Prosopagnosia☆

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Glossary

Affective prosopagnosia An impairment in the appreciation of facial expressions.
Agnosia A recognition disorder that cannot be explained by sensory–perceptual, language, or amnestic deficits or general confusion. Agnosias are often modality-specific, ie, pertaining to one sensory modality (eg, visual agnosia).
Anomia A naming impairment.
Apperceptive agnosia A recognition disorder that is caused by a higher order perceptual impairment.
Associative agnosia A recognition disorder that is caused by a problem in accessing stored information about objects and faces.
Covert face recognition The experimental demonstration of preserved face recognition in prosopagnosia despite a complete absence of acknowledged awareness.
ERP Event-related potential.
fMRI Functional magnetic resonance imaging.
GSR Galvanic skin response.
Metamorphopsia The clinical condition in which patients perceive faces in a distorted manner.
Object agnosia A recognition disorder for common objects. In pure cases, other stimulus categories such as text or faces may remain unaffected.
Person identification deficit The clinical condition in which a patient is able to distinguish between familiar and unfamiliar people but cannot access biographical information.
PET Positron emission tomography.
Priming An experimental procedure in which the processing of a particular stimulus is influenced by a previous presentation of that stimulus (repetition priming) or a related stimulus (semantic priming).
Speech reading The perception of the movements of the lips and the tongue to aid the understanding of spoken speech.
Within-category discrimination The ability to identify specific exemplars of stimulus category with many visually similar items (eg, motor cars).

Background

The term prosopagnosia refers to the inability to recognize familiar people via visual inspection of the face. It is a modality-specific impairment, and other recognition routes, such as via the voice, remain spared. In addition, reading and object recognition may remain unaffected in selective cases. The inability cannot be explained by perceptual, language or amnestic deficits, or by general confusion. Anecdotal observations of face recognition deficits date back to the ancient Greek literature. For instance, the Greek general Thucydides described intriguing behavior, such as the inability to recognize friends, in soldiers who recovered from the plague during the Trojan war. Clinical case studies published in the second half of the 19th century sometimes mentioned problems in this domain in patients who had suffered neurological disease. But it was not until the early 20th century that clinicians suggested that face recognition might be a separate function that can be selectively impaired after brain injury.

In 1947, the German neurologist Joachim Bodamer published a report on a number of patients who experienced a particular selective difficulty in recognizing faces. Bodamer named the condition prosopagnosia with reference to the Greek prosopon (face)

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and agnosis(without knowledge). His patients complained bitterly about the problem. In severe cases, even the faces of family members and close friends were affected and sometimes the patient's own face when seen in a mirror. When Bodamer asked his patient to inspect his own face in the mirror, it was clear that, although he knew he was looking at his own face, there was no sense of familiarity. This problem does not appear to be directly caused by an impairment in the perception of facial features. All of his prosopagnosic patients knew when they were looking at a face, and they could identify and describe separate features such as the eyes and mouth. Thus, although prosopagnosic patients know that they are looking at a human face, it has lost its value as a cue for the identification of a person, and not even a vague feeling of familiarity is evoked. Prosopagnosic patients often describe faces as all looking similar, unattractive, and having lost their individuality. Another common comment from these patients is that faces are not perceived as an integrated whole and appear fragmented.

In order to suggest that prosopagnosia constitutes a separate clinical entity, it is important to look in more detail at the claim that these deficits in the recognition of familiar faces dissociate from visual object agnosia in general. There are many case descriptions suggesting that the patient is unable to recognize any familiar faces, whereas his or her ability to recognize common objects is spared. However, it should be noted that the task demands in face and object recognition are not comparable. Instead of recognizing a hammer as a tool, a more comparable situation would be to recognize one's neighbor's hammer among many other exemplars.

The ability to recognize faces is probably the most difficult visual recognition task that we are able to perform, despite the subjective ease with which we do it. In a lifetime, we learn thousands of faces, most of which we are able to recognize relatively unaffected by changes due to age and variable additions such as spectacles and facial hair. These faces, however, only differ very slightly in visual appearance. This is a point well-illustrated by the general experience that faces from another race than one's own are difficult to remember. When prosopagnosic patients are tested on within-class recognition tasks, such as flower or car recognition, that approach the task demands involved in face recognition, they often show impairments. However, there is evidence that within-class discrimination problems do not always co-occur in prosopagnosia.

