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Processing Faces and Facial Expressions

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This paper reviews processing of facial identity and expressions. The issue of independence of these two systems for these tasks has been addressed from different approaches over the past 25 years. More recently, neuroimaging techniques have provided researchers with new tools to investigate how facial information is processed in the brain. First, findings from "traditional" approaches to identity and expression processing are summarized. The review then covers findings from neuroimaging studies on face perception, recognition, and encoding. Processing of the basic facial expressions is detailed in light of behavioral and neuroimaging data. Whereas data from experimental and neuropsychological studies support the existence of two systems, the neuroimaging literature yields a less clear picture because it shows considerable overlap in activation patterns in response to the different face-processing tasks. Further, activation patterns in response to facial expressions support the notion of involved neural substrates for processing different facial expressions.

KEY WORDS: faces; face recognition; facial expressions; emotion; neuroimaging; fMRI; PET.

Faces constitute perhaps the most important stimuli in social interactions. When we see a face, we need to infer two main types of information. First, the face has to be identified as belonging to a unique individual, taking into account transformations resulting from changes in viewing angle and facial expressions, as well as changes in appearance and aging. Second, the facial expression has to be interpreted for emotional context, which sets the tone for the social interaction. The relative ease and speed with which facial identity and facial expression processing are accomplished suggest the engagement of a highly specialized system or systems. Face recognition has been described as the acme of human visual perception, as such, we really appreciate the elegance and complexity of the system when it fails: when, for example, patients cannot recognize familiar faces or basic facial expressions. This pattern describes an important double dissociation: in some people, the ability to recognize facial expressions is intact but these people fail to identify the person bearing the expression; other people can identify the person but the ability to recognize expressions is impaired. This dissociation between facial identity and facial expression processing has been well documented and together with behavioral observations and experimental evidence led to the development of the well-known cognitive model of face recognition proposed by Bruce and Young in 1986. In the past decade, the study of face processing has benefitted from a new tool. Brain imaging has given cognitive psychologists new insights to the brain's inner workings, thereby changing the "brain black box" approach to cognition. Acronyms such as PET, fMRI, rCBF, BOLD, SPM, and ROIs[3] have become part of our "face" vocabulary. In this review we examine what the brain imaging approach has added to our understanding of the processes involved in face perception and recognition and the processing of facial expressions.

The paper is organized in the following manner. First, we summarize findings from neuropsychological studies of prosopagnosia which clearly established a dissociation between identity and emotion processing. We then move to briefly review findings from psychological studies which lead into a review of the Bruce and Young model of face

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[3]PET (Positron Emission Tomography), fMRI (functional Magnetic Resonance Imaging), rCBF (regional Cerebral Blood Flow), BOLD (Blood Oxygen Level Dependent), SPM (Statistical Parametric Mapping), ROI (Region of Interest).

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recognition. This model has served as a general framework for the study of face processing, not only from a psychological approach but also in event-related potential (ERP) studies, which will also be reviewed. In the second part of the paper, following a brief introduction to neuroimaging techniques, we will examine findings from imaging studies of face and facial expression processing.

A CLEAR DISSOCIATION: NEUROPSYCHOLOGICAL STUDIES OF IDENTITY AND EMOTION PROCESSING

The most striking support for different systems for processing facial identity and facial emotion comes from the double dissociation between recognition of people and identification of emotion. Losing the ability to recognize people results generally from focal damage to selective regions of the brain, although this impairment can also occur in the absence of any obvious brain damage. This condition is called prosopagnosia (from the Greek prosopon "face," and agnosia, "ignorance" or "lack of knowledge"). The neuropsychological literature describes a number of patients with deficiency in face recognition (for detailed reviews, see Damasio et al., 1982, 1990; De Renzi et al., 1991, 1994; Farah, 1991; Wacholtz, 1996; and Sacks, 1985, for a popular case study). The impairment can be so severe that prosopagnosic patients fail to recognize the face of close relatives and even of themselves, and instead rely on other cues such as voice, gait, and other characteristic features to recognize people. Yet, some prosopagnosic patients can still recognize and read emotional clues from faces, as illustrated, for example, by Tranel, Damasio, and Damasio (1988), Etcoff (1984), and Posamentier (2002), who describe prosopagnosic patients with normal performance in their ability to recognize basic facial expressions of emotion.

Generally, the literature distinguishes between "associative" and "apperceptive" prosopagnosia (Damasio et al., 1990; see also Farah, 1991). In "associative" prosopagnosia, the perceptual system seems adequate to allow for recognition, yet recognition cannot take place. Prosopagnosia of the "associative" type is generally caused by bilateral damage in inferior occipital and temporal visual cortices [inferior components of Brodmann areas (BA) 18 and 19], as well as damage to areas in the posterior temporal region (BA 37). In "apperceptive" face agnosia, recognition fails because of an impairment in visual perception. The patient cannot see the face normally, hence cannot recognize it. This type of prosopagnosia is associated with damage to the right visual association cortices within the occipital and parietal regions. Furthermore, the right visual cortices have been shown to be more important for face processing than the left cortices.

Although anatomical evidence from patients with focal brain lesions suggests that certain regions are indispensable for certain behaviors to occur, the interpretation of such data is delicate because the brain becomes reorganized as a result of the damage. Localizations of specialized functional areas are also difficult to assess precisely because brain damage typically affects large and diffuse areas. However, considering prosopagnosia as a pure face disorder remains controversial because several prosopagnosic subjects exhibit also difficulties in object recognition. Therefore some researchers argue that prosopagnosia results from deficits in within-category discrimination (Damasio et al., 1982; Gauthier et al., 1999a). Nevertheless, evidence from neuropsychological studies of prosopagnosic patients does strongly point to the existence of dissociable systems involved in the processing of facial identity and emotion as signaled by facial expressions.

PSYCHOLOGICAL STUDIES

Early experiments in face processing were not explicitly designed to investigate differences in processing of facial identity and facial expressions, but they rather examined the effects of changes in appearance, viewing angle, and expressions on face recognition. As a classic illustration, two experiments by Patterson and Baddeley (1977) investigated the effects of changes in appearance of unfamiliar faces from presentation to test on face recognition. They found that a moderate change in both viewing angle and facial expression (i.e., from frontal unsmiling to 3/4 view smiling) between learning and test did not significantly affect recognition performance. But a change of pose from frontal to profile view clearly impaired recognition. When the target faces displayed changes in appearance, such as hairstyle or facial hair, recognition became seriously impaired. Davies et al. (1978) also showed that subjects' recognition performance was not significantly affected by smaller changes in viewing angle (from frontal to 3/4 and vice versa). These early studies suggested that a single view of a face may contain enough invariant information to allow recognition despite moderate changes in pose and expression. However, in another experiment, Baddeley and Woodhead (1981) observed lowered recognition when changes in pose occurred between learning and testing. Along the same lines, Ellis et al. (1979) suggested that repeated interaction with a face may lead to the establishment of a structural code that emphasizes the internal features of the face. Then, because familiar and

unfamiliar faces differ in their representation, Ellis et al. theorized that these two types of faces need to be analyzed differently. As such, any face-processing theory must integrate familiarity as a factor.

In a departure from earlier studies in face recognition that involved presenting pictures of unfamiliar faces, Bruce (1982) decided to use both familiar and unfamiliar faces to examine the effect of familiarity on recognition accuracy and response latency when the view of faces changed between learning and testing. In one experiment, all the faces were unfamiliar to the subjects. These unfamiliar learned faces were presented at test either unchanged, with a change in viewing angle (frontal to 3/4), in expression (smiling to unsmiling), or in both angle and expression. In agreement with previous findings, Bruce found that unchanged faces were recognized more accurately and faster than faces with a change in viewing angle or expression, which in turn were recognized better than faces with changes in both viewing angles and expressions. In a different experiment, Bruce also examined the effect of familiarity with faces by having subjects learn both familiar and unfamiliar faces. At test, all the faces were tested either unchanged or with a change in both angle and expression. Unfamiliar faces were recognized more slowly and less accurately when a change had occurred, whereas familiar faces were recognized at a slower rate but not less accurately. The results were interpreted as suggesting that subjects' ability to encode any invariant features of a face is limited by exposure time and also influenced by encoding instructions. If an unfamiliar face is tested in a different view, recognition is dependent upon the similarity between the new picture and the old original picture, or upon the extraction of invariant features. Bruce suggested that the critical difference between familiar and unfamiliar faces is that familiar faces are already represented structurally and semantically in long-term memory.

Casting findings from behavioral and neuropsychological observations and the experimental approach into a unified framework, Bruce and Young (1986) developed a now classic model of face recognition expressed in terms of processing pathways and modules for recognition of familiar faces (see Fig. 1). They suggested that seven types of information can be derived from faces: pictorial, structural, visually derived semantics (age and sex), identity-specific semantics, name, expression, and facial speech (movements of the lips during speech production) codes.

The pictorial code provides information about lighting, grain, and possible imperfections in a photograph, as well as information about pose and facial expression. In other words, the pictorial code corresponds to the 2D image of the face. The pictorial code can mediate recognition tasks in studies of episodic memory for faces because the same pictures of previously unfamiliar faces are used at learning and at test. The structural code captures the aspects of the configuration of a face that distinguishes it from other faces. It corresponds to a 3D (view invariant) representation of the face. Bruce and Young distinguished between the structural code for already familiar faces and the tenuous structural code for unfamiliar faces, which is limited to the information that can be extracted from an initial exposure, such as whether the face was seen with varying poses and/or facial expressions. The Bruce and Young model assumes that facial expressions, except for possible characteristic expressions, are not important for face recognition. Facial expressions are treated as view-centered descriptions that vary across encounters with the face, and are consequently analyzed separately by the cognitive system for their affective content. A more abstract, expression-independent description of the face is the input to the face recognition units (FRUs), which contain the structural codes for faces. When a face is presented for recognition, the facial expression is parsed out and the expression-independent description or "normalized" face is forwarded to the FRUs for further identification. The model therefore assumes functional independence between identity and expression processing, and further distinguishes between the processes involved in recognition of familiar and unfamiliar faces.

The Bruce and Young model provided researchers with a general framework for face processing. For example, the Bruce and Young model predicts that processing of familiar faces should be automatic and rapid, whereas processing of unfamiliar faces should require more effort and time. Additionally, the model predicts that judgments about facial expressions should not be influenced by the familiarity of the face. Such predictions were tested by Young et al. (1986), who examined the effect of face familiarity in identity and expression matching tasks. In an identity-matching task, subjects had to decide whether simultaneously presented photographs of faces were pictures of the same or different persons. In agreement with the Bruce and Young model, subjects were faster to decide that the persons were the same when they were familiar than when they were unfamiliar. In an expression-matching task, subjects had to decide whether people in photographs displayed the same or different facial expressions. Again, the prediction of the model held up: There was no difference in reaction times for expression judgment of familiar and unfamiliar faces. These results support independence in processing facial identity and expressions.

However, some studies have challenged the assumption of the Bruce and Young model that facial expressions and identity are processed independently. Endo et al.

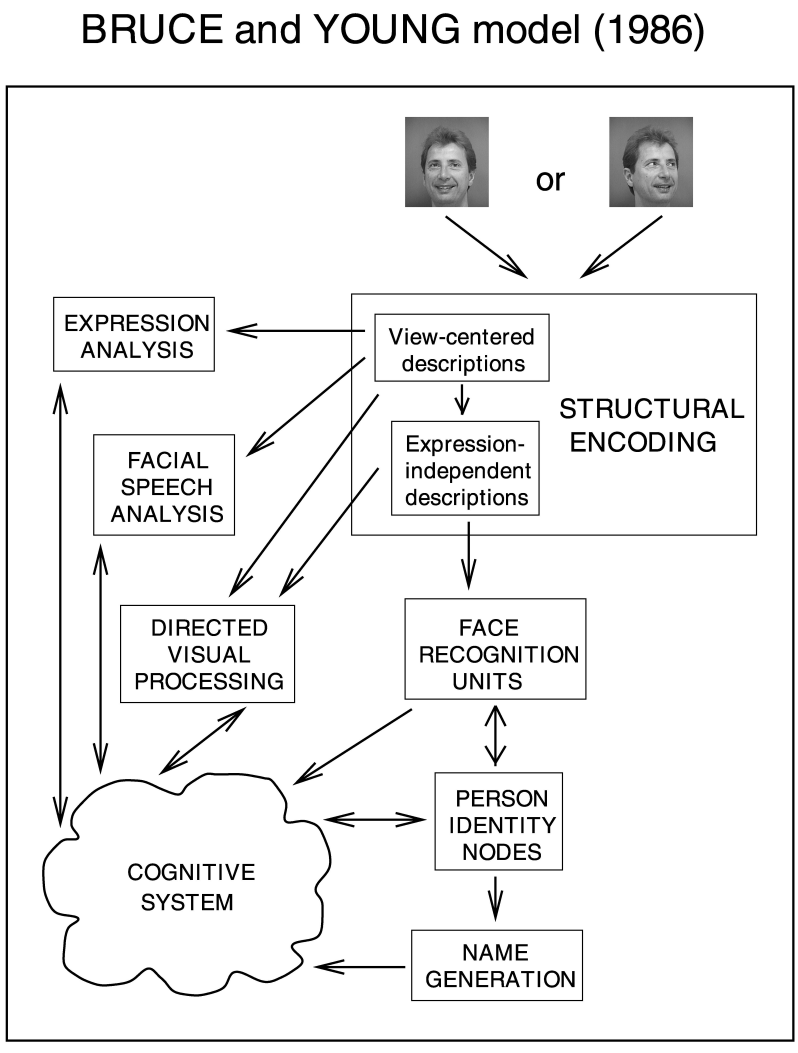

Fig. 1. The Bruce and Young model of face recognition (from Bruce and Young, 1986).

(1992) presented subjects with familiar and unfamiliar faces with three different facial expressions (neutral, happy, and angry). Familiar faces were recognized faster when displaying a "neutral" expression than when displaying a "happy" or an "angry" expression. In another experiment, faces of well-known people with "neutral" and "happy" expressions were used as familiar faces. "Happy" faces were recognized faster than "neutral" faces. Sansone and Tiberghien (1994) also report an effect of facial expressions. Subjects learned faces by viewing five images of each face. In the "unique" encoding condition, subjects saw the face with the same facial expression repeated five times. In the "mixed" encoding condition, the face was shown with the same facial expression presented four times and one additional image with a different facial expression. When subjects were presented with a different facial expression at test, the recognition rate for faces learned in the "unique" encoding condition was lower than that for faces learned in the "mixed" condition. These results point to an effect of facial expression on recognition, and therefore disagree with the Bruce and Young model, which predicts that changes in facial expressions should not affect recognition. But the subjects in the study reported by Sansone and Tiberghien also learned repeated images of the face, which is a departure from other face recognition studies where only one image is usually learned. Seeing a face repeatedly or seeing the face with different expressions may allow subjects to extract more of an invariant representation of the faces at learning. This notion was suggested as early as the late sixties by Dukes and Bevan (1967), who suggested that repeated exposures to different views of unfamiliar faces may cause human

observers to extract some invariant information about the face, thereby allowing for better recognition from a new view of the face.

To summarize, the emergent picture from psychological studies indicates that the factors that contribute to successful face recognition are quite complex. Some studies show that moderate changes in viewing angle and facial expressions do not affect recognition accuracy. Other work shows that changes in facial expression and angle do affect recognition of unfamiliar faces whereas recognition of familiar faces is not affected by such changes. This suggests that different processes are engaged in the recognition of familiar and unfamiliar faces. Identification/recognition of familiar faces appears to be relatively robust against changes in viewing angle, whereas unfamiliar faces are affected by rotation of the head. But whether identity and expressions are processed independently is still not answered satisfactorily because several of these studies manipulated both changes in viewing angle and/or facial expressions.

NEUROPHYSIOLOGICAL STUDIES

The earliest neurophysiological investigations into face processing were done using single-cell recordings in the temporal cortex in monkeys. Such studies have found that cells are preferentially tuned to the identity and expression of the face. Although the results from these studies cannot be taken as direct evidence of similar processes in humans, they provided direction for further studies with human populations.

Evidence for face-specific processing from single-cell recordings in monkeys has been established in a number of studies, as reviewed by Perrett et al. (1992), and Desimone (1991). Populations of cells in the fundus of the superior temporal sulcus were shown to be selectively responsive to human and monkey faces. Most cells responded equally to faces of different individuals across different orientations (in particular frontal and profile views), but some cells also showed of sensitivity to the identity of specific faces. Cells also responded to face components, but not to other complex stimuli such as snakes, spiders, and food. Further, the cells were more sensitive to natural-looking stimuli than line drawings or schematic representations. The responses to inverted faces were weaker with longer latency than to upright faces.

Single-cell recordings have also shed further light on the presumed independence of facial identity and expressions. Hasselmo et al. (1989) investigated the role of expression and identity in face-selective responses of neurons in the temporal visual cortex of macaque monkeys. Hasselmo et al. recorded the responses of 45 neurons to a stimulus set of pictures of three monkeys displaying three different facial expressions (calm, slight threat, and full threat). Fifteen neurons responded to identity independently of expression, and nine neurons responded to the different expressions independently of identity. Hasselmo et al. further found a differential response to the different expressions, with a stronger response to expressions of full threat. The neurons responsive to expressions were found primarily in the superior temporal sulcus, whereas neurons responding to identity were located in the inferior temporal gyrus. Although most single-cell responses have been recorded in the visual cortex, this is not the only area that has shown specific responses to faces. Leonard et al. (1985) found population of cells in the amygdala of macaque monkeys to be also responsive to faces.

From primate studies we then have an emergent picture of neuronal activation patterns in response to faces in the temporal region of the brain. Would a similar pattern be observable in human populations? Single cell recordings in humans are usually done on epileptic patients who undergo craniotomies in an attempt to find the location of their seizures. In the sixties, Penfield and Perot (1963) used this technique to chart large regions of the cerebral cortex and gave use of the now famous picture of the homunculus illustrating how the body surface is represented on the brain surface. The first single-cell recordings of face processing in humans were conducted by Ojemann et al. (1992) on 11 patients. Neuronal activity was measured in 21 neural populations at 13 sites in the right superior and middle temporal gyri. Identity matching and expression labeling tasks showed significant neural activation changes of 62% and 52%, respectively. Further, the facial expression task showed more localized response patterns in the middle temporal gyrus, suggesting some specificity of neuronal responses to faces. These findings parallel Hasselmo et al.'s findings of differential activation patterns (Hasselmo et al., 1989) to identity and expressions in primates.

