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REVIEW

Prosopagnosia: current perspectives

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Abstract: Prosopagnosia is a selective visual agnosia characterized by the inability to recognize the identity of faces. There are both acquired forms secondary to brain damage and developmental forms without obvious structural lesions. In this review, we first discuss the diagnosis of acquired and developmental prosopagnosia, and the challenges present in the latter case. Second, we discuss the evidence regarding the selectivity of the prosopagnosic defect, particularly in relation to the recognition of other objects, written words (another visual object category requiring high expertise), and voices. Third, we summarize recent findings about the structural and functional basis of prosopagnosia from studies using magnetic resonance imaging, functional magnetic resonance imaging, and event-related potentials. Finally, we discuss recent attempts at rehabilitation of face recognition in prosopagnosia.

Keywords: face recognition, perception, fusiform gyrus, anterior temporal, review

Introduction

Face recognition is usually effortless and rapid. In different places and times, despite changes in expression, hairstyle, and clothing, we easily recognize colleagues, friends, and family. Our visual expertise with faces likely exceeds that for any other type of object, and this ability to identify people is a cornerstone of our social interactions as human beings. Subjects with prosopagnosia, however, cannot recognize that they have seen a face before, an impairment that affects both faces well known to them and those recently encountered. This is not due to more general problems with vision, object recognition, or memory. The term "impaired face recognition" should be used rather than "prosopagnosia" when this symptom is part of a broader problem, as with macular degeneration, general memory problems in Alzheimer's disease, and cognitive issues in schizophrenia, for example. These subjects realize that a face is a face and not a car or a tree, but simply cannot say whether they have seen it before or whose face it is. These subjects rely on other cues to identity, such as hairstyle, gait, or voice, and make mistakes if these cues change (eg, hairstyle). They relate surprising and sometimes embarrassing stories, such as not recognizing themselves in a mirror, or walking past siblings or spouses as if they were strangers.

Prosopagnosia can be either acquired or developmental. In acquired prosopagnosia, poor face recognition is the result of brain injury. While the first case of acquired prosopagnosia was reported 150 years ago,[1,2] the modern study of this condition began with Bodamer's[3] report in 1947, which described impaired face recognition in wounded soldiers. Subsequently, it has been recognized that acquired prosopagnosia can arise

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from many different pathologies, including trauma, stroke, encephalitis, tumors, degenerative atrophy, or temporal lobe resections.[4]

Developmental prosopagnosia has been more recently described and is less well understood. Subjects with this condition fail to develop face recognition skills despite otherwise normal vision and memory, and do not have obvious lesions on brain imaging.[5,6] Developmental prosopagnosia may have a genetic basis. It can run in families, with some pedigrees showing as many as ten affected members across two generations,[7–9] observations that parallel findings that normal face recognition skills also have a heritable component, with monozygotic twins having more similar face recognition abilities than dizygotic twins.[10,11] While the acquired form is rare, the developmental form may be relatively common. Some suggest that as many as 2.5% of the population has developmental prosopagnosia,[12,13] although this number will vary with the statistical criteria used and may confound those subjects with a developmental problem with those on the low end of normal face recognition ability (see Barton and Corrow[14] for a discussion on the prevalence and diagnosis of developmental prosopagnosia).

Prosopagnosia has significant implications for those who have it. Adults with developmental prosopagnosia often report that their failure to recognize others creates traumatic social experiences, leading to chronic anxiety, feelings of embarrassment and guilt, and a limited social circle.[15] Our subjects with acquired prosopagnosia acknowledge similar difficulties. Children with developmental prosopagnosia and their parents describe the same problems, but with additional implications for the school environment and safety.[16]

Models of face recognition

Face recognition is a multistage process ending with the identification of a person. These stages are reflected in cognitive models of face recognition, the most influential being that of Bruce and Young[17] (Figure 1). Each box in the model represents a distinct cognitive process: while it is not necessary that these different stages occur in separate anatomic structures, some neuroanatomic models suggest that this may be the case.[4,18] The model begins with creating a "facial percept", the encoding of the structural information about the face. This percept is matched to stores of face memories, termed "face recognition units", to determine whether the face has been seen before. Some argue that a correct match at this stage produces a feeling of familiarity with the face.[19–21] A correct match also activates a "person identity node", which allows access to semantic information and the name of the person to whom the face belongs. This model continues to be useful and has been elaborated to incorporate parallel sources of information from other cues (eg, voice),[22,23] hemispheric lateralization of these cues,[20] and more extensive bidirectional influences between modules.[19,22–25]