An extensive investigation of prosopagnosia by the Italian neurologist Ennio de Renzi is illustrative here. He described a prosopagnosic patient who was able to perform recognition tasks that are visually very demanding. For instance, this patient was able to recognize his own car in the car park, his own wallet from an array of similar wallets, and his own handwriting amid that of others. In addition, he was very apt at distinguishing Italian coins from foreign ones. Until very recently, the controversy about a dedicated cortical system for face recognition continued because the other half of the necessary double dissociation was still lacking, despite a number of clinical reports that described a statistical trend toward relatively more severe problems in recognizing objects than faces. Convincing evidence for this position was provided by a number of reports on a patient who performed normally on tests of familiar face recognition while remaining severely impaired in the recognition of common objects.

Other indirect evidence comes from the clinical phenomenon of metamorphopsia, where the patient sees faces, and only faces, in a distorted manner. For example, one patient reported that his visual world appeared to be completely unaffected by his brain damage apart from the fact that all faces looked like "fish heads." Despite this distortion, he was able to recognize most familiar faces without any problems. The fact that this phenomenon is restricted to faces supports the notion of a separate cognitive system that is dedicated to the perception of faces.

Prosopagnosia and Sensory Status

Visual recognition disorders as a result of brain disease have created theoretical controversies since they were first described in the latter half of the 19th century. Regarding impairments in object recognition, Heinrich Lissauer suggested a distinction between apperceptive and associative Seelenblindheit, where the former results from visuosensory deficits, whereas the latter was thought to represent a difficulty in associating meaning with a relatively intact percept. A few years later, Sigmund Freud coined the term agnosia to describe these recognition disturbances. It is the viability of the concept of associative agnosia, or as Hans-Lukas Teuber put it, "a normal percept stripped of its meaning" that has been questioned. Notably, Eberhard Bay claimed that the so-called higher order recognition deficits are secondary to sensory impairment, general intellectual loss, language problems, or a combination of these factors. More recent investigations have convincingly demonstrated that visual recognition disorders can occur in patients with normal, or even above average intelligence. Also, it has now been clearly established that language difficulties are not instrumental in causing associative agnosias. However, the question of whether subtle sensory impairment or a certain constellation of sensory deficits can produce the clinical symptoms of associative agnosia remains controversial.

The debate is fuelled by the fact that even the most "pure" cases of agnosia often show some mild problems on tasks of visual perception. A seminal study by George Ettlinger in 1956 set out to test Bay's hypothesis. He carried out a careful assessment of sensory status in patients with and patients without recognition deficits, and he argued that sensory status alone could not explain the presence or absence of a recognition disorder. The crux of his argument is that, although patients with a recognition deficit may have sensory impairments, other patients who do not experience recognition problems can show equal or worse impairments on the sensory tests.

George Ettlinger was subsequently criticized for using a composite sensory score and for using tests that might not have covered all of the relevant sensory abilities for visual recognition. It has now been shown that there are highly selective disorders of color, luminance, shape, texture and glossiness that might interfere with visual recognition. In a study with three densely prosopagnosic patients, the evaluation of primary visual proficiency was done with a screening battery comprising all of the relevant visual cues, and the patients did show subtle impairments on several of the screening tasks. These problems were, however, insufficient to

explain the pronounced recognition problems, because other patients with unilateral brain lesions who do not have recognition deficits are at least as impaired on these sensory tasks as the agnosics.

The conclusion is, therefore, that the visuosensory impairments cannot explain the agnosic problem. These results support the notion of an associative type of prosopagnosia, which can be distinguished from a perceptive type. This is in line with more recent studies in which it is concluded that there are both perceptual and amnestic forms of prosopagnosia. Thus, prosopagnosia is modality-(visual) and stimulus-specific (faces) impairment that may arise in the context of preserved visuoperceptual processing.

Prosopagnosia and Other Face Processing Deficits

Faces form the source for a multitude of inferences. From a perceived face, we are able to determine gender and age and make more subjective judgments regarding attractiveness, etc. We can assess the emotional state of the person (eg, happy, sad) by analyzing the facial expression, and by observing the movements of the lips and tongue, we gain additional information regarding the verbal message that the speaker is trying to convey. Finally, the face constitutes the principal cue for visual identification of people we know.

