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Face Perception in Health and Disease

Andrew J Logan BSc PhD FHEA MCOptom DipTp(IP)

Department of Vision Sciences, Glasgow Caledonian University, Cowcaddens Road, Glasgow, G4 0BA

Abstract

For most of us, face recognition is a rapid and effortless process which enables us to identify people who are familiar to us. Underlying this remarkable- but perhaps overlooked- aspect of visual function is a network of interconnected brain regions which processes the raw visual information provided by the eyes. As a result, face recognition is vulnerable to both ocular disease (e.g. AMD) which compromises visual input and neurological impairments, such as prosopagnosia (face-blindness), which disrupt activity within higher-level brain regions. This article reviews current evidence about these causes of impaired face perception and outlines the clinical implications for affected patients.

An Introduction to Face Perception

Faces are amongst the most common, yet complex, objects that humans use vision to recognise. A brief glimpse of a face is typically sufficient to make judgements about a person's age, gender, ethnicity, mood and whether they are familiar to us, or not. Humans recognise familiar faces both accurately and rapidly. For example, Ramon and colleagues reported that participants with healthy vision correctly identified a face on 98% of times that they were tested and required, on average, 380ms to recognise the face as familiar.[1] This ability is particularly impressive because faces appear so similar: all faces are based upon the same template (two eyes, above a nose, above a mouth). As a result, human vision needs to identify subtle differences in the shape (e.g. broader nose) and position (e.g. narrower inter-pupillary distance) of individual face features in order to recognise a face that we have seen before.[2]

Our ability to rapidly and accurately recognise faces is put to good use by society. For example, face identification is the primary method used by authorities to establish an individual's identity (e.g. passport photograph inspection by a border control officer, photographic driving licence for personal identification). Further, juries consider eyewitness identification to be one of the most compelling forms of evidence in criminal trials.[3] Despite many years, and considerable research effort, computer algorithms are, as yet, unable to out-perform the face recognition accuracy of human vision.[4] While automated face recognition is becoming increasingly sophisticated, and finding applications within the fields of surveillance and security, face identification by humans is still relied upon to verify possible matches suggested by computer algorithms.[5]

From the clinician's perspective, the importance of face recognition is, perhaps, best illustrated by reports from patients who, due to injury or disease, experience significant impairments of face perception. These patients explain that, due to being unable to recognise faces, they encounter difficulties with social interactions and experience associated feelings of isolation and depression.[6] Later sections of this article will describe common causes of impairments of face perception and their clinical implications.

Our impressive ability to recognise faces is dependent upon both input from the eyes and functioning within specific brain regions.[7] In order to investigate which brain areas are involved in face perception, researchers have employed functional magnetic resonance imaging (fMRI) to monitor patterns of brain activity while participants view different objects.[8] This work has revealed that faces provoke a strong response from certain brain regions, and these regions are consistent from person-to-person. Whilst faces activate many of the areas of the brain that process a broad range of visual information (e.g. colour, orientation, texture), there are certain brain regions which seem to respond *selectively* to faces, and not other types of visual information. For example, the *Fusiform Face Area*, located within the temporal lobe, is strongly activated when participants are shown images of faces.[9] When participants were shown images of other objects (e.g. cars, houses), brain activity within the same regions fell to baseline level.[8] These results suggest that humans have developed brain areas which are specialised for processing faces, and these are distinct from brain areas recruited to process other objects.[10] Consistent with this premise, clinical case studies indicate that brain damage within these areas gives rise to specific symptoms of impaired face recognition.[11] Later sections of this article will explore this *Acquired Prosopagnosia* in more detail.