In the decade since Ojemann et al.'s study, several groups have used ERPs to determine both the time course and localization of face processing. Some of these studies have been done intercranially on epileptic patients being evaluated for surgery, whereas other studies have used scalp recordings on normal subjects. As we will see, electrophysiological recordings have shown a consistent pattern of results.

In three papers, Allison, McCarthy, Puce, and colleagues report their findings from a number of electrophysiological studies of face processing in a large group of epileptic patients. The 98 subjects included in these studies had electrodes placed directly on the cortical surface

to monitor epileptic seizures. In the first paper, Allison et al. (1999) established the existence of differential ERPs generated by faces, sinusoidal gratings, objects, and letter strings. Compared with other categories, the response to faces is characterized as complex and reflected by at least four types of activity in the ventral occipitotemporal region (N200, P290, P350, and N700, of which the N200 is further explored in the next two papers of the series). The responses to faces were found bilaterally but with a slightly higher response in the right hemisphere.

In the second paper in the series, McCarthy et al. (1999) examined the face-specific responses in greater detail. In summary, they found the previously established N200 response to be insensitive to color, size, and spatial frequency manipulation. Inverted faces evoked a later N200 amplitude than upright faces. The right hemisphere responded faster to upright faces, whereas the inverted faces evoked a more rapid response in the left hemisphere. Additionally, the N200 amplitude was largest for full faces and decreased when eyes, face contours, lips, and noses were presented in isolation. Faces with averted eye gaze or closed eyes evoked a larger potential than straightforward-looking faces. There was no difference in the response to full and three-quarter views of face, but the amplitude for these views was significantly larger than the response to profile views.

The work reported in the third paper in the series (Puce et al., 1999) investigated the influence of top-down processing by examining the effect of emotionally charged stimuli, repetition, priming, familiarity, and face identification. The N200 amplitude was significantly larger for faces than affective stimuli. The N200 did not show an effect of habituation nor semantic priming, and was also unaffected by familiarity with the presented faces. Taken together, the N200 response appears to be relatively constant for faces, as well as invariant to experimental manipulations. The authors suggest that the N200 is the earliest evidence for face-specific processing and may reflect an instantiation of the "structural encoding" stage of the Bruce and Young model.

Using scalp recordings, a number of studies have also examined electrophysiological responses to faces in normal populations. A consistent finding across studies is a negative evoked potential around 170 ms in response to faces. In a series of experiments, Bentin et al. (1996) used scalp recordings on normal subjects to examine the face-specific response properties established in the subdural recordings reviewed above. Bentin et al. found that faces consistently evoked a negative potential around 170 ms (N170), which was absent for other nonface stimuli. The N170 was largest in the posterior temporal scalp in the right hemisphere. The response to inverted faces was delayed, but the amplitude was the same as for upright faces. Similar response to inverted faces has been reported by Rossion et al. (1999, 2000), Eimer (2000a), and Itier and Taylor (2002), as well as McCarthy et al. (1999) as reviewed above. Bentin et al. also report that distorted human faces elicited an N170 amplitude similar to that for normal faces. When presented in isolated, eyes actually elicited a larger N170 than whole faces, whereas noses and lips elicited later and smaller negative ERPs. Bentin et al. also cast the scalp recorded N170 as the "structural encoder" suggested by Bruce and Young, which is also the interpretation of the subdurally recorded N200. The relationship between these two responses is uncertain. One could speculate that the placement of the electrodes (subdurally on the fusiform gyrus vs. the T6 location on the scalp) could account for the differences. Another interpretation takes into account the sensitivity of the N170 to eyes alone. This may reflect the activation of an eye-sensitive region, which possibly serves as an orientation mechanism for the "face structural encoder."

Bentin and Deouell (2000) also examined a possible effect of top-down processing in the encoding of faces. In one experiment, subjects viewed pictures of famous faces, nonfamous faces, and butterflies. Subjects responded only to the butterfly stimuli, which were used as targets. Therefore, no explicit processing was done on the face stimuli. The N170 did not show a familiarity effect as famous and nonfamous faces evoked similar patterns of scalp distribution and amplitude. But an effect of familiarity was observed later, at around 350 ms, with familiar faces eliciting a more negative response, which was termed the N400 response. In a second experiment, subjects were asked to process explicitly the faces in an identification task. The N170 again did not differentiate between familiar and unfamiliar faces and was largest in the lateral parieto-occipital region of the right hemisphere. A difference between familiar and unfamiliar faces was again observed with a larger latency, where familiar faces evoked a significantly larger negative response at N400. Bentin and Deouell interpret this N400 to reflect the activation of semantic memory or the "personal identity nodes" in the Bruce and Young model. Similar results were also observed by Eimer (2000b). Integrating the results from their ERP studies as well as neuroimaging studies (which will be reviewed in detail later), Bentin and colleagues (Bentin and Carmel, 2002; Carmel and Bentin, 2002) propose a model for face processing where visual stimuli, here faces, that include physiognomic information are distinctively integrated into a perceptual representation. This process is performed by a complex neural mechanism that process faces holistically but also is able to detect physiognomic features (e.g., eyes). The global processing is

accomplished by a neural network located in the middle fusiform gyrus, whereas componential processing is performed by networks located in the superior temporal sulcus and the posterior inferior temporal gyrus. The subdurally recorded N200 reflects the global processing in the fusiform gyrus. The N170 is also associated with global processing as well as component-oriented activity. Bentin thus favors a domain-specific interpretation of the N170 activity as it relates to faces. In brief, Bentin builds his argument for domain specificity on the literature that supports a view of faces as being "special." For example, infants show preference for face-like visual stimuli over other (cf. Morton and Johnson, 1991), faces are processed as a whole (holistical or configural processing), whereas objects are processed as a set of features (cf. Farah et al., 1998), recognition of inverted faces, but not objects, is impaired (Yin, 1969). However, this view of face processing as being special is not unequivocally endorsed. Gauthier and colleagues in a series of publications have strongly argued that faces do not hold a special status (Gauthier and Tarr, 1997; Tarr and Gauthier, 2000; Rossion et al., 2002). Instead they view face processing as a case of within-category discrimination of stimuli that subjects have expertise in processing. These diverging opinions of domain specific vs. subordinate-level expertise account of face processing will be discussed in more detail when reviewing neuroimaging studies that have examined the response to faces in the fusiform gurus.

Event-related potentials have also been used to study processing of facial expressions. In a study designed to examine both processing of facial identity and facial expressions in normal subjects, Münte et al. (1998) recorded ERPs from multiple scalp locations to assess the timing and distribution of effects related to the processing of identity and expressions. The earliest ERP differences in an identity matching task were found around 200 ms. The earliest ERP effects in an expression matching task came in later, around 450 ms. The tasks also differed in their localization or scalp distribution: Identity was associated with a fronto-central effect, whereas expression was associated with a centroparietal effect.

Streit et al. (2000) evaluated differences in ERPs in emotional and structural face processing. In the emotional processing task, subjects were asked to identify which one of six facial expressions was present on the face. In the structural encoding task, blurred images of faces and five other categories were presented and subjects had to identify the category of each stimulus. Pictures of facial expressions and blurred facial images evoked similar event potentials around 170 ms, which should be specifically related to processing of facial stimuli and thus replicating previous findings. However, the facial expressions decoding task evoked a peak around 240 ms, whereas such a response was absent for the blurred facial images. According to the authors, this peak latency of 240 ms might then represent specific processes underlying the decoding of facial expressions. This 240-ms peak is different from the time sequence reported by Münte et al. for expression matching. The discrepancy may be due to task differences: a matching task is probably more effortful than an encoding task and would therefore take more time. Herrmann et al. (2002) replicated these findings.

Taking into account that prosopagnosia is characterized by a disproportionate deficiency in face recognition compared with other stimulus categories, we should expect to observe different patterns of evoked response potentials in prosopagnosic subjects. This idea has recently been supported by findings from two studies. First, Bentin et al. (1999) examined face processing in a developmental prosopagnosic subject. In a group of normal control subjects, faces exclusively evoked the now familiar N170. But in the prosopagnosic subject, the N170 was elicited by both faces and objects, thus showing no selectivity. The nonselective N170 suggests that the impairment is at the level of structural encoding or reflects a failure to extract face-specific information as input to FRUs. In addition, Eimer and McCarthy (1999) found a similar pattern in a severely prosopagnosic subject who was compared with 24 normal controls. The control subjects showed an enhanced N170 in response to faces as compared with houses. This effect was absent in the prosopagnosic subject whose N170 did not discriminate between faces and houses. When presented with inverted faces and houses, the N170 effect was present for the control subjects, but absent in the prosopagnosic subject. Thus, the nonselective N170 and the absent N170 in these two prosopagnosic subjects indicate a dissociation between face and nonface processing. Neither of these studies examined ERPs in response to facial expression processing which usually remains intact in prosopagnosic subjects. This is unfortunate because preserved emotion processing still provides the strongest evidence for a dissociation between the processing of identity and emotion.

In summary, findings from ERP studies have revealed dissociations between various face-processing tasks that can be cast in the framework of the Bruce and Young model. The N170 shows the dissociation between processing of face and nonface stimuli and is thought to be the instantiation of the processes involved in the structural encoding of faces. Further, the Bruce and Young model predicts differential processing of familiar and nonfamiliar faces, which is supported by the modulation of later responses. The N400 response, in particular, is thought to reflect the activation of the personal identity nodes.

Additionally, the different time course for processing the emotional content from a face suggests that different processes underlie the processing of facial identity and facial expressions, which is a dissociation clearly evidenced in prosopagnosic subjects.

So far, we have accumulated evidence from neuropsychological, behavioral, experimental, and neurophysiological approaches to face processing that support dissociations between the various types of information that can be extracted from faces, in particular identity and emotion. The next sections of the paper will review findings from the relatively new field of brain imaging that has already made significant contributions to our understanding and knowledge of how faces and facial expressions are processed.

BRAIN IMAGING OVERVIEW

The last decade has seen rapid developments in functional neuroimaging methods which provide a measure of the brain at work. Positron Emission Tomography (PET) and functional Magnetic Resonance Imaging (fMRI) are the two main techniques used to image face-processing tasks. Any change in behaviors and cognitive demands causes a change in neural activity. Neural activation can be measured indirectly through changes in the hemodynamic response or certain properties of the blood, such as blood flow or oxygen content. PET uses a radioactive tracer to measure rCBF, whereas fMRI measures changes in the oxygen content in the blood through the Blood Oxygenation Level Dependent contrast mechanism, referred to as the BOLD signal. There are pros and cons to both imaging methods. PET requires the injection of a radioactive tracer and this limits the number of scans that can be obtained from a given subject. Being a noninvasive technique, fMRI is considered safe for both children and adults, but the environment in the scanner is quite noisy and restrictive. The fMRI signal is also extremely sensitive to motion of any kind. With PET, the signal averages approximately 10 mm in spatial resolution and 30 s to 1 min in temporal resolution. High-speed fMRI (so-called echo planar imaging) gives both better spatial and temporal resolution, with 3–8 mm and 100 ms respectively. Whereas techniques such as ERPs can detect neuronal responses as early as 170 ms after stimulus onset, as we saw in the case of faces, the time course for BOLD signal is actually rather sluggish. Using current fMRI technology, the first detectable change in hemodynamics appears after a delay of 2 s, reaches its peak around 7–10 s, and remains elevated as long as the activity continues, and returns to baseline 8–11 s after the activity ceases. In a recent paper, Logothetis et al. (2001) report on a study that simultaneously recorded intracortical neural signals and fMRI responses in anesthetized monkeys' visual cortex. The BOLD signal actually correlated most strongly with the local field potentials (LFPs), not single- or multiunit activity which represents "neural spiking" (neurons firing or action potentials). The LFPs reflect the aggregate activity from a population of neurons located in close vicinity of the recording electrode tip. Logothetis et al. conclude that their results unequivocally show that an increase in the BOLD contrast directly and monotonically reflects an increase in neural activity, and propose further that the statistical analysis and thresholding methods currently applied to fMRI data probably underestimate the neural activity related to a stimulus or task. In a commentary, Bandettini and Ungerleider (2001) acknowledge the findings by Logothetis et al. as landmark, but caution consideration because the results were obtained in anesthetized monkeys, and that the variance explained by the LFP was only 7.6% larger compared with multiunit activity. A further discussion of the BOLD signal comes from Attwell and Iadecola (2002), who consider the hemodynamic response to be driven by neurotransmitter-related signaling and not directly by the local energy needs of the brain. These new insights into the interpretation of the BOLD contrast are important, but whether these results would require a complete reinterpretation of past fMRI results would probably be taking these findings too far. Clearly, as will be evidenced in the following review, the brain does show different patterns of activation in response to different types of stimuli and tasks.

During a typical PET or fMRI scanning session, subjects attend to repeated blocks of various types of sensory, motor, or cognitive tasks for periods lasting up to several minutes. However, with increasingly more refined scanning technology and better temporal resolution of fMRI, random stimulus presentation or event-related fMRI is used increasingly in experimental designs. The concept of random stimulus presentation is quite significant, because the ability to analyze the response to randomly presented stimuli allows for the import of traditional experimental paradigms into imaging studies. Additionally, one also can hope to avoid the possible confounds of habituation and possible strategies adopted while attending to blocks of repeated stimuli (Buckner and Logan, 2001; D'Esposito et al., 1999).

Before delving into the brain imaging literature on face processing, we present a brief overview of the statistical techniques currently used in the analysis of imaging data. Prior to statistical analyses, the brain images go through a series of preprocessing steps that includes coregistration, spatial normalization, and smoothing. In general,

the areas of activation are reported by giving the coordinates of a standard brain space called the Talairach and Tournoux coordinates (Talairach and Tournoux, 1988). Several strategies can be used to perform the statistical analyses of brain imagery data. These include a voxel- (a 3D pixel) by-voxel-based analysis [e.g., as implemented by SPM, cf. Friston et al., 1995; Friston, Holmes, Poline, Price, and Frith, 1996a], an ROI approach, and subtraction. In a voxel-by-voxel-based analysis, only those voxels that exceed a set criterion value are kept in the subsequent contrast analyses. A Bonferroni correction is routinely performed to correct for multiple comparisons by dividing the α level by the number of voxels. When applied to a complete brain image, this correction drastically reduces the power of the statistical test (i.e., $\alpha = 0.05/40,000$ voxels gives a new significance level of 0.000,001,25). When researchers have a specific hypothesis as to what regions of the brain should activate, they can increase the power of the test by defining *a priori* an ROI, which is a subset of voxels covering a specific brain area. This reduces the number of voxels subjected to statistical testing and so increases statistical power. Another way to identify patterns of activation is to use the subtraction method. The basic premise of so-called cognitive subtraction is that a cognitive task is made up of separable, but additive components. By holding all but one component constant, any observed changes in activation should be due only to the experimental manipulation of the factor of interest. The signal or pattern of activation is averaged on a voxel-per-voxel basis for each experimental condition and the two averages are subtracted. Ideally, the resulting image should then reveal neural activation by the factor of interest. For example, in assessing activation in response to fearful facial expressions, the activation observed when presenting neutral expressions would be subtracted from the activation by fearful expressions. The activation pattern obtained from the subtraction should then reveal the areas most active in processing fearful expressions. Despite its wide use, it should be mentioned that the basic premise of cognitive subtraction has been questioned by researchers who suggest that the brain is not a simple linear system with response properties consistent with pure insertion and additivity (Friston et al., 1996b; Price et al., 1997; Sidtis et al., 1999).

To date, a number of brain imaging studies have examined neural activation patterns in response to a variety of face-processing tasks. So far, we have established that in particular the middle region of the fusiform gyrus is involved in the perception of faces, but we still do not have a complete picture of which structures or functional systems are involved when having to recognize our frowning neighbor with a new hairstyle. The next two sections of the paper will review findings from brain imaging studies that examine neural activation patterns in response to faces and facial expressions.

BRAIN IMAGING STUDIES OF FACES

One of the earliest works to use faces as stimuli was a PET study conducted by Haxby et al. in 1991, who showed a dissociation between object (in this case, faces) and spatial visual processing. Face matching activated a pathway in the ventral/occipitotemporal cortex, whereas location matching activated a dorsal/occipitoparietal pathway. One of the first, and by now, widely cited, neuroimaging study to deliberately examine activation by different face-processing tasks was conducted by Sergent et al. (1992), who identified specific regions activated by faces. Since then, several research groups have used both PET and fMRI to investigate various aspects of face processing. In particular, the research groups led respectively by Kanwisher and Gauthier have examined the role of the fusiform gyrus in early face perception and these two groups have ended up with divergent interpretations. The results from these studies, as well as other studies that have examined more complex face-processing tasks, such as encoding, recognition, and the effect of face familiarity, will be evaluated in turn. Finally, a recent model of face processing based on brain imaging data proposed by Haxby and colleagues will be introduced.

Face Perception

Lesion studies of prosopagnosic subjects had early implicated the importance of the occipital and temporal lobes in face processing. In 1991, Haxby et al. used PET to show that faces did activate the occipitotemporal region. However, the functional aspects of these areas related to face processing had not yet been properly defined. Using PET, Sergent, Otha, and MacDonald's study directly examined the functional neuroanatomy of face and object processing (Sergent et al., 1992). They measured rCBF in six normal adults who performed a number of tasks involving sinusoidal gratings, faces, and objects. Comparisons of activation between tasks used the subtraction method. A gender categorization task (gender discrimination minus grating) revealed strongest activation in the right occipital gyrus (inferior, medial, and lateral) and right lingual gyrus (medial occipitotemporal gyrus), and lesser activation in the left lateral occipital gyrus. A face identity task (face identity minus gender discrimination) revealed additional activation in the fusiform gyrus, anterior temporal cortex,

and temporal poles of both hemispheres. Object recognition (objects minus gratings) showed activation in the left occipitotemporal cortex and the left fusiform gyrus, and did not involve the right hemisphere regions activated during the face identification task. In addition, a high level of activation was observed in the parahippocampal gyrus during the face identification task. The parahippocampal formation is part of the limbic system, and is viewed as an area of convergence for perceptual and memory information. Sergent et al. proposed that face identification requires the functional integrity of three specific cortical regions: the right lingual and fusiform gyrus, the right parahippocampal gyrus, and the anterior temporal cortex.