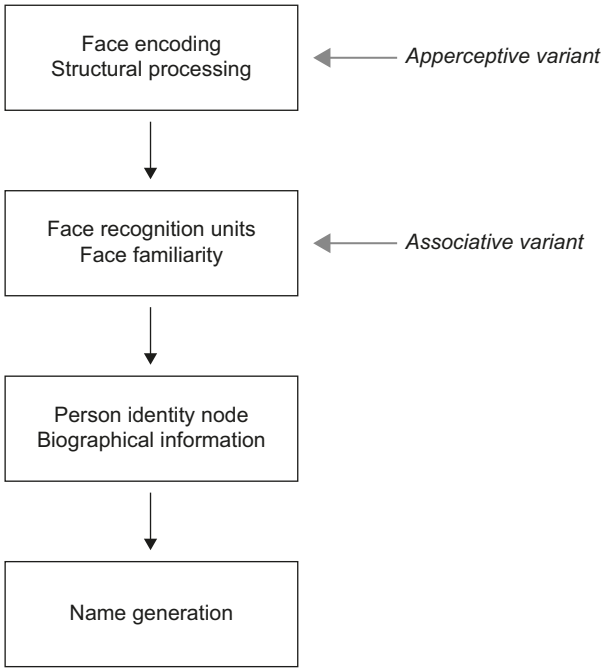

Figure 1 Adaption of the Bruce and Young model.
Notes: In the associative variant of prosopagnosia, face encoding is thought to be intact, represented by the ability to accurately discriminate between faces. However, faces are not seen as familiar suggesting a failure to activate face recognition units, subsequently affecting later stages in the face-processing stream. In the apperceptive variant, face encoding is thought to be impaired, affecting all later stages in the model when faces are the cue to identity. Gray arrows indicate the first stage of the model showing the greatest deficit in apperceptive and associative variants of prosopagnosia, respectively. ©1986 The British Psychological Society. Adapted from Bruce V, Young A. Understanding face recognition. *Br J Psychol.* 1986;77(Pt 3):305–327 with permission John Wiley and Sons.[17]

These models are reflected in our concepts about prosopagnosia. There are functional variants that may correspond to dysfunction of different cognitive stages.[26] Impairments in the ability to see differences between faces, or their structures, suggest an "apperceptive variant", a failure in encoding the facial percept. Other prosopagnosic subjects can perceive facial structure accurately but on tests of facial imagery cannot recall the faces of familiar people, indicating an "associative or amnestic variant". However, this is a relative rather than absolute dichotomy: subtle defects in face perception can be seen in patients with an associative variant,[27–30] while those with an apperceptive variant have milder deficits on face imagery tests.[4] Nonetheless, this distinction remains useful, and these variants have distinct neural correlates (see "Neuroimaging" section).[4]

Diagnosis

Tests of face familiarity

The hallmark of prosopagnosia is the reduced ability of subjects to realize that they have seen a face before: hence, key diagnostic tests probe the sense of familiarity for previously seen faces. Earlier tests of face recognition may have been less sensitive because their stimuli could allow subjects to use alternative strategies to circumvent poor face recognition, such as remembering hairstyles and clothing.[31,32] Newer tests have addressed those limitations by minimizing those extraneous cues. The most commonly used test of familiarity for recently viewed faces is the Cambridge Face Memory Test (CFMT),[33] a test with high internal reliability.[34] While the original version of this test used only adult Caucasian faces, other versions have been created, such as the CFMT-Chinese,[35] CFMT-Australian,[36] and pediatric versions, such as the CFMT-C[37] and the CFMT-Kids.[38]

Tests that use anonymous faces like the CFMT have the advantage that, as none of the faces are familiar to subjects prior to learning, all subjects taking the test have the same degree of short-term familiarity with the faces seen during the test. Tests of familiarity for famous faces are also used, but such tests depend on the person having seen those celebrities before, and are therefore affected by age, education, and cultural background. In prosopagnosic subjects, this can be compounded by the fact that these subjects may lose interest in films and television because they cannot keep track of the characters, thus limiting their exposure to newer celebrities.