The results from neuroimaging studies outlined above suggest that the human brain treats faces differently from other objects. This is also suggested by the way in which humans perform on tests of face recognition. For example, recognition accuracy for most objects (e.g. houses, cars, animals) is reduced to some extent when images are shown upside-down.[12] Recognition of faces, however, is impaired to a considerably greater extent (see Figure 1 for an illustration).[12, 13] This *disproportionate face inversion effect* marks faces out as being processed differently to other objects by the visual system. It has been suggested that this phenomenon is a product of our visual experience: humans rarely see a face presented upside-down and, therefore, have had no need to develop the skills in order to recognise them.[14]

Figure 1. Inverted faces and the Thatcher Illusion. On cursory viewing, this image appears to be a face photograph presented upside-down. The face has, however, been extensively manipulated to appear grotesque. Turning the page upside-down will allow you to appreciate the extent of this transformation, which was not obvious when the face was presented in the original, inverted orientation. This phenomenon, labelled 'the Thatcher illusion' after its first subject, demonstrates that human vision is considerably poorer at recognising face information when faces are shown upside-down, compared to upright.[15]

The effects of visual experience are evident from other aspects of our ability to recognise faces. In particular, it is well established that humans typically recognise faces of their own race more accurately than those of other races. For example, Chiroro and Valentine found that Black participants recognised photographs of Black people more accurately than photographs of Caucasian people.[16] The opposite pattern was found for Caucasian participants. This *own race bias* does not appear to be genetically hard-wired, but can be shaped by visual experience. This is demonstrated by studies which report that contact with individuals belonging to other races weakens the own race bias.[17] As with other aspects of visual development, plasticity seems to be greatest during childhood.[18] For example, Asian children adopted into Caucasian families recognise Asian and Caucasian faces with equivalent accuracy,[19] or may even demonstrate a reversal of the own race bias (i.e. improved recognition of Caucasian, relative to Asian, faces).[20]

Prosopagnosia: A Specific Impairment of Face Perception

As outlined above, face perception relies upon a network of brain regions. Some of the most compelling evidence in support of this proposal is the clinical finding that damage within these brain regions gives rise to *prosopagnosia*; an inability to recognise familiar faces (sometimes referred to as '*face-blindness*').[11] Patients with prosopagnosia may be unable to recognise the faces of their partners, friends and family.[21] In some cases, prosopagnosia means that the patient does not recognise their own reflection in a mirror.[22] Importantly, these symptoms cannot be explained by general visual impairment; patients with prosopagnosia often demonstrate good visual acuity.[23]

It is certainly true that, in cases of widespread brain damage, prosopagnosia can present alongside other neurological impairments, such as alexia (an inability to read)[24], agraphia (an impairment of writing ability)[25] and apraxia (inability to control specific movements).[26] In a few rare cases, however, prosopagnosia exists in isolation.[11] Brain damage in these patients is confined to the regions which are specialised for face perception. As a result, other aspects of vision and cognitive functioning remain intact, yet the patient cannot recognise faces. For instance, visual acuity, contrast sensitivity and colour vision may be within normal limits.[27, 28] Similarly, patients with prosopagnosia can demonstrate memory, cognitive and intellectual abilities which are equivalent to those of healthy patients without brain damage.[11]

The first published case reports of prosopagnosia- in the wake of the second world war- featured brain damage caused by bullet wounds.[29] Nowadays, the leading causes of acquired prosopagnosia include cerebrovascular accidents (stroke), traumatic brain injury, encephalitis and surgery for intractable epilepsy.[11]

A series of case reports indicate that patients with acquired prosopagnosia can recognise a diverse range of non-face objects (including cars, fruit, coins, monuments, dog breeds, shapes and animal faces) with the same accuracy as healthy control participants.[30] In one example, a farmer developed prosopagnosia following a cerebrovascular accident. Although he was unable to recognise familiar faces, he retained the ability to recognise individual members of his flock of sheep.[31] These reports suggest that specific patterns of brain damage can selectively impair face perception, without impacting upon other aspects of visual function.

Developmental Prosopagnosia

Prosopagnosia attributable to selective brain damage- known as *acquired* prosopagnosia- is very rare. There is, however, another form of prosopagnosia which is considerably more prevalent. This *developmental* prosopagnosia is found in patients without any evident

structural brain damage and is thought to result from under-development of the brain regions which are specialised for face processing.[32] While the precise aetiology of the condition is not yet fully understood,[33] a number of reports indicate that developmental prosopagnosia tends to run in families,[34] which suggests that genetics play a significant role.[35] Current understanding of the genetic basis of developmental prosopagnosia is limited, but the results of recent studies point to the conclusion that the condition's inheritance pattern is complex, multi-factorial and may involve multiple genes.[36, 37]

