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Family resemblance: Ten family members with prosopagnosia and within-class object agnosia

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We report on neuropsychological testing done with a family in which many members reported severe face recognition impairments. These 10 individuals were high functioning in everyday life and performed normally on tests of low-level vision and high-level cognition. In contrast, they showed clear deficits with tests requiring face memory and judgements of facial similarity. They did not show deficits with all aspects of higher level visual processing as all tested performed normally on a challenging facial emotion recognition task and on a global–local letter identification task. On object memory tasks requiring recognition of particular cars and guns, they showed significant deficits so their recognition impairments were not restricted to facial identity. These results strongly suggest the existence of a genetic condition leading to a selective deficit of visual recognition.

Participant F39 is an intelligent 39-year-old woman with normal visual acuity, normal cognitive abilities, and no history of brain damage. Yet when F39 was presented with the image of Elvis Presley shown in Figure 1, she identified the face as Brooke Shields, the woman shown next to Elvis. Despite familiarity with most of the 60 celebrities presented to her in a famous-face test discussed below, F39 was able to identify only 6. F39's difficulties indicate that she has face recognition deficits, and her case is particularly interesting because many of her genetic relatives also show severe deficits in the context of normal cognitive abilities.

Prosopagnosia is a condition characterized by face recognition impairments. In acquired prosopagnosia, face recognition abilities are impaired after brain damage, whereas developmental prosopagnosics (DPs) fail to develop normal face recognition abilities. Until recently, developmental prosopagnosia (DP) appeared to be a rare condition. However, our laboratory has been contacted by more than 2,200 self-identified DPs through our web site (http://www.faceblind.org), and a recent study estimated that prevalence could be as high as 2% (Kennerknecht et al., 2006). Most DPs show visual recognition impairments without general cognitive deficits (McConachie, 1976; Nunn, Postma, & Pearson, 2001), and in some cases impairments appear to affect face processing without affecting object processing (Bentin, Deouell, & Soroker, 1999;

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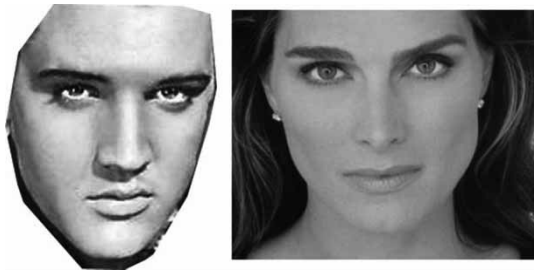

Figure 1. *Spitting image? In the famous-faces task, F39 identified the image of Elvis Presley on the left as Brooke Shields (shown on the right).*

Duchaine & Nakayama, 2005; Duchaine, Yovel, Butterworth, & Nakayama, 2006; Nunn et al., 2001).

Investigations into the neural and cognitive basis of prosopagnosia have sometimes mentioned that DP participants have reported genetic relatives who share their face recognition difficulties (Behrmann, Avidan, Marotta, & Kimchi, 2005; Duchaine & Nakayama, 2005; Duchaine, Nieminen-von Wendt, New, & Kulomaki, 2003a; McConachie, 1976). Further hints to a genetic basis in some cases of DP come from a report of three relatives with poor famous-face recognition (de Haan, 1999) and a report of seven families with a total of 37 members who self-identified as prosopagnosic (Grueter et al., in press). Of the DPs who have contacted us, approximately 20% report relatives with face recognition difficulties, and we have tested a number of families with two or three affected relatives. However, solid behavioural evidence for the heritability of prosopagnosia has not been documented. Poor famous-face recognition may result from a lack of exposure to celebrities (which probably runs in a family), and self-reports are not reliable as we have tested many self-identified prosopagnosics who show no impairments. Small numbers of DPs from the same family may simply be a coincidence especially if the 2% estimate is accurate. In addition, the presence of a condition in multiple individuals in a family increases the likelihood that they will come to the attention of researchers.

Herein, we establish with extensive behavioural testing that DP runs in a family, and we explore the perceptual and cognitive profile of these family members. Many neurocognitive deficits result from genetic conditions, but most have widespread effects. Only a few genetic conditions have relatively selective neurocognitive effects (Fisher & DeFries, 2002; Hurst, Baraitser, Auger, Graham, & Norrell, 1990; Lai, Fisher, Hurst, Vargha-Khadem, & Monaco, 2001). These conditions have led to significant insights into a range of genetic, developmental, neural, cognitive, and evolutionary issues (Fisher & Marcus, 2006).

