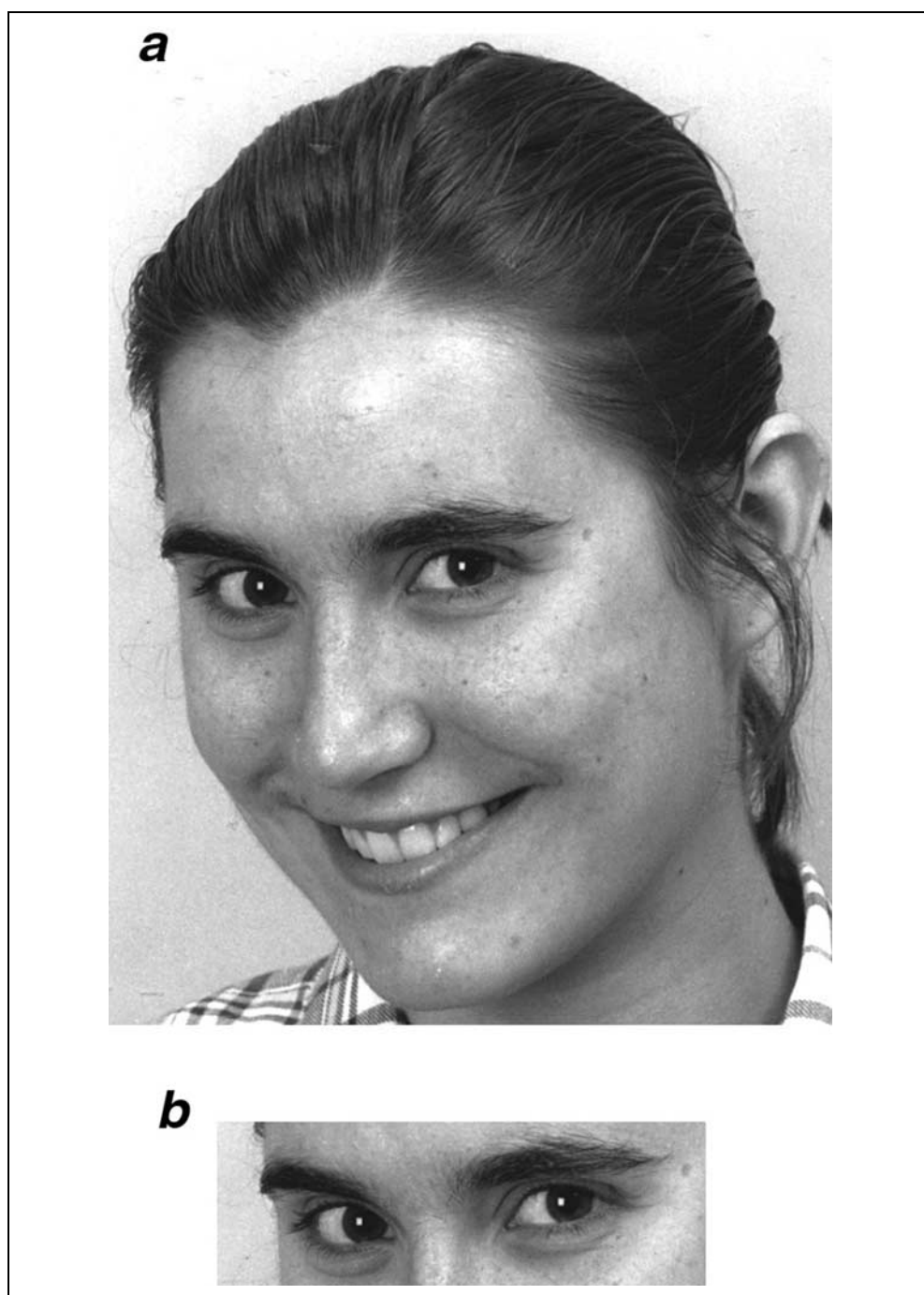
We carried out additional analyses to investigate whether amygdala damage might lead to an impairment that was disproportionately severe for the recognition of social emotions as compared with the recognition of basic emotions. Using the stimuli developed by Baron-Cohen et al. (1997, 2001), we studied groups of subjects with unilateral amygdala damage ( $n = 16$  left and 14 right), and two rare patients with complete bilateral amygdala damage (Figure 1). Subjects were shown pictures of the face stimuli and asked to match them to a list of words for emotions. Our initial analysis used a  $2 \times 2 \times 2$  factorial design: the type of emotion shown in the face stimulus (basic or social), the type of emotion described in the list of words, and the subject group (amygdala lesion or brain-damaged control). This design permitted us to examine independently the effect of emotion type shown in the face stimulus and the effect of emotion type that subjects were asked to match on the word labels, as well as their interaction.

