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Advances in developmental prosopagnosia research

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Developmental prosopagnosia (DP) refers to face recognition deficits in the absence of brain damage. DP affects ~2% of the population, and it often runs in families. DP studies have made considerable progress in identifying the cognitive and neural characteristics of the disorder. A key challenge is to develop a valid taxonomy of DP that will facilitate many aspects of research.

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Introduction

Developmental prosopagnosia (DP) [1], also known as congenital prosopagnosia, is a specific neurodevelopmental disorder of face recognition despite normal intelligence, low-level vision, and broader social cognition [2,3]. In contrast to those with acquired prosopagnosia [4], individuals with DP have no history of brain injury. DP can lead to elevated rates of anxiety and chronic stress [5]. Prevalence estimates suggest ~2% of the population suffer from DP [6,7] (See Box 1).

Overview of DP

Although formal diagnostic criteria have not been agreed upon, DP is typically diagnosed when an individual who complains of face recognition problems in daily life is impaired on standardized tests of face recognition, such as the Cambridge Face Memory Test (CFMT) [8], as well as on tests of famous face recognition appropriate for the individual. Deficits in DP are often as severe as those in acquired prosopagnosia [9]. For example, 17 DP individuals tested in our laboratory averaged 49% correct (range 36–60%) on the CFMT, which was substantially lower than the control mean of 80% (SD = 11%) [10•], and comparable to five recently reported acquired prosopagnosics who averaged 54% (range 42–60%) [11]. Performance on famous face memory tests is usually far below the control range: the mean for the 17 DP individuals above was 39% (range 2–62%), while the control mean was 89% (SD = 9%) [10•].

DP is a heterogeneous disorder, with individual cases showing varied behavioral profiles. Some individuals were impaired with facial identity memory but were able to match faces side-by-side [12], while others were impaired with both tasks [12,13]. Some individuals were even impaired at detecting the presence of a face in a complex image [14]. DP individuals have also shown deficits at processing non-identity aspects of the face including expressions [10•,15,16], sex [9,15], attractiveness [15,17], and trustworthiness [18].

A long-running controversy in the face recognition literature concerns the face-specific hypothesis, which holds that faces are processed by dedicated mechanisms [19–21]. Although some individuals with DP had problems recognizing nonface objects [9,22,23], some cases exhibited deficits only for faces [22,24]. A notable case is Edward [15,25], who was tested with a variety of face and nonface tasks to evaluate multiple alternatives to the face-specific hypothesis, such as the within-class discrimination [26] and the expertise [27] hypotheses. Edward's normal performance with the nonface tasks was inconsistent with each of the alternatives and could only be accounted for by the face-specific hypothesis. Further evidence consistent with the face-specific hypothesis came from the opposite developmental disorder: AW was able to recognize faces normally but not objects [28•]. Together, Edward and AW constitute a double dissociation between developmental disorders of face and object recognition.

Cognitive characteristics of DP

Unlike most types of objects, faces are represented as a perceptual whole [21,29,30]. This style of representation, referred to as holistic or configural face processing, raises the possibility that face recognition deficits in DP may result from abnormal holistic face processing. This issue has been investigated by assessing the inversion effect (i.e. disproportionately poor recognition when faces are seen upside-down [20]) and the composite effect (i.e. perception of one-half of a face is influenced by the other unattended half [31]). Results are mixed for both inversion [9,13,14,17,24,32–35] and composite [17,33,35–37] effects, indicating that holistic processing is not always impaired in DP.

A revealing insight into holistic face representation in DP was recently provided by a study [38•] of the part-whole

effect (i.e. recognition of a face part is much better in context of the whole face than in isolation [39]). This study examined holistic processing for eyes, nose, and mouth separately in the largest DP sample to date (38 individuals). The authors found a lack of holistic processing for eyes but not for mouths (Figure 1). This finding suggests that atypical processing of the eyes may be a critical factor in DP, similar to what has been proposed in acquired prosopagnosia [40].