Family background

Our investigation involves 10 members of an extended family (seven siblings, their parents, and a maternal uncle). Most testing was done in person, but a few follow-up tests were run remotely. The pedigree in Figure 2 uses black symbols for prosopagnosic members that we have tested and grey symbols for individuals believed by the family to be prosopagnosic. Each participant is labelled with their sex and age. Their occupations (siblings: dental student, graduate student, programmer, university administrator, physician, engineer, geologist; parents: truck driver,

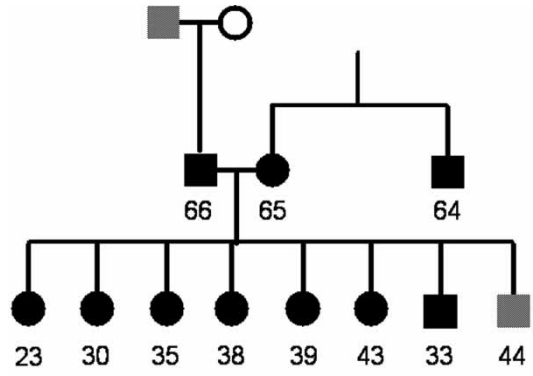

Figure 2. *Pedigree of the family with ages. Everyone tested had face recognition problems, and they are shown in black. Individuals who we were unable to test yet who are believed to be or to have been prosopagnosic are shaded in grey. □ = males, ○ = females.*

registered nurse; uncle: engineer) demonstrate that this is a family of above-average intelligence. They routinely have face recognition difficulties, including incidents involving family members and even their own faces in photographs and mirrors. The paternal grandfather of the siblings is deceased, but many family stories indicate that he had severe face recognition impairments. For example, at his 50th wedding anniversary while still in excellent mental and physical condition, his wife walked a step behind him so she could state people's names as they approached him. One male sibling was unavailable for testing, but he reports that he has difficulty recognizing people when they change their hairstyle or glasses. One sibling believes her daughter may have face recognition problems, but we were unable to test her because our tests are designed for adults. Interestingly, F39 reports that nametags are worn at family reunions.

None of the family members have any history of early visual problems, head trauma, or birth complications, any of which can lead to prosopagnosia (Farah & Rabinowitz, 2000; Le Grand, Mondloch, Maurer, & Brent, 2001). Acuity was tested in the seven siblings and in the maternal uncle, and it was normal or corrected-to-normal in all. This same group of participants also performed normally on the Pelli-Robson contrast sensitivity test (Pelli, Robson, & Wilkins, 1988). On a range of cognitive tests, the siblings performed normally. The National Adult Reading Test–U.S. edition uses word pronunciation to estimate vocabulary size, and with the exception of F30, who had reading difficulties as a child, all scored well above the mean (Grober & Sliwinski, 1991). The forward and backward digit span tests from the Wechsler Adult Intelligence Scale (Wechsler, 1997) measure short-term auditory memory, and all siblings scored normally. Finally, all were normal on the short version of Raven's Advanced Progressive Matrices (Raven, Court, & Raven, 1976), which estimates nonverbal intelligence with items requiring visual pattern completion. Scores for each family member for these tests and all tests discussed below are presented in the Appendix.

Method and results

Memory for facial identity

Famous faces. To assess memory for facial identity, participants were presented with 60 famous faces for 5 s each (Duchaine & Nakayama, 2005). The faces were closely cropped so that little or no clothing or hair was visible. Participants were asked to name the face or report uniquely identifying information if they could not recall the name. For example, "The King", "Hound Dog", or "Married to Priscilla" would be scored as correct for Elvis whereas generic descriptions such as "Singer" or "Actor" would not. Although stimulus duration was fixed, participants could take as long as necessary to respond. To check whether participants were familiar with the individuals presented, each participant was asked after the test whether they had substantial exposure to the faces that they failed to identify.

