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Developmental prosopagnosia: a window to content-specific face processing

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Developmental prosopagnosia is characterized by severely impaired face recognition. Individuals with this disorder, which often runs in families, have no history of brain damage and intact early visual processing systems. Recent research has also demonstrated that many developmental prosopagnosics have normal or relatively good object recognition, indicating that their impairments are not the result of deficits to a unitary visual recognition mechanism. To investigate the nature of the impaired mechanisms, extensive testing was done on an individual with especially pure face processing deficits. The results ruled out all extant explanations of prosopagnosia except one that proposed that faces are recognized by a content-specific face processing mechanism. fMRI and MEG studies show that there are a variety of neural profiles in developmental prosopagnosia, which is consistent with behavioral studies demonstrating that it is a heterogeneous disorder.

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Introduction

In a landmark paper, Bodamer [1] coined the term ‘prosopagnosia’ to describe the selective degradation of face perception and face memory in three individuals suffering from brain damage. Since the work of Bodamer, a small yet steady trickle of reported cases of acquired prosopagnosia has sustained the notion that human recognition of faces is carried out by content-specific (also called domain-specific) mechanisms that are specialized for face processing. Support for this idea also comes from an extraordinary condition that displays this is the opposite pattern from prosopagnosia, that is, object agnosia with normal face recognition [2,3]. The existence of face-specific processing mechanisms is consistent both with

the presence of cells selectively tuned to faces in the temporal lobes of monkeys [4••] and humans [5] and with findings that specific regions in the human temporal lobes become active when presented with facial images [6].

Prosopagnosia can be a devastating loss to patients, because face recognition is an integral part of our social lives. Its onset is usually dramatic, because the afflicted person becomes painfully aware that something so effortlessly achieved in the past is no longer possible.

Although there are a number of documented cases of individuals who acquire prosopagnosia during their lifetime, it appears that the number who have developmental prosopagnosia (DP), that is, individuals who never acquired the ability to recognize faces, might be much larger. Developmental prosopagnosics (DPs) have not suffered any obvious brain damage, yet they have deficits in face recognition that can be as severe and as selective as those seen in acquired prosopagnosics. Until recently, it appeared to be a very rare condition, but a number of research groups have tested DPs in recent years [7,8,9•,10], and our website has been contacted by more than 500 self-diagnosed DPs (<http://www.faceblind.org>).

For example, JK, a woman in her early 30s who has recently completed her Ph.D., discusses a recent episode. “This week I went to the wrong baby at my son’s daycare and only realized that he was not my son when the entire daycare staff looked at me in horrified disbelief”. Social embarrassment and consequent social isolation are common. “A few years ago I introduced myself to a woman at a wedding only to find that I knew her quite well... she worked in the lab down the hall and we frequently had lunch and chatted together in the lounge. She, incidentally, has never spoken to me again”. Interestingly, JK’s sister and father also have face recognition deficits.

Despite these dramatic difficulties associated with developmental prosopagnosia, the existence of and the nature of their impairment is often not evident to those with DP. Having no obvious standards of comparison, those who become aware of their deficits do so gradually in adolescence and adulthood. In addition, many cope as best as they can by relying on hairstyles, clothing, context and voices.

In this review, we only discuss individuals who have no apparent brain damage. We do not discuss individuals who suffered brain damage as children, and who are sometimes categorized as developmental prosopagnosics

[11,12]. We expect that this group of subjects will be an important source of information about face recognition and its development, but they are likely to have a different profile to those prosopagnosics without brain damage.

Because a number of research groups are now working with DPs, there has been a substantial increase in the number of papers on DP. They have addressed the cognitive, neural and developmental basis of the condition. Here, we discuss these results and their contribution to our understanding of DP and face processing more generally.

Developmental prosopagnosia and inferences to functional organization

During the past several years, DP has been investigated at a number of different levels, but the most fruitful work has addressed the cognitive basis of the disorder. A correct cognitive description of DP is undoubtedly a crucial step in our understanding of the condition, but we believe it will also contribute importantly to theories of normal face processing. Although some have argued that developmental disorders are unlikely to shed light on normal functional organization [13], we believe that some conditions are likely to be informative. We appreciate that many developmental disorders result from widespread problems (e.g. neurotransmitter dysfunction, deficits in long distance connections), which will impair many functional systems. We also expect, however, that some disorders result from failures to develop the specific circuitry necessary for particular computations correctly.