The stimuli used by Baron-Cohen et al. (1997, 2001) consisted of faces expressing a variety of emotional and more complex mental states. We initially used the same categories used by Baron-Cohen et al.: (1) faces showing basic emotions (happiness, sadness, etc.), (2) faces showing complex states other than basic emotions (including states such as thoughtfulness, boredom, arrogance, flirtatiousness). An analysis using these classes of stimuli permits comparisons with prior studies that have used the same stimuli in people with autism. However, we were interested in further subdividing category 2 into those states that are social emotions, and thus undertook additional analyses using a subset of category 2 (including flirtatiousness and arrogance, but excluding states like thoughtfulness and boredom). A final issue of interest was whether the possible impairments found relied on configural processing of the whole face or whether they might be attributable to the abnormal processing of information



**Figure 1.** Neuroanatomy of brain-damaged subjects. Images were rendered using BRAINVOX and encode lesion density by color (see scale). (a) Lesions of brain-damaged control subjects. (b) Lesions of subjects with unilateral amygdala damage. (c) Lesions of two subjects with bilateral amygdala damage: S. M. (top) and R. H. (bottom).

**Figure 2.** Examples of stimuli showing the whole face (a) and the eye region (b). Subjects were asked questions about both basic emotions (“Is it happy, sad, etc.?”) and social emotions (“Is it flirtatious, guilty, etc.”). Note that there are appropriate answers in both cases: In this example, the face can be described as happy and also as flirtatious, depending on the list of choices available.



from only a restricted region of the face. Following Baron-Cohen et al., we repeated our experiment twice: with whole faces and with only the eye region of the face (cf. Figure 2 for an example of the stimuli used).

## RESULTS

### Background Neuroanatomy and Neuropsychology

The neuroanatomical distribution of lesions of brain-damaged subjects is shown in Figure 1, demonstrating that lesions were restricted to anteromedial temporal lobe including the amygdala in the case of temporal lobectomies, and excluded the amygdala in the case of

brain-damaged controls. S. M. had bilateral damage restricted to the amygdala, whereas R. H. had bilateral amygdala damage as well as extensive damage to temporal cortex.

The neuropsychological background data (Table 1) showed that none of the subject groups and no individual subject within a group had visuo-perceptual impairments. All subjects performed in the normal range on the Benton Faces task, a sensitive measure of visuo-perceptual ability in processing faces. Subjects with unilateral amygdala damage were significantly younger ( $t = -5.7$ ,  $p < .0001$ ), but did not differ on performance IQ ( $t = -0.3$ ,  $ns$ ) or verbal IQ ( $t = -0.9$ ,  $ns$ ) from brain-damaged

**Table 1.** Background Demographic and Neuropsychological Data for the Brain-Damaged Subjects (Means and *SD*)

	<i>Gender</i>	<i>Age</i>	<i>Education</i>	<i>VIQ</i>	<i>PIQ</i>	<i>Benton</i>	<i>Aphasia</i>	<i>Depression</i>
L	7 females/9 males	38 ± 9	14 ± 2	101 ± 15	107 ± 13	44 ± 3	0	0.5 ± 0.6
R	7 females/7 males	34 ± 10	13 ± 2	94 ± 11	97 ± 16	43 ± 4	0	0.7 ± 0.9
BDC	25 females/22 males	52 ± 14	14 ± 3	101 ± 20	103 ± 14	45 ± 4	0.3 ± 0.6	0.2 ± 0.5
S. M.	female	32	12	86	95	42	0	0
R. H.	male	44	16	110	116	45	0	0

Subject groups are abbreviated as follows: L: left amygdala damage; R: right amygdala damage; BDC: brain-damaged controls; S. M. and R. H. are the two subjects with bilateral amygdala damage; VIQ/PIQ: verbal and performance IQ calculated from the WAIS-R or WAIS-III; Benton: score on the Benton Facial Recognition Task (in the normal range for all subjects); Aphasia/depression: composite scores for language impairment and residual depression. See Methods for details.