Figure 3 displays the results for the 10 family members and 20 controls, 12 of whom were age matched with the siblings (average age = 32.6 years) and 8 who were age matched with the parents and uncle (average age = 63.9 years). The scores for the two control groups were not significantly different. Younger controls correctly identified 53.7 faces ($SD = 3.2$) while older controls identified 52.6 faces ($SD = 5.2$). The ordinate shows how many faces each participant correctly identified while the abscissa shows the number of faces to which each participant acknowledged substantial exposure. Family members clearly identified far fewer faces than did controls, $F(2, 27) = 40.9$, $p < .001$. Using Crawford and Howell's (1998) modified t tests for comparing single cases to small control samples, we found that all family members were significantly impaired relative to controls. They had substantial exposure to most of the individuals, though like many DPs they are not as familiar with celebrities as are individuals with normal face recognition. The performance of the family members was quite variable, with 6 somewhat below the control group while the other 4 identified 15 or fewer famous faces.

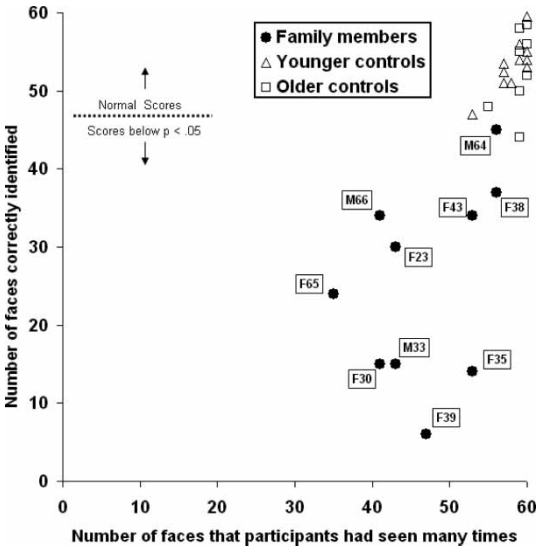

Figure 3. *Famous-face results. A total of 60 famous faces were presented to 12 younger controls (Δ), 8 older controls (□), and 8 members of the family (●). Each symbol displays the number of faces identified by each participant and the number of celebrities that each participant had substantial exposure to.*

The family members showed clear deficits with famous-face identification, but because exposure to famous faces varies for each participant, we next examined their face memory with a test using unfamiliar faces in which exposure was equal for each participant.

Cambridge Face Memory Test. In the Cambridge Face Memory Test (CFMT) (Duchaine & Nakayama, 2006), participants must recognize images of six target faces in progressively more difficult stages. Each target face was introduced by presenting participants with three different study views for 3 s each (See Figure 4a, top row). Immediately after presentation of the study images for a particular target face, participants were presented with three forced-choice items, each of which consisted of one of the study images paired with two other faces in the same pose (See Figure 4a, second row). This study and test cycle was repeated for all six target faces. Thus, the introduction consisted of 18 items (6 faces × 3 test items per face). After the introductory phase, participants were tested with 54 forced-choice items. Each consisted of novel views of one of the six target faces along with two nontarget faces (See Figure 4a, third row). These items were much more difficult than the introductory test items, because novel views were used, and participants did not know which of the six target faces would be present. Noise was added to the final 24 items presenting novel views to make them even more difficult (See Figure 4a, fourth row).

Figure 4d displays modified *t* scores for each family member on the CFMT, computed using Crawford and Howell's method (1998). A dashed line was placed at the .05 significance level. Eight age-matched controls (average age = 45.1) averaged 59.6 correct ($SD = 7.6$) out of 72 items, which is very close to a previously published mean for college-aged controls (Duchaine & Nakayama, 2006). The family members averaged 38.3 ($SD = 5.7$), which is significantly worse than the scores of controls, $t(28) = -7.8$, $p < .001$. As Figure 4d shows, all but two family members were significantly impaired on the CFMT, and the *t* scores for those who were not, F23 and F38, were close to significance. Only two controls scored below 52, so the family's scores were very poor.

Perception of facial similarity

The two face memory tests above demonstrate that the family members have severe face memory deficits. These deficits may result from impairments restricted to memory or they may begin at an earlier stage of facial identity processing. To examine this question, we assessed the family members' facial identity perception with the Cambridge Face Perception Test (CFPT).