Since the Sergent et al. study, a number of research groups have applied both PET and fMRI to explore how, where, and why faces activate the fusiform gyrus in particular. In a series of publications, Kanwisher and colleagues have examined the role of the fusiform gyrus in face perception. Kanwisher argues that this area can be viewed as a specialized module for face perception and therefore termed this area as the Fusiform Face Area (FFA). In a first paper, Kanwisher et al. (1997), using fMRI, initially identified an area in the right fusiform gyrus that was significantly more active (in 12 of 15 subjects) when viewing faces than common objects. Defining the fusiform gyrus as their region of interest, Kanwisher et al. then ran a series of experiments testing for face-specific activation with a subset of five subjects. The right fusiform gyrus or the FFA responded stronger to passive viewing of intact than to scrambled faces, frontal views of faces than frontal views of houses (another subordinate-level category), and three-quarter view of faces than human hands (passive viewing as well as consecutive matching task). Further examining the response properties of the fusiform gyrus, Tong et al. (2000) presented various types of face stimuli, including animal faces. The FFA response was weakest for nonface objects and houses, but was equally large for cat, cartoon, and human faces. The response was equal for frontal and profile views of faces, which is of interest in light of the results from single-cell recordings that also show stronger response to frontal and profile views. Activation of the FFA seems also to be dependent on the level of attention paid to the face stimuli: When the face stimuli appear outside the focus of attention (covert visual attention), the activity is reduced (Wojciulik et al., 1998). On the basis of their findings, Kanwisher and colleagues have concluded that the FFA is selectively involved in the perception of faces, and that these results refute alternative accounts for activation such as subordinate-level classification.

Kanwisher et al. (1998) also investigated the well-known face inversion effect. As documented in numerous studies, inversion of faces disrupts the holistic or configural processing of faces (Bartlett, Searcy, and Abdi, 2003; Bartlett and Searcy, 1993; Farah et al., 1995, 1998; Moscovitch et al., 1997; Rhodes et al., 1993; Valentine, 1988; Yin, 1969, 1970). In their fMRI study, Kanwisher et al. found that the average percent signal change in response to inverted grayscale faces was small and was also not consistent across subjects. This suggests that the FFA response is correlated with the perception of faces, rather than low-level visual features present in faces. Similarly, Haxby et al. (1999) also found that inverted faces produced small and nonselective activation changes in the face-selective region. Interestingly, inverted faces actually produced increased activation in a house-selective region, which suggests that inverted faces recruit the object perception system. Such a pattern of results is consistent with behavioral findings in prosopagnosic subjects who often do not show an inversion effect and are thought to process faces in an object fashion. Along the same lines, Aguirre et al. (1999) also report only small effects of face inversion.

Activation of the fusiform gyrus in response to faces has consistently been replicated. For example, McCarthy et al. (1997) found activation in bilateral regions of the posterior fusiform gyrus when faces were viewed among a montage of phase-scrambled objects. When faces were viewed among normal objects, only the right fusiform region was activated. To see whether another set of familiar within-category stimuli would activate the same regions, subjects viewed flowers presented in the same conditions as faces. Flowers presented along with phase-scrambled objects showed bilateral fusiform activation (greater in left hemisphere), but no activation was obtained when flowers were presented among objects. On the basis of these activation patterns, McCarthy et al. also conclude that faces specifically activate the right fusiform gyrus. Similar activation patterns have been reported by Clark et al. (1996); Haxby et al. (1994); Hoffman and Haxby (2000); and Puce et al. (1995, 1996).

From all these studies it seems that the designation of the fusiform gyrus (primarily the right) as the FFA is warranted. However, this view has been met with challenges. Specifically, Gauthier and colleagues argue that the activation patterns in the fusiform gyrus in response to faces is due to the very high similarity between faces and subjects' expertise with such stimuli. Therefore, activation in this particular area of the fusiform gyrus may reflect processing of highly similar objects rather than faces *per se*. Recall from the discussion of ERP studies that Gauthier and colleagues take the same position when interpreting the N170 response to faces. Going back to Rosch's work on categorization (Rosch, 1978), objects can be classified into a hierarchical set of categories. The superordinate categories are the largest, composed of general categories

such as furniture. Within this category are basic-level categories, such as tables and chairs, which in turn contain subordinate categories, such as kitchen table and living-room table. Most object recognition takes place at the basic level, and identifying an object as being a face (as opposed to another object) also takes place at the basic level. Face recognition, on the other hand, takes place at the subordinate level between subsets of the basic-level category "faces." In other words, Gauthier and colleagues view the fusiform gyrus as optimal for subordinate-level categorization or within-category discrimination with the important caveat that the observer has considerable expertise in processing the particular category of stimuli under consideration.

Gauthier and colleagues contend that studies that have concluded that the fusiform gyrus is a "special-purpose cortical machinery" for face recognition failed to consider experimental designs or stimuli that might refute such a conclusion. In their view, no attempts were made to engage the "face module" by presenting only nonface objects. Specifically, in an fMRI study, Gauthier et al. (1997) investigated the effects of category level of the stimuli in a matching task of nonface objects. Subjects performed two tasks, one visual and one semantic at the basic and subordinate category levels. In the visual task, subjects judged whether simultaneously presented pictures and words matched. In the semantic tasks, subjects decided if the object could move by its own power. Three regions of interest were defined *a priori*: fusiform and inferior temporal gyri, lingual gyrus, and occipital cortex. The strongest activation associated with subordinate-level visual recognition was found in the fusiform and inferior temporal gyri for seven out of eight subjects (bilateral for all but one subject who showed left hemisphere activation), with additional activation in the left occipital lobe. The occipital lobe showed left activation in four subjects. Subordinate-level processing for the semantic task showed strongest activation in the occipital lobe (two subjects bilaterally, five subjects on the left only). The second most activated area was the fusiform and inferior temporal region (two subjects bilaterally, two subjects on the left and right). To test specific activation in the fusiform and inferior temporal region shown in other studies to be responding to faces, a double subtraction [visual(subordinate–basic)–semantic(subordinate–basic)] showed activation of the fusiform and inferior temporal gyri for subordinate-level processing.

Gauthier et al. (1999b) in an fMRI study further investigated whether another subordinate-level class of objects would activate the FFA. The stimuli they used were "Greebles." First introduced by Gauthier and Tarr (1997), Greebles constitute a novel class of computer-generated stimuli specifically designed to be similar to faces along several dimensions. All Greebles are made up of the same number of parts set in the same configuration and displays individual characteristics. As a face, each Greeble also belongs to a particular gender and family. Subjects were trained to become experts at recognizing Greebles and reached expertise level after an average of 3240 trials. Their recognition patterns then showed effects typically associated with face recognition, such as configural and holistic processing. In their imaging study, Gauthier et al. used the Greebles to examine activation in an *a priori* selected ROI (bilateral middle and anterior fusiform gyrus) usually activated by face processing. Subjects were scanned across six sessions when they performed sequential-matching judgments of unfamiliar faces and Greebles (upright and inverted). By the end of the sixth session, Greebles activated the right middle fusiform gyrus as much as faces. The same area was also more activated by "Greeble experts" during passive viewing of Greebles. Gauthier et al. (2000) further extended this finding to subjects who were experts at recognition of other homogeneous categories: birds and cars. The bilateral FFA and the right "occipital face area" were chosen as ROIs. The FFA showed higher activation in response to birds and cars than to familiar objects. In addition, both regions of interest showed significant expertise effects. It is therefore the opinion of Gauthier and colleagues that the level of categorization (subordinate-level discrimination) and expertise are the determining factors for activation of the putative FFA rather than faces *per se*. In other words, the FFA is specialized in processing stimuli that display high within-group similarities and further that the subject is highly specialized in processing such stimuli.

Thus, from the studies reviewed so far, we are left with two different interpretations of fusiform gyrus activation in response to facial stimuli. The positions of these two groups are further outlined in a set of commentaries (Kanwisher, 2000; Tarr and Gauthier, 2000). In brief, on the basis of behavioral, neuropsychological, and neuroimaging data, Kanwisher argues for a domain-specific view of face perception. Gauthier and Tarr argue that the FFA should be considered a flexible (as opposed to face) fusiform area for subordinate-level visual processing that is automated by expertise.

However, a third account of the observed activation patterns in the fusiform gyrus in response to different categories of objects (including faces) has been put forward by Haxby and colleagues. As earlier reported, Haxby et al. (1991) showed differential activation patterns for faces and objects, with faces generally activating a ventral visual pathway and objects activating a dorsal visual pathway. Several studies by this group have also reported activation

by faces in the fusiform gyrus (Haxby et al., 1994, 1996, 1999). Ishai et al. (1999) propose that the ventral visual pathway is characterized by a distributed representation of objects. To support their view, subjects in an fMRI study were scanned during passive viewing and delayed match-to-sample tasks of faces, houses, and chairs. Patterns of activation in response to the different categories varied across the ventral temporal cortex in a highly consistent topological arrangement, which was similar in both hemispheres. Houses elicited greatest activation in the medial fusiform gyrus and chairs elicited greatest activation in the inferior temporal gyrus. Faces elicited greatest activation in the lateral fusiform and occipitotemporal gyri. Ishai et al. acknowledged that indeed these patterns of results can be interpreted as evidence for modularity, but each of the stimuli categories also showed significant patterns of activation in secondary areas that responded maximally to a different category. Houses also elicited activation in the area responding maximally to chairs and vice versa. Faces also activated the house and chair regions, but to a lesser extent than houses and chair activated other regions, which the authors attribute to faces being processed more automatically. Activation in the secondary regions was also modulated by additional attention, as was required in the matching task, which suggests that the secondary regions are recruited to enhance perception of highly similar stimuli.

Similar distributed patterns of response to faces, houses, and chairs were also found in bilateral regions of the ventral occipital cortex (Ishai et al., 2000). The response to faces again appeared more restricted in spatial extent and the face area was also activated more automatically than the areas responsive to houses and chairs. Additionally, a region in the superior temporal sulcus was identified that responded almost exclusively to faces. These patterns of results lead Ishai et al. to propose that the functional architecture of the ventral visual pathway, including both the occipital and temporal regions, is based on a distributed representation of objects. Objects, including faces, that share attributes or are common in their form tend to cluster together. This creates a consistent topographical arrangement and activation pattern. Using a larger number of object categories, Haxby et al. (2001) confirmed their previous findings of distinct patterns of response for different stimulus categories. When excluding the area that responded maximally to each object category, the category being viewed could still be identified on the basis of the pattern of response in the other areas with a mean accuracy of 94%. Taken together, Haxby and colleagues propose that the representations of faces and objects of the ventral temporal cortex are widely distributed and overlapping. In their view, activation patterns in the FFA does not solely represent the perception of the spatial arrangement of human faces, but that the FFA is part of a more extended system of representation for all objects. Spiridon and Kanwisher (2002) replicated these findings and further found that the pattern of activation extended to within-category stimuli that differed in viewpoint, exemplar, or image format.

The studies reviewed so far have examined activation patterns in normal subjects. However, the study of subjects with cognitive impairments have provided value insights into cognitive processes, a case in point being the amnesic patient HM (Milner, 1966), whose pattern of deficits contributed to the study and understanding of human memory. As previously discussed, the study of impaired face-processing skills in prosopagnosic subjects has demonstrated a dissociation between face and non-face processing as well as facial identity and facial expression processing. Acquired prosopagnosia occurs after bilateral damage to the inferior port of the temporal cortex, including the fusiform gyrus, although only right hemisphere damage can also produce face recognition deficits. One explanation offered for the observed face recognition deficits in prosopagnosic patients are that impaired at processing faces as a whole, rather they are thought to rely on a time-consuming feature- or parts-based analysis of faces. Holistic faces processing is thought to be a right hemisphere function, whereas featural analysis is supported by the left hemisphere (Rhodes, 1985, 1993). Rossion et al. (2000) found evidence for such differential processing of face stimuli in their PET study. The right middle fusiform was more activated when subjects matched whole faces, whereas the left fusiform activated more when subjects matched facial features. These lateralized differences were not observed when subjects processed objects as wholes or parts. If face recognition then relies on holistic processing of faces involving the right fusiform gyrus, and holistic face processing is impaired in prosopagnosics, one should expect to see increased activation in the left fusiform which now appears to support more of a feature-based analysis of faces. This is the pattern that Marotta et al. (2001) observed in an fMRI study of two prosopagnosic subjects. The prosopagnosic subjects showed activation in the fusiform gyrus, but more voxels activated in a posterior region than what was observed for the control subjects. One patient showed surprising evidence of posterior activation in the fusiform gyrus in the left hemisphere, which led the authors to suggest that this posterior activation reflects compensatory processes when more anterior regions of the fusiform are damaged. This compensatory process could then be indicative of a feature-based approach to face processing. The three prosopagnosic subjects in Hadjikhani and de Gelder's fMRI study (Hadjikhani and de Gelder,

2002) failed to show the normal pattern of higher activation to faces than houses in the mid-fusiform and the inferior occipital gyrus. None of these prosopagnosic subjects had any difficulty in a face detection task (face presented in a noise pattern or whether the stimulus is a face or not). This raises the issue of the necessity of these brain regions for face recognition but not for face detection.

Another group of subjects that show impaired face processing are individuals with autism spectrum disorders (ASD), including Asperger syndrome. Deficits in social cognition which includes the inattentiveness to faces of others and failure to make appropriate eye contact suggest that individuals with ADS probably have not developed typical face expertise (Schultz et al., 2000b). Such individuals generally rely on a feature-based analysis of the face rather than configural processing, which is one of the hallmarks of normal face processing. Schultz et al. (2000a,b) used fMRI to study face and object perception in such a group of subjects. Regions of interest were first located in two groups of normal subjects to whom the activation patterns in the patients groups were then compared. In the first experiment, the activation patterns in the patient groups were compared with the first control group on a face discrimination task. The autistic group showed significantly more activation in the right inferior temporal gyrus than the right fusiform gyrus. A second experiment compared the patient group to the second control group and generally replicated these initial findings. The autistic group showed larger activation in the left inferior temporal gyrus compared with the controls. No consistent differences were found between the control groups and the autistic group on a subordinate-level matching task of objects. The authors concede that the laterality differences need to be further clarified, but overall, the pattern of results lends support to the claim that autistic subjects process faces using an object-based processing strategy, which activates the inferior temporal gyrus. Further, Grelotti et al. (2002) propose that impaired face processing in ASD individuals can best be cast in the framework of the expertise model rather than the FFA model. Cortical face specialization failed to develop in ASD individual because of their reduced social interest. Although processing of facial expressions will be reviewed in detail later, it is at this point relevant to mention that high-functioning autistic adolescent were found to show impaired perception of facial expressions (Teunisse and de Gelder, 2001). Their deficits in processing facial expressions is obviously part of the pattern of impaired social cognitive skills, but more directly, the deficits can also be due in part to how autistic individual process facial expressions. A recent study found that autistic subjects focused on the mouth areas of actors whereas normal controls focused more on the eyes (Klin et al., in press). Whereas a correlation was found in normal subjects between fusiform and amygdala activation when attending to the gaze direction of faces (George et al., 2001), the reported lack of attention to the eyes in autistic individuals may then explain the low activation patterns in the fusiform gyrus (Grelotti et al., 2002).

Encoding, Recognition, and Face Familiarity

So far, we have established that faces, when used in relatively simple visual tasks, consistently activate the fusiform gyrus. However, the complexity of face processing in real life and even in the laboratory goes beyond passive viewing or matching tasks. Our knowledge about the processes underlying face recognition comes from behavioral studies that employ more complex testing paradigms than the relatively simple visual tasks that have established activation patterns in the fusiform gyrus—whether it should be designated as a special face-processing area or not. Typically, face recognition has been investigated by having subjects first learn a set of faces and then recognize them in a later testing session. Further, as we have seen, familiarity with faces is an important factor in face recognition. How informative have neuroimaging techniques been in uncovering the areas activated with such experimental manipulations?

Haxby et al. (1996) used PET and a traditional behavioral testing paradigm to examine areas of activation in response to encoding and recognition of faces. In the first scan, subjects viewed 32 faces presented sequentially three times in different orders for a total viewing time of 8 min. The subjects were informed that they would be tested on a recognition task in a later scan. Face perception and a sensorimotor task were included as control tasks. The activation patterns observed during encoding and recognition showed a dissociation between the neural systems involved in these processes. As no ROI was identified *a priori*, the voxel-by-voxel statistical analysis revealed a number of regions involved in these tasks. Comparison of the memory tasks with the perceptual tasks showed little overlap. Encoding of faces, compared with the perceptual task, indicated increased rCBF in a number of areas: left prefrontal cortex, right medial temporal region, hippocampus, anterior cingulate cortex, and left inferior temporal gyrus. Face recognition compared with the perception control task activated the right prefrontal cortex, anterior cingulate cortex, bilateral inferior parietal cortex, bilateral ventral occipital cortex, and the cerebellum. When compared with the sensorimotor control task, encoding and recognition also showed activation in a large bilateral region of the ventral occipitotemporal cortex, in addition to the areas already mentioned. Of

particular interest is the switch between hemispheres in the prefrontal cortex depending on the task: left prefrontal cortex for encoding and right prefrontal cortex for recognition. The predominance of the right prefrontal cortex in recognition has also recently been reported by Wiser et al. (2000), who, in their PET study, compared memory for novel vs. well-learned faces. Subjects were asked to recognize faces that had been learned immediately before scanning and faces that had been learned to the point of faultless recognition 1 week before scanning. Both tasks did engage highly similar neural circuits, but memory for novel faces involved more the frontal lobe areas, whereas memory for well-learned faces engaged more the posterior regions. Additionally, recognition of well-learned faces resulted in smaller activation nodes, suggesting less effortful retrieval processes in recognition of familiar faces.