We conceptualize developmental disorders on a continuum ranging from those with widespread cognitive effects to those with more specific effects. Autism [14], William's syndrome [15] and Down's syndrome [16] are on the widespread end of this continuum, whereas developmental conditions such as dyslexia [17], dyscalculia [18] and prosopagnosia are on the specific end. It is difficult to make inferences about normal functional organization from conditions with widespread effects, but conditions with local effects can reveal a lot about functional organization. Regardless of the outcome of this debate, evidence from DP is likely to play a crucial role in it, and we hope that it will provide a model for the study of other specific developmental disorders.

Demonstrating that prosopagnosia and agnosia are separate conditions

Prosopagnosia is characterized by impaired face recognition, but it is often accompanied by object agnosia [9^o,19]. Prosopagnosia and agnosia could stem from deficits to the same mechanisms, and if this were the case, there would be no theoretical reason to differentiate between prosopagnosia and agnosia. Gauthier, Behrmann, and Tarr [20] were the most recent to take this position. They pointed

out that nearly all past reports of dissociations between impaired face and normal object recognition relied exclusively on accuracy measures. Failure to collect response times leaves open the possibility that prosopagnosics achieved normal accuracy on non-face object tests by trading speed for accuracy, so accuracy measures alone might mask actual deficits. Early reports of developmental prosopagnosia indicated that face recognition could be selectively, or at least relatively selectively, impaired compared with object recognition [21,22], but response times were not collected so these dissociations were not definitive.

To investigate whether face and object recognition are truly dissociable in DP, we tested seven prosopagnosics who reported lifelong problems with face recognition [9^o]. Their poor performance on face memory tests demonstrated that they were severely prosopagnosic, and we next tested them with a battery of old–new discriminations requiring recognition of exemplars within a class. We compared performance on faces with performance on cars, tools, guns, horses, houses and natural landscapes. As expected, all seven subjects performed well below the normal range with faces. However, prosopagnosics scored in the normal range on the majority of the tests, with a number of subjects scoring in the normal range on all, or nearly all, of the non-face tasks. Crucially, inspection of the response times showed that speed–accuracy trade-offs could not account for accuracy dissociations. Hence, although some cases of developmental prosopagnosia are accompanied by developmental agnosia, other cases demonstrate the existence of prosopagnosia with normal recognition of objects.

Impairments in developmental prosopagnosia: face-specific or general purpose?

The previous results demonstrate that deficits to an assumed unitary visual recognition system cannot account for developmental prosopagnosia. More recent work has focused on the mechanisms that failed to develop in DP. In particular, these studies examined whether faces are handled by face-specific mechanisms or mechanisms used with a wider range of object classes, such as mechanisms for processing the precise spatial configuration of the features of an object or processing of object classes for which an individual has expertise. Evidence from neuropsychology [2,23], cognitive experiments with normal subjects [24–26], neuroimaging [6,27] and neurophysiology [4^o,28] has indicated that face-specific processing mechanisms exist, however, other evidence has been taken to support alternative theories [29–31]. Recent studies of DP have examined predictions made by different theories of face recognition. However, face recognition appears to involve a number of hierarchical stages [32] and a variety of parallel processes [33^o]. Therefore, prosopagnosia is likely to be a heterogeneous disorder,

and in fact different cases have demonstrated different types of impairments [8,9,34]. As a result, if evidence from one case of developmental prosopagnosia is inconsistent with a certain theory, it does not mean that the theory should be discounted, because that same theory could be correct in another case of prosopagnosia [33].

Behavioral experiments with normal participants demonstrate that upright face processing involves a representation that more precisely encodes the configuration of the parts (e.g., distance between the eyes, distance from the nose to the mouth) than other types of object recognition [26,35]. Two recent experiments have investigated whether deficits in general-purpose configural processing mechanisms account for developmental prosopagnosia. Behrmann and co-workers [7] used the Navon task to investigate non-face configural processing in five DPs. Compound stimuli were presented in the task consisting of a global letter created by the configuration of small letters (e.g. a large 'S' made of small 'H's) [36]. The compound stimuli were consistent when the large and small letters were the same and inconsistent when they were different. Subjects attended to either the global letter or the local letter depending on the block, and made keypress responses as quickly as possible to indicate whether an S or an H was presented. Controls' response times for global discriminations and local discriminations were nearly identical, whereas some of the DPs responded more slowly to the global letters than the local letters. In addition, DPs showed more interference from local letters on inconsistent global trials than controls did [7]. These results provide some support for the idea that some DPs might have deficits with configural processing across a range of object classes. However, some DPs performed normally in the Navon task, and all showed only minor slowing with the global discriminations. In addition, we have tested 10 DPs with a nearly identical task, and they have not shown any deficits in global processing.