Tests of face perception

Tests of face perception – that is, the ability to perceive differences between faces – do not establish the diagnosis of prosopagnosia. What they can do is demonstrate if prosopagnosia is due to impaired encoding of the facial structure, and therefore is an apperceptive variant, or if such encoding is intact, which would point to an associative variant. Deficits in face perception have been measured by the Cambridge Face Perception Test[39] and the Glasgow Face Matching Test,[40] which involve sorting or matching faces by their identity with minimal demands on memory. The Dartmouth Face Perception Test is useful for children.[41]

Questionnaires of social impact

Questionnaires can evaluate everyday experiences with face recognition. There is a 15-item self-report questionnaire[13] that contains questions on face recognition, attractiveness judgments, and expression recognition, and a more recent 20-item questionnaire for face identity (Prosopagnosia Index, "PI20",[42]). However, these should be supplemented by objective tests for diagnosis.

Exclusionary tests

Establishing impaired face recognition is not sufficient for the diagnosis of prosopagnosia. One must also show that this is not due to more general problems with vision and memory. The assessment of acuity and visual fields can exclude low-level impairments of vision as a cause of poor face recognition: indeed one of the problems of subjects with macular degeneration is difficulty recognizing faces.[43] Beyond this, to exclude a more general visual agnosia, subjects with prosopagnosia should have normal object recognition at a "basic" level (ie, identifying that an object is a face, a bicycle, a lamp, etc). Some may have difficulties identifying specific examples of these objects (ie, which bicycle or which lamp): this is not grounds for rejecting a diagnosis of prosopagnosia, but is relevant to the debate about whether the recognition problem in prosopagnosia is truly specific for faces alone (see "Face Specificity" section). For this reason, challenging tests of object recognition that include measures of reaction time and premorbid expertise[44] are useful (see "Objects" section).

Finally, face identity recognition deficits can occur in the context of other disorders, and the diagnostic process should consider whether any of these are present. In children, this includes conditions such as autism[45–48] and Turner's syndrome,[49] while in adults impaired face recognition has been reported in schizophrenia,[50,51] Alzheimer's disease,[52–54] and Parkinson's disease,[55] for example. The diagnosis of prosopagnosia should be reserved for cases in which poor face recognition cannot be explained by one of these other conditions. Suggested criteria for the diagnosis of acquired and developmental prosopagnosia are outlined in Table 1. Greater detail regarding guidelines and available tests can be found in a recent review.[56]

Face specificity

Are prosopagnosic subjects impaired in the recognition of faces only? Here, we comment on four aspects of this question about specificity. First, a long-standing debate in face research is whether the mechanisms used to process faces are dedicated to faces alone, ie, "face specific", or if they are involved in processing other objects, particularly those for which we possess perceptual expertise.[57–59] Second, new theories have proposed that words and faces, two visual classes for which literate humans have great expertise, share and compete for resources, leading to predictions that prosopagnosic subjects may have subtle deficits in word

Table 1 Suggested inclusion and exclusion criteria for the diagnosis of acquired and developmental prosopagnosia

Inclusion criteria	Exclusion criteria	Clarification questions
• Difficulty with faces evident in everyday life (PI20) • Impairment on at least two measures of face familiarity (CFMT) • Confirmation of lesion by MRI or CT scan (AP cases only)	• Low-level visual impairment that could otherwise explain prosopagnosia • General visual agnosia • General memory impairment • Neuropsychological disorders associated with face recognition impairment • Visible lesion on MRI (DP cases only)	• Does the individual have associative or apperceptive subtype? (Cambridge Face Perception Test or Glasgow Face Matching Test) • Is the disorder prosopagnosia or a multimodal person recognition disorder? (Tests of name and voice familiarity)

Note: Suggested tests for adults are indicated in parentheses, and brackets indicate criteria specific to either acquired prosopagnosia (AP) or developmental prosopagnosia (DP).
Abbreviations: CFMT, Cambridge Face Memory Test; MRI, magnetic resonance imaging; CT, computed tomography.

processing.[60,61] Third, questions have arisen as to whether some prosopagnosic subjects may actually have a multimodal problem in recognizing people.[19,62,63] If so, they should also have impairment of recognition of people by voice and name; however, voice recognition has seldom been objectively evaluated in prosopagnosia. Finally, an issue of less theoretical but some practical interest is the array of other visual deficits that likely reflect damage to neighboring structures and networks, particularly with acquired prosopagnosia.