Bernstein et al. (2002) also examined the effect of encoding strategy using PET. Subjects were scanned during encoding of faces by using either a "deep" task (pleasantness judgment), a "shallow" task (face orientation), or intentional learning, and were also scanned during a subsequent face recognition task. Encoding activated primarily a ventral system including bilateral temporal and fusiform regions and left prefrontal cortices. Recognition activated primarily a dorsal set of regions which included the right prefrontal and parietal areas. An effect of encoding strategy was observed with deep encoding activating the amygdala and left anterior cingulate. Differential activity was observed in the fusiform gyrus, which suggests that this area was also modulated by controlled processes.

The hemispheric switch between activation during encoding and retrieval processes observed by Haxby et al., Wiser et al., and Bernstein et al. is consistent with the predictions from Tulving's hemisphere encoding-retrieval asymmetry model (Tulving et al., 1994; see also Bartlett et al., 2003), which, roughly, postulates that encoding activates the left hemisphere and recognition activates the right hemisphere.

A study conducted by Kuskowski and Pardo (1999) also looked at encoding of faces, but manipulated the number of times subjects saw the faces by presenting the faces one time only or repeatedly. Following the scans, subjects were tested on their memory for the learned faces. Only scans with a postscan recognition rate of 70% correct were included in the subsequent analysis. In the "face memory" condition, subjects were asked to learn 21 unfamiliar faces that were presented individually without repetition. In the "face repeat" condition, subjects learned four unfamiliar faces presented individually and repeatedly. The design also included "face watching" and "scrambled face" conditions as controls. Again, the results showed activation across several regions during face encoding, but the areas of activation also appeared to be dependent on the encoding condition. For the "face memory minus scrambled face" comparison, the largest magnitude of responses was located in the right mid-fusiform gyrus, with additional activation in both anterior fusiform gyri, and prefrontal cortex. "Face repeated minus scrambled face" showed activation in the right precentral gyri, left cingulate gyrus, left lateral cerebellum, prefrontal cortex, and temporal cortex (left anterior fusiform foci). "Face watching minus scrambled face" revealed activation in the bilateral temporal pole regions, bilateral anterior fusiform regions, left parietal cortex, left prefrontal, right inferior frontal, and precuneus. The strongest positive correlations with recognition test scores were found in the right mid-fusiform regions. No hippocampal or parahippocampal activation was recorded, which contrasts with previous findings (Haxby et al., 1996; Kapur et al., 1995; Sergent et al., 1992).

As previously mentioned, familiarity is a strong mediating factor in face recognition. According to the Bruce and Young model, recognition of familiar faces is mediated by an established structural code in long-term memory. By contrast, recognition of unfamiliar faces is, to a large degree, dependent upon the strength of a structural code that can be extracted from the initial exposure to the face, as well as upon the pictorial code. Dubois et al. (1999) investigated the effect of face familiarity in a PET study. Subjects performed a gender categorization task on both familiar and unfamiliar faces, as well as face recognition and pattern discrimination tasks, which were included as control tasks. All face tasks showed bilateral activation in the fusiform gyrus, with activation patterns in the right anterior area closely matching the mean coordinates reported by Kanwisher et al. (1997). Dubois et al. also found differential activation patterns for known and unknown faces, with rCBF increasing in the left amygdala in categorization of unknown faces (subtraction of known from unknown faces). The amygdala activation in response to the gender categorization task was unexpected. For example, Sergent et al. (1992) did not observe any activation in the amygdala in response to this task. On the other hand, it is well established that the amygdala is activated by processing of threatening stimuli, as well as a variety of facial expressions. For example, the facial expression of fear consistently activates the amygdala (Morris et al., 1996, 1998; Whalen et al., 1998). The amygdala also responds to happy and neutral facial expressions, but shows rapid habituation effects (Breiter et al., 1996). Therefore, unknown faces may initially have been processed as possible threatening stimuli, whereas the known faces had been encountered without negative implications during the familiarization process and therefore did not present a "threat" that would activate the amygdala. Additionally,

processing of known faces led to a decrease in activation in the posterior part of the calcarine sulcus and middle occipital gyrus. Differential activation patterns for familiar and unfamiliar faces have also been reported by George et al. (1999), Katanoda et al. (2000), Leveroni et al. (2000), and Tempini et al. (1998).

Toward an Integrative Model

As should be evident by now, brain imaging studies of different face-processing tasks have yielded a wealth of information, but not until recently did we see a serious effort to consolidate the findings into a coherent framework. On the basis of their own work and other groups' imaging studies of face processing, ERP studies, as well as animal models and studies, Haxby et al. (2000, 2002) took another step forward by proposing a model for a "distributed human neural system for face perception" (see Fig. 2).

The Haxby model takes into account the two important tasks that an effective face-processing system must accomplish. First, the system must be able to establish an invariant face representation that allows for recognition across encounters. Second, the system must also be able to effectively interpret changeable aspects of faces, such as facial expressions, eye gaze, and lip movement, which mediate social interactions and communication (cf. O'Toole et al., 2002, for an expanded discussion of recognition of moving faces). The model is hierarchical and is divided into a core system and an extended system. The core system is composed of three bilateral regions in the occipitotemporal visual extrastriate cortex and includes the inferior occipital gyri, the lateral fusiform gyrus, and the superior temporal sulcus. The inferior occipital gyri are involved in the early perception of facial features and provide input to the lateral fusiform gyrus and the superior temporal sulcus. The fusiform gyrus analyzes the invariant aspects of faces or unique identity, whereas the changeable aspects of faces are mediated by face-responsive regions in the superior temporal sulcus. The extended system supplements further face processing in concert with other neural systems: Emotional content is processed by the amygdala, the insula, and the limbic system, the auditory cortex is recruited in processing speech-related mouth movements, and the intraparietal sulcus processes spatially directed attention such as eye gaze. Personal identity, name, and biographical information are accessed in the anterior temporal region.

Summary: Face Processing

Although the studies reviewed in this section show great potential for finally gaining access to the "magic black box," a recent study can serve as a reminder that neuroimaging is still a young discipline. Farah and Aguirre

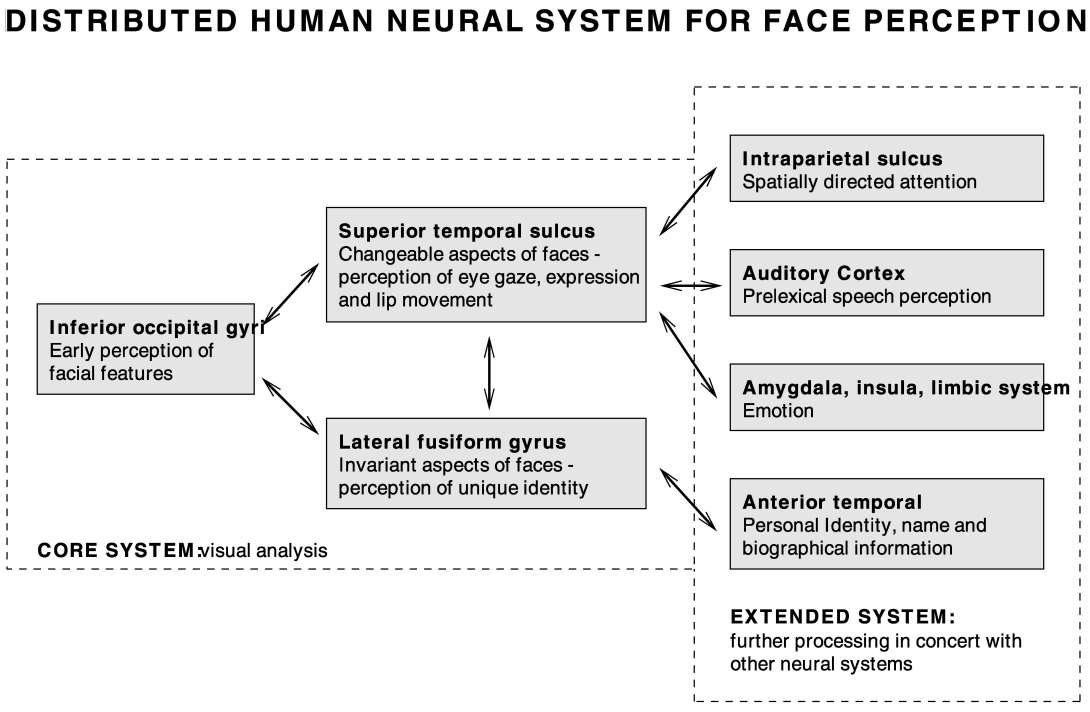

Fig. 2. The Haxby model of a "distributed human neural system for face perception" (from Haxby et al., 2000).

(1999) addressed some of the difficulties in interpreting activation patterns in imaging studies aimed at identifying neural substrates of visual recognition. They performed a meta-analysis of 20 tasks from 17 different imaging studies of visual recognition of words, objects, and faces. A total of 84 local maxima of activation were plotted using the Talairach and Tournoux coordinates. Failing to observe a focal clustering of activation even within each stimulus class, the authors reached the conclusion that "visual recognition is a function of the posterior half of the brain" (p. 181). On the other hand, when isolating the points of activation for passive viewing of faces only, the coordinates of activation do fall close to those reported by Kanwisher et al. (1997). The general failure to observe a consensus among the studies included in the meta-analysis was attributed to methodological issues, spatial normalization techniques, statistical analysis, and individual differences in subjects' brain structure.

As we have seen, the fusiform gyrus has been strongly implicated in face processing. Recall that there are divergent opinions as to whether the fusiform gyrus (right hemisphere in particular) may be considered as the FFA as illustrated by the exchange between Kanwisher and Gauthier. Is it then possible to arrive at a reconciliation of these two views? Perhaps it is worth considering the type of stimuli that is under investigation. Few would disagree that faces constitute the category that the human perceptual and cognitive systems are most highly specialized to process. No other category of visual stimuli comes close to engaging the cognitive system as faces do, unless the observer is an apparent specialist in processing such categories at a subordinate level. Thus, the designation of the fusiform gyrus as the FFA can be considered a "default option" which remains in effect until the system operator overrides or complements the specialization for faces with another category of stimuli. Of course, the results from the Haxby group indicate that activation in the fusiform gyrus in response to face stimuli is part of a distributed and overlapping system that processes both faces and objects.

Other studies have gone beyond the fusiform gyrus and attempted to identify regions and chart patterns of activation in response to more challenging face memory tasks, such as encoding and recognition. As expected, when face-processing tasks become more demanding, the activation patterns become more complex. Far more areas light up beyond the now expected activation in the fusiform gyrus. The model proposed by Haxby et al. for a distributed neural system for face processing provides a sound framework for future investigations. The challenges we will face in the next decade will be to further refine our investigative methods to answer questions: What is the functionality of the areas of activation? How do the various areas interact? What is the temporal sequence of activation? And how do we establish an invariant representation of faces that can also accommodate the processing of changeable aspects, such as facial expressions?

BRAIN IMAGING STUDIES OF FACIAL EXPRESSIONS

Facial expressions have been studied for more than 100 years: As early as in the 1870s, Charles Darwin wrote of emotion and facial expressions. A century later, the work by Ekman (1972, 1992) and Izard (1971, 1977) led the field in the study of perception and categorization of facial expressions. It is generally accepted that there exist at least six basic categories of emotion that can be conveyed through facial expressions: happiness, surprise, fear, sadness, anger, and disgust. The perception and assignment of facial expressions to these categories are claimed to be universal, as members of both literate and preliterate cultures categorize facial expressions along the same dimensions. However, this approach has not escaped criticism related mainly to the problem of ecological validity. For example, Russel (1997) argues that the kinds of expressions seen in Ekman and Friesen's photographs (Ekman and Friesen, 1976) are rare occurrences in daily interactions. Setting controversies aside, the database of facial expressions compiled by Ekman and Friesen has been widely used by a number of research groups in the area of perception and categorization of facial expressions, as well as face recognition studies.

As we have previously seen, lesion studies suggest that some specific brain structures or different neural substrates are involved in processing different types of face-related information. In fact, we are looking at a double dissociation: prosopagnosic patients are impaired at face recognition but have intact facial expression processing, and other subjects are impaired at facial expression processing but can still recognize faces. Results from studies of facial expression processing suggest that different emotions are also processed by different regions of the brain. For example, Adolphs et al. (1996) investigated facial expression recognition in a large number of subjects with focal brain damage. The authors hypothesized that cortical systems primarily responsible for recognition of facial expressions would involve discrete regions of higher-order sensory cortices. Recognition of specific emotions would depend on the existence of partially distinct systems. This predicts that different patterns of expression recognition deficits should depend upon the lesion site. In general, none of the subjects showed impairment in processing happy facial expressions, but several

subjects displayed difficulty in recognizing negative emotions (especially fear and sadness). The authors propose that deficits in processing negative emotions can be due to the fact that the number of negative emotions is larger than the number of positive emotions. In fact, there is only one positive emotion, namely happiness. As such, a happy smile should be easily recognizable. It is also possible that negative emotions show a deficit because they can be easily confused, such as mistaking fear for surprise, or anger for disgust.

So far, a general consensus has been reached on the anatomical structures involved in the processing of fear and disgust, with findings coming from both lesion and brain imaging studies. We also have an emerging picture of the perception of happy facial expressions, because this expression has often been used as a basis for comparison in studies of different expressions. Other facial expressions such as anger and sadness have received more limited attention. So far, no brain imaging study has examined the expression of surprise.

The following sections will review processing of the six basic emotions in turn: fear, disgust, sadness, anger, happiness, and surprise. Each type of facial expression will be examined from both human subjects and neuroimaging studies.

Fear: Behavioral Studies

Close links have been established between the emotion of fear and the amygdala. For example, LeDoux (1996) proposes that the fear reaction system involves parallel transmissions to the amygdala from both the thalamus and the sensory cortex, which he refers respectively to as the "low" and "high" roads. The direct pathway from the thalamus to the amygdala is the shortest and fastest transmission route. This "low road" provides the amygdala with a crude representation of the stimulus and allows for immediate response to the present danger. The "high road" involves elaborated processing in the sensory cortex before the input reaches the amygdala and is slower but more precise. In agreement with LeDoux's theorization, neurons in the monkey amygdala have been shown to respond to the affective significance of sensory stimuli, and lesions of the amygdala render the animal insensitive to stimuli that normally evoke fear. In humans, the amygdala has been studied as a result of surgical lesions and electrical stimulation, in particular in epileptic patients. The role of the amygdala in processing fear and aggression in social behaviors has been firmly established (cf. Aggleton, 1992; Davis and Whalen, 2001, for reviews). Animals studies have shown that the amygdala also receives highly processed visual input, and contains neurons that respond selectively to faces (Rolls, 1992).

The role of the amygdala in processing facial expressions has been highlighted in three interesting case studies. In all cases, the perception of fear is particularly compromised. The first case of SM was reported by Adolphs et al. (1994, 1995). A female in her early thirties at the time of testing, SM suffers from Urbach-Wiethe disease, which has resulted in bilateral calcification and atrophy of the amygdala. Her performance on facial expression perception was evaluated in a series of experimental tasks and compared with the performance of several control groups (unilateral amygdala damage, other brain damage, and normal subjects). When SM judged faces showing fear, her ratings of fear were less intense than any of the control groups' ratings. In addition, SM's ratings for afraid faces correlated poorly with normal ratings, and she often described faces displaying a fearful expression as surprised or angry. Multidimensional scaling of perceived similarity judgments of facial emotions revealed that SM is less able to interpret similarity or blends between expressions than are normal controls. When asked to draw various facial expressions, SM was unable to draw the expression of fear, stating that she did not know what an afraid face looks like. Yet, she understands the verbal concept of fear. SM showed no difficulty in recognizing people from their faces, providing additional evidence for a dissociation between recognition of emotion and identity in faces (Damasio et al., 1990; Humphreys et al., 1993; Tranel et al., 1988). Adolphs et al. conclude that the amygdala is required for linking visual representation of facial expressions with representations that constitute the concept of fear.

The second case, DR, was first reported by Young et al. (1995). DR, a female in her early fifties at time of testing, became epileptic at the age of 28. Brain imaging showed extensive lesions in left amygdala and smaller lesions in the right amygdala, resulting from a series of surgical procedures to the amygdala. Although DR has some problems in recognizing familiar people out of context as well as faces encountered postoperatively, her unfamiliar face matching capability (as evaluated by the Benton test) was unimpaired. By contrast, her performance on facial expression matching and expression recognition tasks was severely impaired, but as SM, DR can describe and define emotions.

In a follow-up study, Young et al. (1996) further evaluated DR's facial expression processing. In a facial expression-matching task, DR experienced difficulties when the same face was shown with different expressions, and when different faces were shown with the same expression. In an identity-matching task, DR exhibited

problems whenever the same face was presented with different expressions. In addition, she was poor at imagining facial expressions.

Calder et al. (1996) also tested DR's recognition of facial expressions, using morphed facial expressions between faces taken from the Ekman and Friesen series of faces. This testing paradigm uses photographic images of six prototype emotions that are interpolated or morphed to create a hexagonal continuum (running from happiness to surprise to fear to sadness to disgust to anger back to happiness). Control subjects identified these morphed images of facial expressions as belonging to distinct regions of the continuum, corresponding to the nearest prototype expression, which was not shown. DR was impaired on this task, in particular at identifying morphed images of fear, and to a lesser extent disgust and anger. By contrast, DR showed no impairment in an identification task based on morphed faces of famous people.

The third case was reported by Anderson and Phelps (2000). SP, a 54-year-old female at time of testing, had the right amygdala removed, showed a lesion in the left amygdala, and had a right temporal lobe resection. SP's facial recognition was intact and she could correctly judge sex and age from faces. Compared with normal controls, SP was severely impaired in her appraisal of fear, as well as disgust, sadness, and to a lesser extent happiness. Ratings of surprise and anger were not impaired. As both SM and DR, SR's lexical affect identification was normal. When asked to generate facial expressions (which were videotaped for later ratings), SR's facial expressions were rated as accurate as those generated by normal controls. Thus, she cannot perceive a face as fearful but she can herself produce the expression of fear. This is in contrast to SM, who was not able to produce a drawing of a fearful face.

Taken together, the above findings (Adolphs et al., Anderson and Phelps, Calder et al., and Young et al.) strongly support the amygdala's involvement in recognition of emotion, and fear in particular. Furthermore, these studies also support the existence of a dissociation between processes underlying facial identity and facial affect. Insofar as these studies indicate that some emotions have specific pathways, they also militate for the existence of a more general dissociation between facial identity and facial affect.