Evidence for a dissociation between the recognition of familiar faces and the processing of facial expressions can be traced back to neuropsychological studies in the 1960s. It was observed that some patients with severe impairments of facial identity recognition showed relatively intact ability to recognize facial expressions. Conversely, a group study with patients suffering from a degenerative illness showed that they were impaired at both recognizing facial expressions and recognizing American Presidents. However, the identity and expression recognition impairments did not correlate with each other. The conclusion is that some of these patients must have had intact familiar face recognition but poor facial expression perception, and vice versa.

There is also evidence for distinct processing pathways involved in the recognition of facial expressions and the matching of unfamiliar faces. For example, in a 1985 study by Dawn Bowers and colleagues, patients who had left and right hemisphere damage or no neurological disease were assessed using a series of facial affect tests and tasks for unfamiliar face perception. The patients with right hemisphere lesions were significantly impaired on both types of tasks, but the impairments on the affect tests remained significant even when the face perception deficit was statistically partialed out. Several reports subsequently supported the notion of dissociable impairments of facial expression recognition and unfamiliar face matching after brain injury. A selective deficit in the analysis of facial expressions is sometimes referred to as affective prosopagnosia. Regarding the neuroanatomical substrate of expression analysis, functional imaging studies suggest that the right posterior (superior temporal) hemisphere is instrumental in the perceptual processing of facial information, but the amygdala (bilaterally) plays a crucial role in the experience of emotions, especially for fear and anger.

In 1986, Ruth Campbell and colleagues described two Swiss patients. One patient was able to recognize faces but could no longer read lips, and the other showed the opposite pattern. Lip reading is tested using a recognition task in which the patient is shown photographs of the face of an actor making a speech sound, for instance "ee" or "aa". The other method uses what is known as the McGurk illusion. This involves the videotape recording of a face making a speech sound (eg, da) and the simultaneous sound recording of a different speech sound (eg, ta). A normal observer confronted with such a stimulus would "hear" a blend of these two speech sounds (eg, ga). Subjects who can no longer read lips will report the auditory stimulus and not the blend. Apart from this single paper there is little neuropsychological evidence for this dissociation, although there is some convergent evidence from cognitive psychology.

The literature also supports the notion of distinct pathways for the processing of familiar and unfamiliar faces. Prosopagnosia patients may perform normally on tasks of matching unfamiliar faces. Conversely, patients with unfamiliar face matching impairments often are not clinically prosopagnosic. One of the first clear examples of a double dissociation came from a study by Daniel Malone. Case one initially was unable to recognize familiar faces, and he was also impaired on the matching of unfamiliar face photographs. Later testing revealed that the familiar face recognition impairment had improved significantly, whereas his unfamiliar face matching problem remained. Case two was still unable to recognize both relatives and various familiar personalities by their faces alone 6 weeks after initial testing. There was a significant improvement, however, in his initially impaired unfamiliar face matching performance, which now fell within the normal range. The idea of independence of familiar face recognition from the perception of unfamiliar faces in terms of gender decision is supported by studies with normal subjects.

In short, prosopagnosia is a selective deficit in the recognition of familiar faces that may arise in the context of preserved recognition of other types of information that can be read from faces, such as expressions and speech gestures.

Toward a Taxonomy of Prosopagnosia

It is now becoming clear that there are different forms of prosopagnosia that can be distinguished in terms of the underlying functional deficit. The distinction between apperceptive and associative prosopagnosia has already been introduced. Another important form of face recognition deficit could be termed "person identification deficit." The patient M.E. was a right-handed female who, after a vasculitic disorder, suffered from a selective memory deficit. She performed normal to above normal on tasks for the

perception of faces (eg, Benton face test) and on tasks that required her to choose the familiar face from an array with unfamiliar faces. However, she was very poor at retrieving personal information about the people whose faces she had correctly recognized as familiar. Often she would claim that a face looked familiar but that was all she could say about it. These findings provide a strong indication that her face recognition system is unimpaired to the level of familiarity recognition. The dissociation between face familiarity and access to biographical information was confirmed on formal testing.

A related impairment concerns a specific anomia. In 1989, Brenda Flude and colleagues studied a patient with an impairment at the last stage of face recognition, that is, retrieval of the name. Their patient, E.S.T., showed a preserved ability to (a) match unfamiliar faces on identity, (b) distinguish between familiar and unfamiliar faces and names, and (c) access semantic information from faces and written names of familiar people. However, E.S.T. was severely impaired in naming familiar faces. The finding of this selective functional deficit supports the idea that the mechanism dealing with name retrieval (which was severely impaired for E.S.T.) is separable from mechanisms responsible for the sense of familiarity and access to appropriate semantic information (which were both well-preserved for EST). Flude's investigation of E.S.T., then, demonstrates that name retrieval can be impaired when access to identity-specific semantic information is preserved.