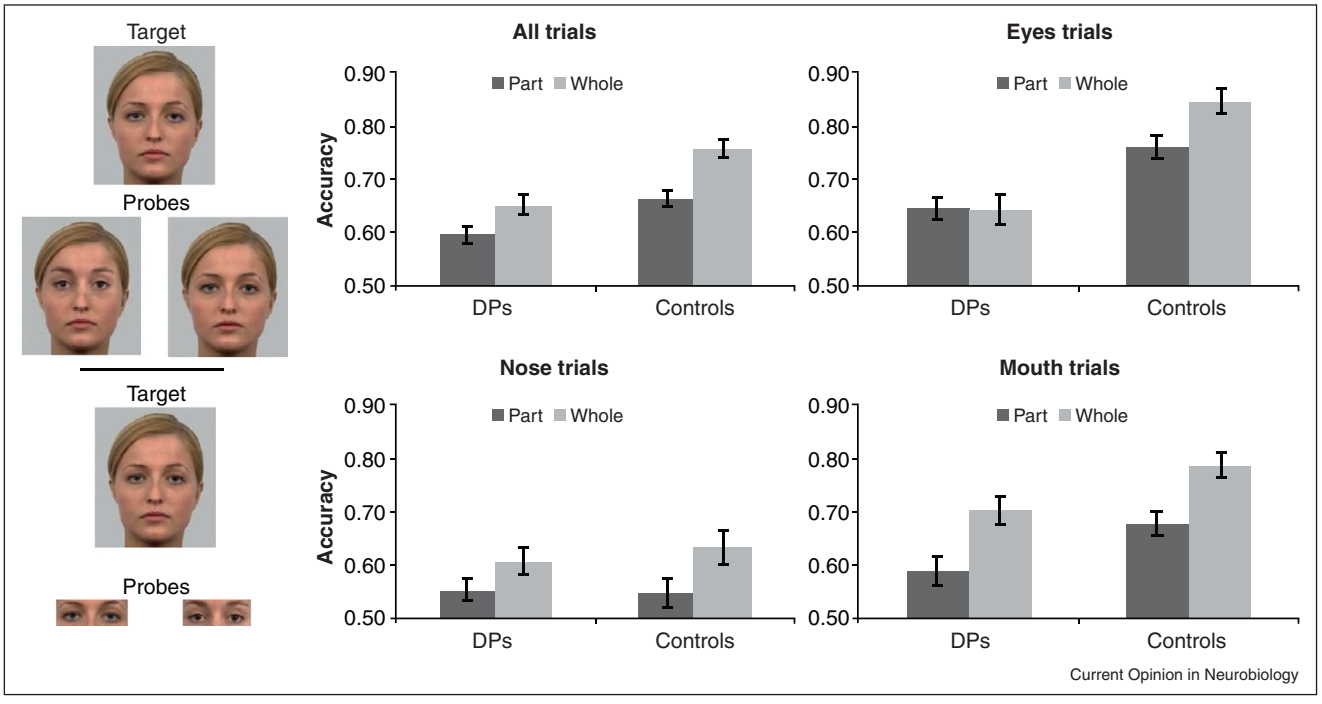
Face recognition is often conceptualized as relying on face space: a space in which facial identities are mapped according to their values on the multiple dimensions used to represent facial features [41]. The status of face space in DP has been examined in seven individuals, all of whom showed normal representation [36,42], suggesting that recognition problems in DP may originate in later processes that read the output from face-space representations.

Some DP individuals can extract an ''average identity'' from a set of identities which they failed to recognize [43], consistent with several reports of covert face processing [44–46]. Indeed, a recent event-related potential (ERP) study [47] found that six out of 12 DP individuals showed a stronger N250 response, a component believed to reflect the matching of a percept to a memory, to unrecognized famous faces than to unrecognized novel faces matched in appearance. In contrast, no difference was found in their response to the two sets of faces in a later component, the P600f, which is considered an index of the activation of semantic person representations. These results suggest that unconscious recognition of identity in the visual system was not fed forward to semantic mechanisms. Similar findings of covert processing in dyslexia [48] and amusia [49] indicate that failures to access conscious representations may commonly be a factor in selective neurodevelopmental disorders.

Neural findings in DP

The neural basis of face processing has received extensive research attention in the last two decades. Several cortical regions show much stronger functional magnetic resonance imaging (fMRI) response to faces than to control stimuli, most notably in fusiform gyrus, inferior occipital gyrus, and superior temporal sulcus [50]. Often considered the core system, these face-selective regions constitute the front end of a broader network of areas responsible for different aspects of face processing [51]. As a group, DP individuals have shown reduced face-

Figure 1



The part-whole effect in DP [38•]. (Left) The part-whole paradigm. Participants are briefly shown a target face, and then must discriminate which of two simultaneously presented images shows the target face, either in the context of the whole face (the whole condition) or when only parts of the face are shown (the part condition). The part-whole effect refers to better discrimination performance in the whole than in the part condition. (Right) Part-whole effects in 38 individuals with DP and 38 controls. Overall, DP and control individuals exhibited part-whole effects, although mean performance of the DP group was lower However, an interesting pattern emerged when performance was analyzed separately for eyes, nose, and mouth: DP individuals only showed part-whole effects for mouth but not eyes (performance for nose is difficult to interpret because of floor effects), in contrast to controls who showed part-whole effects for all face parts.

Box 1 Developmental prosopagnosia or congenital prosopagnosia?

Throughout this article we use the term developmental prosopagnosia (DP) [1,23,24,75,76] for a specific reason: we conceptualize DP as a disorder caused by anomalies occurring at any time during the development of the mechanisms used for face recognition. Other researchers however prefer the term congenital prosopagnosia (CP) [37,57,77–79], describing the disorder as lifelong and, by definition, assuming its presence at birth. Are DP and CP synonymous or are there substantive differences between them?