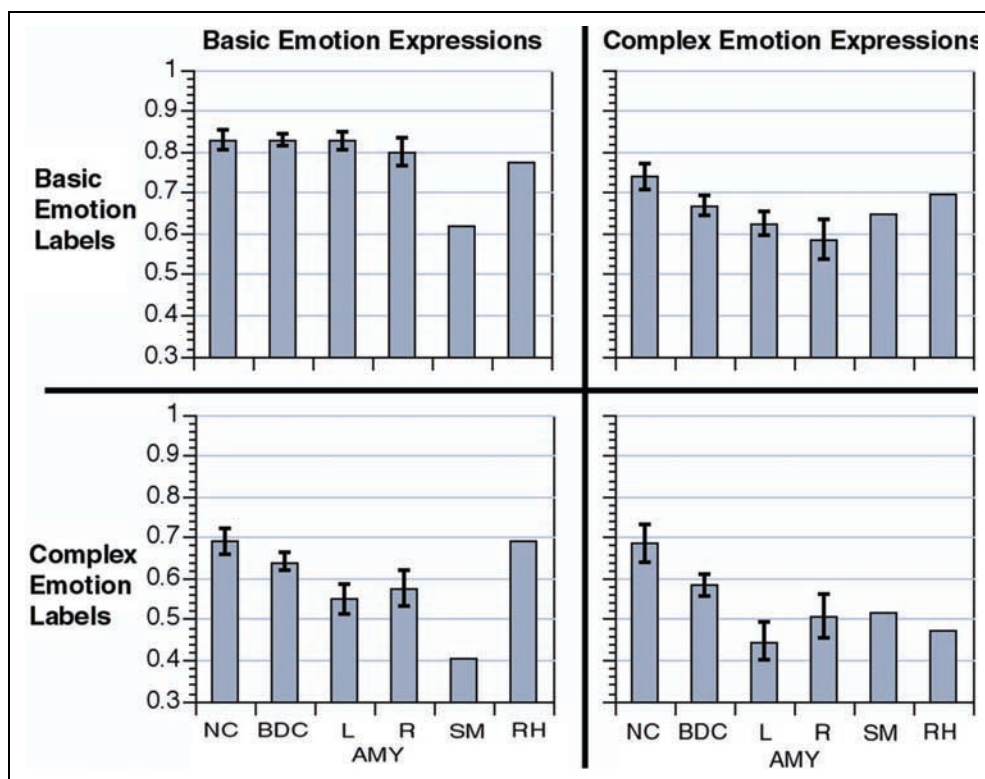
controls. Gender ratios, education, aphasia, and depression were similar across groups. We calculated the ANOVAs reported below also with a subset of brain-damaged controls of similar mean age to the subjects with unilateral amygdala damage to ensure that age differences could not account for the patterns of performance we observed: There were no differences in the direction or magnitude of the findings, and only a loss of statistical power due to smaller samples when such analyses were carried out, confirming that age differences do not account for the findings we report below.

### Recognition of Basic Emotions and Complex Mental States from the Whole Face

We first undertook analyses that corresponded to those in prior studies, namely, of the categories of basic

emotions and complex mental states. As noted, the design of the study allowed us to examine the effects of the emotion shown in the face and the emotion given in the label independently. As Figure 3 shows, all subject groups gave lower performances when asked to match faces to labels that stood for complex mental states than to labels that stood for basic emotions; likewise, subjects gave somewhat lower performances when matching faces expressing complex mental states than when matching faces expressing basic emotions. Brain-damaged controls performed identically to normal controls when recognizing basic emotions (83% correct when matching faces showing basic emotions with a list of the labels for the basic emotions; upper left in Figure 3), but had a lower mean accuracy score when recognizing complex mental states (58% correct when matching faces showing complex mental states

**Figure 3.** Mean performances in judging the whole face. The two columns show performances on two different subsets of the stimuli: those faces expressing only basic emotions and those faces expressing complex mental states. The two rows show performances according to the labeling that was required of subjects: Either assign a label to the face from a list of the basic emotions (top row) or from a list of labels for complex mental states (bottom row). Bars indicate means and *SEM*, from left to right, for normal controls (NC), brain-damaged controls (BDC), subjects with left (L) or right (R) unilateral amygdala damage (AMY), and the two subjects with bilateral amygdala damage (S. M. and R. H.).



with a list of the labels for these; lower right in Figure 3). Subjects with unilateral amygdala damage performed comparably to brain-damaged controls when recognizing basic emotions, whereas one of the subjects with bilateral amygdala damage (S. M.) had lower scores. When recognizing complex mental states, subjects with unilateral amygdala damage gave lower performance scores than brain-damaged controls, as did subjects with bilateral amygdala damage.

The above findings were examined in a  $2 \times 2 \times 2$  repeated measures ANOVA with factors of type of expression shown in the face (basic emotion or complex mental state), category of the label (basic emotion or complex mental state), and subject group (brain-damaged control vs. unilateral amygdala damage). Each subject thus had four scores in this analysis (corresponding to the four quadrants of Figure 3); subjects with bilateral amygdala damage were omitted. We found the following results: Across all subjects, performances on the faces showing complex mental states, or on the labels for complex mental states, were worse than on the basic emotion faces or on the basic emotion labels ( $ps < .0001$  for both faces and labels). Subject groups differed overall in that subjects with amygdala damage performed worse than brain-damaged controls ( $p < .005$ ). Most interesting were significant interactions of subject group and label ( $p < .01$ ) and subject group and face factors ( $p < .05$ ). Post hoc Scheffe corrected  $t$  tests showed that in both cases the difference between controls and amygdala subjects was not significant