It is concluded that there are several forms of prosopagnosia that can be distinguished on the basis of the functional locus of the deficit. First, higher order perceptual impairments may lead to face recognition problems. In line with Lissauer's suggestion some 100 years ago, there is also an amnestic or associative form. Subsequently, patients may be impaired in accessing biographical information about familiar people, and finally there are patients who are unable to retrieve the names of familiar people. Finally, it is useful to point out that prosopagnosia entails, in fact, two impairments: the inability to recognize familiar faces and the inability to learn new faces or relearn old ones. These problems have subsequently been described in isolation. Transient forms can be interpreted as retrograde prosopagnosia, whereas anterograde prosopagnosia refers to those who are no longer able to learn new faces but can recognize faces learned before the onset of the condition.

The Anatomy of Face Perception

Face processing disorders have been described in a wide range of patient populations. Patients suffering from diffuse brain damage such as dementia or closed head injury have been shown to suffer from severe deficits in face processing. Progressive visual recognition problems, notably for faces, are the prevailing clinical symptom in patients with a degenerative illness that specifically affects the posterior areas of the cortex. Several studies have shown that autistic patients demonstrate problems in the perception and recognition of faces and facial expressions. A number of patients with visual recognition deficits, including prosopagnosia, have been described after carbon monoxide poisoning. Face processing disorders have also been described in patients with neurological illnesses resulting in more localized damage. For instance, temporal and frontal lobectomy may impair the perception of faces and facial expressions. In addition, the famous prosopagnosic case H.J.A., who has been studied extensively by Glyn Humphreys and Jane Riddoch, had suffered a large posterior stroke.

Prosopagnosia is, in most cases, the result of bilateral posterior damage to the cortex. The left and right occipitotemporal junctions have traditionally been identified as the substrate for face recognition. Most patients with prosopagnosia (and all patients who have come to post mortem investigation) had lesions in those areas. In humans, the fusiform and lingual gyri are most often implicated. These observations are somewhat at odds with the primate single-cell recording studies that localize the cells sensitive to face identity much more anteriorly in the upper bank of the superior temporal sulcus. These differences could be explained as differences between species, and it might be prudent to keep this in mind when comparing the human and the primate data on face perception. In this context it is interesting to note that one study found face-specific cells in the prefrontal area of the macaque monkey.

A number of reports have claimed that a unilateral right hemisphere lesion might be sufficient to cause prosopagnosia, It is known from clinical practice that transient or partial prosopagnosia can occur after a unilateral lesion but that full-blown prosopagnosia appears to require bilateral damage.

The upsurge in functional neuroimaging studies has largely served to confirm the clinical evidence from neuropsychology. MEG has supported the notion of bilateral processing as well as the importance of the right inferior temporal lobe for familiar face recognition. Work using PET and fMRI scanning have identified a number of areas involved in the perception of familiar faces in the posterior, lateral-occipital and occipital temporal areas of the brain, and again the fusiform gyrus, especially on the right side, appears to play a major role.

In 1999, using fMRI, Nancy Kanwisher and colleagues provided further evidence for a specific face processing system, demonstrating that the face area is not especially activated when the subjects are presented with pictures of animals. Perhaps the most interesting result to come out of neuroimaging until now is that the area involved in face processing is actually adjacent to the area that is involved in object recognition. This raises the possibility that this area processes visual primitives that happen to be important for face recognition. If these observations are replicated and extended, they might well form the basis for a more tractable definition of what is specific about the face area.

The general consensus is that prosopagnosia is caused by bilateral damage to the ventral fusiform gyrus. There are a number of controversial issues, such as the possibility that a unilateral lesion may be sufficient, and whether there is a specific face area or whether it is just a part of a general visual object processing area. However, the main challenge for future research must be to use the functional subdivisions of prosopagnosia in order to arrive at a more fine-grained localization.