The association between prosopagnosia and slower global processing is consistent with a general configural processing impairment, but Yovel and Duchaine [37] found conflicting results. Subjects were tested with two parallel same-different tasks, one with faces and one with houses. On different trials, faces and houses differed either in the configuration of the parts or in the shape of the parts. Configural differences in the faces were created by manipulating the distance between the two eyes and the distance between the nose and the mouth. Similarly, house configural items differed in the spacing of the door and windows. Importantly, the features themselves were identical when the configural information was varied. By contrast, feature differences were created by pasting different eyes and mouths in the faces and different doors and windows in the houses. Although the features were changed, their spacing did not change. DPs performed poorly with face configural items as expected, but they

also performed poorly with the face feature items. By contrast, the DPs had normal performance on house configural and the house feature items, and their house performance suggests that DP does not result from a general impairment with configural processing. Furthermore, their impaired performance with the face feature items suggests that their problems with faces are not limited to face configural information but apply more generally to facial information.

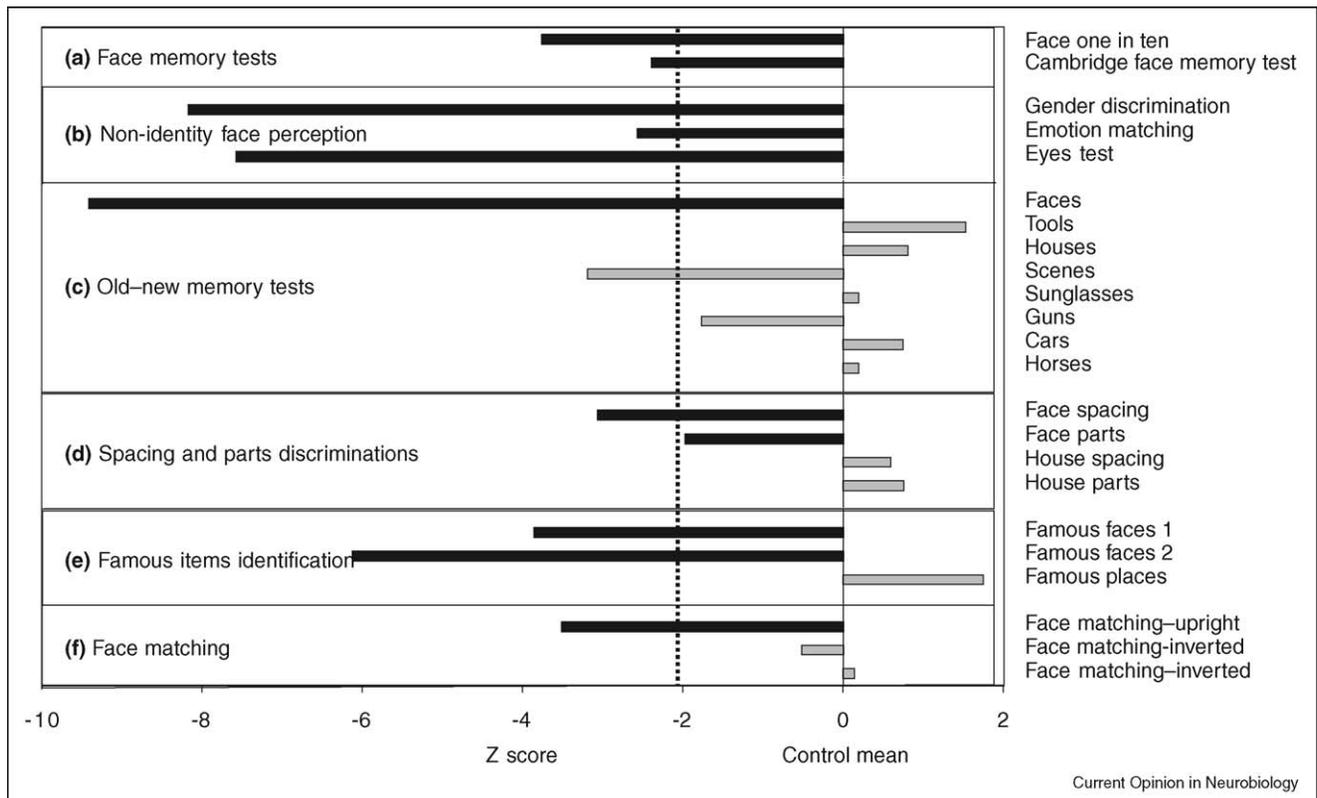
Systematic testing implicates deficits to content-specific mechanisms

The experiments discussed above shed light on the nature of the impaired face processing mechanisms in DP, but they do not enable us to infer that a particular explanation is the best account of DP. Many explanations of prosopagnosia have been proposed, and previous studies have addressed only a few explanations. To assess which explanation best accounts for an individual case, the predictions of each explanation must be tested. In addition, this testing must be carried out exhaustively with an individual prosopagnosic, because, as discussed above, the basis of DP might be different in different prosopagnosics, so ruling out an account in one subject does not rule it out in another. If extensive testing with a prosopagnosic rules out all explanations except one, the remaining explanation would appear to be the correct one for that DP.

Recently we tested the predictions of all extant accounts of prosopagnosia with Edward, an individual with severe developmental prosopagnosia [33,38]. The results were inconsistent with all except the face-specific account, which proposes that faces are represented by mechanisms specialized for specific visual content, not specific visual processing. This content-specific account proposes that the content for face-specific processes is upright faces. Edward's performance on the tasks testing the predictions of the explanations is presented in Figure 1 and Figure 2.

Figure 1 shows that Edward did very poorly on tests involving face memory, face gender discrimination, and facial expression recognition. By contrast, he did well on object memory tasks using a wide range of classes. On the configural and featural same-different tasks with faces and houses, Edward did poorly with both tasks involving faces but normally with both house tasks. Edward was also tested on a task requiring face matching across viewpoint changes. With upright faces, Edward performed much more poorly than controls, but his score with inverted faces was normal. Most strikingly, Edward's percent correct with upright faces was nearly identical to his percent correct for inverted faces. All controls were substantially worse when faces were inverted. Edward's similar performance with upright and inverted faces suggests that he processes both in the same manner.

Figure 1



A summary of Edward's face and non-face test results [33**]. Results are presented as z scores, and the dashed line was placed two standard deviations below the mean, a traditional cut off in neuropsychology. Scores from tests involving upright faces are black, whereas other test scores are gray. Parallel tests are grouped together so that the dissociations between Edward's impaired face processing and normal non-face processing are evident.

Figure 2 displays data comparing the performance of Edward with that of the controls on a task using greebles [33**,38*]. Greebles are an artificial stimulus class designed to place similar demands on visual recognition as faces. It has been claimed that extended training with greebles leads to activation of expert recognition processes and that these same processes are also used for face recognition [39]. This hypothesis predicts that DPs should show deficits when trained in a standard greeble training procedure. However, Edward performed normally throughout a typical greeble training paradigm [33**,38*], so problems with expertise do not account for his prosopagnosia.

The poor performance of Edward with different types of face processing (identity, emotion, gender, etc.) suggests that he failed to develop a mechanism early in the face-processing stream that typically represents the face for a wide range of face processing tasks. His neural response to faces is consistent with an impairment early in the stream. Most DPs show face-selective voxels, but in two fMRI sessions with Edward, we failed to find any voxels that responded more strongly to faces than objects despite

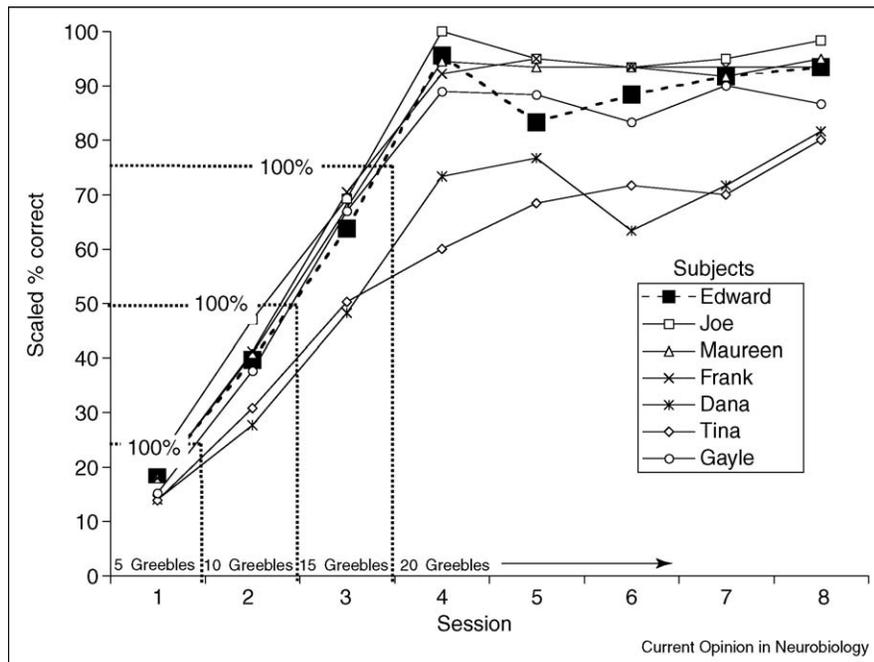
finding normal selectivity for objects, bodies, and places [33**].