Despite the lack of structural brain damage, the effect of developmental prosopagnosia on the ability to recognise faces can be as severe as that in the acquired form of the condition. For example, the average score of individuals with healthy vision on a well-established, lab-based test of face recognition is approximately 80%.[38] Average performance of patients with developmental and acquired prosopagnosia, however, was 49%[39] and 54%[40] respectively.[36]

As described earlier, faces enable us to effortlessly and rapidly recognise people who are familiar to us, and this ability is critical for social interactions. It is perhaps unsurprising, then, that some patients with developmental prosopagnosia report that an inability to recognise familiar faces has a profound impact upon their quality of life.[6] A number of studies have used patient self-report questionnaires and structured interviews to investigate the experiences of people with developmental prosopagnosia.[6, 41, 42] A common theme is that failing to recognise a colleague or friend leads to patients being unfairly labelled as rude, arrogant or aloof.[43] Patients further report that developmental prosopagnosia leads to social isolation, with associated anxiety and depression.[6] The results of one study suggests that children (aged 5-14 years old) with the condition are at particular risk of long-term psychosocial impairments and may experience difficulty maintaining friendships.[41]

While some patients undoubtedly find developmental prosopagnosia to be a debilitating condition which severely impacts upon their quality of life, other patients appear to be considerably less affected.[44] This is consistent with the finding that developmental prosopagnosia is not uncommon; a handful of studies have estimated that the prevalence of the condition is approximately 2-3%.[44-46] Whilst this implies that developmental prosopagnosia affects more people worldwide than glaucoma,[47] '*difficulties with face recognition'* are neither routinely reported to healthcare professions nor widely recognised by society as a common condition (although awareness is steadily increasing). One explanation for this paradox is that developmental prosopagnosia is thought to be present from birth.[48] As a result, since their experience of face perception has always been atypical, some patients may be unaware of the extent to which their face recognition ability is impaired, relative to that of a typically-developed individual. In the same way, some patients are unaware of a congenital colour vision deficiency until the condition is uncovered by formal testing.[49]

Another explanation for the apparent under-reporting of developmental prosopagnosia is that there is a range of coping strategies which enable some patients to minimise the impact of the condition on their daily lives. Most strategies rely upon making use of alternatives clues to an individual's identity, such as voice, posture, hairstyle, spectacles or distinctive articles of clothing (e.g. a patterned jacket).[50] Others include directing the conversation to reveal identifying details (e.g. *"How are things at work?"*) and priming partners to use a friend's name at the beginning of a conversation (e.g. *"How are you, Susan?"*).[51] A number of patients explain that context is critical: it might be possible to identify a co-worker when they are seated at their usual desk within the office, but not upon chance meeting in the supermarket.[6] Patients with developmental prosopagnosia report considerable difficulty

when the clues upon which their coping strategies are based are removed (e.g. a friend changes their hairstyle).[52]

Prosopagnosia: Diagnosis and Management Options

In the UK, acquired prosopagnosia is typically diagnosed by clinical neuropsychologists.[53] The developmental form of the condition is less likely to be medically investigated; many cases of suspect developmental prosopagnosia are formally tested within university-based research centres. Testing for prosopagnosia typically involves asking the patient to complete a range of face recognition tests. Patients also complete recognition tests for objects other than faces (e.g. cars, houses) to determine if any impairment is specific to faces, or could be explained by an impairment of vision, memory or general object recognition (i.e. object agnosia).[50]

No specific treatment for either acquired or developmental prosopagnosia is currently available. In general, management of prosopagnosia is primarily focussed upon rehabilitative measures, such as supporting patients in learning to use the coping strategies outlined above. Researchers are, however, investigating the viability of novel treatment options for prosopagnosia. For example, recent results from a lab-based study suggest that administration of oxytocin[54]- a hormone which plays a key role in social bonding- significantly improves familiar face recognition ability in patients with developmental prosopagnosia.[55] Further, engaging with an online training programme over an 11 week period significantly improved face perception ability in patients with the condition.[56] Encouragingly, participants reported that the benefits translated to improve face recognition ability in everyday life, and were still evident several months after completing the training. These results suggest that face perception may be amenable to *perceptual learning*- improved sensitivity following repeated practice with a specific task (i.e. face perception).[57] This possibility is particularly exciting because treatments based on perceptual learning are non-invasive, low-cost, portable (patients can undertake training on their own laptop or similar electronic device) and carry minimal risk of adverse effects. Considerable future work is required, however, to develop an effective face training programme for widespread use with patients.

Face Perception in Ocular Disease

As outlined earlier, there are several, high-level brain regions which appear to be specialised for the processing of faces. These specialised regions receive their input from earlier areas of the visual system, such as the retina and primary visual cortex, which process a considerably broader range of visual information. As a result of this hierarchical organisation, visual impairment due to retinal disease (e.g. age-related macular degeneration, glaucoma) feeds-forward to impact upon more complex visual functions, including face perception. In this way, patients with central visual impairment may experience considerable difficulty with face recognition, despite an intact network of face processing brain regions.[58] This mirrors the case of developmental prosopagnosia in which patients with good visual acuity and full visual fields nevertheless experience considerable difficulty with identifying faces.

Age-Related Macular Degeneration

Age-related macular degeneration (AMD) is a chronic and progressive retinal disease which gives rise to central visual impairment. AMD is a leading cause of irreversible visual impairment in the UK[59] and the number of patients affected by the disease is projected to increase significantly over the coming decades.[60]

Difficulty with aspects of face perception is often reported by patients with AMD.[61] In particular, identifying the faces of family and friends is frequently highlighted as a day-to-day activity which poses a problem for these patients.[62] For example, Tejeria and colleagues[63] found that 29 of the 30 patients with AMD that they questioned reported difficulty recognising familiar faces in the street. In a recent study, Lane and colleagues[64] recruited 21 patients with bilateral AMD and specifically interviewed them about their experiences with impaired face perception. The patients initially reported that AMD made it difficult to recognise familiar people, understand facial expressions and make eye contact. The patients then explained that this, in turn, limited their ability to enjoy social interactions, led to feelings of isolation and reduced their quality of life.

These descriptions are supported by the results of lab-based studies which have provided evidence of significantly impaired ability to recognise both face identities[63, 65, 66] and facial expressions[63] in patients with AMD, relative to participants of the same age with healthy vision. For instance, Barnes and colleagues found that patients with AMD performed a face matching test (i.e. which of these photographs show the same person?) significantly slower and less accurately than control participants of the same age.[66] Similarly, Johnson et al.[67] reported that patients with AMD performed poorly on a task which asked participants to recognise facial expressions (e.g. happy, angry).

It should be borne in mind, however, that difficulties with face perception are not experienced by all patients with AMD. As with many other aspects of vision, face perception ability varies considerably, and some patients with AMD score as highly as age-matched controls with healthy vision.[66] In line with this premise, Taylor and colleagues[68] found that patients in the later stages of the disease are considerably more likely to demonstrate impaired face recognition than those in the earlier stages. In particular, Taylor et al.'s data suggest that geographic atrophy which has compromised the fovea increases the likelihood of impaired face recognition. This is supported by the finding that face recognition ability is associated with performance on other tests of central visual function, such as distance visual acuity[69], reading acuity[65] and contrast sensitivity.[68, 70] For example, poor VA increases the likelihood that a patient with AMD will experience difficulty with face perception.

There is some evidence to suggest that the performance of patients with AMD on tests of face perception can be significantly improved through use of a bioptic telescope device.[63] While these data are encouraging, Tejeria and colleagues[63] pointed out that not all patients find these devices easy to use, owing to the highly magnified image and limited field of view. Current research is exploring the possibility that electronic image enhancement might offer a route to improving face perception ability in patients with central visual impairment. Researchers have demonstrated that digital enhancement of faces to make them appear more distinctive (i.e. 'caricaturing') significantly improves recognition accuracy in patients with AMD.[71] Whilst technical barriers remain, the hope is that this image enhancement technique could be employed within an augmented reality platform (e.g. via a smartphone or smart-glasses) which would enable patients to benefit from image enhancement while viewing the real world.[72] Indeed, wearable low vision aids which incorporate face recognition technology have recently become available to patients.[73]