DP is a more general label whereas CP is a term that implies evidence of prosopagnosia or some correlate of the disorder at birth or at least early in infancy. Collecting such evidence would be challenging but in principle possible [80,81]. For example, newborn children from families with a history of DP could be assessed if infant-friendly behavioral or neurophysiological measures that predict prosopagnosia later in life become available.

In addition, it is important to note that DP likely has multiple etiologies and onsets. Perhaps certain types of DP are caused by disturbance during prenatal development (See Box 2), and thus are appropriately classified as CP. The onset of other types of DP may be postnatal, resulting from a failure to develop typical face processing mechanisms in infancy or childhood.

selectivity in core face regions [52], though it is worth noting that some of these individuals show typical face-selectivity [52,53]. Another functional signature of face processing is an ERP component called the N170, which shows a larger response to faces than nonfaces [54]. Some individuals with DP showed N170 with normal face-selectivity [16,55,56] whereas others did not [55–57]. What lies behind these mixed results is unclear, but is likely related to the heterogeneity of DP.

The existence of face-selective regions and N170 in DP may, however, mask subtle impairments. For example, case C [58] exhibited normal face-selective regions, but the regions did not show repetition suppression (i.e. reduction in fMRI response to repeated stimuli [59]). Similarly, an ERP study of 16 DP individuals [56] found normal face-selectivity for the N170 at the group level but observed that the N170 component was not enhanced for inverted faces as it was in controls (Figure 2). Extending previous reports [23,60], this study suggests that individuals with DP process upright and inverted faces similarly. These examples illustrate that studies of neural functions in DP may benefit from not only examining the existence of signatures of face processing, but also whether they exhibit the properties characteristic of normal face recognition.

Structural correlates of DP have also been identified. A diffusion tensor imaging study found reduced connectivity in two major tracts that project through the fusiform region to more anterior areas, indicating abnormal integrity of white matter in ventral cortex [61•]. Other investigations using voxel-based morphometry found gray matter reduction in cortical regions implicated in face processing including fusiform gyrus, inferior temporal gyrus, and superior temporal sulcus [10•,62]. Of note, similar gray matter reduction in regions involved in phonological processing has been reported in developmental dyslexia [63,64], hinting at a common etiology for DP and developmental dyslexia (see Box 2).

In sum, neural studies have begun to characterize functional and structural correlates of DP. An important next step will be to map particular neural correlates onto specific cognitive deficits. This step is challenging because the relationship between neural and cognitive mechanisms in face processing remains unclear. Despite substantial effort, attempts to localize specific cognitive operations onto focal neural regions have met with limited success [50].

Genetic factors in DP

Consistent with the strong heritability of face recognition in the general population [65••,66], DP tends to run in families [33,67–69]. These findings indicate that DP is a disorder with a genetic component.