Unfortunately, the picture may not be as clear as the previous studies indicate. Specifically, Hamann et al. (1996) found intact recognition of emotions in two patients (ages 73 and 59) with bilateral amygdala lesions resulting from herpes simplex encephalitis after age 50. These patients were tested on the same material as patient SM (refer to case study already described earlier in this section). The inconsistent results can be attributed to two possibilities. First, structures outside the amygdala may be implicated in recognition of facial emotions. SM did show some damage to areas outside the amygdala (anterior entorhinal cortex). Second, and more importantly, impaired recognition of emotions in facial expressions may result only if the amygdala lesions occurred early in life, suggesting a developmental or learning component in the ability to process facial expressions as socially relevant stimuli. This would be consistent with SM's congenital condition caused by Urbach-Wiethe disease.

Fear: Neuroimaging Studies

As we have seen, lesions of the amygdala lead to selective deficits in processing emotional facial expressions. Several groups of researchers have turned to neuroimaging techniques to investigate how normal subjects process facial expressions. In general, the testing paradigm adopted in such studies has been to present faces displaying fear, happy, and/or neutral expression, and examine differential activation through subtraction. This section will concentrate on the activation patterns obtained for the expression of fear.

Morris et al. (1996), using PET, measured rCBF in five subjects who viewed fearful and happy faces. Each face within the emotion category showed six levels of intensity of expression (obtained with morphed images), resulting in a 2×6 factorial design. As predicted, presentation of fearful faces (as assessed by an *a priori* contrast with happy faces) showed activation in the left amygdala and left peri-amygdaloid cortex. No activation was observed in the right amygdala. Other areas of activation included the left cerebellum, the right superior frontal gyrus, and the left cingulate gyrus. The presentation of happy faces (contrasted with fearful) showed activation in the right medial temporal gyrus, right putamen, left superior parietal lobule, and left calcarine sulcus. The contrast of emotional faces (both happy and fearful) vs. neutral faces produced activation in the left pulvinar and the right orbitofrontal cortex. Additionally, orthogonal contrasts comparing neutral and emotional intensity level showed that the pattern of activation was affected by the level of emotional intensity (increasing with increasing level of fear and decreasing with increasing level of happiness). Areas responsive to increasing intensity of fear were the left anterior insula, left pulvinar, and the right anterior cingulate, as well as the amygdala. The left amygdala was also responsive to decreases in the intensity of happy expression. No explicit recognition or classification of the emotional expressions was required as the subjects classified the faces by gender. Therefore, activation

of the amygdala does not appear to require explicit attention (e.g., classification of the expression) being paid to the facial expression. Further, regression analysis indicates that activation in the amygdala appears to modulate expression-specific neural activity in the extrastriate cortex.

Breiter et al. (1996) used fMRI and presented standardized facial expressions of fear and happiness to examine activation in the amygdala. Their first experiment examined visual processing of fearful and happy vs. neutral faces in normal subjects. Presentation of the face stimuli involved a fixed order (first fear, then happy, to optimize activation by fearful faces) both within and across runs. Fearful expressions activated the amygdala bilaterally, but showed higher activation for the left (0.68 signal change) than the right (0.52). No significant signal changes were associated with happy faces compared with neutral faces in the amygdala. The second experiment counterbalanced the expression condition within and across runs to control for order effects. An ROI comprising the left amygdala again showed significant activation for the fear vs. neutral contrast. Breiter et al. also found posterior amygdala activation for each type of facial expression against a low-level baseline, which suggests that the amygdala might respond to faces in general. Post hoc analysis of the within-run signal changes across runs showed a significant order or habituation effect in an ROI comprising the amygdala for both fearful and happy expressions. The fusiform gyrus, known to be activated during the processing of faces, was chosen *a priori* as a control region. This area showed the same general activation for both fearful vs. neutral and happy vs. neutral comparisons, and did not show a within-run signal decrement (habituation) pattern. Wright et al. (2001) found differential habituation patterns in the amygdala with the right amygdala showing greater habituation to emotionally valenced stimuli than the left amygdala. Feinstein et al. (2002) found evidence of habituation outside the amygdala, using a test/retest paradigm with different facial expression (including fear). With the apparent salience of the affective stimuli decreasing at the second presentation, habituation was observed in areas in a right hemispheric network (right frontal and precentral gyrus, right insula, right postcentral gyrus, and right inferior parietal lobule). However, a left hemispheric network (angular gyrus, posterior superior temporal gyrus, precentral gyrus, and insula) showed increased activation at the second presentation of the stimuli. This left network is involved in object recognition, and it is thought that the faces were encoded as unfamiliar objects during the first exposure and recognized as being familiar during the second exposure, which was reflected by the increased activation pattern. This study did not observe any significant activation in the amygdala, but the significant activation in the prefrontal cortex may have attenuated the response of the amygdala.

As we have already seen, the amygdala is activated in response to fearful facial expressions without any explicit processing instructions (Morris et al., 1996, 1998). Whalen et al. (1998) investigated whether the amygdala is activated in response to emotional stimuli, even when the subjects do not explicitly know that such stimuli are being presented. They used a backward masking procedure consisting of 33-ms presentation of fearful and happy expressions followed by 167-ms presentation of neutral facial expressions. Eight of 10 subjects reported that they only saw neutral faces. Activation in the amygdala was significantly different when subjects viewed masked fearful expressions than happy faces. Fearful faces increased the response significantly and happy faces decreased the response, a pattern which is consistent with the directional pattern found by Morris et al. (1996). In addition, signal intensity in response to masked fearful faces showed a similar habituation pattern as the one reported by Breiter et al. (1996). The experimental manipulation also produced activation in the substantia innominata in response to both fearful and happy expressions. The substantia innominata activation might represent a more generalized response to the salience or arousal level of the stimuli, whereas the amygdala activation is based on the valence of the stimuli. A number of studies report activation in the amygdala in response to visual stimuli with strong negative valence, including faces (Hamann, 2001; Irwin et al., 1996; Taylor et al., 1998, 2000).

Iidaka et al. (2001), using fMRI with a 3T magnet, revealed a differential role of the left and right amygdala and their neural connections. The analysis showed that the left amygdala was predominantly involved in the processing of negative expressions but the right amygdala did not show any differentiation for the expression conditions. The activation in the left amygdala covaried with activity in the left ventrolateral and right dorsolateral prefrontal cortices, suggesting that processing of negative expression is based on activation of a loop between the amygdala and the prefrontal cortex. The activation in the right amygdala interacted with the hippocampus and the temporal cortex in the right hemisphere. Further, Hariri et al. (2000) investigated the existence of a neural network that can control and modulate instinctive emotional responses (in this case labeling an emotional experience). Subject either matched the facial expression of two faces to a target (perceptual task) or identified the expression by choosing one of two presented linguistic labels (intellectual task). The matching task showed increased activation in both the left and right amygdala. However, the labeling task was

associated with decreased activation in the amygdala, but this decrease was correlated with an increase in activation in the right prefrontal cortex which is implicated in regulating emotional responses.

The neuroimaging studies reviewed so far have used adult subjects (mean age across four studies was 29.9 years). A recent fMRI study conducted by Baird et al. (1999) examined activation of the amygdala in a group of adolescents (mean age = 13.9 years) when fearful facial expressions were presented. This study found no significant difference between left and right amygdala activation. The data were therefore combined and showed a significant activation compared with looking at a fixation point. An obvious limitation of this study is that subjects saw only one type of facial expression. By including several emotions in the same study, it would be possible to compare differences in activation patterns involved in processing the different expressions. Nevertheless, the finding demonstrates that the amygdala is involved in processing fearful expressions before adulthood. However, because the children studied are relatively old, the data reported cannot eliminate a learning factor in the involvement of the amygdala in processing facial expressions. Such learning may occur earlier in development. Using a group of younger children as well as young adults, Thomas et al. (2001) report an fMRI study that assessed amygdala activation in response to a passive viewing task using fearful and neutral faces. The mean age of the children was 11 years and the mean age of the adults was 24 years. The adults showed increased left amygdala activation for fearful faces compared with neutral faces. This pattern was not found in the children who actually showed greater activation in the amygdala in response to neutral faces when compared with a fixation point. Behavioral data showed that the children experienced problems in categorizing facial expressions as fearful and neutral, which suggest that children may not reach adult levels in such discrimination tasks until early adolescence, which is the pattern observed with regard to face recognition. Thus, these results do point to a learning component. The higher activation for neutral faces was attributed to a perceived ambiguity in the stimuli, which may result in increased vigilance and thereby increasing the activity of the amygdala. Further, gender differences were observed in the children: Boys showed a decrease in response with repeated presentations of fearful faces, but the response in girls remained stable. These results are quite intriguing as they point to not only a developmental effect but also a gender effect. Kesler-West et al. (2001) also found gender differences in their subject pool of adult men and women. Men showed greater bilateral activation while viewing angry faces than happy faces; greater right hemisphere activation while viewing angry faces than sad faces; and greater left hemisphere activation to sad faces than happy faces. No such differences were reported for the female participants.

In summary, results from both human subjects and animals, lesion, and neuroimaging studies concur on the importance of the amygdala in the processing of fearful stimuli, including facial expression. On the other hand, the amygdala is also activated by other facial expressions and even faces in general, and shows rapid habituation effects. One possible explanation for this generalized response to faces is that there may be differential responses by subsets of nuclei within the amygdala structure. The amygdala is a small structure situated deep within the brain and therefore is hard to image. However, the use of higher field strength magnets (3T vs. 1.5T magnets) and more refined image acquisition techniques would increase the ability to detect more subtle activation changes in deep brain structures.

Disgust: Behavioral Studies

Evidence for a specific neutral substrate dedicated to the processing of stimuli involving disgust comes from nonhuman primate studies. Yaxley et al. (1988) identified the primate anterior insular cortex as the gustatory cortex, because this structure contained neurons that respond to pleasant and unpleasant tastes. Can a similar area be identified in the human brain that would respond to "disgusting" stimuli, in particular the perception of disgusted facial expressions? In what follows, impaired perception of disgust is illustrated different patient groups [Huntington's, obsessive–compulsive disorder (OCD), and Tourette's syndrome patients with and without OCD]. Then, evidence from neuroimaging studies supporting a specific neural substrate for disgust is presented.

Among the many symptoms of Huntington's disease are deficits in visual and auditory perception of social stimuli. Patients suffer generalized intellectual deterioration, including impaired face and facial expression processing (Jacobs et al., 1995). Initial neural degeneration affects the basal ganglia and the caudate nucleus. Postmortem studies have also revealed tissue loss in the amygdala region. Sprengelmeyer et al. (1996) investigated face perception and emotion recognition in a group of patients with Huntington's disease. The patient group performed a series of tasks that explored perception of age, sex, unfamiliar face identity (the Benton test), and gaze direction. They showed a marked deficit in evaluating gaze direction, and their performance on the Benton test was significantly below the controls. Using morphed facial images, subjects' perception of male–female continua, identity (i.e.,

Grant-Bogart) continua, and emotion continua (sadness–happiness and fear–anger) were evaluated. Patients with Huntington's disease performed the same as controls on all tasks except the fear–anger continua. To further evaluate emotion recognition, subjects identified morphed images of facial expressions from an "emotional hexagon." The same task was employed by Calder et al. (1996) in the case study of DR reviewed previously. The only emotion that was not impaired was happiness. Subjects had difficulty in recognizing anger, fear, and particularly disgust (63%, 57%, and 24% correctly identified, respectively). Identification of disgust was below chance level. Subjects with Huntington's disease also committed a significant number of so-called "remote prototype errors" (misclassifying emotions far apart on the emotional hexagon, such as mistaking surprise for anger). A study by Gray et al. (1997) further confirms the selective impairment for disgust in Huntington's disease. They studied a group of subjects who carried the gene for Huntington's disease but otherwise were clinically presymptomatic with no cognitive deterioration. Identification of famous faces, unfamiliar face matching, recognition memory for faces were normal. Facial expression recognition showed a selective deficit in recognition of disgust with some impairment for fear and anger. Interestingly, a study involving schizophrenic patients and their unaffected first-degree relatives found impaired face recognition in both the schizophrenics and the relatives (Conklin et al., 2002). Impaired-face-processing skills have been consistently demonstrated in schizophrenic patients (Archer et al., 1994; Baudouin et al., 2002; Heimberg et al., 1992; Mandal et al., 1998). Further studies are of course warranted, but impaired processing of facial information may provide insight into the etiology and symptomatology of different disorders. Sprengelmeyer et al. (1997) report that patients suffering from OCD are also selectively impaired in recognizing the expression of disgust, but that they understand the verbal meaning of disgust. Interestingly, groups of patients with generalized anxiety disorder tested in the same study showed an enhanced recognition of fear and anger.

Because the study conducted by Sprengelmeyer et al. of patients with Huntington's disease used the same test procedure and material as Calder et al. (1996) in assessing bilateral amygdala damage, the performance of these two patient groups can be compared. Both groups showed selective impairment in processing fear, which implicates the involvement of the amygdala. But the disproportionate impairment in recognition of fear in patients with amygdala damage and the selective deficit in recognition of disgust in patients with Huntington's disease and OCD points to a double dissociation, which suggests that different emotions may have dedicated neural substrates. In addition to the involvement of the basal ganglia structure, the processing of the facial expression of disgust would likely take place in a neural structure or network that includes the insular cortex and the orbitofrontal cortex. This network should be able to integrate visual, auditory, as well as olfactory information. Strengthening the support for a specific region dedicated to processing of disgust is the report a patient who suffered focal damage to the left insula and putamen and was impaired at the recognition of the facial expression of disgust and the experience of disgust (Calder et al., 2000). Interestingly, Harmer et al. (2002) found that a group of euthymic patients with bipolar disorder actually showed enhanced recognition of the expression of disgust. This finding raises the possibility that the study of facial expression recognition may provide insights into the neural networks that are involved in the regulation of mood as well as other disorders.

Disgust: Neuroimaging Studies

Following up the clinical evidence that suggests dedicated neural substrates for different emotions, Phillips et al. (1997) sought to find distinct neural substrates for the perception of fear and disgust. An fMRI study was designed to replicate amygdala activation in fear perception, and to explore the possibility of a separate substrate for the perception of disgust. Subjects viewed faces from the Ekman and Friesen face set displaying disgusted, fearful, and neutral expressions. The disgusted and fearful expressions were morphed to show two levels of intensity (75% and 150%). Four conditions (presented randomly) incorporated an alternating (neutral/emotional) design for each emotion (fear/disgust) and intensity of expression (75% and 150%). During scanning, subjects performed a gender identification task on the faces presented. A generic brain map showed activation of the left insula and amygdala for 75% fearful faces vs. neutral faces. No activation was observed at the 150% level of fearful expressions, possibly due to a type II error or to a rapid habituation of amygdala response (cf. Breiter et al., 1996). Adopting an ROI approach of 18 voxels representing the amygdala region bilaterally, activation was observed in the right amygdala and the putamen for the 150% fear intensity level. The most significant finding for the perception of facial expression of disgust was activation in the right insula, but not in the amygdala. Activation in the anterior insula became greater with increased emotional intensity. Additional areas activated by disgust were the medial frontal cortex, and right putamen (part of the basal ganglia structure) and

thalamus for the 150% intensity level. The anterior insula is connected to ventro-posterior-medial thalamic nucleus, which has been identified as the gustatory cortex in primates. In humans, this area is active during salt tasting and perception of aversive stimuli. Thus, the neural response to facial expressions of disgust appears closely related to the appraisal of distasteful stimuli and may well be served by the same neural substrate.

Sprengelmeyer et al. (1998) further examined cerebral activation in response to disgusted as well as fearful, angry, and neutral facial expressions. Six normal subjects took part in the fMRI study and performed a gender classification task on the face images. A voxel-by-voxel statistical analysis compared each expression (fear, anger, and disgust) against neutral faces as the baseline condition. The areas activated by the expression of disgust closely matched the findings of Phillips et al., with activation of the left insular cortex and the right putamen (basal ganglia area). Thus, converging evidence from clinical populations (Huntington's disease and OCD) and neuroimaging studies supports a dedicated neural substrate for processing the facial expression of disgust, which is a neural network incorporating the basal ganglia and the insular cortex. Contrary to previous findings, this study did not find activation in the amygdala in response to fear. This was attributed to rapid habituation effects in the amygdala (cf. Breiter et al., 1996). Fearful expression activated the right fusiform gyrus and the left dorsolateral frontal cortex. Angry expression evoked activation in the left inferior frontal lobe and the posterior part of the left temporal lobe. The results generally showed no overlap in the areas activated by the different emotions with the exception of the inferior frontal cortex, which was equally activated to faces showing anger, fear, and disgust as compared with neutral faces. Illustrating impaired facial expression in schizophrenics, Phillips et al. (1999) found that nonparanoid schizophrenics categorized the expression of disgust as either anger or fear more frequently than paranoids, and showed activation in the amygdala in response to disgust. Paranoid schizophrenics were more accurate in recognizing expressions, and showed greater activation patterns than nonparanoids.

A recent study by Gorno-Tempini et al. (2001) also reports activation of in the right putamen and caudate nucleus in response to disgust, in addition to activation in the right thalamus and the left amygdala. Recall that activation in the caudate nucleus and the amygdala was not reported by Phillips et al., which Gorno-Tempini et al. attribute to experimental design issues as they contrasted disgust with happiness and used an explicit expression recognition task. The contrast for happiness vs. disgust showed bilateral activation in the orbitofrontal region.

The study of the expression of disgust further supports the idea that different structures are involved in the perception of different facial expressions. The link between the amygdala and fear has already been established. It now appears that the basal ganglia structure and the insular cortex are involved in the perception of disgust.