Prosopagnosia and Knowledge Without Awareness

In 1994, Russell Bauer published his seminal paper that fuelled the research on face recognition and awareness. He investigated a patient who had become prosopagnosic after a severe closed head injury. First, he asked his patient to select the correct name from five alternatives to match a photograph of a familiar face. All five alternative names were of celebrities and, therefore, familiar to the patient (who had no problem recognizing names). As expected of a prosopagnosic patient, L.F. performed at chance level. However, skin conductance responses recorded during the experiment occurred significantly more often and with higher amplitude to the correct name than to the other four foils. Daniel Tranel and Antonio Damasio have subsequently replicated these surprising findings. They recorded significantly increased autonomic responses when their prosopagnosic patients looked at slides of familiar faces embedded among those of unknown people.

These observations have been corroborated using other psychophysiological techniques. Differential processing of familiar and unfamiliar faces in prosopagnosia has been demonstrated with ERP measures using an odd-ball paradigm in which the patients looks at a long series of faces. Most of the photographs are of completely unfamiliar people, but once in a while the face of a very famous person is embedded in the series. Each time a famous face appears there is a clear increase in the amplitude of the P300 response.

Finally, the recording of eye movements has suggested spared processing of familiar faces in prosopagnosic patients. It has been demonstrated that the way in which normal people look at faces depends on whether they know the person. Unfamiliar faces are scanned in a rather global manner with about an equal amount of attention given to all aspects of the face. However, with familiar faces the emphasis is clearly on the internal part of the face (eyes–nose–mouth region). Although the prosopagnosic patient is unaware of the fact that he is looking is a familiar face, his scanning behavior is as if he is looking at a familiar face.

These demonstrations of differential processing of familiar and unfamiliar faces despite the inability to recognize the familiar faces overtly, thus, appear robust. These data were first interpreted as indicating two separable recognition systems, one for overt and another for covert recognition. The overt system would result in a conscious experience of recognition, whereas the covert system would feed into the limbic system and serve an alerting function. However, there are clear indications that covert recognition might not be restricted to autonomic or physiological measures. These suggestions have been extensively followed up by our research group in a number of studies with the patient P.H., who is completely unable to recognize familiar faces overtly. The extent of P.H.'s recognition problems is well-illustrated by his performance on a forced-choice task where he had to choose the familiar face from an array of two faces. This test procedure is sensitive to small degrees of residual processing as it is not influenced by possible language or memory dysfunction or subject to the common response bias of agnosic patients who respond ''I don't know'' in standard line-up confrontation tasks. P.H. performed this task at chance level (correct on 51% of the trials), indicating that he has no access to familiarity information from faces.

Next, in seeking to demonstrate covert knowledge, we used experiments that have been shown to be sensitive to knowledge of face familiarity in healthy individuals but that do not require overt identification of the famous faces used as experimental stimuli. Such experiments used the procedures of matching, interference, associative priming, and paired-associate learning. For instance, in the matching experiment, subjects are required to decide whether two simultaneously presented photographs of faces are taken from the same person or two different people. Normal subjects are faster to match photographs of familiar than unfamiliar people, and P.H. showed exactly the same effect, yet he was unable to identify any of the faces used. Covert processing can also be demonstrated in associative priming experiments. Such experiments show the influence of previously presented stimuli on a subsequent response. Subjects have to decide whether the targets (ie, written names of familiar and unfamiliar people) are familiar. The responses are influenced by the presentation of a priming stimulus (eg, a face) shortly before the target appears. If there is a strong association between the prime stimulus (eg, a photograph of Prince Charles) and the target (eg, the name of Princess Diana), the subject responds faster than when the prime is either the face of an unfamiliar person or that of a familiar person who is not closely associated with the target (ie, the face of Prince Charles followed by the name of George Bush). The patient, P.H., shows this type of priming effect with faces that he does not overtly recognize. Moreover, we were able to compare the extent of the priming effect with that triggered by name primes (which P.H. recognizes without difficulty). The effects are equivalent: overt recognition of name primes makes no additional contribution to the associative priming effect. This suggests that P.H. not only covertly recognizes faces but that his recognition is normal in the amount of associative priming that it produces.

It should be noted that covert recognition effects are not invariably found in agnosias. Some prosopagnosic patients do not show any covert effects at all, and others have exhibited partial covert recognition effects, such as covert recognition effects for only a subset of the people known to the patient.