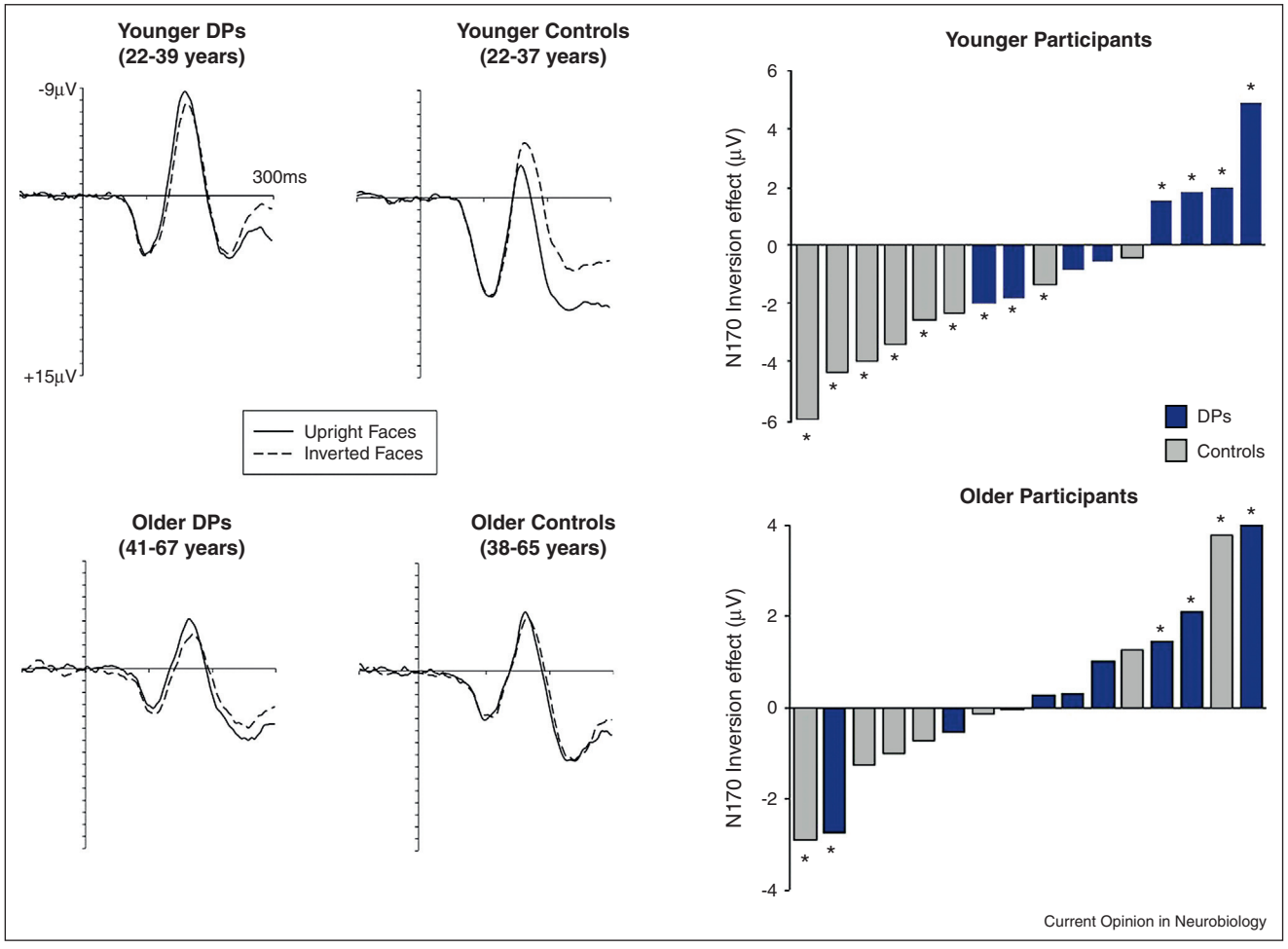
Self-report data suggest that DP may be a monogenic, autosomal dominant disorder [6,7]. This idea is consistent with the profile of the largest family tested in our lab, in which DP is seen in both sexes in about half the members. However, most neurodevelopmental disorders appear to be polygenic because they result from allelic variants that are relatively frequent in the population and are therefore neither necessary nor sufficient for developing the condition. The monogenic view also predicts the existence of large, extended families of prosopagnosics. However, despite contact with more than 7500 self-identified DP individuals over the last decade, we have yet to hear from a family with more than eight affected members.

These considerations suggest that DP may instead result from the cumulative effect of multiple genes. If correct, the probability of DP in an extended family would decrease as genetic distance from the prosopagnosic member increases. Another prediction is that the face recognition ability of the nonprosopagnosic family members would be lower than the population average because these individuals will carry some of the genes associated with DP. Indeed, only one of the nine nonprosopagnosic members of the family above scored better than average on the CFMT.

Intervention in DP

Several intervention attempts have been reported [70–73]. Most noteworthy is a study of case MZ, who practiced classifying hundreds of faces per day based on distances between facial features such as eyes and nose [73]. MZ reported temporary improvement in her daily face recognition following training, which was supported by formal testing and accompanied by the emergence of a face-

Figure 2



N170 inversion effects in DP, adapted with permission from [56]. (Left) Average ERP elicited by upright and inverted faces for DP and control groups. The young but not older control group showed enhanced N170 component to inverted faces. Neither DP group showed an enhanced N170. (Right) Size of N170 inversion effect (defined as N170 amplitude for upright faces minus N170 amplitude for inverted faces) for the 16 DP (blue bar) and 16 control (grey bar) individuals, sorted based on the size and polarity of the effect. Asterisks indicate significant N170 inversion effects based on a bootstrapping analysis, regardless of polarity. For young participants, only two of eight DP individuals exhibited inversion effect in the normal range, in contrast to almost all control individuals.

selective N170 and more typical connectivity between face-selective regions. Two other studies trained children with DP to focus their attention on inner facial features [70,71]. Eye-tracking analyses showed increased fixations on the inner features after training, and recognition of trained faces improved in both cases.

While these efforts suggest certain training regimes may improve face recognition ability in DP, future work will need to use larger samples, explore the generalizability of training to daily life, and most critically, make use of randomized controlled trials.

A taxonomy of DP

A key challenge for researchers is to develop a valid taxonomy of DP, which will help resolve inconsistent findings and facilitate many aspects of research. For example, the mixed results of cognitive and neural studies may result from grouping DP individuals with distinct phenotypes. Similarly, different types of DP are likely to respond to different rehabilitative strategies.

A natural starting point for developing a taxonomy of DP is contemporary models of face recognition [51,74]. These models propose that faces are processed by a network of subsystems, each responsible for analyzing different aspects of the face such as identity, sex, gaze, expression, and trait. Atypical development of particular subsystems would result in deficits for certain aspects of face processing but not others. Future studies should assess these different face aspects simultaneously in a large DP sample to uncover systematic associations and dis-

Box 2 Do neural migration errors contribute to DP?