Sadness and Anger: Behavioral Studies

The expression and perception of emotions such as sadness and anger are closely related to the concept of empathy—the identification with and understanding of another's situation, feelings, and motives. Sociopathy is characterized by disregard for others and aggressive behaviors. In a first study to investigate the relationship between expression recognition and behavioral problems, Blair and Coles (2000) showed that in their sample of adolescent children, the ability to recognize sad and fearful expressions was inversely related to levels of affective–interpersonal disturbance, impulsiveness, and conduct problems. Further, Blair and Cipolotti (2000) reported on a patient with damage to the right frontal region, including the orbitofrontal cortex, who showed a case of what they termed "acquired sociopathy." The patient showed difficulties in the area of social cognition, in particular facial expression recognition of anger and fear. The patient's impairment was attributed to a reduced ability to generate expectation of other's negative emotional reactions and to suppress inappropriate behaviors. The authors propose that the orbitofrontal cortex is implicated in the generation of such expectations and suppression of behaviors. Psychopathic patients generally show reduced autonomic responses to sad and angry expressions, impoverished aversive conditioning, and reduced startle reflexes, which is a syndrome similar to patients with amygdala lesions. Patients with lesions to the temporal lobe, including the amygdala, consistently fail to recognize fear as well as impaired recognition of sad facial expressions (Fine and Blair, 2000). Both animal and human lesion studies have implicated the orbitofrontal cortex in behavioral extinction and reversal learning (see Rolls, 2000). Therefore, the amygdala and orbitofrontal cortex would be likely structures for processing facial expressions for sadness and anger respectively.

Sadness and Anger: Neuroimaging Studies

A recent study by Blair et al. (1999) reports findings of dissociable neural responses to the facial expressions of sadness and anger. Thirteen male subjects took part in the

PET study. During scanning, subjects performed a sex discrimination task on morphed facial expressions of sadness and anger displaying varying degrees of intensity. The statistical analysis identified differential responses to sad and angry faces. Signal intensity also increased as a function of degree of emotional intensity displayed by the face. Sad expressions showed activation in the left amygdala and the right middle and inferior temporal gyrus. Angry expressions showed significant activation in the right orbitofrontal cortex (as predicted) and bilateral activation in the anterior cingulate cortex. Conjoint activation was found in the right temporal pole and anterior cingulate cortex as a function of increased emotional intensity. The unilateral response in the left amygdala to sad expressions parallels the response to fearful faces observed in other imaging studies. Unfortunately, this study did not include the perception of fear as a control stimuli. As such we are left to speculate whether there would be a differential response to sad and fearful expressions in the amygdala. As suggested before, it is possible that different nuclei of the amygdala may respond to different emotions. The activation of the right inferior and middle temporal gyri in response to sad expressions is in line with the findings of Adolphs et al. (1996) who proposed an extended right hemisphere neural system for processing facial expressions. On the basis of their results, Blair et al. suggest the involvement of at least two dissociable, but interlocking systems in the processing of negative facial expressions. One system responds to facial stimuli (sad, fearful) involved in (social) aversive conditions; the other system implicates regions involved in behavioral extinction by responding to angry facial expressions. The differential activation patterns obtained by Sprengelmeyer et al. (1998) for fear, anger, and disgust support this position.

Recent studies have examined perception of facial expressions in schizophrenic patients. Schizophrenics have consistently performed worse than controls in facial expression discrimination tasks (Heimberg et al., 1992). Phillips et al. (1999) replicated activation in the left amygdala in normal controls in response to fearful expressions. Schizophrenics showed no activation in the amygdala in this implicit gender discrimination task. Further, Schneider et al. (1998) found amygdala activation in normal controls in a sad mood induction task, using sad facial expressions. The schizophrenics in this study did not show any activation. Kosaka et al. (2002) had schizophrenics and normal controls perform an emotional intensity judgment task on positive (happy) and negative (angry/disgusted/sad) faces. Here, in contrast, the schizophrenics showed greater activation in the amygdala than did the normal controls. In a study with another type of patient group (manic patients), Lennox et al. (2002) found that normal controls displayed the expected activation pattern of increased activation in the amygdala and the anterior cingulate cortex to increasing intensities of sadness. This pattern of response was absent in the manic patients.

Happiness: Behavioral Studies

Smiling appears to be innate. An infant will produce the first smile anywhere from 2 to 12 hr after birth. In fact, even blind and deaf babies smile. Ekman has identified several different types of smiles; enjoyment, dampened, miserable, qualifier, compliance, coordination, flirtatious, and embarrassed (in McNeill, 1998, pp. 206–208). The type of smile most used in facial expression studies is that of enjoyment or the so-called Duchenne smile. According to the norms published by Ekman and Friesen (1976), mean accuracy for recognition of the facial expression of happiness reached 100% for the subset of faces used by Young et al. (1996), which makes the smile the most easily recognized expression.

So far, no patient groups have displayed problems in recognizing happy facial expression. Both patients with amygdala damage (SM and DR) and patients with Huntington's disease performed at normal levels in processing happy facial expressions. Similarly, Adolphs et al. (1996) did not find any patients that displayed problems in recognizing happy facial expressions. In fact, recognition scores for happy expression did not correlate with the recognition of any other emotion. Taken together, these results suggest that a happy facial expression may be processed differently than all other expressions.

Happiness: Neuroimaging Studies

Neuroimaging studies of the perception of happy facial expressions have usually contrasted this expression with the perception of negative facial expressions (i.e., fear, anger, and disgust). So far, no consistent pattern of activation has been found in response to smiling. Morris et al. (1996) found no activation in the amygdala for the contrast of happy–fearful expressions. Statistical parametric mapping of this contrast showed activation in the right medial temporal gyrus, right putamen, left superior parietal lobe, and left calcarine sulcus. On the other hand, Breiter et al. (1996), in their second experiment, found that the left anterior amygdala responded preferentially to happy vs. neutral faces. This suggests a possible generalized response to emotionally valenced stimuli in the amygdala, but rapid habituation effects to both happy and fearful expressions were observed. Support for a broader role for

the amygdala in modulating the vigilance level during the perception for both positive and negative comes from a recent study by Yang et al. (2002), who found reliable bilateral amygdala activation to happy as well as sad, angry, and fearful faces. Gorno-Tempini et al. (2001) found bilateral activation in the orbitofrontal regions (BA 11 and 47) in both implicit and explicit processing of happy expressions.

A PET study conducted by Dolan et al. (1996) investigated neural activation during covert processing of happy facial expressions. Prior to scanning, subjects were presented with a face that they were explicitly asked to hold in mind for 45 s after which they had to match the target face to one of two faces. Repeated blood flow measures were obtained whereas the subjects held happy and neutral faces in mind. Presentation of happy faces (compared to neutral and a controlled fixation condition) was associated with significant activation in left ventral prefrontal cortex, left anterior cingulate cortex, thalamus, and the right fusiform gyrus. In contrast to other studies, the amygdala was not activated.

It thus appears that there is a differential activation pattern for negative and positive facial expressions. Processing of negative facial expressions involves regions that specifically have been damaged in clinical populations, such as the amygdala for fear, and the basal ganglia and the insula for disgust. No consistent activation patterns in response to a smiling face have been identified, nor did Adolphs et al. (1996) find any patients who were deficient in processing happy facial expressions.

Surprise

So far, no major studies have focused on the facial expression of surprise. The few data available on the perception, classification, and identification of surprised facial expressions have been collected in conjunction with assessment of the five other basic emotions (Adolphs et al., 1994, 1995; Calder et al., 1996; Young et al., 1995, 1996). For example, patient SM rated facial expressions of surprise, anger, and fear as less intense than controls. Bilateral damage to the amygdala has been shown to impair judgment of the intensity of fearful expressions, and also of expressions normally judged to be similar to fear, such as surprise. As the main focus of these studies was fear and the involvement of the amygdala, any observed deficit related to the perception of surprise was not further addressed. The expression of surprise bears strong resemblances with fear: eyes and mouth wide open. Surprise is also the briefest expression, lasting less than 1 s. An important point to consider is that emotional states are dynamic. This is especially important for surprise, because an expression of surprise can quickly turn into fear or happiness, depending upon the nature of the surprise. In fact, *surprise* can be defined as a transitory emotion which leads into the appropriate reaction for the emotional event facing the person. Given the short-lived nature of a surprised expression and given that surprise often transitions into an expression of fear, it is quite likely that the amygdala is also involved in processing the perception of surprise.

Context Effects

The areas involved in the processing of facial expressions have been identified by the use of relatively simple tasks, such as gender categorization and passive viewing, which involves mostly implicit processing. A major point to consider when assessing such activation patterns is how well these tasks replicate face processing in real life. Few would argue that the environment in a scanning room and even psychology laboratories, for that matter, comes close to replicating real-life situations. In the real world, a correct interpretation of facial expressions would also be guided by the situational context, which is usually accompanied by a verbal message and a body language. Thus, we can add factors such as prosody as well as gaze to aid the interpretation of a facial expression. In addition, the salience and valence of the expressions must also be taken into account. Interpretation of facial expressions in real life would therefore generally appear to benefit from additional top-down or explicit processing. Two recent studies have established differences in activation patterns between explicit and implicit processing of facial expression. In an explicit processing task, Critchley et al. (2000) had subjects judge happy, angry, and neutral facial expressions. In an implicit processing task, subjects performed a gender categorization task on faces displaying the same expressions as in the explicit task. Overall, processing of facial expressions increased activity in the fusiform and middle temporal gyri, the hippocampus, the amygdala-hippocampal junction, and the pulvinar nucleus. The neural substrates for explicit and implicit processing were shown to be dissociable, with explicit processing activating the temporal lobe cortex and implicit processing activating the amygdala. The latter result thus replicates findings from previous experiments as reviewed above.

The study by Gorno-Tempini et al. (2001), already referred to in the discussion on processing of disgust, used both explicit and incidental tasks, expression recognition and gender decision respectively, in processing of disgusted, happy, and neutral expressions. Regions of

activation varied by both task and facial expression: the right neostriatum and the left amygdala showed higher activation in explicit processing than in incidental processing. The bilateral orbitofrontal cortex activated to explicit recognition of happy expressions. Common activation for all conditions were found in the right temporal occipital junctions, which would be indicative of visual and perceptual processing, and the left temporal and left inferior frontal cortex, which would be involved in semantic processing. Clearly then, activation is both distributed and task-modulated.

The observed difference in activation patterns between explicit and implicit processing, or what could also be viewed as conscious and unconscious processing, falls in line with LeDoux's concept of the "low and high roads" for processing emotional stimuli (LeDoux, 1996). Recall that the "low road" provides a crude representation of the stimuli to the amygdala, whereas the "high road" involves elaborated processing in the sensory cortex. By adding factors such as social context, prosody, gaze, and body language to the relatively simple visual perception of facial expressions, the conscious processing of facial expression can then be also subsumed under the concept of social cognition (see Adolphs, 1999, 2001, for recent reviews of the field of social cognition). For example, damage to the amygdala goes beyond pure impairments in facial expression recognition and appears to play a role in social decision making. This is illustrated by a study of three subjects with bilateral damage to the amygdala who were impaired at facial expression recognition and who deviated from normal controls in social judgments involving facial stimuli. The subjects with amygdala damage judged faces that normal subjects had deemed most untrustworthy and unapproachable as trustworthy and approachable (Adolphs, 1999; Adolphs et al., 1998). Adolphs et al. (2001) recently extended this finding to a group of high-functioning autistic subjects who were tested on the same material as the bilateral-amygdala-damaged patients. The autistic subjects showed normal social judgments from lexical stimuli, but, as the amygdala patients, showed abnormal social judgments regarding the trustworthiness of faces. These findings support the role of the amygdala in linking visual perception of socially relevant stimuli with the retrieval of social knowledge and subsequent social behaviors.

Summary: Facial Expressions

The goal of imaging studies of the perception of facial expressions has been to evaluate whether there are distinct neural substrates dedicated to processing emotions as displayed by different facial expressions. Evidence from behavioral and lesion studies do suggest that different structures are activated by different emotions. The role of the amygdala in processing fearful stimuli has been well established. Recall that the patients who presented lesions of the amygdala and were impaired at processing negative emotions with fear being most strongly affected. Patients with Huntington's disease display loss of amygdala and basal ganglia tissue, associated with impaired processing of fear, anger, and disgust in particular. However, no subject groups displayed any difficulties in processing happy facial expression. This suggests differential processing of positive and negative emotions. So far, a number of neuroimaging studies have shown differential activation patterns in response to five of the six basic emotions displayed by facial expression. No studies have examined activation by surprised facial expression.

Activation of the amygdala by fearful expressions should come as no surprise as reported by Morris et al. (1996, 1998) and Breiter et al. (1996). But, note that the facial expressions of sadness and happiness also activated the amygdala. Amygdala activation has been also reported in a categorization task of unknown faces (Dubois et al., 1999). Thus, it is quite likely that the amygdala also responds to faces in general with the purpose of assessing the possible threatening valence of a stimuli (e.g., "Is this a friend or foe?"). Further, the results from the imaging studies of disgust implicate the basal ganglia structure as well as the insular cortex in the processing of this emotion (Gorno-Tempini et al., 2001; Phillips et al., 1997; Sprengelmeyer et al., 1997). Interestingly, the two facial expressions for which consistent patterns of activation have been established are fear and disgust. These are emotions that are evoked in direct threats to the system.

The majority of studies that have examined activation in response to different facial expressions also have found activation in other areas (e.g., prefrontal cortex, inferior frontal cortex, right medial temporal region, anterior cingulate cortex, inferior temporal cortex, and orbitofrontal cortex) in addition to the regions of particular interest. The inferior frontal cortex is commonly activated in response to different facial expressions, and may serve as an area for integration or semantic processing of information contained in facial expressions. Additionally, activation in the orbitofrontal cortex in response to facial expressions is quite interesting because this region is implicated in numerous cognitive activities, including decision making, response selection and reward value, behavior control, as well as judgments about olfactory stimuli (Abdi, 2002; Blair and Cipolotti, 2000; Rolls, 2000). Attractive faces produced activation in the medial orbitofrontal cortex, and the response was enhanced if the face was smiling

in addition to being attractive (O'Doherty et al., 2003). However, the areas mentioned above are also activated by other face-processing tasks, such as encoding and recognition. Therefore, the functionality or exact role of these additional areas of activation remains unclear.

According to Haxby et al.'s distributed neural model of face perception (Haxby et al., 2000), some of these additional areas of activation would be considered to be part of the extended network dedicated to the processing of changeable aspects of the face. One approach to further examine the functionality of these extended activation patterns would be to include facial expressions as part of an experimental design that manipulates facial expressions during both encoding and recognition of faces.

In summary, we have accumulated considerable evidence that facial expression processing is supported by highly specialized circuits or neural substrates. According to Haxby et al.'s model, changeable aspects of faces processing that includes the perception of facial expressions, as well as gaze and lip movement, are processed in the temporal sulcus which inputs to the amygdala, insula, and limbic system for processing emotion, the intraparietal sulcus for gaze detection, and the auditory cortex for processing of speech. By specifically addressing the changeable aspects of face processing (including facial expressions), the model of a distributed neural system of face perception proposed by Haxby and colleagues complements the Bruce and Young cognitive model of face recognition, which mainly specified the pathways to successful face recognition. Perhaps the word "distributed" is by far the best descriptor of the neural activity patterns evoked by both faces and facial expressions. Taken together, these two models present a more complete picture of how facial information is processed in the human brain.

CONCLUSION

We began this review by reporting a dissociation in the processing of facial identity and facial emotions as evidenced in certain patient populations. For example, prosopagnosic patients fail to recognize faces, but some show no impairment in processing facial expressions, whereas patients with amygdaloid lesions display problems in processing facial expressions but not facial identity. Although the Bruce and Young model states that identity processing follows a different route than facial expression processing, findings from experimental and behavioral studies alone failed to a certain degree to establish functional independence between the two subsystems, because the testing paradigms employed often confounded facial expression and facial identification tasks. From the field of neurophysiology, single-cell recordings in primates have identified differential neuronal responses to facial identity and expressions, as well as different brain areas. Event-related potential studies have established a different time course as well as different foci for identity and expression processing. Taken together, the findings from experimental, neuropsychological, and neurophysiological approaches strongly support the existence of dissociable systems for processing facial identity and facial expressions, and thus validate the Bruce and Young model, which postulates independent processing of identity and expression.

How successful has the newer brain imaging approach been in supporting the existence of separate systems dedicated to the processing of facial identity and facial expression? So far, activation of the fusiform gyrus has been well established in a number of face-processing tasks. The activation patterns in the fusiform gyrus in response to face perception do correlate with lesion sites resulting in an inability to recognize faces, as seen in prosopagnosic patients. However, the fusiform gyrus is also activated when facial expressions are processed. But because the majority of studies of facial expression processing used an ROI approach, additional areas of activation were largely not evaluated or at least not reported. The role of the amygdala in processing fearful facial expressions is also well established, but the amygdala has also been found to show a generalized response to faces. Thus, it becomes quite difficult to tell whether observed activation patterns are in response to facial identity or facial expression processing. Because the areas of neural activation in response to both face-processing tasks are quite extensive and show considerable overlap, we are still faced with the task of interpreting and assessing the functionality of these areas of activation. Going beyond the fusiform gyrus, the activation patterns in response to different face-processing tasks reveal to a large extent a task-dependent as well as distributed network.

Although neuroimaging studies of face perception are answering questions related to basic research into face processing, the results from neuroimaging studies of facial expression processing in patient populations show perhaps greater promise of having direct applications. As we have seen, deficits in facial expression processing have been found in a number of different disorders, ranging from Huntington's disease, schizophrenia, depression, mania, OCD, sociopathy to autism. Gaining an understanding of the neural networks involved in the processing of affective stimuli may provide further insights into the spectrum of deficits associated with these disorders. For example, Sheline et al. (2001) reported normalized amygdala activation in a group of depressed patients with antidepressant

treatment. One should, however, keep in mind that the findings that have been reviewed in this paper come from studies that report patterns of activation at a group level, sometimes with a relatively small sample size. Extending these findings to clinical applications at an individual level will require more work. Whether having patients somehow being trained in interpreting or attending to facial expressions could become a viable treatment option also remains open for future research. However, a recent case study of a patient with Balint's syndrome showed that cognitive rehabilitation training resulted in improved performance in different areas, including the ability to recognize famous faces (Rosselli et al., 2001).

The Bruce and Young model for face recognition integrated findings from experimental, behavioral, and neuropsychological studies and provided a sound framework for future experimental probes of face processing. As we have seen, 15 years later the Bruce and Young model still guides researchers using new technologies. The model proposed by Haxby and colleagues also integrates the findings from numerous imaging studies of various face-processing tasks and shows promise of providing a similar framework for future work. It is however prudent to remember that the discipline of neuroimaging is still relatively young. Issues pertaining to both imaging techniques, statistical analysis, as well as experimental design, must be carefully evaluated and refined before we can ascertain the reliability of this approach in not only dissociating facial identity and facial expression processing, but also assessing the functionality of the different areas of activation as well as the temporal sequence of face processing.