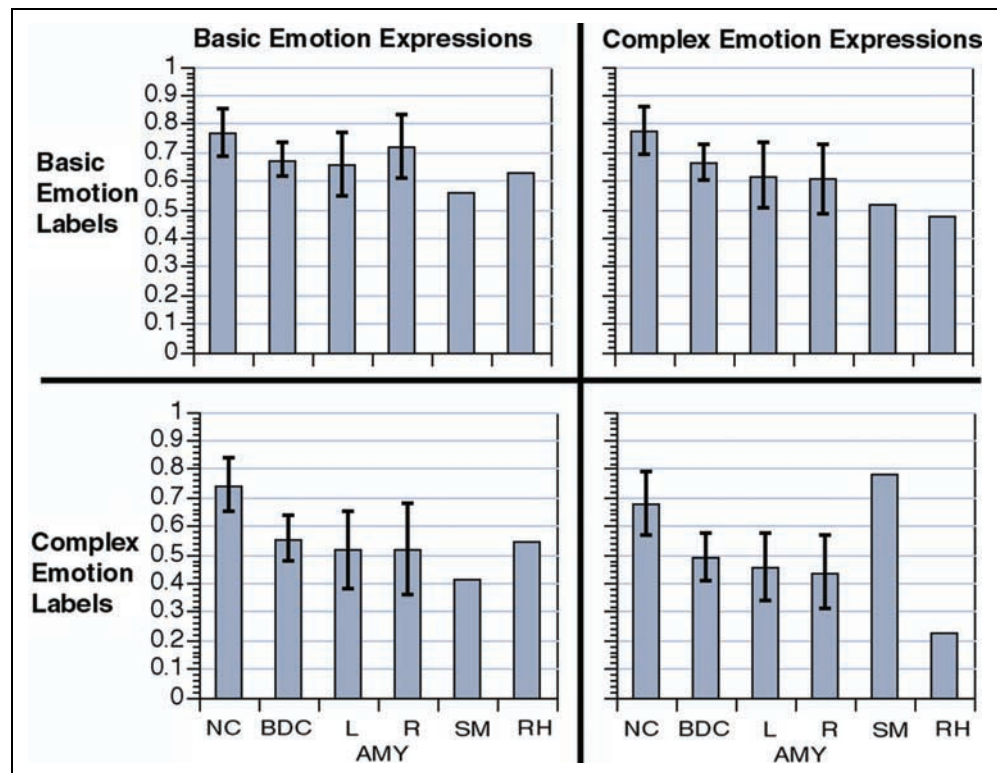
when comparing performances involving basic emotions ( $ps > .2$ ), but was highly significant when involving complex mental states ( $ps < .0001$ ). Although the Group  $\times$  Face  $\times$  Label three-way interaction was not itself significant, an examination of the pairwise comparisons of this interaction corroborated the above findings: Subjects with amygdala damage did not differ from controls when matching faces that expressed basic emotions to labels denoting basic emotions ( $p > .7$ ), but were significantly worse than controls when matching faces that expressed complex mental states to labels denoting complex mental states ( $p < .0001$ ).

### Recognition of Basic Emotions and Complex Mental States from the Eyes

In general, it is more difficult to recognize emotions from only a small region of the face, such as the eyes, than from the whole face. In our analysis, we controlled for this effect since correctness scores were calculated on the basis of the distribution of performances given by normal subjects to the stimuli. Figure 4 summarizes these data, in the same format previously shown for Figure 3.

Some general trends are evident in Figure 4. First, brain-damaged subjects in general perform somewhat worse than normal controls, as might be expected. In particular, brain-damaged subjects perform disproportionately worse when matching to labels for complex mental states, as we saw in the case of whole faces

**Figure 4.** Mean performances in judging the eyes only. Format and abbreviations are as in Figure 3.



(Figure 3). Of special interest, given the prior findings by Baron-Cohen et al. (1997) that recognition of complex mental states requires information about the eye region of the face, was a statistical comparison that included an examination of the whole face as compared to the eyes. We thus analyzed the data from both the eyes and the whole face with a  $2 \times 2 \times 2 \times 2$  repeated measures full-interaction ANOVA with factors of type of visual stimulus (whole face or eyes), emotion type expressed by the visual stimulus (basic emotion or complex mental state), emotion type given in the label (basic emotion or complex mental state), and subject group (brain-damaged control, unilateral amygdala damage). Again, subjects with bilateral amygdala damage were not included in the analysis. All factors showed significance at the  $p < .01$  level (group  $F = 7.3$ , label emotion  $F = 204$ , expression emotion  $F = 75$ , face/eye  $F = 34$ ). In particular, amygdala subjects performed worse than did controls, recognition scores for complex mental states were lower than for basic emotions, and recognition scores from the eyes alone were lower than recognition scores from the whole face. Furthermore, there were significant interactions of group with the label factor ( $F = 4.3$ ,  $p < .05$ ), with the facial expression factor ( $F = 4.7$ ,  $p < .05$ ), and with the eye/face factor ( $F = 4.5$ ,  $p < .05$ ).