Selective neurodevelopmental disorders have been identified for a wide variety of human cognitive abilities including numerical, language, motor, navigation, face recognition, object recognition, voice recognition, visual localization, semantic memory, and many others [82,83], raising the question of whether these disorders share a common etiology. An appealing model based on findings from developmental dyslexia proposes that they might [84]. In this model, phonological deficits in dyslexia are caused by cortical dysplasias in the left perisylvian cortex (LPC) which result from neural migration errors. Consistent with this model, autopsies have observed focal dysplasias in LPC [85,86] and most genes associated with dyslexia are involved in neural migration [87].

Ramus [84] suggests that this model can be generalized to other selective neurodevelopmental disorders. Neural migration errors in focal cortical regions would disrupt specific cognitive abilities subserved by those regions, just as dysplasias in LPC disrupt phonological processing. In the case of DP, neural migration errors in occipital and temporal regions involved in face processing would disrupt face recognition. Highly circumscribed dysplasias would result in face-specific deficits [15], whereas more extended dysplasia would disrupt other abilities such as object recognition and spatial navigation mediated by nearby regions [9,22,88]. It is currently unclear whether neural migration problems contribute to DP, but the dyslexia findings [89•] provide a roadmap for future work in DP.

sociations between different face deficits, which will reveal the dimensions underlying the varied behavioral profiles of face recognition deficits in DP.

References and recommended reading

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

- • of special interest
- •• of outstanding interest

1. McConachie HR: **Developmental prosopagnosia. A single case report**. *Cortex: J Devoted Study Nerv Syst Behav* 1976, **12**:76-82.
2. Duchaine BC: **Developmental prosopagnosia: cognitive, neural and developmental investigations**. In *The Oxford Handbook of Face Perception.* Edited by Calder AJ, Rhodes G, Johnson MH, Haxby JV. Oxford University Press; 2011:821-838.
3. Behrmann M, Avidan G, Thomas C, Nishimura M: **Impairments in face perception**. In *The Oxford Handbook of Face Perception.* Edited by Calder AJ, Rhodes G, Johnson MH, Haxby JV. 2011:799-820.
4. Bodamer J: **Die Prosop-Agnosie**. *Arch Psychiatr Nervenkr* 1947.
5. Yardley L, McDermott L, Pisarski S, Duchaine B, Nakayama K: **Psychosocial consequences of developmental prosopagnosia: a problem of recognition**. *J Psychosom Res* 2008, **65**:445-451.
6. Kennerknecht I, Ho NY, Wong VCN: **Prevalence of hereditary prosopagnosia (HPA) in Hong Kong Chinese population**. *Am J Med Genet A* 2008, **146A**:2863-2870.
7. Kennerknecht I, Grueter T, Welling B, Wentzek S, Horst J, Edwards S, Grueter M: **First report of prevalence of nonsyndromic hereditary prosopagnosia (HPA)**. *Am J Med Genet A* 2006, **140**:1617-1622.
8. Duchaine B, Nakayama K: **The Cambridge Face Memory Test: results for neurologically intact individuals and an investigation of its validity using inverted face stimuli and prosopagnosic participants**. *Neuropsychologia* 2006, **44**:576-585.
9. 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.
10. • Garrido L, Furl N, Draganski B, Weiskopf N, Stevens J, Tan GC-Y, Driver J, Dolan RJ, Duchaine B: **Voxel-based morphometry reveals reduced grey matter volume in the temporal cortex of developmental prosopagnosics**. *Brain* 2009, **132**:3443-3455.
This voxel-based morphometry study of 17 DP individuals identified less grey matter in cortical regions implicated in face recognition including the fusiform gyrus, inferior temporal gyrus, and superior temporal sulcus.
11. Dalrymple KA, Oruç I, Duchaine B, Pancaroglu R, Fox CJ, Iaria G, Handy TC, Barton JJS: **The anatomic basis of the right face-selective N170 IN acquired prosopagnosia: a combined ERP/fMRI study**. *Neuropsychologia* 2011, **49**:2553-2563.
12. McKone E, Hall A, Pidcock M, Palermo R, Wilkinson RB, Rivolta D, Yovel G, Davis JM, O'Connor KB: **Face ethnicity and measurement reliability affect face recognition performance in developmental prosopagnosia: evidence from the Cambridge Face Memory Test-Australian**. *Cogn Neuropsychol* 2011, **28**:109-146.
13. Duchaine B, Yovel G, Nakayama K: **No global processing deficit in the Navon task in 14 developmental prosopagnosics**. *Soc Cogn Affect Neurosci* 2007, **2**:104-113.
14. Garrido L, Duchaine B, Nakayama K: **Face detection in normal and prosopagnosic individuals**. *J Neuropsychol* 2008, **2**:119-140.
15. Duchaine BC, Yovel G, Butterworth EJ, Nakayama K: **Prosopagnosia as an impairment to face-specific mechanisms: elimination of the alternative hypotheses in a developmental case**. *Cogn Neuropsychol* 2006, **23**:714-747.
16. Minnebusch Da, Suchan B, Ramon M, Daum I: **Event-related potentials reflect heterogeneity of developmental prosopagnosia**. *Eur J Neurosci* 2007, **25**:2234-2247.
17. Le Grand R, Cooper PA, Mondloch CJ, Lewis TL, Sagiv N, De Gelder B, Maurer D: **What aspects of face processing are impaired in developmental prosopagnosia?** *Brain Cogn* 2006, **61**:139-158.
18. Todorov A, Duchaine B: **Reading trustworthiness in faces without recognizing faces**. *Cogn Neuropsychol* 2008, **25**:395-410.
19. Diamond R, Carey S: **Why faces are and are not special: an effect of expertise**. *J Exp Psychol Gen* 1986, **115**:107-117.
20. Yin RK: **Looking at upside-down faces**. *J Exp Psychol* 1969, **81**:141-145.
21. McKone E, Kanwisher N, Duchaine BC: **Can generic expertise explain special processing for faces?** *Trends Cogn Sci* 2007, **11**:8-15.
22. Duchaine B, Nakayama K: **Dissociations of face and object recognition in developmental prosopagnosia**. *J Cogn Neurosci* 2005, **17**:249-261.
23. Righart R, De Gelder B: **Impaired face and body perception in developmental prosopagnosia**. *Proc Natl Acad Sci USA* 2007, **104**:17234-17238.
24. Nunn JA, Postma P, Pearson R: **Developmental prosopagnosia: should it be taken at face value?** *Neurocase* 2001, **7**:15-27.
25. Duchaine BC, Dingle K, Butterworth E, Nakayama K: **Normal greeble learning in a severe case of developmental prosopagnosia**. *Neuron* 2004, **43**:469-473.
26. Damasio AR, Damasio H, Van Hoesen GW: **Prosopagnosia: anatomic basis and behavioral mechanisms**. *Neurology* 1982, **32**:331-341.
27. Gauthier I, Williams P, Tarr MJ, Tanaka J: **Training "greeble" experts: a framework for studying expert object recognition processes**. *Vision Res* 1998, **38**:2401-2428.
28. • Germine L, Cashdollar N, Düzel E, Duchaine B: **A new selective developmental deficit: impaired object recognition with normal face recognition**. *Cortex: J Devoted Study Nerv Syst Behav* 2010.
In this first report of developmental object agnosia, AW was able to recognize faces but was impaired when discriminating between exem-