The CFPT is a computerized sorting task in which participants arrange six facial images according to their similarity to a target face. The images were created by morphing six different individuals with the target face. The proportion of the morph coming from the target face is varied in each image. Figure 4b displays a sample sort in correct order. On each trial, participants were presented with a 3/4 profile view of a

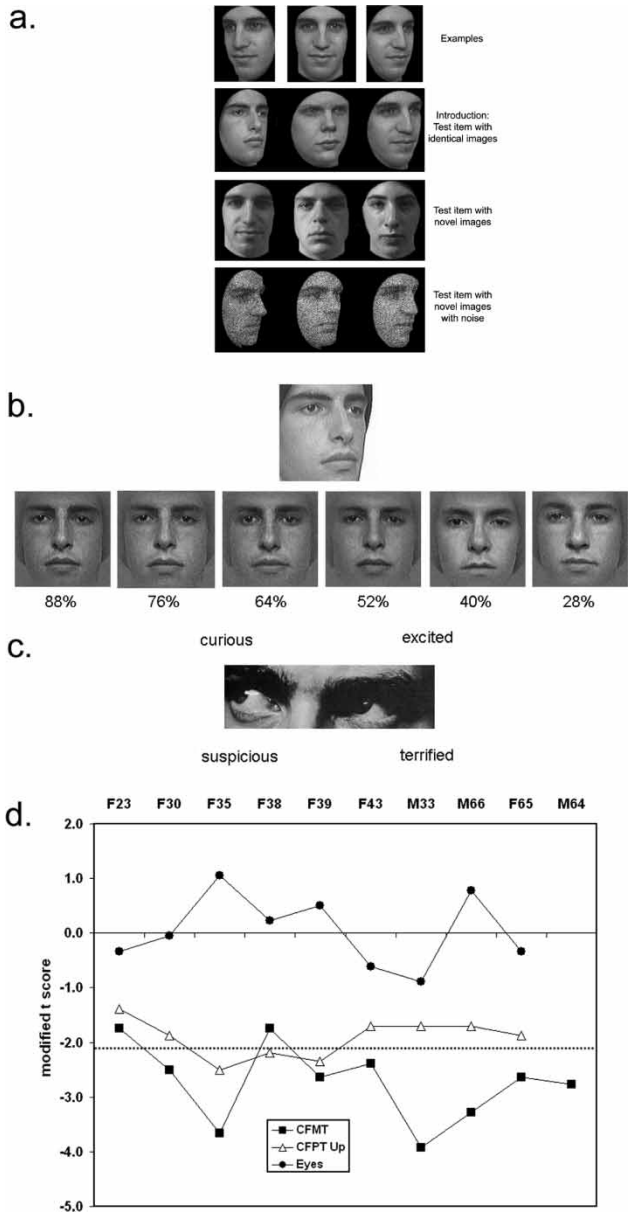

Figure 4. *Face processing stimuli and results. (a) Examples of study images and test items from the Cambridge Face Memory Test. (b) Images from an item in the Cambridge Face Perception Test. Numbers under each image indicate the percentage of the target face in the image. In a test item, the six frontal shots were presented in a random order, and participants sorted based on similarity to the target image (the 3/4 profile view). Turn the page upside down to experience the effect of inversion. (c) Example of Eyes Test item. Participants were presented with an eye region surrounded by four words describing emotional states. The correct answer in this example is suspicious. (d) Face processing scores. Participants' scores are presented as modified t scores (Crawford & Howell, 1998), and the dashed line shows the cut-off for impaired performance. All participants performed poorly with the tests involving facial identity (CFMT and CFPT), but all did well on the test involving facial emotion (Eyes Test). Note that Figure 4d uses the same line of significance for the three tests despite different-sized control samples (20, 21, and 122). The difference between the cut-offs for the CFMT and CFPT samples were negligible, and scores for the Eyes Test were not close to significantly impaired.*

target face above frontal views of six men's faces in a random order. Participants had one minute for each sort. The images contained 88%, 76%, 64%, 52%, 40%, and 28% of the target face. Eight different sorts were created, and each was presented upright once and inverted once. Upright and inverted trials were intermixed, with the upright trial occurring first half the time. One upright and one inverted practice trial were presented at the start of the test. Participants sorted the faces by clicking on a face and then indicating where that face should be moved by clicking in the area between two faces. The chosen face was then moved by the program to the desired location.

Scores for each item were computed by summing the deviations from the correct position for each face. For example, if a face was one position from its correct position, that was one error. If it was three positions away, that was three errors. Scores for the eight upright items and the eight inverted items were added to determine total number of upright and inverted errors. Performance at chance is 93.3 errors. A total of 21 adult controls (average age = 46.5 years) showed a robust inversion effect, with an upright average of 36.7 errors ($SD = 12.2$) and an inverted average of 65.0 errors ($SD = 9.8$). