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Abstract (250 words max)

Face perception is probably the most developed visual perceptual skill in humans, most likely as a result of its unique evolutionary and social significance. Much recent research has converged to identify a host of relevant psychological mechanisms that support face recognition. There has also been substantial recent progress in uncovering the neural mechanisms that mediate rapid and accurate face perception, with specific emphasis on a broadly distributed neural circuit, comprised of multiple nodes whose joint activity supports face perception. This article focuses specifically on the neural underpinnings of face recognition, and reviews recent structural and functional imaging studies that elucidate the neural basis of this ability. In addition, the article covers some of the recent investigations that characterize the emergence of the neural basis of face recognition over the course of development, and explores the relationship between these changes and increasing behavioural competence. This paper also describes studies that describe the breakdown of face recognition in individuals who have an impairment in face recognition, either acquired by virtue of brain damage or as a result of failing to master recognition over the course of development. Finally, information regarding similarities between the neural circuits for face perception in humans and in non-human primates is briefly covered, as is the contribution of subcortical regions to face perception.

Face perception is probably the most developed visual perceptual skill in humans, most likely as a result of its unique evolutionary and social significance. Perhaps surprisingly, in light of the value of face recognition for survival, the discrimination and individuation of faces present extraordinary challenges for the visual system. In terms of image properties, compared to other classes of visual inputs (for example, vehicles or even just different makes of cars) faces are more similar to one another perceptually and are all essentially composed of the same local elements (two eyes, a nose, cheeks and a mouth) in the identical spatial layout (e.g., eyes above the nose). In addition, at any moment in time, faces carry a large amount of information about the individual including their age, gender, emotional state, gaze direction, thereby increasing the complexity of processing the input. Notwithstanding these challenges, human observers can identify individual faces accurately and rapidly even across radically different viewing conditions (e.g. lighting, vantage point) and structural geometric changes as the person ages or conveys different emotional expressions dynamically. While there is some variability in face recognition abilities within the normal population (e.g. (1-5)), most people can represent the identity of an almost unlimited number of faces, and can access the relevant information such as the name and biographical knowledge associated with a particular face. Of interest too, is that these skills are derived in a relatively unsupervised fashion over the course of development (in contrast, for example, with word recognition that requires many hours of directed training for the majority of individuals).

Here, we provide a review of recent studies that explore the neural mechanisms supporting robust and accurate face recognition. In addition, we review findings from studies that explore the emergence of face perception over the course of development, and report results from investigations of individuals who are impaired in face recognition, either as a result of acquired brain damage or of a failure to master this skill. In sum, we argue that the mature face recognition system comprises a distributed network of multiple nodes whose joint activity supports reliable and robust face individuation. This network evolves and is fine-tuned over the course of development as evident by both the emergence of the nodes of the network and their increased structural and functional connectivity. Disrupting the network through damage to the node/s themselves or through compromised connectivity between them results in impairments in face recognition.

Face perception

Neural underpinnings: normal populations

Cortical contributions

The neural underpinning of successful face representation has been of much interest in visual neuroscience likely because of the complexity of the process and the observers' great facility with faces. Face selective cells have been identified in monkey inferior temporal cortex (see (6) for a review) and, more recently, face-selectivity has been confirmed in the same regions in studies using functional magnetic resonance imaging (fMRI) in monkeys (7, 8) and in marmosets (9) [see also (10-14)]. We note, however, that the findings of single unit investigations and fMRI in monkeys are not always consistent (15), and that there is a need to elucidate the relationship between these two domains.

In humans, our understanding of the neural substrate of face recognition has received the greatest boost from the numerous fMRI studies in adult humans investigating this issue. These studies, collectively, point to a number of regions that show a selective response to faces (compared to other stimuli) including the fusiform gyrus (FFA) (16-18), the lateral occipital region (LOF), the superior temporal sulcus (STS), and the occipital face area (OFA) (19-26). In addition to these "core regions" of face processing (adopting the terminology of (21)), there are a number of other regions outside the occipito-temporal cortex that constitute an "extended" face recognition system and play a critical role in other aspects of face perception. These include for example, the anterior temporal lobe, which plays a critical role in processing semantic and biographical information (27-29) as well as identity representations of faces (30), independent of modality (for a recent review, see (31-33). In addition, the precuneus/posterior cingulate cortex and the anterior paracingulate cortex likely play a role in representing some knowledge of faces, consistent with the stronger activation for familiar versus unknown faces in these regions obtained via various paradigms (e.g., generally famous faces [30], personally familiar faces (26), and visually familiar faces [31]). Others have implicated the precuneus/posterior cingulate region in the acquisition of face familiarity [32], and this is also consistent with studies showing selective activation for familiar voices in this region [33].

Largely as a result of the imaging studies in humans and in non-human primates, there is now a growing consensus that face perception is accomplished by the activity of a well-connected face processing network (17, 34). However, considerable debate still continues to revolve around the particular role of the different face-selective regions, with some suggesting even that the regions do not have disparate assigned roles that are distinct and that all regions participate in all types of face perception. Advances in more sophisticated data analysis approaches, including network analyses and multi voxel pattern analysis (MVPA) permit an examination of the properties of the face network as a whole (35) in normal as well as individuals with impaired face processing and the specification of the computational contribution of the different face selective regions within the network at a much finer grain of resolution. For example, using MVPA, several studies have now implicated the anterior temporal cortex as being critical for image invariant representation of face identity (e.g. (27, 28, 36) and, interestingly, these face representations can persist even when the core FFA and OFA regions are damaged (30). This region alone, however, is unlikely to suffice as a lesion to many other regions, including FFA and OFA, result in a deficit in face perception, as does a lesion to the anterior temporal lobe itself (32, 37)(see section on prosopagnosia below for further discussion).

Beyond these advances in understanding the network as well as regional contribution to face processing, it is important to acknowledge that part of the difficulty in establishing whether different regions within the core and extended face system play distinct roles comes from the fact that fMRI studies are correlational rather than causal and it is not obvious how to segregate the relative contribution of the different areas. As we review below, the findings from studies of prosopagnosia can inform our understanding of this issue. Additionally, studies which provide direct electrical stimulation to the human brain can be informative: for example, recent reports in which either the OFA (38) or the FFA (39) have been directly stimulated in patients undergoing mapping prior to neurosurgery have led to temporary distortions in face processing, thereby revealing that these regions play a functional role in normal face processing. Relatedly, a recent study, which also attempted to examine causality used thetaburst transcranial magnetic stimulation (TBS) and then measured the effect of this disruption in local and remote face-selective regions with fMRI. Disruption of the right OFA reduced the neural response to both static and dynamic faces in the downstream face-selective region in the fusiform gyrus whereas disruption of the right pSTS reduced the response to dynamic but not static faces (40). Together, these studies provide evidence for a multi-node cortical network whose integrated function is key to normal face perception.

Subcortical contributions

While much of the research on face recognition has explored the cortical substrate associated with this behaviour, there is also some evidence that subcortical parts of the brain might be contributing functionally, as well. Phylogenetic evidence indicates that the ability to discriminate kin from non-kin is ubiquitous even in species with rudimentary brain structures, such as wasps (41, 42), and honeybees (43), and, along similar lines, some neuroimaging studies in non-human primates have detected activation of lower-order, subcortical structures when monkeys view images of monkey faces and bodies compared with images of their scrambled counterparts (44). Of relevance, one high-resolution imaging study in non-human primates has even succeeded in observing separable activations of subnuclei within the amygdala in response to faces (45). The amygdala, due to its role in processing emotional aspects of face representations (17, 21, 26), is obviously also a critical structure that is engaged in face perception (46).

Ontogenetic evidence in humans also indicates a contribution from more rudimentary neural structures to face perception: even with a rather immature neural system, newborn human infants are able to discriminate perceptually heterogeneous faces, an ability attributed to a primitive subcortical bias to orient towards face-like patterns with relevant configural information (47, 48). Consistent with this, under monocular viewing, infants preferentially orient to images resembling faces to a greater extent in the temporal compared with nasal hemifield (49), a result indicative of retinotectal mediation (50). Despite these findings implicating more rudimentary neural structures in face perception, evidence for the contribution of such structures in adult humans is rather sparse likely because of the difficulty of imaging deep structures. In one recent study using fMRI data from 215 participants viewing faces, Mende-Siedlecki et al. (51) were able to detect robust and reliable responses to neutral faces in the amygdala bilaterally and observed strong functional coupling between the amygdala and posterior face-selective regions (such as FFA). Although the major emphasis of this study is on the amygdala, face-selective responses were also noted in the superior colliculus and hippocampus (see also 52). The results from this large-scale study indicate that, when methodology permits, a substantial contribution from subcortical structures to face perception in adult humans can be uncovered. What remains uncertain from this finding is what aspect of the representation activated in these structures contributes functionally to face perception.

To address these issues, recent studies have explored whether subcortical regions contain representations of face identities (it is well established that representations of facial expression is mediated by subcortical structures such as the amygdala as noted above; (53-55). The technique we adopted to address this takes advantage of the fact that visual input, once received by the retina, is propagated in an eye-specific fashion through the early stages of the visual system. This monocular segregation is retained up to layer IV of striate cortex (56, 57). Because there are relatively few monocular neurons beyond area V1 (58), activation of extrastriate areas is not eye-dependent (see Figure 1). Given that observers are not explicitly aware of the eye to which a visual stimulus is projected (59, 60) and, rather, perceive the images from different eyes as ‘fused’ (see

Figure 2), manipulating the eye-of-origin of the stimulus provides a useful tool for isolating monocular versus binocular neural channels. Thus, the logic of our studies was as follows: if perceptual performance is enhanced when two images are presented sequentially to a single eye versus inter-ocularly to the different eyes, we can infer that the monocular advantage is a product of neural facilitation within lower levels of the visual pathway. This technique has been used successfully in the past to examine plasticity in transferring perceptual learning from one eye to another (61), examination of spatial attention (62) and multi-sensory perception (63).

Insert Figure 1 and Figure 2 approximately here

Adult participants were significantly better at judging the likeness of two faces than the likeness of two cars or of two letter-strings, when the stimuli were presented to the same eye compared to when they were presented to different eyes. Having established that this monocular enhancement was selective for faces; we then demonstrated that the monocular advantage was 1) of equal magnitude for faces presented in the upright and inverted orientations, 2) not present when subjects judged whether the sex of two consecutively presented faces was the same or different, 3) evident only for low- but not high-pass face images and, 4) only observed when the inputs are face-like in their spatial configuration. Taken together, this evidence indicates that subcortical mechanisms are sensitive to face-like configurations and afford a coarse representation comprised of primarily low spatial frequency information. These representations appear to suffice for some aspects of face perception such as matching faces but not for more complex aspects of face perception such as sex differentiation. Clearly, much research is still required to clarify further the nature of the contribution of the subcortical structures and whether such representations are dependent on cortical computations or contribute independently in some fashion.

Development of face perception

The majority of research investigating developmental changes in the neural organization of the face-processing system has focused on understanding how activation within discrete nodes, of the network change as a function of age. Many studies have investigated age-related changes in the magnitude of the face-related neural response within posterior “core” regions (64-70). These results indicate that the response properties of the neural regions supporting face processing change in the transition from childhood to adolescence as well as between adolescence and early adulthood. For example, unlike adults, young children (aged 5-8 years) do not exhibit consistent group-level face-related activation (71, 72) (see Figure 3). When these regions are defined within individual participants, there is a linear relation between the size/volume of these functional regions and age: a result that has been replicated and extended in other studies (66, 67, 73). This increase in volume of the right FFA is reportedly related to face, but not object, recognition behavior in older adolescents (ages 12-16 years) (67). Furthermore, although young adolescents (11-14 years) evince adult-like topography for face-selective core regions in the right hemisphere, the representational capacity of these regions to encode individual faces is not mature until early adulthood (73). Similarly, there are age-related increases during adolescence in the response profile of the fusiform gyrus during emotional expression processing (74).

Insert Figure 3 approximately here

Among the extended regions, there are consistent findings of age-related changes in childhood and adolescence in the response profile of the amygdala during processing of emotional expressions (e.g., (75, 76). Much less work has investigated developmental changes in the properties of the other extended regions (i.e., vmPFC, PCC, anterior temporal pole). Three studies reported that children or adolescents produced larger signal changes in several of these extended regions during implicit processing of faces (i.e., participants button pressed when the background of an image containing a face changed color, or when any image appeared) (77-79). These findings converge to reveal that activation within the core face-processing regions grow in size with age and become more computationally precise during childhood and adolescence. There is a less clear pattern of results indicating how the properties of the extended regions change with development.

These findings of age-related changes in the functional properties of the nodes within the distributed face-processing network lead to predictions that such changes will likely impact the functional interactions among these regions as well. Connectivity analyses are based on the temporal synchrony of the activation patterns between regions. Therefore, as the response properties of these regions change, it is likely that the functional interactions between the regions will changes as well. However, there are only three studies that have investigated age-related changes in the face-processing system from a network level perspective. We evaluated the relation between changing functional properties of the core face processing regions and changing structural properties in the fiber tracts that connect these core regions with extended regions in a sample of participants ranging in age from 6-23 years (72) (see Figure 4). Using diffusion tensor imaging, we observed selective age-related changes in the volume, fractional anisotropy and mean and radial, but not axial, diffusivities of the inferior longitudinal fasciculus (ILF), along which outputs from the OFA and FFA travel to the anterior temporal lobes and amygdala. Critically, these structural changes were tightly and specifically linked with increasing size of the FFA. In other words, individuals with larger sized FFAs also had thicker ILF tracts, even after age was accounted for. These results reveal the relation between developing functional regions and the structural connections that integrate these regions into a distributed network (80, 81).

Insert Figure 4 approximately here

Two existing studies report age-related changes in the functional organization of the face-processing network. Cohen-Kadosh and colleagues (82) used dynamic causal modelling to evaluate whether 7-11 year olds exhibit the adult profile of functional organization within the core regions as reported in Fairhall & Ishai (17). They found that, like adults, children exhibited a functional connection from OFA->FFA, but that it was weaker than in the adults. In contrast to the adults, children did not exhibit the OFA->pSTS connection. Critically, the children did not exhibit modulation of their network organization as a function of face-processing task (expression versus identification) as did the adults. In other words, the child network was less flexibly responsive to variation in face processing task demands. The authors suggested that the functional connections within the core network may be limited by the continued developmental specialization of the functional properties within each of the discrete regions.

A second study used graph theory metrics to characterize the functional organization among the core and (some of the) extended regions in children and young adolescents (ages 5-12 years) and adults as they passively viewed images of faces and objects (83). The researchers reported age-related changes in the functional topography of the network that were largely focused on the integration of the right OFA and FFA into modules (i.e., densely connected subgroups) within the network. In younger children, the OFA was a weaker node and was clustered with other right temporal lobe regions into a module. In contrast, in the older children, the OFA emerged as a stronger node that was integrated with the FFA into a module. In the adults, the OFA and FFA were segregated into separate modules that were densely connected with other modules (including limbic regions). These are the first results of widespread functional re-organization of the face-processing system in late childhood.

To summarize, rather little is known about developmental changes in the functional organization of the face-processing network. The functional and structural connections in and out of the OFA and FFA are likely changing in late childhood and early adolescence. There is some evidence to suggest that the network of younger individuals does not exhibit the same kind of functional flexibility in response to changing task demands/computational requirements that is seen in the adult network. As a result, there are many open questions about how the neural architecture becomes organized and optimized during development to perform the multifaceted computations that enable one of the most essential set of social skills for humans - face processing.

Breakdown of face perception

Prosopagnosia refers to an individual's inability to recognize faces despite normal sensory vision and normal intelligence. The term has been standardly applied to individuals who were pre-morbidly normal but who, following acquired brain damage, lost the ability to recognize faces. In such cases of acquired prosopagnosia (AP), the lesion is typically to the ventral visual cortex and is sustained during adulthood (for reviews, see (84-86). AP has provided a unique window into the psychological and neural substrate of face processing since its initial recognition (87). The disorder has often been differentiated into an apperceptive and an associative form: while the former is characterized by an inability to form an accurate perceptual representation of the face, the latter is characterized by perception that is relatively intact but the association of the face to other related information is impaired (i.e. name, biographical knowledge etc; (88)). Anatomically, these forms of AP roughly coincide with earlier versus later lesions along a caudal to rostral axis of ventral cortex but drawing clear boundaries between these subtypes has often been challenging (89, 90).

Over the past several years, there has been growing recognition of a deficit, analogous to AP, in which there is an impairment in face processing but in which this occurs in the absence of brain damage. This disorder has been termed ‘congenital prosopagnosia (CP)’ or ‘developmental prosopagnosia’ (DP) with the ‘congenital’ label adopted here to reflect the fact that the disorder is apparently life-long in duration, and occurs in individuals with normal intellectual function and who have had adequate opportunity to acquire normal face recognition skills [for recent review, see (91)]. Critically, the CP individuals show no evidence of damage on conventional MRI (unlike some cases of DP; e.g., (92, 93)).

Several studies, some of them conducted by us, have examined the function of the core face network in CP. Consistently, across these different studies, the activation in each of the core regions (FFA, OFA, pSTS) appeared to be largely normal, as determined by a host of various dependent measures such as the extent of face selectivity, the anatomical location (coordinates of peak activation), the number of activated voxels in each region and the extent of the right lateralization of the face activation (94-97).

While, there are some reports of abnormal activation in the core regions in CP (1, 98-100), to a large extent, even if not entirely, activity in these regions in CP appears comparable to that of the controls. The emergent view from these studies is that the differences between CPs and controls only become apparent when large samples are tested and that these neural differences are subtle and are most evident when correlation with behavior is taken into account. Of note is that such differences in core regions, may not necessarily represent inherent abnormality of these regions, but, rather, might result from abnormal feedback propagating back from the extended face system.

We stress that these findings do not undermine the integral role of core regions such as the FFA in face processing, a finding that is strongly supported by numerous lesion studies (89, 101, 102). Rather, we postulate that these core regions, although necessary, may not be sufficient for successful recognition; consequently, additional regions, as well as the connectivity between the core and extended regions are also involved, as we discuss below.

Several characteristics of CP potentially implicate the extended face system but in a specific fashion: The behavioral impairment in CP is mostly related to the detailed perception/recognition and memory of individual faces (although, of course, the memory deficits might stem from impaired encoding due to the perceptual difficulties) while emotional processing in these individuals is largely intact. This differential behavioral profile predicts a selective disruption in the activation of those parts of the extended network that mediate identity recognition and their related connectivity, while regions mediating emotional expression or other properties of faces should be intact.

To examine this prediction, we explored, in detail, the activation profile in CP of two key regions: the anterior temporal cortex, related to identity representation, on the one hand and the amygdala, involved in emotion processing, on the other hand. In addition, we examined other regions belonging to the cluster of the extended system that is involved in person knowledge such as the precuneus/posterior cingulate and the anterior paracingulate cortex. So far only a few studies have systematically explored these regions and this contrasts with the growing number of studies characterizing the core system in CP.

Using an intensive visual stimulation paradigm, which included blocks of famous, unfamiliar, emotional and neutral faces, we obtained sufficient signal in these extended regions to enable us to characterize extended regions in CP. First, we observe activity in the core system that was largely intact (and see also for related results (1)) (see Figure 5). More novel and intriguing was that, relative to controls, we uncovered abnormal activation and connectivity pattern of the right anterior temporal cortex in CP (97) with intact activation and connectivity to the amygdala (see Figure 6).

Insert Figure 5 approximately here

Insert Figure 6 approximately here

These initial findings regarding the role of anterior temporal cortex in CP are certainly intriguing and warrant further investigation using additional sophisticated and sensitive approaches. For example, Multi Voxel Pattern (MVP) analyses would allow better understanding of the face representation in this region in CP (28) and sophisticated network analysis allows further detailed examination of the network structure in CP (103).

Two other regions that are part of the extended system and are presumably involved in the representation of "person knowledge" are the precuneus/posterior cingulate and the anterior paracingulate cortex regions. Activation of these regions is often observed in studies in which responses to famous vs. unfamiliar faces are contrasted (26, 31). Using a taxing, rapid-event related adaptation paradigm, we have shown that these two regions are not activated in CP individuals in response to famous compared to unfamiliar faces (96). Importantly, this result was obtained while, during the same experiment, CP individuals exhibited activation as well as adaptation in the core face system that was equivalent to that measured in the controls. Furthermore, in both groups, this activation was more pronounced for famous compared to unknown faces, indicating that the lack of activation in these extended regions is not due to the lack of statistical power per se (and see (104) for somewhat different results).

Indeed, in sharp contrast to the absence of activation in regions of the extended network in CP, which are involved in identity representation, during the very same study, the amygdala activation was equivalently robust in CP and controls (97, 104). The dissociation between abnormal activation in identity-related regions and the normal activation of the amygdala uncovers the specificity of the impairment in CP and provides a neural candidate for the observed behavioral dissociation between identity and emotion processing in individuals with this disorder.

A final cortical area of interest that has been occasionally described in the CP literature is the prefrontal cortex. While this region was not explicitly defined originally as part of the extended face network by Haxby and colleagues (26, 105), face-selective activation has been repeatedly documented in this region (16, 106). Notably, in our studies, activation in this prefrontal region was stronger and more bilateral in CPs than in controls (95, 97) (but see (104) for evidence of reduced activation in dorsolateral prefrontal cortex (DLPFC) in CPs). While these findings are intriguing and of potential interest, further research is required in order to understand the exact role of this region in CP. One possible explanation for the enhanced activation found in our studies concerns the involvement of this area in working memory (107, 108) as participants were performing a one-back task. Indeed, despite the relative ease of the task, CP participant exhibited impaired performance during the fMRI scans, particularly during the face conditions and might have recruited working memory representations to a greater degree than was true of the controls (95, 97).

Another potential role of the enhanced prefrontal activation, which is not mutually exclusive with that of working memory, might concern the impaired holistic/configural processing in CP (109, 110). Thus, a possible hypothesis is that DLPFC, especially in the right hemisphere, may be inefficiently engaged in face processing tasks in CP. This may be the case even if holistic processing is not explicitly required, thus leading to enhanced, compensatory activation in this region. Clearly, much research is required in order to determine the validity of this interpretation.

The converging results, stemming from the functional studies described above as well as structural neural investigations (111) have led to the hypothesis that CP does not result from a specific lesion or an alteration in the core face system per se, but rather is the result of an abnormal propagation of (feedforward and/or feedback) information between the core and extended regions. This notion is also consistent with the large body of evidence reviewed above showing that face processing, even in the normal brain, relies on the activity of a face network including cortical as well as subcortical regions, rather than on single regions although, of course, damage to a single region can interrupt the propagation of signals, as well. Furthermore, normal development is apparently accompanied by an emerging connectivity pattern in this network (82, 83).

Intervention and treatment of prosopagnosia

Finally, there is growing attention to the development of possible approaches for improving face perception skills in individuals with congenital prosopagnosia. A number of studies have used cognitive interventions (but see (112) below). The findings are somewhat mixed with some initial studies showing some limited improvement in CP (113, 114). The long-lasting impact and the neural correlates of these interventions, are also largely unknown. Building on their previous success, in a recent study, Degutis et al (115) trained 24 congenital prosopagnosics using an online face-training program targeting holistic face processing. Detailed pre- and post-intervention assessments were conducted. Training resulted in moderate but significant overall training-related improvements on measures of front-view face discrimination in the trained individuals compared with individuals who had not undergone the training. A subset of individuals who reached the more difficult levels of training showed most improvements in front-view face discrimination as well as increased holistic face processing. Interestingly, self-report measures also indicated some improvement.

Together, these results challenge the generally accepted view that prosopagnosia is not remediable and, instead, suggest that carefully designed procedures can yield changes in face perception especially in a subset of individuals.

Other approaches to intervention have begun to explore more pharmacologically-based methods. For example, one recent study employed a randomized placebo-controlled double-blind within-subject experimental design (AB-BA), and each participant took part in two testing sessions, one in which they inhaled placebo and the other in which they inhaled 24 IU of oxytocin (116). Participants performed two tasks, one assessing memory for a set of newly encoded faces, and the other measuring the ability to match simultaneously presented faces according to identity. The prosopagnosic individuals, but not the controls, showed improved performance on both tests in the oxytocin condition, suggesting that oxytocin can improve face processing in congenital prosopagnosia. Developing new potential methods for intervention is critical and a broad review of possible new directions is provided in a recent relevant review (112).