Objects

All objects share visual processing in the striate and early extrastriate cortex: whether the processing of faces and objects diverges later is the question. Neuroimaging studies of healthy individuals show that face processing depends on a cortical network of regions that is partially overlapping but distinct from areas involved in object processing.[64–67] Transcranial magnetic stimulation has demonstrated a double dissociation between face and object processing: stimulation of face areas interrupts face processing more than object processing and stimulation of object areas results in the reverse.[68]

The contribution of prosopagnosia research to this debate is mixed. While there are studies that report intact ability to distinguish between members of other object categories,[28,69–76] others describe cases who have difficulty.[63,77] If prosopagnosia is about expert processing, though, a notable omission from many of these studies is the failure to consider the premorbid expertise of the prosopagnosic subject for the objects being used in the testing. A recent advance is the development of a method to use verbal semantic knowledge about a type of object as an index of their premorbid expertise and to adjust visual recognition scores for the degree of expertise. When this was done, nine of ten subjects with acquired prosopagnosia were impaired in expertise-adjusted car recognition.[44,78]

Similar mixed results have been obtained in adults with developmental prosopagnosia, with several studies describing cases in which the recognition deficit affected only faces[9,79–84] and some cases in which the recognition of other objects was also impaired.[8,9,79,80,85–88] This is true for studies of children too.[9,49,89–92] A study of six children with developmental prosopagnosia found face-specific deficits in four, and more general deficits for both faces and objects in one.[93] These differences across cases and studies may reflect a real heterogeneity rather than methodological issues.

One study has also attempted to evaluate the effect of object expertise on recognition ability in developmental prosopagnosia.[34] Using the Cambridge Car Memory Test,[94] this study reported that, at the group level, those with developmental prosopagnosia did not differ from controls after controlling for car expertise. However, individual data were analyzed before controlling for expertise, making it difficult to know whether expertise-adjusted car recognition was intact in each subject.

Words

Next to faces, words may be the stimulus category for which we have the highest degree of visual expertise. Although face processing is more active in the right hemisphere and word processing on the left, both show bilateral networks that overlap.[95] A recent theory proposes that face processing and word processing compete for neural resources during development and that incomplete hemispheric lateralization is a result of this competition.[60,96,97] The prediction of this theory is that prosopagnosic subjects should have subtle impairments in the processing of words, even if their lesions are limited to the right hemisphere.

Several recent studies have tested this prediction in acquired prosopagnosia. One study found subtle impairments in word processing in three subjects,[61] but these may have had a more general integrative visual agnosia rather than prosopagnosia.[81,98] A second study of five subjects found normal performance on seven different reading tasks.[99] A third study[100] found that only prosopagnosic subjects with bilateral fusiform lesions showed an increased word-length effect (the time taken to read a word as a function of the number of letters), and slow sorting of printed cards by their word content. On the other hand, even subjects with right hemisphere lesions alone were impaired when they had to sort the same cards by their

font or handwriting. This suggested that the right hemisphere makes a critical contribution to the processing of stylistic properties of written text, rather than analyzing their word content. However, a similar recent study in developmental prosopagnosia has not found any deficit in processing the words or style of writing.[101] This may indicate that the style-processing impairments in acquired prosopagnosia are related to damage to adjacent processing areas rather than damage to the mechanisms involved in face processing.

Voices

Another question of specificity has examined whether individuals with prosopagnosia have difficulty in only the recognition of faces, or whether they struggle with person recognition more generally, such as the recognition of voices. On the other hand, years of relying on voice cues to recognize others may produce superior voice recognition in prosopagnosic subjects.[102]

A recent study of acquired prosopagnosia found that only subjects with bilateral anterior temporal lesions had deficits in the recognition of voices, and therefore were better classified as having a multimodal disorder of person recognition.[62] However, the recognition deficit was specific to faces and did not involve voices or names in those with right anterior temporal lesions alone or occipitotemporal lesions. A second study of 12 subjects with developmental prosopagnosia found impaired voice recognition in only one subject;[103] nevertheless, this provides more evidence for heterogeneity in the developmental variant.