Edward's dissociation between face and object processing is not only a functional dissociation but also a developmental dissociation, because he appears to have developed normal object mechanisms without normal face mechanisms. His developmental dissociation suggests that the development of face processing mechanisms relies, at least in part, on different developmental processes than object processing does.

Investigating the architecture of face processing through developmental prosopagnosia

Research into the cognitive and neural basis of developmental prosopagnosia has benefited from the models of normal face processing developed during the past twenty years. In turn, we think developmental prosopagnosia provides an opportunity to corroborate and further refine these models, because DPs report considerable variability within face processing. The classic model of Bruce and Young [32] proposes that a number of independent

Figure 2



Performance of Edward and controls on individual verification trials during greeble training [33^{**},38^{*}]. During the training subjects learned the names of individual greebles. To reflect the fact that subjects learned 5 of the 20 greebles in each of the first four sessions, we scaled the percent correct so that the maximum percent correct was equivalent to the percentage of greebles that had been introduced. Contrary to the predictions of the expertise hypothesis [39], Edward was as good as or better than the controls. His performance was also normal for the other two trial types used to assess greeble knowledge.

modules are involved in face processing, with some operating serially and others operating in parallel. The model proposes that upright face processing is carried out in a hierarchical fashion by a number of relatively independent units, each of which performs specific computations. Our working model (Figure 3) inspired by the effort of Bruce and Young shows candidate stages involved in face recognition: face detection, structural encoding and face memory. Edward detects faces normally and, similar to control subjects, he detects faces better when they are upright than when they are inverted. However, he does very badly with tasks requiring structural encoding and those requiring face memory, and he does no better with upright faces than inverted faces on these tasks [33^{**}]. As a result, he appears to be impaired in structural encoding. The serial nature of this model predicts that impairment at a particular stage will produce a deficit at that stage and at all later stages. Edward's results are consistent with this prediction, but results from more participants are needed to properly test this hypothesis.

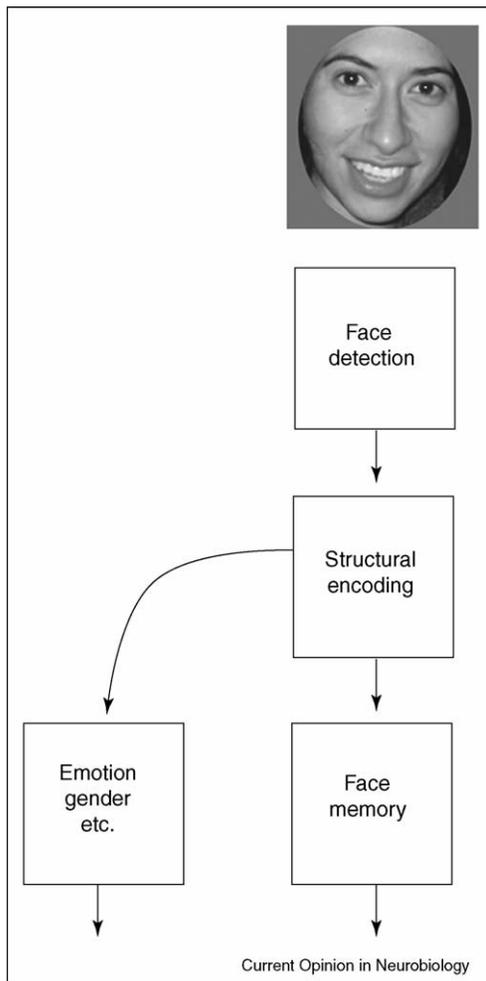
This model holds that facial identity and facial expression are computed by separate modules, though these might rely on common early mechanisms. Some DPs have impaired emotion recognition [33^{**},40,41], whereas others have normal emotion recognition [21,22,42]. However, emotion recognition was incidental to these papers,

so they often included only anecdotes or limited emotion recognition tests. A recent report of a DP directly addressed whether identity and emotion are dissociable, and the results show a clear dissociation [43]. This subject was impaired on five tests of identity recognition, yet she scored normally on four tests of emotion recognition. Tellingly, she reported that she regularly recognizes identity on the basis of characteristic facial expressions. On the basis of testing done with other DPs and self-reports from DPs, we expect that many DPs can recognize emotions normally. It is currently unknown whether other face processing skills, such as gender discrimination, trustworthiness judgments, age estimation and attractiveness judgments, are also dissociable from face recognition and from other face processing abilities.

Neural basis of developmental prosopagnosia

The neural basis of developmental prosopagnosia has recently been investigated with both fMRI and MEG. It is now well established that neurotypical subjects have an area in fusiform gyrus that responds much more strongly to faces than it does to other objects. This area is often called the fusiform face area (FFA), and Avidan *et al.* [44^{*}] and Hasson *et al.* [45] used fMRI to investigate whether DPs showed an atypical FFA. Surprisingly, they found no difference in the FFA between the DPs and the

Figure 3



Simplified version of the Bruce and Young [32] model of face recognition with the addition of a face detection stage. A face is first detected by a specialized module. At the next stage, a mechanism represents the structure of the face. Finally, this representation is matched to a memory representation in the final stage. The box on the left indicates that the perceptual representation is also used for other face processing tasks, such as expression recognition and gender discrimination.

controls. In addition, Avidan *et al.* [44^{*}] also showed that, similar to normal subjects, the FFAs of their four DPs showed a diminished response when repeatedly shown the same face. By contrast, a recent paper that found that an acquired prosopagnosic failed to show a diminished response when the same face was repeatedly shown [46]. As mentioned above, Edward failed to show any face-selective voxels, so there appears to be significant variability in the neural basis of DP. Understanding the behavioral correlates of the different neural profiles in DP is clearly a major outstanding issue.

Neural heterogeneity in DP was also found in a recent experiment using MEG [34]. Neurotypical subjects show a component called the M170 approximately 170

milliseconds after stimulus presentation that responds more strongly to faces than it does to non-faces [47]. In a study examining the M170s of five DPs [34], all controls and two DPs showed greater amplitude to faces when compared with that to houses. However, similar to previous experiments using evoked response potentials (ERPs) [21,48], three DPs did not show a differential response to faces [34].

Developmental course and the genetic basis of developmental prosopagnosia

Currently, little is known about the developmental course of DP. The few studies conducted in children with DP suggest that the deficit is apparent early in life [42]. Our laboratory has been contacted by many parents who are concerned that their young children might have prosopagnosia, and many adult DPs report that their difficulties with faces started very early on in childhood.

De Gelder and Stekelenburg [8] recently investigated whether DP might originate in defective subcortical mechanisms. One of the leading models of the development of face recognition suggests that infants attend to faces because of a subcortical mechanism that directs attention to faces [49]. This attention to faces is hypothesized to lead previously unspecialized areas in posterior cortex to become specialized for face processing. According to this account, infants whose subcortical mechanism is defective will not attend to faces, and hence will not develop specialized processing for faces. Recent research suggests that a subcortical mechanism processes facial information even in adults [50,51], and de Gelder and Stekelenburg found evidence that ERPs detect the operation of this mechanism [49]. In particular, they showed that under monocular viewing conditions normal subjects show a stronger N170 (the ERP equivalent of the MEG M170) when faces are presented in the temporal hemifield than in the nasal hemifield. This asymmetry is believed to occur because the nasal hemiretina, which processes the temporal hemifield, has greater connections to subcortical areas. Intriguingly, a DP failed to show the normal pattern, which suggests that their subcortical pathways for face processing are not operating properly, and thus might not have been operating properly during infancy [49].

Over the years, DPs have often reported genetic relatives who share their impairment [21,40,52], and recent papers have reported more DPs with affected relatives [8,9^{*},41]. Even more interesting was a recent paper that presented self-report data from 38 DPs from seven families [10]. They suggested that the segregation pattern found is consistent with that of a dominant autosomal gene. Clearly, much work needs to be done to understand familial prosopagnosia, but the condition is a very promising model to study the connection between genes and neurocognitive mechanisms.

Conclusions

Recent work with DPs has made significant advances. We now have much stronger evidence than before that DP is a disorder that sometimes results from deficits with content-specific mechanisms specialized for processing faces. Qualitative differences in the cognitive and neural basis of DP in different DPs have shown that the condition has multiple types. However, work on DP has only begun to scratch the surface, and DP appears likely to provide insight into a number of key cognitive, neural, developmental and genetic issues. Recently, researchers have usually approached these issues individually. However, to develop a comprehensive understanding of DP, it will be necessary to tie these different levels of explanation together. Such unification will be challenging, but our relatively sophisticated understanding of face processing and the large population of DPs interested in research participation makes this exciting possibility a tractable task.

Acknowledgments

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References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

1. Bodamer J: **Die prosopagnosie.** *Archiv für Psychiatrie und Nervenkrankheiten* 1947, **179**:6-53.
2. Moscovitch M, Winocur G, Behrmann M: **What is special about face recognition? Nineteen experiments on a person with visual object agnosia and dyslexia but normal face recognition.** *J Cogn Neurosci* 1997, **9**:555-604.
3. McMullen P, Fisk J, Phillips S: **Apperceptive agnosia and face recognition.** *Neurocase* 2000, **6**:403-414.
4. Tsao DY, Freiwald WA, Tootell R, Livingstone MS: **A cortical region consisting entirely of face-selective cells.** *Science* 2006, **311**:670-674.
5. Kreiman G, Koch C, Fried I: **Category-specific visual responses of single neurons in the human medial temporal lobe.** *Nat Neurosci* 2000, **3**:946-953.
6. Kanwisher N, McDermott J, Chun M: **The fusiform face area: a module in human extrastriate cortex specialized for face perception.** *J Neurosci* 1997, **17**:4302-4311.
7. Behrmann M, Avidan G, Marotta JJ, Kimchi R: **Detailed exploration of face-related processing in congenital prosopagnosia: 1. Behavioral findings.** *J Cogn Neurosci* 2005, **17**:1130-1149.
8. de Gelder B, Stekelenburg J: **Naso-temporal asymmetry of the N170 for processing faces in normal viewers but not in developmental prosopagnosia.** *Neurosci Lett* 2005, **376**:40-45.
9. Duchaine B, Nakayama K: **Dissociations of face and object recognition in developmental prosopagnosia.** *J Cogn Neurosci* 2005, **17**:249-261.
10. Grueter M, Grueter T, Bell V, Horst J, Laskowski W, Sperling K, Halligan P, Ellis H, Kennerknecht I: **Hereditary prosopagnosia: the first case series.** *Cortex* in press.
11. Thomas M, Karmiloff-Smith A: **Are developmental disorders like cases of adult brain damage? Implications from connectionist modelling.** *Behav Brain Sci* 2002, **25**:727-750.
12. Frith U, Happe F: **Autism spectrum disorder.** *Curr Biol* 2005, **15**:R786-R790.
13. Barton JJ, Cherkasova MV, Press DZ, Intrilligator JM, O'Connor M: **Developmental prosopagnosia: a study of three patients.** *Brain Cogn* 2003, **51**:12-30.
14. Michelon P, Biederman I: **Less impairment in face imagery than face perception in early prosopagnosia.** *Neuropsychologia* 2003, **41**:421-441.
15. Galaburda AM, Duchaine BC: **Developmental disorders of vision.** *Neurol Clin* 2003, **21**:687-707.
16. Teipel SJ, Alexander GE, Schapiro MB, Moller HJ, Rapoport SI, Hampel H: **Age-related cortical gray matter reductions in non-demented down's syndrome adults determined by MRI and voxel-based morphometry.** *Brain* 2004, **127**:811-824.
17. Ramus F, Rosen S, Dakin S, Day B, Castellote J, White S, Frith U: **Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults.** *Brain* 2003, **126**:841-865.
18. Landerl K, Bevan A, Butterworth B: **Developmental dyscalculia and basic numerical capacities: a study of 8-9-year-old students.** *Cognition* 2004, **93**:99-125.
19. Damasio AR, Damasio H, Van Hoesen GW: **Prosopagnosia: anatomic basis and behavioral mechanisms.** *Neurology* 1982, **32**:331-341.
20. Gauthier I, Behrmann M, Tarr M: **Can face recognition really be dissociated from object recognition?** *J Cogn Neurosci* 1999, **11**:349-370.
21. Bentin S, Deouell L, Soroker N: **Selective streaming of visual information in face recognition: evidence from congenital prosopagnosia.** *Neuroreport* 1999, **10**:823-827.
22. Nunn J, Postma P, Pearson R: **Developmental prosopagnosia: should it be taken at face value?** *Neurocase* 2001, **7**:15-27.
23. Farah M: **Is face recognition "special"? Evidence from neuropsychology.** *Behav Brain Res* 1996, **76**:181-189.
24. Tanaka J, Farah M: **Parts and wholes in face recognition.** *Q J Exp Psychol A* 1993, **46**:225-245.
25. Yin R: **Looking at upside-down faces.** *J Exp Psychol* 1969, **81**:141-145.
26. Young A, Hellawell D, Hay D: **Configurational information in face perception.** *Perception* 1987, **16**:747-759.
27. McCarthy G, Puce A, Gore JC, Allison T: **Face-specific processing in the human fusiform gyrus.** *J Cogn Neurosci* 1997, **9**:605-610.
28. Perrett D, Rolls E, Caan W: **Visual neurones responsive to faces in monkey temporal cortex.** *Exp Brain Res* 1982, **47**:329-342.
29. de Gelder B, Bachoud-Levi A, Degos J: **Inversion superiority in visual agnosia may be common to a variety of orientation polarized objects besides faces.** *Vision Res* 1998, **38**:2855-2861.
30. Diamond R, Carey S: **Why faces are and are not special: an effect of expertise.** *J Exp Psychol Gen* 1986, **115**:107-117.
31. Tarr M, Gauthier I: **FFA: a flexible fusiform area for subordinate-level visual processing automatized by expertise.** *Nat Neurosci* 2000, **3**:764-769.
32. Bruce V, Young AW: **Understanding face recognition.** *Br J Psychol* 1986, **77**:305-327.