Sidebar 1

Homologs of the distributed face network in humans and non-human primates

Just as there has been considerable progress on understanding the neural basis of face perception in humans, so too has there been advances in understanding the face perception system in non-human primates and comparisons between the species have begun to be conducted (11). The macaque visual system consists of over three dozen different areas specialized for different aspects of vision. Of interest here is that there is a set of six regions in the temporal lobe, the “face patches,” that show greater activation in response to faces compared to non-face objects in fMRI scans (117, 118). These six face-selective regions are strongly and specifically connected to each other, and the regions are functionally distinct. Neurons in the middle lateral and middle fundus face patches are view-specific; neurons in anterior lateral patch are tuned to identity mirror-symmetrically across views, thus achieving partial view invariance; and neurons in anterior medial patch, the most anterior face patch, achieve almost full view invariance (119) [but note some discrepancy in the findings from fMRI in macaque and single unit recording (15), as alluded to above]. A clear comparison between the non-human and human primate neural circuits has not yet been done but some initial investigations indicate that they may not be fully identical (120). For example, in comparing neural activation to static versus dynamic faces, in monkeys, face areas outside of the superior temporal sulcus fundus responded more to facial motion than to general object motion. Human face areas, processing the same stimuli, exhibited specializations for facial motion as well, yet the spatial patterns of facial motion selectivity differed across species, suggesting that facial dynamics are analyzed differently in humans and macaques. As in human imaging, studies have begun to adopt MVPA analytic methods to explore representational selectivity in non-human primates, as well (121). For example, MVPA analyses have uncovered response patterns to individual exemplars in the inferior temporal (IT) cortex, especially area TE and especially the anterior face patches, encoded the animate-inanimate categorical division, with a subordinate cluster of faces within the animate category. This was not true in V4, the amygdala or prefrontal cortex. These results reveal that there are responses in non-human cortical activation that show face-selectivity and within-face exemplar-selectivity.

In addition to characterising the regions that are activated by faces (see sidebar 1), there has also been recent interest in understanding the dynamics of the activation patterns in this region. A recent study studying responses of single units in cortex has revealed that the visual responses of face-selective cells in macaque inferotemporal cortex evince robust responses, showing virtually no change in their patterns over time periods as long as one year. Using chronically implanted microwire electrodes guided by functional MRI targeting, McMahon et al. (122) obtained distinct profiles of selectivity for face and nonface stimuli that served as fingerprints for individual neurons in the anterior fundus (AF) of the superior temporal sulcus. Longitudinal tracking over a series of daily recording sessions revealed that face-selective neurons maintained consistent visual response profiles across months-long time spans despite the influence of ongoing daily experience. These findings led the authors to conclude that neurons in the AF face patch are specialized for aspects of face perception that demand stability as opposed to plasticity.

Conclusion

Face recognition is perhaps the most challenging task confronting the visual system – individual identity must be determined rapidly and precisely and, this process is repeated hundreds or thousands of times over the course of the day. Here, we review recent findings that explore the neural basis of face recognition and we describe the results in several domains including investigations of normal face recognition and of the developmental emergence of face recognition. We also review data from studies of individuals with an impairment in face recognition (‘prosopagnosia’) and we consider the outcome of recent attempts to remediate this impairment. We also briefly describe the homologies between human and non-human face perception and we explore findings related to the functional contribution of subcortical structures to face recognition.

The key conclusion from this review echoes the growing consensus that normal face recognition is accomplished through the concerted activity of a number of cortical regions (‘face patches’). These face patches may contribute somewhat different functional roles to the process and their integrated (structural and functional connectivity) circuitry is critical for normal face perception. This circuit emerges over developmental time and when it is compromised, it results in prosopagnosia. While much remains to be done to understand further the relative contribution of the different cortical regions, much progress has been made and the application of techniques, such as adaptation, and of analytic methods, such as MVPA analysis, has been helpful in this regard.

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Figure captions

Figure 1: A schematic depiction of the experimental apparatus and visual pathways from the eyes to the brain (shown in axial plane). Each monitor provided visual information to a different eye. The visual information first passes through monocularly segregated subcortical regions (left eye-dashed lines right eye – solid lines), which is then projected to the pulvinar, lateral geniculate nucleus (LGN) and superior colliculus en route to the striate and then binocular extrastriate regions. Note that we have excluded the amygdala from this schematic depiction as the focus is on face (and car and letter string) perception rather than on perception of facial emotional expression. For simplicity, we depict only the input from the contralateral eye to each superior colliculus.

Figure 2. (A) A typical different-eye trial in which the first image is presented to the left eye (left column) and the second image is presented to the right eye (right column). The middle column represents the participant's fused perception. A "same" response is required. (B) An example of a face, car, and letter string stimulus in the low-pass filter, original, and high-pass filter condition. (C) An example of face-like and non-face-like images.

Figure 3: Ventral stream category-specific topography within each age group. Contrast maps for each object category ($p < .05$ corrected) from the group-level random-effects GLM mapped onto the ventral projection (a.) and the lateral right hemisphere (b.) of a single representative inflated brain in order to show consistency, or lack thereof, across the age groups in category-selective activation. FFA = fusiform face area, OFA = occipital face area, STS = superior temporal sulcus, LO = lateral occipital object area, PPA = parahippocampal place area. (From (71) with permission).

Figure 4: Age-related differences in the macro- and microstructural properties of the ILF. The volume of both the right (a) and left (e) ILF, as indexed by the mean cubic volume within the fasciculus, increased significantly with age. Similarly, the microstructural properties of the ILF exhibited age-related differences, such that the MD and RD decreased significantly with age in both the right (b and c) and left (f and g) hemispheres. In contrast, the AD was stable across the age range (d and h). This pattern of results suggests that the right and left ILF are becoming increasingly more myelinated with age. (From (72) with permission).