plars of multiple non-face objects.

29. Maurer D, Grand R, Le Mondloch CJ: **The many faces of configural processing**. *Trends Cogn Sci* 2002, **6**:255-260.

30. Rossion B: **Picture-plane inversion leads to qualitative changes of face perception**. *Acta Psychol (Amst)* 2008, **128**:274-289.

31. Young AW, Hellawell D, Hay DC: **Configurational information in face perception**. *Perception* 1987, **16**:747-759.

32. De Gelder B, Rouw R: **Configural face processes in acquired and developmental prosopagnosia: evidence for two separate face systems?** *Neuroreport* 2000, **11**:3145-3150.

33. Schmalzl L, Palermo R, Coltheart M: **Cognitive heterogeneity in genetically based prosopagnosia: a family study**. *J Neuropsychol* 2008, **2**:99-117.

34. Russell R, Duchaine B, Nakayama K: **Super-recognizers: people with extraordinary face recognition ability**. *Psychon Bull Rev* 2009, **16**:252-257.

35. Avidan G, Tanzer M, Behrmann M: **Impaired holistic processing in congenital prosopagnosia**. *Neuropsychologia* 2011, **49**:2541-2552.

36. Susilo T, McKone E, Dennett H, Darke H, Palermo R, Hall A, Pidcock M, Dawel A, Jeffery L, Wilson CE *et al.*: **Face recognition impairments despite normal holistic processing and face space coding: evidence from a case of developmental prosopagnosia**. *Cogn Neuropsychol* 2011, **27**:636-664.

37. Palermo R, Willis ML, Rivolta D, McKone E, Wilson CE, Calder AJ: **Impaired holistic coding of facial expression and facial identity in congenital prosopagnosia**. *Neuropsychologia* 2011, **49**:1226-1235.

38. DeGutis J, Cohan S, Mercago RJ, Wilmer JB, Nakayama K:
• **Holistic processing of the mouth but not the eyes in developmental prosopagnosia.** Cogn Neuropsychol n.d.
This study tested the largest sample of DP to date (38 individuals) with the part-whole paradigm and found a specific impairment of holistic processing for the eyes but not the mouth.