33. Duchaine B, Yovel G, Butterworth E, Nakayama K:

- **Prosopagnosia as an impairment to face-specific mechanisms: elimination of the alternative hypotheses in a developmental case.** *Cogn Neuropsychol* in press.

The authors present a systematic study that tests the predictions of all extant alternative explanations to the face-specific explanation of prosopagnosia. Results were inconsistent with all the explanations and, therefore, implicated impairment to a domain-specific face processing mechanism.

34. Harris A, Duchaine B, Nakayama K: **Normal and abnormal face selectivity of the M170 response in developmental prosopagnosics.** *Neuropsychologia* 2005, **43**:2125-2136.

35. Freire A, Lee K, Symons L: **The face-inversion effect as a deficit in encoding of configural information: direct evidence.** *Perception* 2000, **29**:159-170.

36. Navon D: **Forest before trees: precedence of global features in visual-perception.** *Cognit Psychol* 1977, **9**:353-383.

37. Yovel G, Duchaine B: **Specialized face perception mechanisms extract both part and spacing information from faces: evidence from developmental prosopagnosia.** *J Cogn Neurosci* in press.

The authors used tasks that required DPs to decide whether sequentially presented pairs of faces or houses were the same or different. Faces and houses differed in either the spacing of parts or the parts themselves. Prosopagnosics were impaired in both face spacing and face part discriminations, but were normal with both types of house discrimination.

38. Duchaine B, Dingle K, Butterworth E, Nakayama K: **Normal greble learning in a severe case of developmental prosopagnosia.** *Neuron* 2004, **43**:469-473.

The authors tested the claim that the same mechanisms process faces and other objects for which individuals have expertise. This theory predicts that prosopagnosics will be impaired on tasks requiring expertise, but this DP subject had no difficulty with training claimed to require expertise.

39. Gauthier I, Williams P, Tarr M, Tanaka J: **Training "greble" experts: a framework for studying expert object recognition processes.** *Vision Res* 1998, **38**:2401-2428.

40. de Haan EH, Campbell R: **A fifteen year follow-up of a case of developmental prosopagnosia.** *Cortex* 1991, **27**:489-509.

41. Duchaine B: **Developmental prosopagnosia with normal configural processing.** *Neuroreport* 2000, **11**:79-83.

42. Jones R, Tranel D: **Severe developmental prosopagnosia in a child with superior intellect.** *J Clin Exp Neuropsychol* 2001, **23**:265-273.

43. Duchaine B, Parker H, Nakayama K: **Normal emotion recognition in a prosopagnosic.** *Perception* 2003, **32**:827-838.

44. Avidan G, Hasson U, Malach R, Behrmann M: **Detailed exploration of face-related processing in congenital prosopagnosia: 2. Functional neuroimaging.** *J Cogn Neurosci* 2005, **17**:1150-1167.

The authors present fMRI evidence that five DPs had normal FFAs and normal FFA adaptation.

45. Hasson U, Avidan G, Deouell L, Bentin S, Malach R: **Face-selective activation in a congenital prosopagnosic subject.** *J Cogn Neurosci* 2003, **15**:419-431.

46. Schiltz C, Sorger B, Caldara R, Ahmed F, Mayer E, Goebel R, Rossion B: **Impaired face discrimination in acquired prosopagnosia is associated with abnormal response to individual faces in right middle fusiform.** *Cereb Cortex* 2005, Advance access.

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

48. Kress T, Daum I: **Event-related potentials reflect impaired face recognition in patients with congenital prosopagnosia.** *Neurosci Lett* 2003, **352**:133-136.

49. Morton J, Johnson M: **Conspic and Conlern: a two-process theory of infant face recognition.** *Psychol Rev* 1991, **98**:164-181.

50. de Gelder B, Vroomen J, Pourtois G, Weiskrantz L: **Non-conscious recognition of affect in the absence of striate cortex.** *Neuroreport* 1999, **10**:3759-3763.

51. Vuilleumier P, Armony J, Driver J, Dolan R: **Distinct spatial frequency sensitivities for processing faces and emotional expressions.** *Nat Neurosci* 2003, **6**:624-631.

52. de Haan E: **A familial factor in the development of face recognition deficits.** *J Clin Exp Neuropsychol* 1999, **21**:312-315.