Post hoc tests revealed some further patterns. From the Group  $\times$  Label interaction, subjects with unilateral amygdala damage performed worse than brain-damaged controls only when matching the visual stimuli to labels for complex mental states ( $p < .0001$ ), but not when matching them to labels for basic emotions ( $p > .05$ ); likewise; from the Group  $\times$  Facial expression interaction, subjects with amygdala damage performed worse than brain-damaged controls only when matching to visual stimuli expressing complex mental states ( $p < .0001$ ), but not when matching to those expressing basic emotions ( $p > .1$ , post hoc Scheffe corrected  $t$  tests in all cases). In regard to recognition from the eyes compared with the whole face, subjects with unilateral amygdala damage performed worse than brain-damaged controls only when processing whole faces ( $p < .0001$ ), but not when recognizing the eyes alone ( $p > .05$ ), a pattern that likely resulted from the fact that the means and variances in control performance were considerably worse for the eyes alone than for the whole face, compromising statistical power in detecting any significant differences between groups.

Although higher order interactions were not themselves significant, corrected pairwise contrasts from them corroborated the above patterns. The three-way interaction of Group  $\times$  Label  $\times$  Facial expression showed that amygdala subjects performed worse than brain-damaged controls when matching expressions of complex mental states to labels that stood for complex mental states ( $p < .0001$ ), when matching expressions of complex mental states to labels that stood for basic

emotions ( $p < .01$ ) or when matching expressions of basic emotions to labels that stood for complex mental states ( $p < .05$ ), but not when matching expressions of basic emotions with labels denoting the basic emotions ( $p > .9$ ). Especially noteworthy is thus the consistently impaired performance of subjects with unilateral amygdala damage, when asked to utilize information pertaining to complex mental states, either from the visual stimulus or from the label.

### **Is the Amygdala Specialized for Recognizing Social Emotions?**

The above analyses partitioned the stimuli into the two broad classes originally given by Baron-Cohen et al. (1997, 1999): basic emotions and complex mental states. However, as described in the Methods, it seems reasonable to subdivide the second category into states that would not normally be considered emotions (namely, interested, scheming, thoughtful, quizzical, bored) and those that would normally be considered social emotions, states that are emotions but that only make sense in an explicitly social relation (namely, arrogant, guilty, admiring, and flirtatious). Specifically, we investigated the hypothesis that amygdala damage might not impair the recognition of all complex mental states, but perhaps disproportionately impair recognition of social emotions.

For this analysis, full face and eyes alone stimuli were broken down into the two groups expressing basic emotions and expressing social emotions. Although all subject groups had lower mean scores on social as compared to basic emotions, subjects with unilateral amygdala damage performed close to brain-damaged controls on basic emotions, but were impaired when matching faces that express social emotions. We analyzed the data as before, with a  $2 \times 2 \times 2 \times 2$  repeated-measures ANOVA with factors of visual stimulus type (whole or eyes), emotion expressed in the visual stimulus (basic or social), emotion type provided in the label (basic or social), and subject group (brain-damaged control or unilateral amygdala damage). All factors showed significance at the  $p < .005$  level (group:  $F = 10$ , label emotion:  $F = 205$ , visual stimulus emotion:  $F = 26$ , face/eye:  $F = 50$ ). There was a significant Group  $\times$  Label interaction ( $F = 11.1$ ,  $p < .001$ ) but no other significant interactions.

The significance of the interaction term led us to carry out post hoc corrected  $t$  tests that were especially informative. When examining the Group  $\times$  Label interaction, there was no difference between controls and amygdala subjects when matching to labels for basic emotions ( $p > .3$ ), but amygdala subjects were significantly worse than controls when matching to labels for social emotions ( $p < .0001$ , Scheffe tests). Although none of the other interactions with group were significant, we examined pairwise contrasts with corrected  $t$  tests. For

the interaction of group with the emotion expressed, we found that amygdala subjects were significantly worse than controls when matching expressions showing social emotions ( $p < .0001$ ), but not when matching expressions of basic emotions ( $p > .1$ ). The three-way interaction of Group  $\times$  Label emotion  $\times$  Expression emotion summarizes these effects best: Amygdala subjects performed worse than controls only in one out of the four meaningful comparisons: when matching visual stimuli expressing social emotions to labels that stood for social emotions ( $p < .0001$ , all other  $ps > .2$ ). Moreover, this difference held up equally in regard to just the eyes ( $p < .0001$ ) or the whole face ( $p < .001$ , obtained from the four-way interaction among all factors).