Glaucoma

Glaucoma is a progressive optic neuropathy which gives rise to irreversible visual impairment. The classic view has been that, at least in the early stages, glaucoma predominately affects peripheral vision, with the fovea typically spared until the very advanced stages of the disease.[74] It is worth noting that this traditional view is challenged by evidence that careful examination of macular visual function in early cases of glaucoma

reveals significantly reduced visual sensitivity.[75] In any case, glaucoma is, perhaps, a less intuitive cause of impaired face perception than diseases, such as AMD, which are characterised by severe impairments of central vision. In the first formal report of face perception in glaucoma, Glen and colleagues[76] invited patients with early, moderate and advanced glaucomatous visual field loss to undertake a lab-based test of face recognition. The results showed that the performance of those patients with advanced visual field defects was considerably poorer than that of either people with healthy vision or patients with less advanced visual field loss. Consistent with the premise that accurate face recognition requires the fine resolution of foveal vision, Glen et al. found that patients with significant visual field defects within the central 10° performed particularly poorly. In a subsequent study, Schafer and co-workers demonstrated that, relative to participants with healthy vision, patients with glaucoma required to be significantly closer to a face image in order to accurately categorise an individual's gender or facial expression.[77] These findings are supported by reports from patients with glaucoma which include descriptions of difficulty recognising familiar faces at a distance and identifying characters in television programmes.[78]

Atypical Visual Development

While AMD and glaucoma primarily affect older adults, children with atypical visual development are also at risk of impaired face perception. For example, Robbins and colleagues[79] assessed face recognition in a group of children and young adults who had been born with bilateral congenital cataracts. In all cases, the cataracts had been surgically removed within the first year of life and refractive error had been corrected. Although the participants were now 11-27 years old, all demonstrated a significant impairment of face recognition. Intriguingly, the same participants showed no impairment on an almost identical task which assessed the ability to recognise other objects, such as houses and animal faces. Accordingly, the difficulty recognising human faces cannot be explained by general visual impairment (e.g. impoverished VA or contrast sensitivity). Rather, these findings suggest that the significant visual deprivation associated with the congenital cataracts was sufficient to disrupt the development of human face recognition ability. Similar results have been reported elsewhere.[80, 81] It seems that visual experience is a requirement for the development of the effortless ability to recognise differences between human faces.

Unilateral amblyopia- typically caused by either strabismus or anisometropia- is a considerably more common presentation in clinical practice.[82] Little is currently known, however, about the impact of unilateral amblyopia on face perception.[83] The results of a single study suggest that patients with strabismic amblyopia demonstrate an impairment of recognising different face identities, but in their amblyopic eye only.[84] Further research is needed to explore the implications of this preliminary finding.

Clinical Testing of Face Perception

Many prevalent conditions (e.g. AMD, glaucoma, developmental prosopagnosia) place patients at risk of difficulties with face recognition. A recurring theme is that there is considerable variation in the extent to which these conditions impair face perception. As described earlier, whilst some patients with developmental prosopagnosia or AMD describe a severe and debilitating inability to recognise faces, others report no specific symptoms of impaired face perception. Currently, however, clinicians are without a standardised test to directly assess face perception ability. While a number of face recognition tests are used by researchers to study prosopagnosia, these require considerable time to administer (10-15 minutes per test)[85, 86], which makes them impractical for use in a clinical setting. The lack of

a suitable clinical test raises the risk that impairments of face perception could be under-diagnosed.[87]

There is evidence, however, that a diagnosis of impaired face perception is valued by patients, even when treatment is unavailable.[51] In a recent study, patients with developmental prosopagnosia reported that earlier diagnosis would be helpful because it would empower them to explain their condition to friends, family and employers.[51] This, in turn, might reduce the risk of patients with developmental prosopagnosia being misunderstood as 'rude' or self-important when they fail to recognise friends or acknowledge co-workers.[51] The latter is of particular importance since patients with developmental prosopagnosia are at risk of career limitations due to difficulties with social interactions.[6]