Figure 5: a. Examples of the stimuli used in the visual stimulation experiment. b. Averaged activation maps for controls (left panel) and CPs. The activation maps are overlaid on a group-averaged folded cortical mesh of each group and are presented in a lateral view (top row) and a ventral view (bottom row). The maps for the face activation were obtained by the contrast all faces>buildings (red to yellow colors). Note the similarity of the activation maps across groups in the core face network including bilateral OFA, LOS, FFA, and pSTS. This is in sharp contrast to the activation in anterior temporal cortex in the right hemisphere that is clearly evident in controls but is completely lacking in the CP map. Also shown is the building selective activation obtained from the contrast buildings>all faces (blue to green colors) in the PPA and TOS which is also very similar across groups. The two group maps and both contrasts are presented in the same statistical threshold. Abbreviations: Ant. temp. – anterior temporal cortex. (From (97) with permission).

Figure 6: Activation maps and profiles in anterior temporal cortex and amygdala: a. Activation maps in right anterior temporal cortex obtained for the contrast all faces > buildings; maps are projected on a horizontal slice. Robust activation can be seen in controls (left panel) in the right anterior temporal cortex, while only very weak activation is observed in CPs when applying the same statistical threshold. Note that in the activation map shown in Figure 1b, no activity is evident in this region at the group level in the CP. When examined individually, only three CP individuals exhibited activation in this region and

contributed to the activation profile presented here. b. Activation profiles obtained from anterior temporal cortex in controls (left) and CP (right). c. Activation maps obtained in right amygdala for each group projected on a coronal slice. Given that the maps presented in figure 1b only exhibit cortical activation, averaged activity of the amygdala could not be observed and it is therefore projected on a coronal slice for each group. d. Activation profiles obtained from individually defined right amygdala in each participant in each group. Robust and comparable amygdala activation was found in both groups as evident from the activation maps and profiles. (From (97) with permission).

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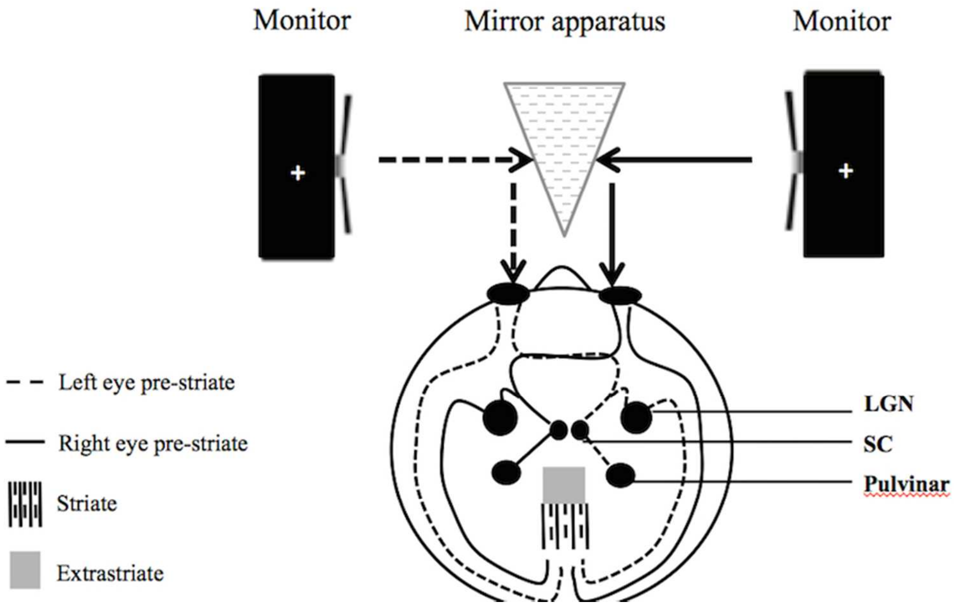

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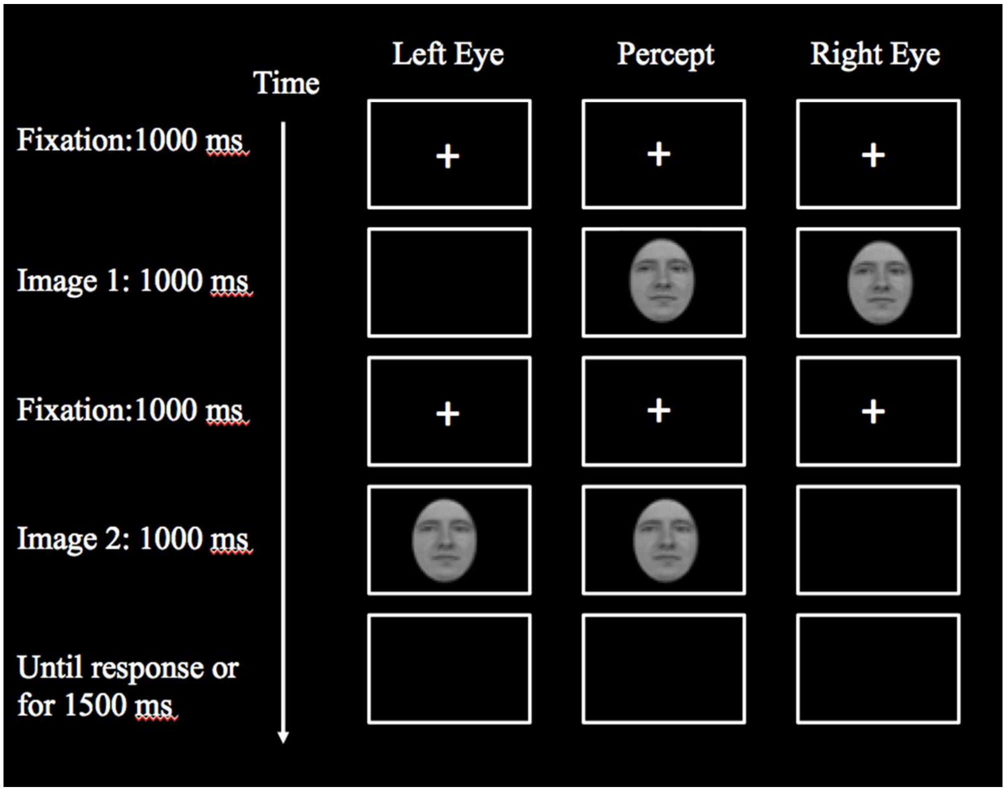