As a final analysis, we compared, within each subject, how well the subject scored when matching expressions to labels for the basic or for the social emotions. Table 2 shows a summary of these difference scores for all the subject groups, for the eyes, and whole face, broken down this time with respect to expressions of basic emotions, expressions of social emotions (the four noted above), and expressions that were neither basic nor social emotions (i.e., the complex mental states minus the social emotions). Subjects with amygdala damage in general performed comparably or better than brain-damaged controls in regard to expressions of basic emotions, but showed impairments in regard to expressions of social emotions. Especially striking are the performances on the eye stimuli showing social emotions, for which subjects with either left or right unilateral amygdala damage, as well as both S. M. and R. H., were worse than brain-damaged controls in matching such stimuli to labels for the social emotions, than to

labels for the basic emotions. This pattern was not seen for the third category of expressions (the complex mental states minus the social emotions). In fact, for this latter class of stimuli, subjects with amygdala damage tended to perform better than brain-damaged controls. This result argues that the previous impairment we found in subjects with amygdala damage in recognizing complex mental states resulted not from a general impairment in recognizing all complex mental states, but rather from a more specific impairment in recognizing social emotions. To corroborate this impression, we carried out a  $2 \times 2 \times 3$  ANOVA with factors of subject group (brain-damaged control or unilateral amygdala damage), emotion type given in the label (basic or complex mental state), and emotion type expressed by the eyes (basic emotion, social emotion, or complex mental states other than the social emotions). We specifically examined the interaction of Group  $\times$  Emotion expressed by the eyes. Although this interaction term was not itself significant ( $F = 2.0, p = .1$ ), examination of its pairwise contrasts was very informative. Subjects with amygdala damage did not differ from brain-damaged controls when processing eyes that expressed basic emotions ( $p > .9$ ) or when processing eyes that expressed complex mental states that were not social emotions ( $p > .2$ ), but were significantly worse than brain-damaged controls when processing eyes that expressed social emotions ( $p < .05$ , Scheffe corrected  $t$  tests). It is thus only when processing information regarding social emotions, not when processing basic emotions or complex mental states that are not social emotions, that amygdala damage impairs recognition of facial expressions. Moreover, this specific pattern of

**Table 2.** Performance Differences (Means Shown) When Matching Visual Stimuli to Labels for Basic Emotions or to Labels for Complex Mental States

<i>Expression Category</i>	<i>Controls</i>		<i>Amygdala</i>		<i>S. M.</i>	<i>R. H.</i>
	<i>Normal</i>	<i>Brain-Damaged</i>	<i>Left</i>	<i>Right</i>		
<i>Eyes</i>						
Basic	0.02	0.12	0.13	0.2	0.03	0.08
Social	0.11	0.12	0.25	0.23	0.25	0.29
Other	0.09	0.18	0.09	0.13	-0.1	0.22
All	0.07	0.14	0.16	0.19	0.06	0.2
<i>Face</i>						
Basic	0.13	0.19	0.27	0.24	0.22	0.09
Social	0.10	0.18	0.29	0.25	0.51	0.56
Other	0.02	0.03	0.11	0.00	-0.12	0.00
All	0.09	0.14	0.23	0.17	0.17	0.16

The visual stimuli (eyes or whole face) are broken down according to their expression of basic emotion, social emotion, and other (nonbasic and nonsocial). The last row (all) gives the mean difference score for all stimuli. Positive numbers indicate that subjects performed better when matching the stimuli to labels for basic emotions than to labels for complex mental states.

impairment is evident when subjects perceive only the eye region of the face.

## DISCUSSION

All subjects in this study were neuroanatomically and neuropsychologically very well characterized, permitting us to exclude several confounds, such as systematic differences in neuropsychological background abilities. Specifically, differences in age, IQ, and visuo-perceptual abilities cannot explain the findings. Nor were the lesions of subjects with amygdala damage simply more extensive than those who did not have amygdala damage (an exception to this is subject R. H. who had bilateral amygdala damage as well as extensive damage to other temporal lobe structures). The statistically significant effects we report therefore should be robust indicators of real differences due to the site of lesion between subject groups and should permit inferences regarding the role of the amygdala in processing our stimuli.

We used an established set of stimuli (Baron-Cohen et al., 1997, 2001) to investigate the recognition of basic emotions, complex mental states, and social emotions from whole faces and eyes. The analyses showed statistically significant patterns of impairment in subjects with damage to the amygdala compared with brain-damaged controls who did not have amygdala damage. The main conclusions can be summarized as follows.

1. Amygdala damage impairs recognition of complex mental states more than it impairs recognition of basic emotions, on average. This impairment was evident either when matching to faces expressing complex mental states or when matching to labels denoting complex mental states (Figure 3). There were statistically highly significant differences demonstrating impaired recognition of complex mental states from faces in subjects with unilateral amygdala damage.

2. The above impairment in recognizing complex mental states was evident both when subjects were shown whole faces and when they were shown only the eye region of the face. Thus, recognition of complex mental states from the eye region of the face is disproportionately impaired in subjects with amygdala damage (Figure 4), a pattern of impairment that parallels impairments reported in subjects with autism (Baron-Cohen et al., 1997, 1999).