39. Tanaka JW, Farah MJ: **Parts and wholes in face recognition**. *Q J Exp Psychol A: Hum Exp Psychol* 1993, **46**:225-245.

40. Ramon M, Rossion B: **Impaired processing of relative distances between features and of the eye region in acquired prosopagnosia — two sides of the same holistic coin?** *Cortex: J Devoted Study Nerv Syst Behav* 2010, **46**:374-389.

41. Valentine T: **A unified account of the effects of distinctiveness, inversion, and race in face recognition**. *Q J Exp Psychol A: Hum Exp Psychol* 1991, **43**:161-204.

42. Nishimura M, Doyle J, Humphreys K, Behrmann M: **Probing the face-space of individuals with prosopagnosia**. *Neuropsychologia* 2010, **48**:1828-1841.

43. Leib AY, Puri AM, Fischer J, Bentin S, Whitney D, Robertson L: **Crowd perception in prosopagnosia**. *Neuropsychologia* 2012.

44. Avidan G, Behrmann M: **Implicit familiarity processing in congenital prosopagnosia**. *J Neuropsychol* 2008, **2**:141-164.

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

46. Rivolta D, Palermo R, Schmalzl L, Coltheart M: **Covert face recognition in congenital prosopagnosia: a group study**. *Cortex: J Devoted Study Nerv Syst Behav* 2012, **48**:344-352.

47. Eimer M, Gosling A, Duchaine B: **Electrophysiological markers of covert face recognition in developmental prosopagnosia**. *Brain: J Neurol* 2012, **135**:542-554.

48. Ramus F, Szenkovits G: **What phonological deficit?** *Q J Exp Psychol (2006)* 2008, **61**:129-141.

49. Peretz I, Brattico E, Järvenpää M, Tervaniemi M: **The amusic brain: in tune, out of key, and unaware**. *Brain: J Neurol* 2009, **132**:1277-1286.

50. Kanwisher N, Barton JJS: **The functional architecture of the face system: integrating evidence from fMRI and patient studies**. In *The Handbook of Face Perception.* Edited by Calder A.J., Rhodes G., Johnson M.H., Haxby J.V.. Oxford University Press; 2011.

51. Haxby JV, Gobbini MI: **Distributed neural systems for face perception**. In *The Handbook of Face Perception.* Edited by Calder AJ, Rhodes G, Johnson MH, Haxby JV. Oxford University Press; 2011:93-110.

52. Furl N, Garrido L, Dolan RJ, Driver J, Duchaine B: **Fusiform gyrus face selectivity relates to individual differences in facial recognition ability**. *J Cogn Neurosci* 2011, **23**:1723-1740.

53. Avidan G, Behrmann M: **Functional MRI, reveals compromised neural integrity of the face processing network in congenital prosopagnosia**. *Curr Biol* 2009, **19**:1146-1150.

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

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

56. Towler J, Gosling A, Duchaine B, Eimer M: **The face-sensitive N170 component in developmental prosopagnosia**. *Neuropsychologia* 2012, **50**:3588-3599.

57. Bentin S, Degutis JM, D'Esposito M, Robertson LC: **Too many trees to see the forest: performance, event-related potential, and functional magnetic resonance imaging manifestations of integrative congenital prosopagnosia**. *J Cogn Neurosci* 2007, **19**:132-146.

58. Williams MA, Berberovic N, Mattingley JB: **Abnormal FMRI adaptation to unfamiliar faces in a case of developmental prosopamnesia**. *Curr Biol* 2007, **17**:1259-1264.

59. Grill-Spector K, Malach R: **fMR-adaptation: a tool for studying the functional properties of human cortical neurons**. *Acta Psychol (Amst)* 2001, **107**:293-321.

60. De Gelder B, Stekelenburg JJ: **Naso-temporal asymmetry of the N170 for processing faces in normal viewers but not in developmental prosopagnosia**. *Neurosci Lett* 2005, **376**:40-45.

61. Thomas C, Avidan G, Humphreys K, Jung K, Gao F, Behrmann M:
• **Reduced structural connectivity in ventral visual cortex in congenital prosopagnosia**. *Nat Neurosci* 2009, **12**:29-31.
Using diffusion tensor imaging and tractography, the authors tested 6 DP individuals and revealed structural anomalies in two major tracts that project from posterior face-selective regions to more anterior regions.

62. Behrmann M, Avidan G, Gao F, Black S: **Structural imaging reveals anatomical alterations in inferotemporal cortex in congenital prosopagnosia**. *Cereb Cortex* 2007, **17**:2354-2363.

63. Richardson FM, Price CJ: **Structural MRI studies of language function in the undamaged brain**. *Brain Struct Funct* 2009, **213**:511-523.