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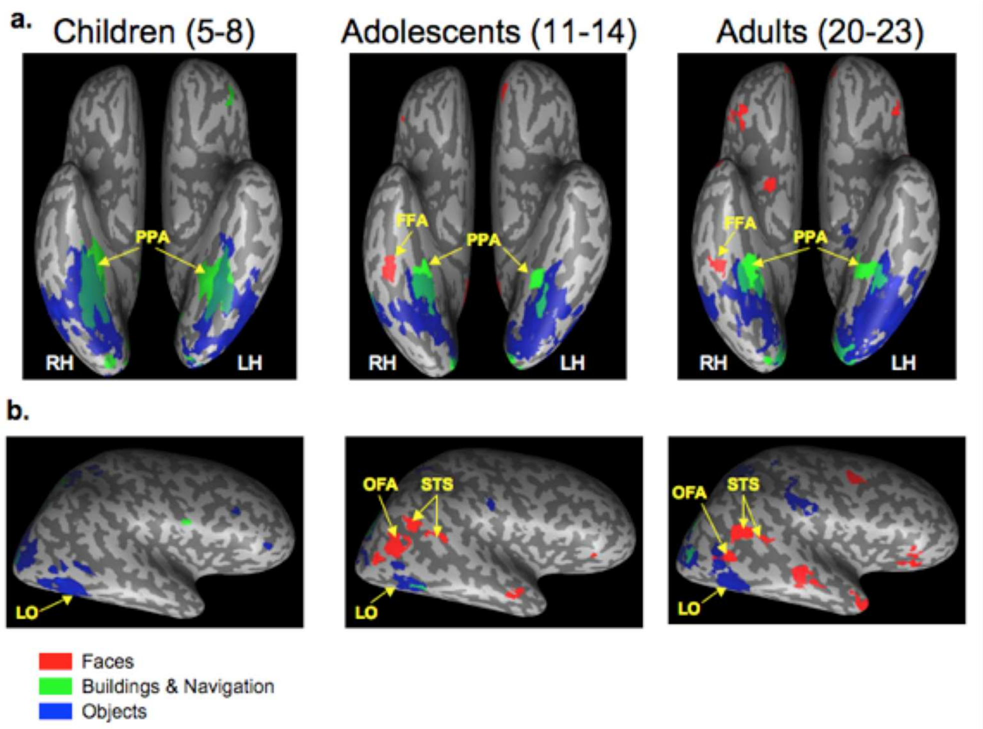

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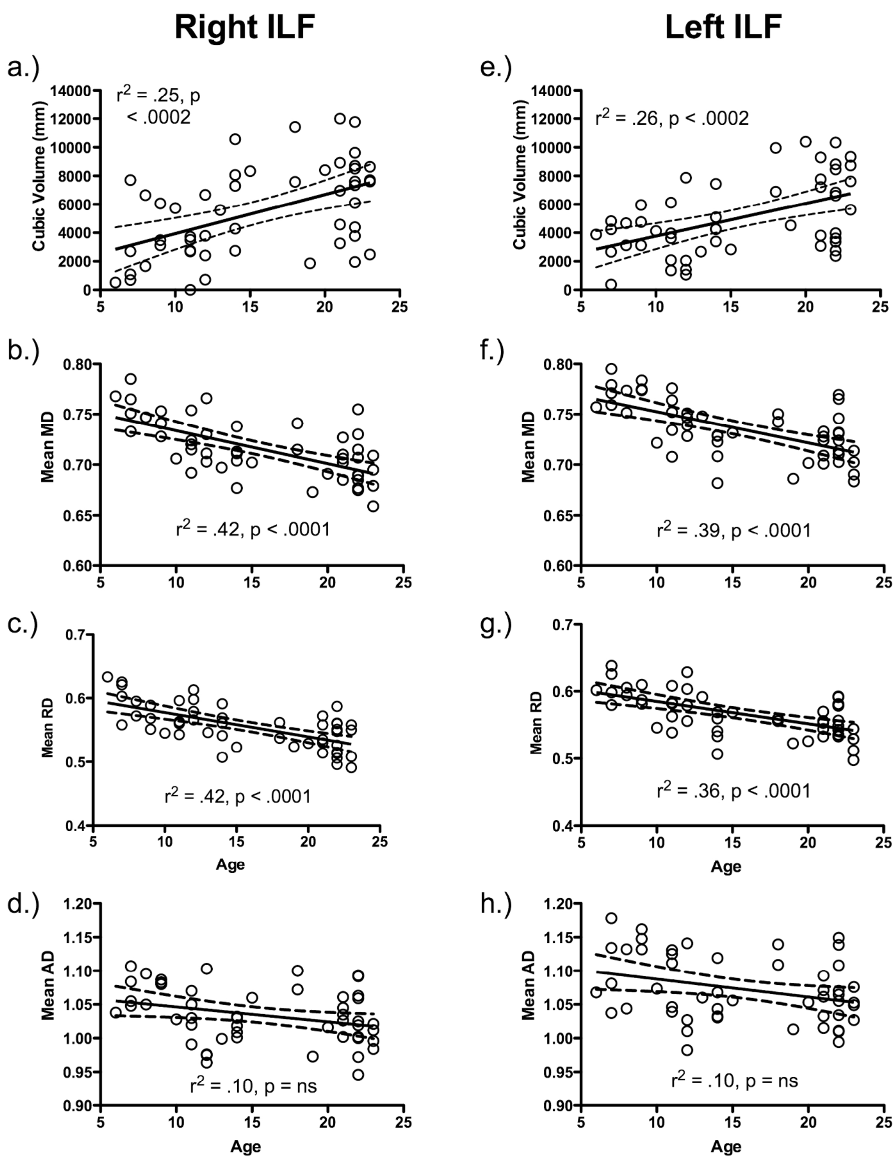