3. A further analysis showed that amygdala damage impairs recognition of social emotions (guilt, arrogance, admiration, and flirtatiousness), again both from the whole face and from the eyes. Subjects with amygdala damage showed a relative impairment in matching to labels of the social emotions compared with labels of the basic emotions that was striking only for faces or eyes expressing social emotions, but not for expressions of basic emotions or complex mental states other than the social emotions (interest, boredom, thoughtfulness, scheming, and quizzical) (Table 2).

The above set of findings supports our initial hypothesis that the amygdala is important to recognize social emotions from faces. Furthermore, they indicate that the impairment in recognition of social emotions we found is due not to a broader impairment in recognizing all complex mental states, but rather to a specific impairment in recognizing those complex mental states that are social emotions. We found this pattern of impairment both in subjects with unilateral and with bilateral amygdala damage. Our findings are in line with several studies reviewed in the Introduction, which implicate the amygdala in processing complex social stimuli and in regulating social behaviors, and we therefore suggest that the human amygdala may be relatively specialized to process explicitly social emotions rather than other information from facial expression.

A final issue of interest concerns the implications of our findings for a neurobiological understanding of autism. Arguably, the evidence for dysfunction in a particular structure as a contributor to autism is strongest in the case of the amygdala: Morphological, neurotransmitter-related, and functional imaging studies all support the so-called amygdala theory of autism (Baron-Cohen et al., 2000). An additional approach, which we took in a prior study, compares performances between people with autism and neurological subjects with gross lesions of the amygdala (Adolphs, Sears, & Piven, 2001). Although such concordant findings cannot unequivocally demonstrate a link between the amygdala and autism, they do suggest that such a link is likely to be at least part of the story, especially if the pattern of impairments that are shared is quite specific. The findings from the present study can be compared with those from Baron-Cohen et al. in people with autism. Like autistic people, subjects with amygdala damage showed impaired recognition of social emotions from the eye region of the face, a task that also activates the amygdala in normal individuals. Taken together, the results provide further support for the idea that amygdala dysfunction contributes to impaired social cognition in subjects with autism.

## METHODS

### Subjects

Subjects with both unilateral and bilateral amygdala damage participated in our study. A total of 30 post-operative subjects who had undergone unilateral temporal lobectomy for the treatment of epilepsy (16 left, 14 right) participated; all of these had single, focal, stable (nonprogressive) neurosurgical lesions confined to the anterior temporal lobe on one side, including part of the amygdala and all of temporal pole (Figure 1b). Although some of these subjects were taking anti-epileptic medications, post hoc investigations showed that there was no systematic association between either

dosage or type of medication and task performances in our experiments.

We also tested two subjects who had complete bilateral amygdala damage (Figure 1c): S. M. and R. H. S. M. is a 34-year-old woman with Urbach–Wiethe disease, which resulted in destruction of the entire amygdala as well as minor damage to anterior entorhinal cortex (Adolphs & Tranel, 2000; Tranel & Hyman, 1990). R. H. suffered from herpes simplex encephalitis, which resulted in complete bilateral destruction of all medial temporal lobe structures, encompassing both amygdalae, as well as regions of temporal neocortex (Tranel, Damasio, & Damasio, 2000; Adolphs et al., 1998).

Performances from subjects with amygdala damage were compared with those from 47 brain-damaged control subjects who had lesions distributed throughout various brain regions, but not in the amygdala (Figure 1a). Demographic and background neuropsychological information for all subjects is given in Table 1. All brain-damaged subjects were selected from the Patient Registry of the Division of Cognitive Neuroscience, and had been fully characterized neuropsychologically and neuroanatomically (Tranel, 1996; Damasio & Frank, 1992). All data presented here were collected during the chronic epoch from brain-damaged participants, after the individuals had achieved a stable cognitive profile (>4 months post lesion acquisition).

Normative data were obtained from a group of 19 normal controls with no history of neurological or psychiatric disease. All subjects had given informed consent to participate in these studies as approved by the Human Subjects Committee of the University of Iowa.

### **Background and Control Tasks**

For all brain-damaged subjects, we collected background neuropsychological data regarding IQ, basic visual perception, speech and language, and depression (Table 1). Specifically, data were derived from the following tasks.

#### *Verbal and Performance IQ*

These measures were obtained from the WAIS-R or WAIS-III for all subjects.

#### *The Benton Facial Recognition Task*

This task requires subjects to match the faces of identical individuals taken under different views and lighting conditions and provides a sensitive measure of basic visuo-perceptual function (Benton, Hamsher, Varney, & Spreen, 1983).

#### *Speech and Language Functioning*

Speech and language were assessed with the Multilingual Aphasia Examination (Benton & Hamsher, 1989)

and the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983). On the basis of data from these two instruments and on observations recorded in the neuropsychological reports, a neuropsychologist blind to the hypotheses of the current study rated each subject on a scale from 0 (“normal”) to 3 (“severe impairment”) in terms of speech and language functioning. These scores thus represent summary measures of the overall degree of speech/language impairment in each subject.

#### *Depression*

Depression was assessed by the Beck Depression Inventory (Beck, 1987), the MMPI (Hathaway & McKinley, 1951), and MMPI-2 (Butcher et al., 1989) as well as interviews with the subject and relatives, on the basis of which a neuropsychologist blind to the hypotheses of the current study rated each subject on a scale from 0 (“normal”) to 3 (“severely depressed”).

### **Experimental Tasks**

We used a set of stimuli that has been used previously with brain-damaged and normal populations (Baron-Cohen et al., 1997, 1999, 2001). Stimuli were photographs of the same woman showing various facial expressions. There were 20 different facial expressions. As described in the prior studies (Baron-Cohen et al., 1997, 1999, 2001), two different versions of these photographs were generated: (1) the section of the photograph showing the eyes and surrounding region only and (2) whole face. Examples of stimuli are given in Figure 2.

Baron-Cohen et al. had identified two categories of facial expression in their original studies: basic emotions and complex mental states. We consequently carried out two tasks in which subjects were asked to recognize these two categories. We thus showed subjects four blocks corresponding to the two types of stimuli and the two types of task: In blocks 1 and 2, subjects were shown 20 pictures of the eyes and were asked to match each picture to a list of basic emotions in one block (the labels listed under number 1 below) and to a list of complex mental states in the other block (the labels listed under number 2 below). In blocks 3 and 4, subjects were shown 20 pictures of the entire face under the two different task conditions. Blocks 1 and 2 were always shown prior to blocks 3 and 4 to avoid the possibility that the experience with the full face could prime judgment of the eyes seen alone.

For the purpose of data analysis, we divided the stimuli into three categories, two of them identical to the categories specified by Baron-Cohen et al. and used in the tasks above (basic emotions and complex mental states) and a third derived from one of the other

categories in order to provide a specific exploration of social emotions:

1. Basic emotions: There were 10 photographs showing basic emotions, as originally defined by Baron-Cohen et al. (1997): two each of happiness, anger, and surprise, and one each of fear, disgust, sadness, and distress. The rationale for this category was that the stimuli corresponded to the basic emotions found in other studies; with the possible exception of “distress,” they in fact correspond exactly to those six basic emotions shown by Ekman to be recognized cross-culturally from the face (Ekman, 1992, 1994) and proposed to rely on innately specified mechanisms (Ekman, 1973).

2. Complex mental states: There were 10 photographs corresponding to what Baron-Cohen et al. (1997) had termed “complex mental states”: two photographs of interested and one each of scheming, thoughtful, quiz-zical, bored, arrogant, guilty, admiring, and flirting. The rationale for this category was twofold: First, they were specified by exclusion—they were not basic emotions. Second, these were precisely those expressions shown previously to depend most critically on information signaled by the eye region of the face, and shown to be recognized abnormally by subjects with autism, suggesting that they draw on neural processes partly distinct from those used to process basic emotions.

3. Social emotions: There were four photographs showing social emotions, which were a subset of the stimuli in category 2: arrogant, guilty, admiring, and flirting. This was not a category previously used by Baron-Cohen et al. (1997), but one that we explicitly wanted to explore in the present study. The rationale for this category was that we wanted a set of emotions that depended on complex social context for their specification. Thus, basic emotions, such as fear, disgust, and so forth, while they certainly figure in social communication, can also arise outside of any social context (e.g., when falling off a cliff or smelling rotten eggs). By contrast, it is impossible to understand social emotions outside of a social context: They only arise in our interactions with other people.

All data were scored in relation to the relative frequencies of occurrence of responses given by normal control subjects. A subject always obtained a score of 1.0 on whatever label received the highest proportion of responses in the normal control group. Thus, if 100% of normal subjects called a happy face “happy,” a subject would get a score of 1.0 for choosing the label “happy” and 0.0 for all other choices. On the other hand, if 50% of normal subjects called a surprised face “surprise,” 40% called it “afraid,” and 10% called it “sad,” a subject would receive a score for that face of 1.0 if choosing the label “surprise,” 0.8 if choosing the label “afraid,” and 0.2 if choosing the label “sad.” In this way, correctness was made a parametric function solely of the distribution of responses that normal subjects gave to the face: High scores correspond to

relatively better performance, low scores to relatively worse performance.

## Acknowledgments

This work was supported by grants from the National Institute of Neurological Disorders and Stroke, the National Institute of Mental Health, and the Klingenstein Fund.

Reprint requests should be sent to Ralph Adolphs, Department of Neurology, Division of Cognitive Neuroscience, University of Iowa College of Medicine, 200 Hawkins Drive, Iowa City, IA 52242, USA, or via e-mail: ralph-adolphs@uiowa.edu.

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