64. Vinckenbosch E, Robichon F, Eliez S: **Gray matter alteration in dyslexia: converging evidence from volumetric and voxel-by-voxel MRI analyses**. *Neuropsychologia* 2005, **43**:324-331.

65. Wilmer JB, Germine L, Chabris CF, Chatterjee G, Williams M,
•• Loken E, Nakayama K, Duchaine B: **Human face recognition ability is specific and highly heritable**. *Proc Natl Acad Sci USA* 2010, **107**:5238-5241.
The authors showed that identical twins are much more similar in their face recognition performance than fraternal twins, controlling for general intelligence, indicating that face recognition is a specific cognitive ability with strong heritability.

66. Zhu Q, Song Y, Hu S, Li X, Tian M, Zhen Z, Dong Q, Kanwisher N, Liu J: **Heritability of the specific cognitive ability of face perception**. *Curr Biol* 2010, **20**:137-142.

67. Duchaine B, Germine L, Nakayama K: **Family resemblance: ten family members with prosopagnosia and within-class object agnosia**. *Cogn Neuropsychol* 2007, **24**:419-430.

68. Grüter T, Grüter M, Carbon C-C: **Neural and genetic foundations of face recognition and prosopagnosia**. *J Neuropsychol* 2008, **2**:79-97.

69. Lee Y, Duchaine B, Wilson HR, Nakayama K: **Three cases of developmental prosopagnosia from one family: detailed neuropsychological and psychophysical investigation of face processing**. *Cortex: J Devoted Study Nerv Syst Behav* 2010, **46**:949-964.

70. Brunsdon R, Coltheart M, Nickels L, Joy P: **Developmental prosopagnosia: a case analysis and treatment study**. *Cogn Neuropsychol* 2006, **23**:822-840.

71. Schmalzl L, Palermo R, Green M, Brunsdon R, Coltheart M: **Training of familiar face recognition and visual scan paths for faces in a child with congenital prosopagnosia**. *Cogn Neuropsychol* 2008, **25**:704-729.

72. DeGutis J, DeNicola C, Zink T, McGlinchey R, Milberg W: **Training with own-race faces can improve processing of other-race faces: evidence from developmental prosopagnosia**. *Neuropsychologia* 2011, **49**:2505-2513.

73. DeGutis JM, Bentin S, Robertson LC, D'Esposito M: **Functional plasticity in ventral temporal cortex following cognitive rehabilitation of a congenital prosopagnosic**. *J Cogn Neurosci* 2007, **19**:1790-1802.

74. Young AW, Bruce V: **Understanding person perception**. *Br J Psychol* 2011, **102**:959-974.

75. Duchaine BC, Nakayama K: **Developmental prosopagnosia: a window to content-specific face processing**. *Curr opin neurobiol* 2006, **16**:166-173.

76. Bentin S, Deouell LY, Soroker N: **Selective visual streaming in face recognition: evidence from developmental prosopagnosia**. *Neuroreport* 1999, **10**:823-827.

77. Behrmann M, Avidan G: **Congenital prosopagnosia: face-blind from birth**. *Trends cogn sci* 2005, **9**:180-187.

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

79. Carbon C-C, Grüter T, Weber JE, Lueschow A: **Faces as objects of non-expertise: processing of thatcherised faces in congenital prosopagnosia**. *Perception* 2007, **36**:1635-1645.

80. Morton J, Johnson MH: **CONSPEC and CONLERN: a two-process theory of infant face recognition**. *Psychol rev* 1991, **98**:164-181.

81. Meltzoff AN, Moore MK: **Imitation of facial and manual gestures by human neonates**. *Science* 1977, **198**:75-78.

82. Bishop DVM: **Which neurodevelopmental disorders get researched and why?** *PLoS ONE* 2010, **5**:e15112.

83. Grüter T, Carbon C-C: **Neuroscience. Escaping attention**. *Science* 2010, **328**:435-436.

84. Ramus F: **Neurobiology of dyslexia: a reinterpretation of the data**. *Trends Neurosci* 2004, **27**:720-726.

85. Galaburda AM, Sherman GF, Rosen GD, Aboitiz F, Geschwind N: **Developmental dyslexia: four consecutive patients with cortical anomalies**. *Ann Neurol* 1985, **18**:222-233.

86. Humphreys P, Kaufmann WE, Galaburda AM: **Developmental dyslexia in women: neuropathological findings in three patients**. *Ann Neurol* 1990, **28**:727-738.

87. Galaburda AM, LoTurco J, Ramus F, Fitch RH, Rosen GD: **From genes to behavior in developmental dyslexia**. *Nat Neurosci* 2006, **9**:1213-1217.

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

89. • Giraud A-L, Ramus F: **Neurogenetics and auditory processing in developmental dyslexia**. *Curr Opin Neurobiol* 2013, **23**: 37-42.
This article summarizes genetic and neurophysiological findings in dyslexia relevant to the neural migration model of selective developmental disorders.