The observation of covert face recognition, in its different forms, has extended the taxonomy of prosopagnosia. Apart from the difference between apperceptive and associative prosopagnosia, we can now distinguish between prosopagnosia with and without covert recognition. This difference is possibly related to the issue of whether the stored representations of familiar faces are still spared in a prosopagnosic patient. Several authors have commented on the use of mental imagery tests to evaluate the integrity of the stored representations. A number of patients have been described who are able to ''picture faces in their mind'' that they cannot recognize overtly. Obviously testing of such a subjective ability as mental imagery is not easy, but a number of useful procedures have been developed. The method of odd-one-out, in which the patient has to decide which one of three people looks most unlike the other two (eg, the patient is given the names: Charlie Chaplin, Adolf Hitler, and Bill Clinton) has been used successfully to demonstrate clinical dissociations between impaired recognition and spared mental imagery of familiar faces.

Developmental Prosopagnosia

The ability to recognize familiar faces can, thus, be characterized as modular in terms of the ideas put forward by Jerry Fodor. It is a highly specialized function with a well-circumscribed neurological substrate that is functional soon after birth. Faculties are modular as a consequence of their importance to the individual and, therefore, are designed to mature and develop in an efficient and relatively protected way. This modular nature of face recognition suggests that the variability in capacity in the general population can be attributed largely to biological factors. A genetic basis for language processing is now generally accepted. Developmental face recognition problems have been described in a number of case studies. By definition, the problem is present from early childhood. In addition, there should be no known neurological history that could explain the recognition problem. The first case of developmental prosopagnosia was described by McConachie in 1976 and has been followed up extensively. This is an intelligent and verbal woman, who experienced very severe difficulties in recognizing familiar faces. Reading and object recognition were preserved, but she encountered problems on most tasks that depend on the use of facial information, such as the visual analysis of expressions and the short-term memory of unfamiliar faces. Her problem is best described as an inability to form an adequate internal representation of faces. As a result, faces have never gained the significance for her that they have for most of us. Instead, she relies heavily on voices for the recognition of familiar people.

An interesting observation, mentioned in some case studies on developmental face recognition deficit, concerns the anecdotal reference to other family members who are supposedly also poor at recognizing faces. An early study identified a family of two parents, three daughters, and one son. Two of the daughters and the father were very poor at recognizing familiar faces. The other family members did not have any problems. Thus, the face recognition problems occurred not only in two family members of the same generation but also in family members of different generations. These observations, that have now been confirmed in large-scale studies, present clear evidence for a familial factor in the development of face recognition problems.

Conclusions

In clinical terms, it has become clear that, although pure prosopagnosia is rare, face recognition impairments are much more common than has been previously suggested. The reasons that these problems often remain undiagnosed are due to the fact that patients do not spontaneously complain either as a result of lack of insight or because the difficulties are ascribed to a general memory deficit and/or because the assessment of face recognition deficits is often not included in standardized neuropsychological screening. In most cases, face recognition deficits occur in the context of more widespread perceptual problems. The effects on daily living are often underestimated. Prosopagnosia severely reduces the professional and social possibilities of the patient and in many cases has led to social isolation. Finally, there have been several attempts at developing rehabilitation programs, but until now without much success. Memory training has been successfully applied in patients who can recognize faces but have problems with retrieving the name. However, even patients with intact covert recognition do not appear to benefit from cognitive training.

In terms of understanding the underlying mechanisms of face recognition, the study of prosopagnosic patients has produced a number of intriguing and often counterintuitive results during the past two decades. The fractionation of face and object recognition has remained controversial. On the other hand, we have now seen a general acceptance of a specialized face processing system that itself fractionates further into a system responsible for familiar face recognition, expression analysis, face perception skills, such as gender decision and age estimation, and perhaps lip reading. Within the processing route involved in familiar face recognition, separate sequential processing stages have been identified for the processing of the incoming visual image and access to stored representations of faces, autobiographical information, and names. These processing stages are largely automatic and escape conscious introspection as demonstrated by the phenomenon of covert face recognition where these automatic processes continue to operate despite the fact that the output never reaches conscious awareness. Functional imaging techniques have produced new data suggesting that the apparent selectivity of face processing can actually be traced back to the functional organization of the fusiform gyrus, where there may be a continuous representation of form information that has a highly consistent and orderly topographical arrangement. The face area constitutes the part that entails the form detectors that are particularly important for defining faces. The challenge for future research is to refine the functional architecture of face recognition in order to understand the neuroanatomical basis of the different forms of prosopagnosia that can be distinguished.

Further Reading

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