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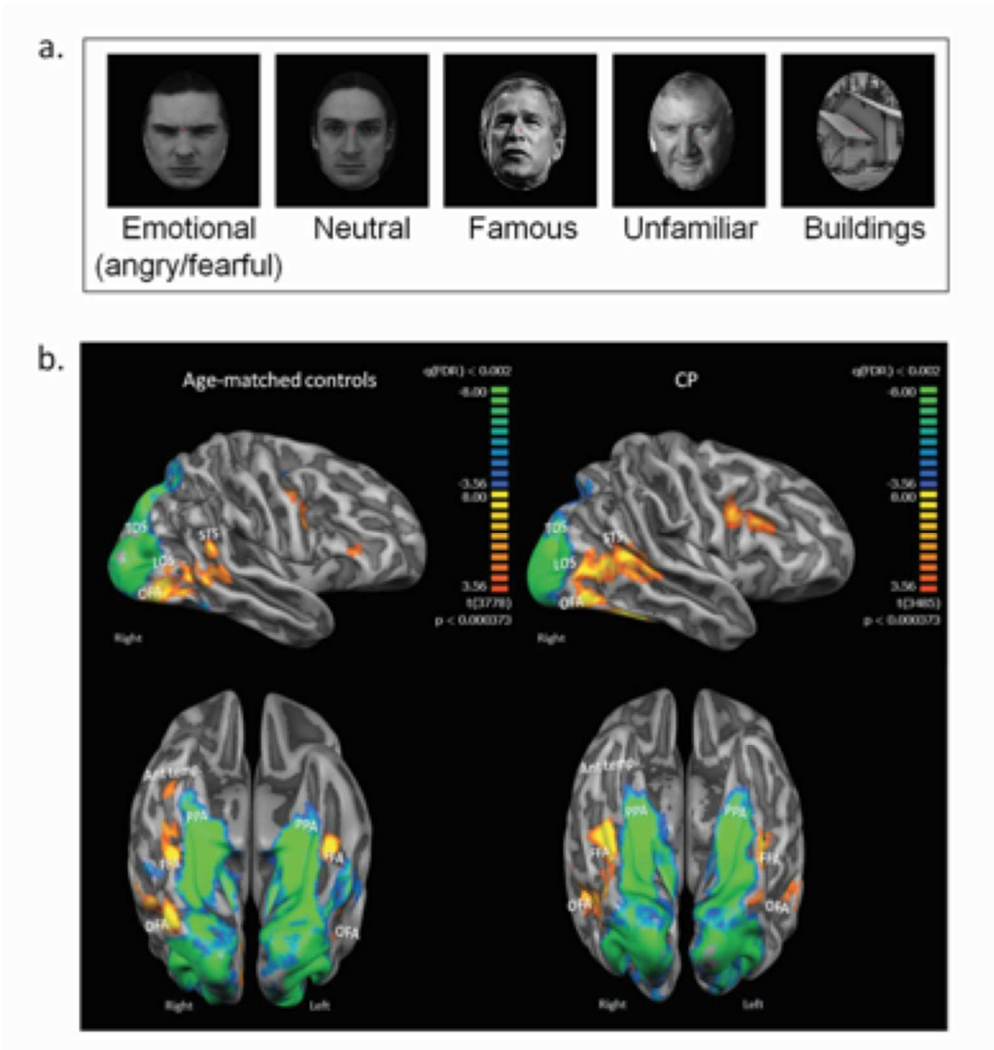

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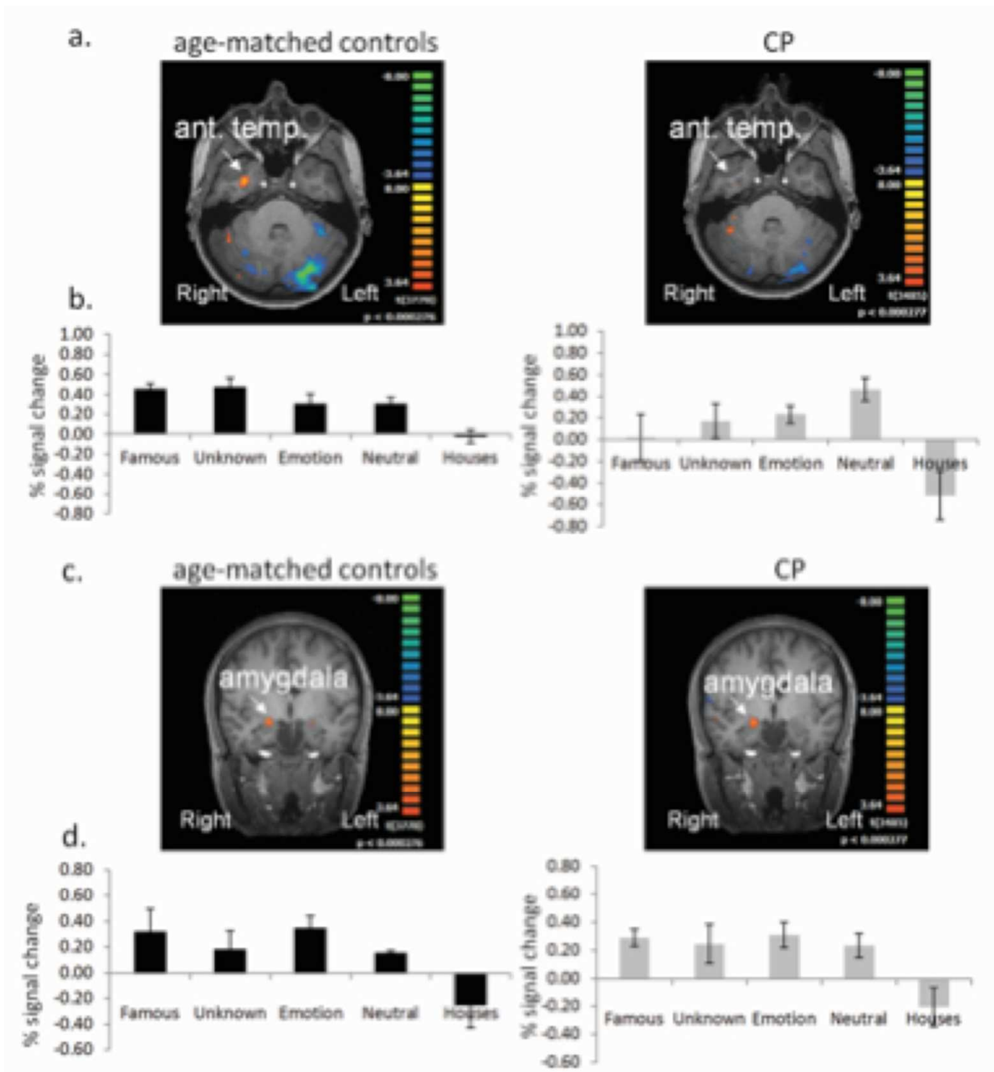

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