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REFERENCES

Abdi, H. (2002). What can psychology and sensory evaluation learn from each other? *Food Qual. Pref.* **13:** 445–451.

Adolphs, R. (1999). Social cognition and the human brain. *Trends Cogn. Sci.* **3:** 469–479.

Adolphs, R. (2001). The neurobiology of social cognition. *Curr. Opin. Neurobiol.* **11:** 231–239.

Adolphs, R., Tranel, D., Damasio, H., and Damasio, A. R. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature* **372:** 613–614.

Adolphs, R., Tranel, D., Damasio, H., and Damasio, A. R. (1995). Fear and the human amygdala. *J. Neurosci.* **15:** 5879–5891.

Adolphs, R., Damasio, H., Tranel, D., and Damasio, A. R. (1996). Cortical systems for the recognition of emotion in facial expressions. *J. Neurosci.* **16**(23): 7678–7687.

Adolphs, R., Tranel, D., and Damasio, A. R. (1998). The human amygdala in social judgment. *Nature* **393:** 470–474.

Adolphs, R., Sears, L., and Piven, J. (2001). Abnormal processing of social information from faces in autism. *J. Cogn. Neurosci.* **13**(2): 232–240.

Aggleton, J. P. (1992). The functional effects of amygdala lesions in humans: A comparison with findings from monkeys. In Aggleton, J. P. (ed.), *The Amygdala*, Wiley-Liss, New York, pp. 485–503.

Aguirre, G. K., Singh, R., and D'Esposito, M. (1999). Stimulus inversion and the response of face object-sensitive cortical areas. *Neuroreport* **10:** 194–198.

Allison, T., Puce, A., Spencer, D. D., and McCarthy, G. (1999). Electrophysiological studies of human face perception. I: Potentials generated in occipitotemporal cortex by face and non-face stimuli. *Cereb. Cortex* **9:** 415–430.

Anderson, A. K., and Phelps, E. A. (2000). Expression without recognition: Contributions of the human amygdala to emotional communication. *Psychol. Sci.* **11:** 106–111.

Archer, J., Hay, D. C., and Young, A. W. (1994). Movement, face processing, and schizophrenia: Evidence of a differential deficit in expression analysis. *Br. J. Clin. Psychol.* **33:** 517–528.

Attwell, D., and Iadecola, C. (2002). The neural basis of functional brain imaging signals. *Trends Neurosci.* **25:** 621–625.

Baddeley, A. D., and Woodhead, M. (1981). Techniques for improving eyewitness identification skills. Paper presented at the *SSRC Law and Psychology Conference*, Trinity College, Oxford.

Baird, A. A., Staci, M. A., Gruber, E. M., Fein, D. A., Maas, J. C., Steingard, R. J., et al. (1999). Functional magnetic resonance imaging of facial affect recognition in children and adolescents. *J. Am. Acad. Child Adolesc. Psychiatry* **38:** 195–199.

Bandettini, P. A., and Ungerleider, L. G. (2001). From neuron to BOLD: New connections. *Nat. Neurosci.* **4:** 864–866.

Bartlett, J. C., and Searcy, J. (1993). Inversion and configuration of faces. *Cognit. Psychol.* **25:** 281–316.

Bartlett, J. C., Searcy, J., and Abdi, H. (2003). What are the routes to face recognition? In Peterson, M., and Rhodes, G. (eds.), *Analytic and Holistic Processes in the Perception of Faces, Object, and Scenes*, Oxford University Press, Oxford, pp. 21–52.

Baudouin, J.-Y., Martin, F., Tiberghien, G., Verlut, I., and Frank, N. (2002). Selective attention to facial emotion and identity in schizophrenia. *Neuropsychologia* **40:** 503–511.

Bentin, S., and Carmel, D. (2002). Accounts for the N170 face-effect: A reply to Rossion, Curran, & Gauthier. *Cognition* **85:** 197–202.

Bentin, S., and Deouell, L. Y. (2000). Structural encoding and identification in face processing: ERP evidence for separate mechanisms. *Cogn. Neuropsychol.* **17:** 35–54.

Bentin, S., Allison, T., Puce, A., Perez, E., and McCarthy, G. (1996). Electrophysiological studies of face perception in humans. *J. Cogn. Neurosci.* **8:** 551–565.

Bentin, S., Deouell, L. Y., and Soroker, N. (1999). Selective visual streaming in face recognition: Evidence from developmental prosopagnosia. *Neuroreport* **10:** 823–827.

Bernstein, L. J., Beig, S., Siegenthaler, A. L., and Grady, C. L. (2002). The effect of encoding strategy of the neural correlates of memory for faces. *Neuropsychologia* **40:** 86–98.

Blair, R. J., and Cipolotti, L. (2000). Impaired social response reversal. A case of "acquired sociopathy." *Brain* **123:** 1122–1141.

Blair, R. J., and Coles, M. (2000). Expression recognition and behavioural problems in early adolescence. *Cogn. Dev.* **15:** 421–434.

Blair, R. J. R., Morris, J. S., Frith, C. D., Perrett, D. I., and Dolan, J. R. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain* **122:** 883–893.

Breiter, H. C., Etcoff, N., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., et al. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron* **17:** 875–887.

Bruce, V. (1982). Changing faces: Visual and non-visual coding process in face recognition. *Br. J. Psychol.* **73:** 105–116.

Bruce, V., and Young, A. (1986). Understanding face recognition. *Br. J. Psychol.* **77:** 305–327.

Buckner, R. B., and Logan, J. M. (2001). Functional neuroimaging methods: PET and fMRI. In Cabeza, R., and Kingstone, A. (eds.), *Handbook of Functional Neuroimaging of Cognition*, The MIT Press, Cambridge, pp. 27–48.

Calder, A. J., Young, A. W., Rowland, D., Perrett, D. I., Hodges, J. R., and Etcoff, N. L. (1996). Facial emotion recognition after bilateral amygdala damage: Differentially severe impairment of fear. *Cogn. Neuropsychol.* **13:** 699–745.

Calder, A. J., Keane, J., Manes, F., Antoun, N., and Young, A. W. (2000). Impaired recognition and experience of disgust following brain injury. *Nat. Neurosci.* **3:** 1077–1078.

Carmel, D., and Bentin, S. (2002). Domain specificity versus expertize: Factors influencing distinct processing of faces. *Cognition* **83:** 1–29.

Clark, V. P., Keil, K., Maisog, J. M., Courtney, S., Ungerleider, L. G., and Haxby, J. V. (1996). Functional magnetic resonance imaging of human visual cortex during face matching: A comparison with positron emission tomography. *Neuroimage* **4:** 1–15.

Conklin, H. M., Calkins, M. E., Anderson, C. W., Dinzeo, T. J., and Iacono, W. G. (2002). Recognition memory for faces in schizophrenia patients and their first-degree relatives. *Neuropsychologia* **40**(13): 2314–2324.

Critchley, H., Daly, E., Phillips, M., Brammer, M., Bullmore, E., Williams, S., et al. (2000). Explicit and implicit neural mechanisms for processing of social information from facial expressions: A functional magnetic resonance imaging study. *Hum. Brain Mapp.* **9:** 93–105.

Damasio, A. R., Damasio, H., and Van Hoesen, G. W. (1982). Prosopagnosia: Anatomic basis and behavioral mechanisms. *Neurology* **32:** 331–341.

Damasio, A. R., Tranel, D., and Damasio, H. (1990). Face agnosia and the neural substrates of memory. *Annu. Rev. Neurosci.* **13:** 89–109.

Davies, G. M., Ellis, H. D., and Shepherd, J. W. (1978). Face recognition accuracy as a function of mode of representation. *J. Appl. Psychol.* **63:** 180–187.

Davis, M., and Whalen, P. J. (2001). The amygdala: Vigilance and emotion. *Mol. Psychiatry* **6:** 13–34.

De Renzi, E., Faglioni, R., Grossi, D., and Nichelli, P. (1991). Apperceptive and associative forms of prosopagnosia. *Cortex* **27:** 213–222.

De Renzi, E., Perani, D., Carlesimo, G. A., Silveri, M. C., and Fazio, F. (1994). Prosopagnosia can be associated with damage confined to the right hemisphere. An MRI and PET study and a review of the literature. *Neuropsychologia* **32:** 893–902.

Desimone, R. (1991). Face selective cells in the temporal cortex of monkeys. *J. Cogn. Neurosci.* **3:** 1–8.

D'Esposito, M., Zarahn, E., and Aguirre, G. K. (1999). Event-related functional MRI: Implications for cognitive psychology. *Psychol. Bull.* **125:** 155–164.

Dolan, R. J., Fletcher, P., Morris, J., Kapur, N., Deakin, F. W., and Frith, C. D. (1996). Neural activation during covert processing of positive emotional facial expressions. *Neuroimage* **4:** 194–200.

Dubois, S., Rossion, B., Schiltz, C., Bodart, J. M., Michel, C., Bruyer, R., et al. (1999). Effect of familiarity on the processing of human faces. *Neuroimage* **9:** 278–289.

Dukes, W. D., and Bevan, W. (1967). Stimulus variation and repetition in the acquisition of naming responses. *J. Exp. Psychol.* **74:** 178–181.

Eimer, M. (2000a). Event-related brain potentials distinguish processing stages involved in face perception and recognition. *Clin. Neurophysiol.* **111:** 694–705.

Eimer, M. (2000b). Effects of face inversion on the structural encoding and recognition of faces: Evidence from event-related brain potentials. *Cogn. Brain Res.* **10:** 145–158.

Eimer, M., and McCarthy, R. A. (1999). Prosopagnosia and structural encoding of faces: Evidence from event-related potentials. *Neuroreport* **10:** 255–259.

Ekman, P. (1972). Universals and cultural differences in facial expressions of emotion. In Cole, J. (ed.), *Nebraska Symposium on Motivation*, University of Nebraska Press, Lincoln, pp. 207–283.

Ekman, P. (1992). Facial expression and emotion. *Am. Psychol.* **48**(4): 384–392.

Ekman, P., and Friesen, W. (1976). *Pictures of Facial Affect*, Consulting Psychological Press, Palo Alto, CA.

Ellis, H. D., Shepherd, J. W., and Davies, G. M. (1979). Identification of familiar and unfamiliar faces from internal and external features: Some implications for theories of face recognition. *Perception* **8:** 431–439.

Endo, N., Endo, M., Kirita, T., and Maruyama, K. (1992). The effects of expression on face recognition. *Tohoku Psychol. Folia* **52:** 37–44.

Etcoff, N. L. (1984). Selective attention to facial identity and facial emotion. *Neuropsychologia* **22:** 281–295.

Farah, M. J. (1991). Patterns of co-occurrence among the associative agnosias: Implications for visual object representation. *Cogn. Neuropsychol.* **8:** 1–19.

Farah, M. J., and Aguirre, G. K. (1999). Imaging visual recognition: PET and fMRI studies of the functional anatomy of human visual recognition. *Trends Cogn. Sci.* **3:** 179–186.

Farah, M. J., Wilson, K. D., Drain, M., and Tanaka, J. N. (1995). The inverted face effect in prosopagnosia: Evidence for mandatory, face-specific perceptual mechanisms. *Vision Res.* **35:** 2089–2093.

Farah, M. J., Wilson, K. D., Drain, M., and Tanaka, J. N. (1998). What is "special" about face perception? *Psychol. Rev.* **105:** 482–498.

Feinstein, J. S., Goldin, P. R., Stein, M. B., Brown, G. G., and Paulus, M. P. (2002). Habituation of attentional networks during emotion processing. *Neuroreport* **13:** 1255–1258.

Fine, C., and Blair, R. J. R. (2000). Mini review: The cognitive and emotional effects of amygdala damage. *Neurocase* **6:** 435–450.

Friston, K. J., Holmes, A., Worsley, K. J., Poline, J.-B., Frith, C. D., and Frackowiak, R. S. J. (1995). Statistical parametric maps in functional imaging: A general linear approach. *Hum. Brain Mapp.* **2:** 189–210.

Friston, K. J., Holmes, A., Poline, J.-B., Price, C. J., and Frith, C. D. (1996a). Detecting activations in PET and fMRI: Levels of inference and power. *Neuroimage* **40:** 223–235.

Friston, K. J., Price, C. J., Fletcher, R., Moore, C., Frackoviak, R. S. J., and Dolan, R. J. (1996b). The trouble with cognitive subtraction. *Neuroimage* **40:** 97–104.

Gauthier, I., Anderson, A. W., Tarr, M. J., Skudlarski, P., and Gore, J. C. (1997). Levels of categorization in visual recognition studied with functional MRI. *Curr. Biol.* **7:** 645–651.

Gauthier, I., Behrmann, M., and Tarr, M. (1999a). Can face recognition really be dissociated from object recognition? *J. Cogn. Neurosci.* **11:** 349–370.

Gauthier, I., Tarr, M. J., Anderson, A. W., Skudlarski, P., and Gore, J. C. (1999b). Activation of the middle fusiform "face area" increases with expertise in recognizing novel objects. *Nat. Neurosci.* **2:** 568–573.

Gauthier, I., Skudlarski, P., Gore, J. C., and Anderson, A. W. (2000). Expertize for cars and birds recruits brain areas involved in face recognition. *Nat. Neurosci.* **3:** 191–197.

Gauthier, I., and Tarr, M. J. (1997). Becoming a "greeble" expert: Exploring mechanisms for face recognition. *Vision Res.* **37:** 1673–1682.

George, N., Dolan, R. J., Fink, G. R., Baylis, G. C., Russell, C., and Driver, J. (1999). Contrast polarity and face recognition in the human fusiform gyrus. *Nat. Neurosci.* **2:** 574–580.

George, N., Driver, J., and Dolan, R. (2001). Seen gaze-direction modulates fusiform activity and its coupling with other brain areas during face processing. *Neuroimage* **13:** 1102–1112.

Gorno-Tempini, M. L., Pradelli, S., Serafini, M., Pagnoni, G., Baraldi, P., Porro, C., et al. (2001). Explicit and incidental facial expression processing: An fMRI study. *Neuroimage* **14:** 465–473.

Gray, J. M., Young, A. W., Barker, W. A., Curtis, A., and Gibson, D. (1997). Impaired recognition of disgust in Huntington's disease gene carriers. *Brain* **120:** 2029–2038.

Grelotti, D. J., Gauthier, I., and Schultz, R. T. (2002). Social interest and the development of cortical face specialization: What autism teaches us about face processing. *Dev. Psychobiol.* **40:** 213–225.

Hadjikhani, N., and de Gelder, B. (2002). Neural basis of prosopagnosia: An fMRI study. *Hum. Brain Mapp.* **16:** 176–182.

Hamann, S. (2001). Cognitive and neural mechanisms of emotional memory. *Trends Cogn. Sci.* **5:** 394–400.

Hamann, S. F., Stefanacci, L., Squire, L., Adolphs, R., Tranel, D., Damasio, H., et al. (1996). Recognizing facial emotion. *Nature* **379:** 497.

Hasselmo, M. E., Rolls, E. T., and Baylis, G. C. (1989). The role of expression and identity in the face-selective responses of neurons in the temporal visual cortex of the monkey. *Behav. Brain Res.* **32:** 203–218.

Hariri, A. R., Bookheimer, S. Y., and Mazziotta, J. C. (2000). Modulating emotional responses: Effects of neocortical network on the limbic system. *Neuroreport* **11:** 43–48.

Harmer, C. J., Grayson, L., and Goodwin, G. M. (2002). Enhanced recognition of disgust in bipolar illness. *Biol. Psychiatry* **51:** 298–304.

Haxby, J. V., Grady, C. L., Horwitz, B., Ungerleider, L. G., Mishkin, M., Carson, R. E., et al. (1991). Dissociation of spatial as visual processing pathways in the human extrastriate cortex. *Proc. Natl. Acad. Sci. U.S.A.* **88:** 1621–1625.

Haxby, J. V., Horwitz, B., Underleider, L. J., Maisog, J. M., Pietrini, P., and Grady, C. L. (1994). The functional organization of human extrastriate cortex: A PET-rCBF study of selective attention to faces and locations. *J. Neurosci.* **14:** 6336–6353.

Haxby, J. V., Ungerleider, L. G., Horwitz, B., Maisog, J. M., Rapoport, S. I., and Grady, C. L. (1996). Face encoding and recognition in the human brain. *Proc. Natl. Acad. Sci. U.S.A.* **93:** 922–927.

Haxby, J. V., Ungerleider, L. G., Clark, V. P., Schouten, J. L., Hoffman, E. A., and Martin, A. (1999). The effect of face inversion on activity in human neural systems for face and object perception. *Neuron* **22:** 189–199.

Haxby, J. V., Hoffman, E. A., and Gobbini, I. M. (2000). The distributed human neural system for face perception. *Trends Cogn. Sci.* **4:** 223–233.

Haxby, J. V., Gobbini, I. M., Furey, M. L., Ishai, A., Schouten, J. L., and Pietrini, P. (2001). Distributed and overlapping representation of faces and objects in the ventral temporal cortex. *Science* **293:** 2425–2430.

Haxby, J. V., Hoffman, E. A., and Gobbini, I. M. (2002). Human neural systems for face recognition of social communication. *Biol. Psychiatry* **51:** 59–67.

Heimberg, C., Gur, R. E., Erwin, R. J., Shtasel, D. L., and Gur, R. C. (1992). Facial emotion discrimination: III. Behavioral findings in schizophrenia. *Psychiatry Res.* **42:** 253–265.

Herrmann, M. J., Aranda, D., Ellgring, H., Mueller, T. J., Strik, W. K., Heidrich, A., et al. (2002). Face-specific event-related potential in humans is independent from facial expression. *Int. J. Psychophysiol.* **45:** 241–244.

Hoffman, E. A., and Haxby, J. V. (2000). Distinct representations of eye gaze and identity in the distributed human neural system for face perception. *Nat. Neurosci.* **3:** 80–84.

Humphreys, G. W., Donnelly, N., and Riddoch, M. J. (1993). Expression is computed separately from facial identity, and it is computed separately for moving and static faces: Neuropsychological evidence. *Neuropsychologia* **31:** 173–181.

Iidaka, T., Omori, M., Murata, T., Kosaka, H., and Yonekura, Y. (2001). Neural Interaction of the Amygdala with the Prefrontal and Temporal Cortices in the Processing of Facial Expressions as Revealed by fMRI. *J. Cogn. Neurosci.* **13:** 1035–1047.

Irwin, W., Davidson, R. J., Lowe, M. J., Mock, B. J., Sorenson, J. A., and Turski, P. A. (1996). Human amygdala activation detected with echo-planar functional magnetic resonance imaging. *Neuroreport* **7:** 1765–1769.

Ishai, A., Ungerleider, L. G., Martin, A., Schouten, J. L., and Haxby, J. V. (1999). Distributed representation of objects in the human ventral visual pathway. *Proc. Natl. Acad. Sci. U.S.A.* **96:** 9379–9384.

Ishai, A., Ungerleider, L. G., Martin, A., and Haxby, J. V. (2000). The representation of objects in the human occipital and temporal cortex. *J. Cogn. Neurosci.* **12:** 35–51.

Itier, R. J., and Taylor, M. J. (2002). Inversion and contrast polarity reversal affect both encoding and recognition processes of unfamiliar faces: A repetition study using ERPs. *Neuroimage* **15:** 353–372.

Izard, C. E. (1971). *The Face of Emotion*, Appleton Century Crofts, New York.

Izard, C. E. (1977). *Human Emotions*, Plenum, New York.

Jacobs, D. H., Shuren, J., and Heilman, K. M. (1995). Impaired perception of facial identity and facial affect in Huntington's disease. *Neurology* **45:** 1217–1218.

Kanwisher, N. (2000). Domain specificity in face perception. *Nat. Neurosci.* **3:** 759–763.

Kanwisher, N., McDermott, J., and Chun, M. M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for face perception. *J. Neurosci.* **17**(11): 4302–4311.

Kanwisher, N., Tong, F., and Nakayama, K. (1998). The effect of face inversion of the human fusiform face area. *Cognition* **68:** B1–B11.

Kapur, N., Friston, K. J., Young, A., Frith, C. D., and Frackowiak, R. S. J. (1995). Activation of human hippocampal formation during memory for faces: A PET study. *Cortex* **31:** 99–108.

Katanoda, K., Yoshikawa, K., and Sugishita, M. (2000). Neural substrates for the recognition of newly learned faces: A functional MRI study. *Neuropsychologia* **38:** 1616–1625.

Kesler-West, M. L., Andersen, A. D., Smith, C. D., Avison, M. L., Davis, C. E., Kryscio, R. J., et al. (2001). Neural substrates of facial emotion processing using fMRI. *Cogn. Brain Res.* **11:** 213–226.

Klin, A., Jones, W., Schultz, R., Volkmar, F., and Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Arch. Gen. Psychiatry* **59**(9): 809–816.

Kosaka, H., Omori, M., Murata, T., Iidaka, T., Yamada, H., Okada, T., et al. (2002). Differential amygdala response during facial recognition in patients with schizophrenia: An fMRI study. *Schizophr. Res.* **57:** 87–95.

Kuskowski, M. A., and Pardo, J. V. (1999). The role of the fusiform gyrus in the successful encoding of face stimuli. *Neuroimage* **9:** 599–610.

LeDoux, J. (1996). *The Emotional Brain*, Simon & Schuster, New York.

Lennox, B., Jacob, R., Calder, A., Lupson, V., and Bullmore, E. (2002). Behavioural insensitivity to sad facial expressions is associated with attenuated activation in amygdala and cingulate regions in patients with mania. *Bipolar Disord.* **4:** 89.

Leonard, C. M., Rolls, E. T., Wilson, F. A. W., and Baylis, G. C. (1985). Neurons in the amygdala of the monkey with responses selective for faces. *Behav. Brain Res.* **15:** 159–176.

Leveroni, C. L., Seidenberg, M., Mayer, A. R., Mead, L. A., Binder, J. R., and Rao, S. M. (2000). Neural systems underlying the recognition of familiar and newly learned faces. *J. Neurosci.* **20**(2): 878–886.

Logothetis, N., Pauls, J., Augath, N., Trinath, T., and Oeltermann, A. (2001). Neurophysiological investigation of the basis of the fMRI signal. *Nature* **412:** 150–157.

Mandal, M. K., Pandey, R., and Prasad, A. B. (1998). Facial expressions of emotion and schizophrenia. *Schizophr. Bull.* **24:** 399–412.

Marotta, J. J., Genovese, C. R., and Behrmann, M. (2001). A functional MRI study of face recognition in patients with prosopagnosia. *Neuroreport* **12:** 1581–1587.

McCarthy, G., Puce, A., Gore, J. C., and Allison, T. (1997). Face-specific processing in the human fusiform gyrus. *J. Cogn. Neurosci.* **9:** 605–610.

McCarthy, G., Puce, A., Belger, A., and Allison, T. (1999). Electrophysiological studies of human face perception. II: Response properties of face-specific potentials generated in occipitotemporal cortex. *Cereb. Cortex* **9:** 431–444.

McNeill, D. (1998). *The Face*, Little, Brown, Boston.

Milner, B. (1966). Amnesia following operation on the temporal lobes. In Whitty, C. W. M., and Zangwill, O. L. (eds.), *Amnesia*, Butterworths, London, pp. 109–133.

Morris, J. S., Frith, C. D., Perrett, D. I., Rowland, D., Young, A. W., Calder, A. J., et al. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature* **383:** 812–815.

Morris, J. S., Friston, K. J., Buchel, C., Frith, C. D., Young, A. W., Calder, A. J., et al. (1998). A neuromodulatory role of the human amygdala in processing emotional facial expressions. *Brain* **121:** 47–57.

Morton, J., and Johnson, M. H. (1991). CONSPECT and CONLERN: A two-process theory of infant recognition. *Psychol. Rev.* **98:** 164–181.

Moscovitch, M., Winocur, G., and Behrmann, M. (1997). What is special about face recognition? Nineteen experiments on a person with visual object agnosia and dyslexia but normal face recognition. *J. Cogn. Neurosci.* **9:** 555–604.

Münte, T. F., Brack, M., Grootheer, O., Wieringa, B. N., Matzke, M., and Johannes, S. (1998). Brain potentials reveal the timing of face identity and expression judgments. *Neurosci. Res.* **30:** 25–34.

O'Doherty, J., Winston, J., Critchley, H., Perrett, D., Burt, D. M., and Dolan, R. J. (2003). Beauty in a smile: The role of medial orbitofrontal cortex in facial attractiveness. *Neuropsychologia* **41:** 147–155.

Ojemann, J. G., Ojemann, G. A., and Lettich, E. (1992). Neural activity related to faces and matching in human right nondominant temporal cortex. *Brain* **115:** 1–13.

O'Toole, A. J., Roark, D. A., and Abdi, H. (2002). Recognizing moving faces: A psychological and neural synthesis. *Trends Cogn. Sci.* **6:** 261–266.

Patterson, K. E., and Baddeley, A. D. (1977). When face recognition fails. *J. Exp. Psychol. [Hum. Learn.]* **3:** 406–417.

Penfield, W., and Perot, P. (1963). The brain's record of auditory and visual experience. *Brain* **86:** 595–696.

Perrett, D. I., Hietanen, J. K., Oram, M. W., and Benson, P. J. (1992). Organization and functions of cells responsive to faces in the temporal cortex. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **335:** 23–30.

Phillips, M. L., Young, A. W., Senior, C., Brammer, M., Andrew, C., Calder, A. J., et al. (1997). A specific neutral substrate for perceiving facial expressions of disgust. *Nature* **389:** 495–498.

Phillips, M. L., Williams, L., Senior, C., Bullmore, E. T., Brammer, M., Andrew, C., et al. (1999). A differential neural response to threatening and non-threatening negative facial expressions in paranoid and non-paranoid schizophrenics. *Psychiatry Res.: Neuroimag.* **92:** 11–33.

Posamentier, M. T. (2002). Developmental Prosopagnosia: A Case Study. PhD dissertation, The University of Texas at Dallas.

Price, C. J., Moore, C. J., and Friston, K. J. (1997). Subtractions, conjunctions, and interactions in experimental design of activation studies. *Hum. Brain Mapp.* **5:** 264–272.

Puce, A., Allison, T., Gore, C., and McCarthy, G. (1995). Face-sensitive regions in human extrastriate cortex studied by functional MRI. *J. Neurophysiol.* **74:** 1192–1199.

Puce, A., Allison, T., Asgari, M., Gore, J. C., and McCarthy, G. (1996). Differential sensitivity of human visual cortex to faces, letterstrings, and textures: A functional magnetic resonance imaging study. *J. Neurosci.* **16:** 5205–5215.

Puce, A., Allison, T., and McCarthy, G. (1999). Electrophysiological studies of human face perception. III: Effects of top-down processing on face-specific potentials. *Cereb. Cortex* **9:** 445–458.

Rhodes, G. (1985). Lateralized processes in face recognition. *Br. J. Psychol.* **76:** 249–271.

Rhodes, G. (1993). Configural coding, expertise, and the right hemisphere advantage for face recognition. *Brain Cogn.* **22:** 19–41.

Rhodes, G., Brake, S., and Atkinson, A. P. (1993). What's lost in inverted faces? *Cognition* **47:** 25–57.

Rolls, E. T. (1992). Neurophysiological mechanisms underlaying face processing within and beyond the temporal cortical visual areas. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **335:** 11–21.

Rolls, E. T. (2000). The brain and emotion. *Behav. Brain Sci.* **23:** 177–234.

Rosch, E. (1978). Principles of categorization. In Rosch, R., and Lloyd, B. (eds.), *Cognition and Categorization*, Erlbaum, Hillsdale, NJ, pp. 27–48.

Rosselli, M., Ardila, A., and Beltran, C. (2001). Rehabilitation of Balint's syndrome: A single case report. *Appl. Neuropsychol.* **8:** 242–247.

Rossion, B., Campanella, S., Gomez, C. M., Delinte, A., Debatisse, D., Liard, L., et al. (1999). Task modulation of brain activity related to familiar and unfamiliar face processing: An ERP study. *Clin. Neurophysiol.* **110:** 449–462.

Rossion, B., Gauthier, I., Tarr, M. J., Despland, P., Bruyer, R., Linotte, S., et al. (2000). The N170 occipito-temporal component is delayed and enhanced to inverted faces but not to inverted objects: An electrophysiological account of face-specific processes in the human brain. *Neuroreport* **11:** 69–74.

Rossion, B., Curran, T., and Gauthier, I. (2002). A defense of the subordinate-level expertise account for the N170 component. *Cognition* **85:** 189–196.

Russel, J. A. (1997). Reading emotions from and into faces: Resurrecting a dimensional–contextual perspective. In Russel, J. A., and Fernandez-Dols, J. M. (eds.), *The Psychology of Facial Expression*, Cambridge University Press, New York, pp. 295–320.

Sacks, O. W. (1985). *The Man Who Mistook His Wife for a Hat*, Harper-Collins, New York.

Sansone, S., and Tiberghien, G. (1994). Traitement de l'expression faciale et reconnaissance des visages: Ind'ependence ou interaction? *Psychol. Fr.* **39**(4): 327–344.

Schneider, F., Weiss, U., Kessler, C., Salloum, J. B., Posse, S., Grodd, W., et al. (1998). Differential amygdala activation in schizophrenia during sadness. *Schizophr. Res.* **34:** 133–142.

Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R. K., Anderson, A. W., Volkmar, F., et al. (2000a). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. *Arch. Gen. Psychiatry* **57:** 331–340.

Schultz, R. T., Romanski, L. M., and Tsatsanis, K. D. (2000b). Neurofunctional models of autistic disorder and Asperger syndrome. In Klin, A., Volkmar, F. R., and Sparrow, S. S. (eds.), *Asperger Syndrome*, Guilford Press, New York, pp. 172–209.

Sergent, J., Otha, S., and MacDonald, B. (1992). Functional neuroanatomy of face and object processing. *Brain* **115:** 15–36.

Sheline, Y. I., Barch, D. M., Donnelly, J. M., Ollinger, J. M., Snyder, A. Z., and Mintun, M. A. (2001). Increased amygdala response to masked emotional faces in depressed subjects resolves with antidepressant treatment: An fMRI study. *Biol. Psychiatry* **50:** 651–658.

Sidtis, J. T., Strother, S. C., Anderson, J. R., and Rottenberg, D. A. (1999). Are brain functions really additive? *Neuroimage* **9:** 490–496.

Spiridon, M., and Kanwisher, N. (2002). How distributed is visual category information in human occipito-temporal cortex? An fMRI study. *Neuron* **35:** 1157–1165.

Sprengelmeyer, R., Young, A. W., Calder, A. J., Karnat, A., Lange, H., Hömberg, V., et al. (1996). Loss of disgust: Perception of faces and emotions in Huntington's disease. *Brain* **119:** 1647–1665.

Sprengelmeyer, R., Young, A. W., Pundt, I., Sprengelmeyer, A., Calder, A. J., Berrios, G., et al. (1997). Disgust implicated in obsessive–compulsive disorder. *Proc. R. Soc. Lond. B Biol. Sci.* **264:** 1676–1773.

Sprengelmeyer, R., Rausch, M., Eysel, U. T., and Przuntek, H. (1998). Neural structures associated with recognition of facial expressions of basic emotions. *Proc. R. Soc. Lond. B Biol. Sci.* **265:** 1927–1931.

Streit, M., Wulver, W., Brinkmeyer, J., Ihl, R., and Gaebel, W. (2000). Electrophysiological correlates of emotional and structural face processing in humans. *Neurosci. Lett.* **278:** 13–16.

Talairach, J., and Tournoux, P. (1988). *Co-Planar Stereotaxic Atlas of the Human Brain*, Thieme Medical, New York.

Tarr, M., and Gauthier, I. (2000). FFA: A flexible fusiform area for subordinate-level visual processing automatized by expertise. *Nat. Neurosci.* **3:** 764–769.

Taylor, S. F., Liberzon, I., Fig, L. M., Decker, L. R., Minoshima, S., and Koeppe, R. A. (1998). The effect of emotional content on visual

recognition memory: A PET activation study. *Neuroimage* **8:** 188–197.
Taylor, S. F., Liberzon, I., and Koeppe, R. A. (2000). The effect of graded aversive stimili on limbic and visual activation. *Neuropsychologia* **38:** 1415–1425.
Tempini, M. L. G., Price, C. J., Josephs, O., Vandenberghe, R., Cappa, S. F., Kapur, N., et al. (1998). The neural systems sustaining face and proper-name processing. *Brain* **121:** 2103–2118.
Teunisse, J.-P., and de Gelder, B. (2001). Impaired categorical perception of facial expressions in high-functioning adolescents with autism. *Child Neuropsychol.* **7:** 1–14.
Thomas, K. M., Drevets, W. C., Whalen, R. J., Eccard, C. H., Dahl, R. E., Ryan, N. D., et al. (2001). Amygdala response to facial expressions in children and adults. *Biol. Psychiatry* **49:** 309–316.
Tong, F., Nakayama, K., Moscovitch, M., Weinrib, O., and Kanwisher, N. (2000). Response properties of the human fusiform face area. *Cogn. Neuropsychol.* **17:** 257–279.
Tranel, D., Damasio, A. R., and Damasio, H. (1988). Intact recognition of facial expression, gender, and age in patients with impaired recognition of face identity. *Neurology* **38:** 690–696.
Tulving, E., Kapur, S., Craik, F. I., Moscovitch, M., and Houle, S. (1994). Hemispheric encoding/retrieval asymmetry in episodic memory: Positron emission tomography findings. *Proc. Natl. Acad. Sci. U.S.A.* **96:** 2016–2020.
Valentine, T. (1988). Upside-down faces: A review of the effect of inversion upon face-recognition. *Br. J. Psychol.* **79:** 471–491.
Wacholtz, E. (1996). Can we learn from the clinically significant face processing deficits, prosopagnosia and capgras delusion? *Neuropsychol. Rev.* **6:** 203–258.
Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., and Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *J. Neurosci.* **18:** 411–418.
Wiser, A. K., Andreasen, N. C., O'Leary, D. S., Crespo-Facorro, B., Boles-Ponto, L. L., Watkins, G. L., et al. (2000). Novel vs. well-learned memory for faces: A positron emission tomography study. *J. Cogn. Neurosci.* **12:** 255–266.
Wojciulik, E., Kanwisher, N., and Driver, J. (1998). Covert visual attention modulates face-specific activity in the human fusiform gyrus: fMRI study. *J. Neurophysiol.* **79:** 1574–1578.
Wright, C. I., Fischer, H., Whalen, P. J., McInerney, S. C., Shin, L. M., and Rauch, S. L. (2001). Differential prefrontal cortex and amygdala habituation to repeatedly presented emotional stimuli. *Neuroreport* **12:** 379–383.
Yang, T. T., Menon, V., Eliez, S., Blasey, C., White, C. D., Reaid, A. J., et al. (2002). Amygdalar activation associated with positive and negative facial expressions. *Neuroreport* **13:** 1737–1741.
Yaxley, S., Rolls, E. T., and Sienkiewicz, Z. J. (1988). The responsiveness of neurons in the insular gustatory cortex of the macaque money is independent of hunger. *Physiol. Behav.* **43:** 223–229.
Yin, R. K. (1969). Looking at upside-down faces. *J. Exp. Psychol.* **81:** 141–145.
Yin, R. K. (1970). Face recognition by brain-injured patients: A dissociable ability? *Neuropsychologia* **8:** 395–402.
Young, A. W., McWeeny, K. H., Hay, D. C., and Ellis, A. W. (1986). Matching familiar and unfamiliar faces on identity and expression. *Psychol. Res.* **48:** 63–68.
Young, A. W., Aggleton, J. P., Hellawell, D. J., Johnson, M., Broks, P., and Hanley, J. R. (1995). Face processing impairments after amygdalotomy. *Brain* **118:** 15–24.
Young, A. W., Hellawell, D. J., van de Wal, C., and Johnson, M. (1996). Facial expression processing after amygdalotomy. *Neuropsychologia* **34:** 31–39.