Other deficits from damage to adjacent structures

The classic tetrad found with acquired prosopagnosia, particularly when due to occipitotemporal lesions, is superior field deficits, dyschromatopsia and topographic disorientation. Cerebral dyschromatopsia is associated with damage to the lingual and fusiform gyri, in the vicinity of the collateral sulcus, almost always with bilateral but rarely with right unilateral lesions.[104,105] It is characterized primarily by an accentuation of the tritanopic-like patterns seen in healthy subjects.[104] This last study did not find color impairments in subjects with developmental prosopagnosia.

Topographic disorientation, a disorder in which subjects get lost in familiar surroundings, is commonly reported with acquired prosopagnosia. A recent review found mention of topographic difficulty in 29% of 147 cases.[106] One possible explanation is the close proximity of the parahippocampal place area,[107] an area activated when viewing scenes, to the fusiform face area (FFA),[108] which is activated by viewing faces. In developmental prosopagnosia, there are anecdotal reports of both impaired[94–96,109] and preserved[84,110,111] navigational abilities. A recent study found that most patients with acquired prosopagnosia, regardless of lesion location, were impaired in scene and landmark recognition, while those with occipitotemporal lesions were also impaired in the ability to form cognitive maps,[112] and either deficit was rare in developmental prosopagnosia.

Neuroimaging

The advent of functional imaging has revolutionized cognitive brain science. In face research, it has delineated networks of regions active during face perception. This includes a core face network that includes the FFA,[108] the occipital face area (OFA), and the posterior portion of the superior temporal sulcus[113,114] (Figure 2). There is also an extended network that includes the anterior temporal face area and other regions such as the inferior frontal gyrus and precuneus.[4,115] While faces activate these areas in both hemispheres,[95,114] the effect is stronger on the right.[108]

Studies of acquired prosopagnosia have been advanced by the improved functional and structural capabilities of magnetic resonance imaging (MRI). A key fact is that a variety of lesions can cause prosopagnosia,[116] an observation that makes sense when one considers the widely distributed networks involved in face processing. Two key observations have been made from the study of acquired prosopagnosia. First, lesions may be bilateral or unilateral, and when unilateral they are far more likely to be on the right.[4,117,118] A few prosopagnosic subjects with left-sided lesions have been described, but most have been left-handed,[119–121] raising the possibility that they may have had anomalous hemispheric lateralization to begin with. Second, there is a useful division between occipitotemporal and anterior temporal damage. Recent functional MRI work has shown that occipitotemporal damage is associated with loss of activation of core components such as the FFA and OFA,[78] while activation in anterior areas may be spared.[122]

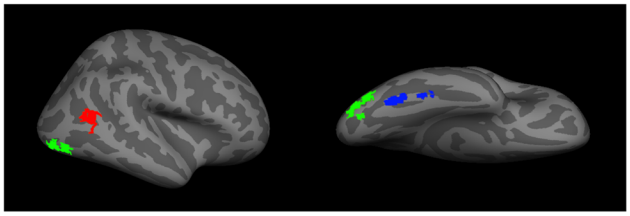

Figure 2 A representation of the core face network – including the fusiform face area (blue), the occipital face area (green), and the posterior superior temporal sulcus (red).

Conversely, activation of the FFA and OFA may be spared in individuals with anterior temporal lesions.[78]

These modern neuroimaging observations have generated structural correlates for functional variants of prosopagnosia that had long been hypothesized (see "Models of face recognition" section and Figure 1).[26] Recent studies show that those with fusiform lesions are more likely to have the apperceptive variant,[4,78,123] whereas those with anterior temporal lesions are more likely to have the associative variant[4,78,124,125] (Figure 3). The main conclusion is that acquired prosopagnosia is not a single disorder, but a family of disorders with different mechanisms and different lesions that nevertheless lead to the same end result of impaired face recognition.[78]

The structural correlates of developmental prosopagnosia are still debated. By definition, there is no obvious structural lesion, and early studies examining the evidence of abnormal activation of the core face network produced mixed results, with some reporting normal activation[126,127] and another reporting activation for faces that did not differ from activation for other object types.[126–128] Recent work with more advanced imaging methods has begun to uncover both structural and functional anomalies in developmental prosopagnosia, but there is disagreement. Some have suggested that there are anatomical[84] or functional[129–131] abnormalities in the FFA and localized differences in white matter fibers around the right FFA.[132,133] Others maintain that the core face network is largely normal and that abnormalities lie instead in the anterior temporal cortex,[134,135] other regions of the extended face network,[136] or the long-range white matter tracts that connect the core regions in occipitotemporal cortex with the anterior temporal face area, namely the inferior longitudinal fasciculus.[134,137] Both groups claim that the degree of altered white matter connectivity in their results correlated with behavioral measures of impaired face recognition.[133,137] Whether these discrepancies reflect a real heterogeneity that exists in developmental prosopagnosia remains to be determined.

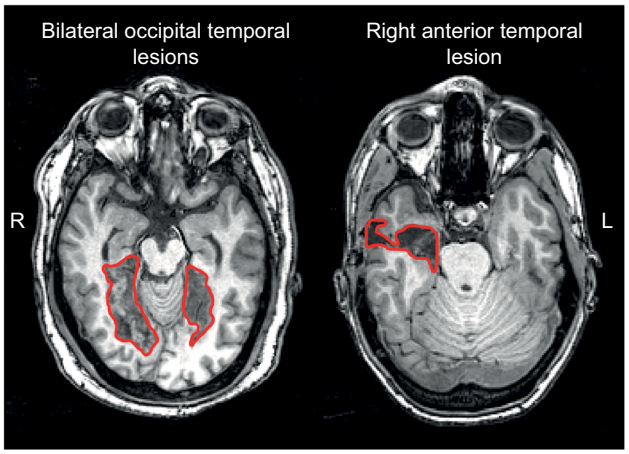

Figure 3 Examples of lesions that produce acquired prosopagnosia.
Notes: Approximate lesions, as can be seen on a single slice, are outlined in red. Patients with bilateral occipital temporal lesions (left) often experience apperceptive prosopagnosia and those with anterior temporal lesions (right) often experience associative prosopagnosia. These cases demonstrate that acquired prosopagnosia is a family of disorders with different mechanisms and different lesion locations that lead to the same end result of prosopagnosia.

Event-related potentials

While event-related potentials do not have as good spatial resolution as MRI, they have a much finer resolution in time and can advance our understanding of the temporal dynamics of face recognition. Studies of face recognition in healthy subjects identify three components. The N170 component is prominent in right lateral occipitotemporal areas: it shows larger responses to faces than other objects and is associated with perceptual aspects of face processing.[138–140] The N250 is also right-dominant and is the first component to show effects linked to the appearance of a specific facial identity, rather than just faces in general.[18,141,142] The P600 is seen when subjects can recognize a person by stating their name or providing information about them.[142,143]

Studies of acquired prosopagnosia support the association of the N170 with both an occipitotemporal location and perceptual aspects of face processing. Dalrymple et al[144] found that the face-selective aspect in the N170 was absent in subjects with apperceptive prosopagnosia whose lesions included at least two components of the core network (eg, FFA and OFA). However, it was intact in those with associative prosopagnosia whose lesions were restricted to anterior temporal cortex. Another study supported this finding by demonstrating preserved N170 face-selectivity in a subject with a right OFA lesion but preserved right fusiform gyrus,[145] and other studies reported its absence in a subject with impaired face perception.[143,146]

The findings in developmental prosopagnosia are less straightforward.[139] There are reports of both normal[147–149] and abnormal N170 components,[111,129,147,150] including one on the analogous M170 component detected by magnetoencephalography.[151] Larger studies have found heterogeneous results across subjects that can explain this inconsistency.[147,152–154] It is also possible that there are more subtle abnormalities in the N170 component. For example, the amplitude of the N170 component is usually larger when viewing upside-down faces, likely because it is harder to process them, but one study found that the majority of 16 subjects with developmental prosopagnosia failed to show an orientation effect in the N170 amplitude.[152]

With regard to the later potentials, a study of 12 subjects with developmental prosopagnosia found normal N250 and

P600 components on the few trials on which these subjects did identify a face, suggesting relatively normal processing when face recognition is successful. About half also exhibited N250 components for famous faces they did not recognize, which may indicate some unconscious processing. There was no P600 component under those circumstances, implying that this potential reflects conscious face identification.[155] Even though these studies indicate that the processes indexed by these later potentials can still be activated in developmental prosopagnosia, a recent Event Related Potential study claimed that these components are delayed[156] (see Towler et al[157] for a recent review of ERP findings in developmental prosopagnosia).

Treatment and rehabilitation

Can training improve prosopagnosia? The answer may be that it depends. In acquired prosopagnosia, one might speculate that the efficacy of any training could be affected by age at onset, duration since onset, and lesion size, laterality, and location, particularly with regard to how much of the face network and its connections are compromised.[158] Given the rarity of acquired prosopagnosia, it will be very difficult to establish the impact of each of these factors. To date, there have been few remedial attempts for the acquired variant, and most focus on enhancing coping strategies to circumvent poor face recognition.[158,159] Only one published study attempted to improve face recognition, in a child with diffuse damage after meningococcal meningitis: 18 months of training did not improve matters.[160] More recently, two training studies have been reported at conferences. DeGutis et al[159,161] attempted to train a 46-year-old with a right occipital-temporal lesion to categorize faces based on the distances between face parts. Unfortunately, this did not help. A second study trained 12 subjects to discriminate increasingly subtler differences in face shape across variations in expression and viewpoint, over 11 weeks. Some improvement was found, but this was more modest for the recognition of faces not used during training.[162]

Given the lack of overt brain damage, one might wonder whether training may be more effective in developmental prosopagnosia. One group trained 25 subjects with developmental prosopagnosia[163,164] to perceive the spacing between facial features and found improvements that generalized to new faces but did not help recognition when viewpoint varied.[164] A different therapeutic approach was used in a randomized, placebo-controlled, double-blind study examining the effect of intranasal inhalation of oxytocin, a drug associated with the regulation of social behaviors, on face identity processing.[165] The authors reported transient improvement of face perception and recognition in ten subjects with developmental prosopagnosia after oxytocin administration.

While these reports are encouraging, there may be limitations. Given the heterogeneity of deficits in prosopagnosia, it may be that a specific training program will not be appropriate for all subjects. How much of a residual face network one needs in acquired prosopagnosia to benefit from training is unknown. The belief that the subtler structural alterations of developmental prosopagnosia imply a better chance of having the neural substrate to generate benefit from training is unproven.

Conclusion

A prevailing theme in prosopagnosia research is the heterogeneity of findings across both acquired and developmental prosopagnosia. There is heterogeneity in the mechanism of prosopagnosia (ie, apperceptive versus associative), the location, lateralization, and extent of structural damage in the acquired form, and the presence or absence of impairments in other perceptual domains (eg, object, word, and voice processing). Heterogeneity is expected when one is dealing with a complex process such as person recognition, but it does create challenges that require particular care and rigor in experimental study and analysis.

For one, it is important to ensure that heterogeneity is not the inadvertent result of experimental factors. To this end, care is required in establishing the diagnosis of prosopagnosia and excluding other conditions (Table 1). First, besides excluding more general failures in object recognition and memory, tests of voice and name recognition are needed to establish where a patient is more accurately characterized as having a multimodal disorder of person recognition, whose mechanisms may differ from prosopagnosia. Second, uniform diagnostic criteria are needed. This is particularly an issue for developmental prosopagnosia. Currently, there is no diagnostic consensus: inclusion criteria range from purely self-report measures[12,13] to various conglomerations of self-report, behavioral tests of face familiarity, and tests of face naming/identification,[156,166] and few require imaging to exclude brain lesions that would point to an early-onset acquired variant rather than developmental prosopagnosia. Another diagnostic issue that reflects the current lack of definitive genetic or radiologic markers for the developmental form is the challenge of distinguishing subjects with true pathology resulting from aberrant development of face recognition networks from those who are simply at the low end of a spectrum of normal face-processing skill (see Barton and Corrow[14] for a discussion).

Nevertheless, it remains a possibility that there is real heterogeneity in developmental prosopagnosia, just as there is in acquired prosopagnosia. This accounts for the current trend to use single-subject methods of analysis, using Crawford's *T* tests, for example. However, it may be difficult for subtle anomalies to achieve statistical significance at the individual level, as illustrated by recent ERP work.[151,152] Further work may benefit from the definition of more homogeneous variants, and supplementing the single-subject methods with group analyses on these subgroups. This will necessitate the collection of larger samples of these patients. Such efforts should advance our knowledge of the neuroanatomic and functional origins of these intriguing conditions.

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