My colleagues and I recently co-developed a computer-based test which provides an accurate and efficient assessment of face perception. *The Caledonian Face Test*[87] asks patients to view four faces on a computer screen and identify the 'odd-one-out'. Since the patient does not need to memorise individual faces, a poor test result cannot be explained by memory impairment, such as that found in Alzheimer's disease.[88] The test automatically adjusts the difficulty level in response to the patient's performance. This enables the test to rapidly measure face perception (average test time is 3-5 minutes) and makes it suitable for all patients, including those with either exceptional or severely impaired face discrimination ability. The results of our initial validation suggest that *The Caledonian Face test* may be more sensitive to impairments of face perception than currently available tests. Our current work, funded by the College of Optometrists, aims to adapt the test to make it suitable for measuring face perception in patients with AMD.[89]

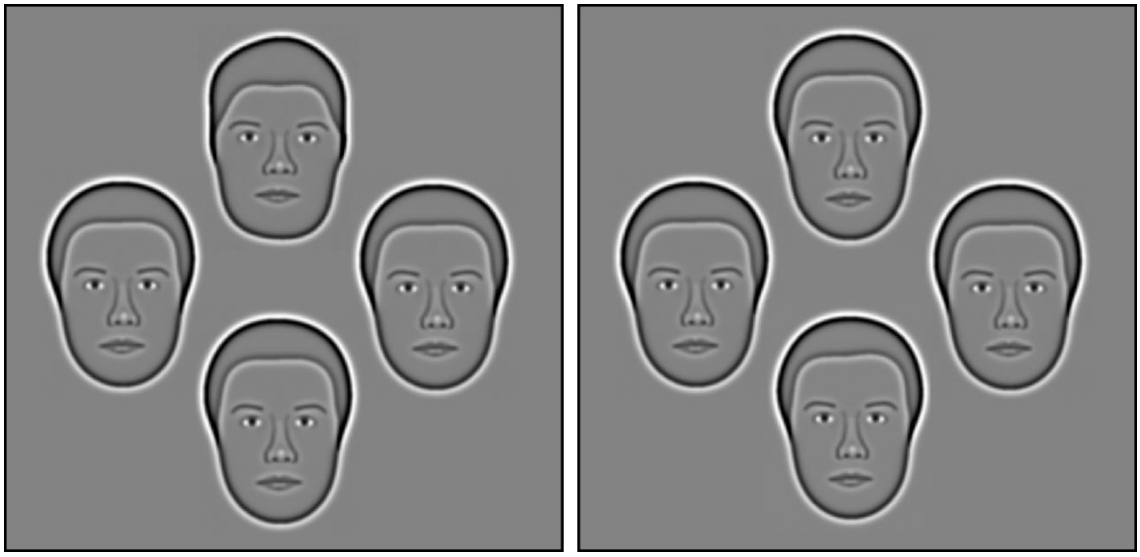

Figure 2. The Caledonian Face Test. The left and right images provide example trials of the Caledonian face test. In both cases, the task is to identify which of the four faces is different to the other three. Left: In this example, the difference between the faces has been set at a supra-threshold level for typical participants. As a result, most observers can identify that the top face is different from the remaining three. Right: The level of face difference has been set below the threshold for most people. Accordingly, only a minority of observers recognise that the bottom face is the odd-one-out. The Caledonian face test dynamically adjusts the level of task difficulty- based on participant performance- throughout the test in order to zone-in on the face discrimination threshold (the minimum difference required between faces for an observer to reliably tell them apart).

Conclusion

Symptoms of impaired face perception can be indicative of a wide range of conditions. Patients with undiagnosed developmental prosopagnosia may become aware of long-standing difficulties with recognising familiar faces and seek an explanation. At the other extreme, in rare cases, an inability to recognise faces can result from severe neurological conditions such as a cerebrovascular accident or brain aneurysm. As Optometrists, we are well-placed to exclude common ocular causes of impaired face perception by carrying out a dilated fundus examination to investigate for signs of diseases such as age-related macular degeneration and glaucoma. Current evidence indicates that patients with AMD- particularly those with advanced disease- are at significant risk of impaired face perception. It may be helpful, therefore, when eliciting a history from patients with advanced AMD, to ask specific questions about any difficulties with face perception which the patient might have experienced. On a related point, children who suffer visual deprivation during their early years are at risk of irreversible impairments of face perception which may be detrimental to their social development. Current research efforts are working towards improving diagnosis of impaired face perception through development of standardised tests. The future may also see availability of innovative management options, including medical treatment of prosopagnosia and application of virtual-reality technology for patients with visual impairment.

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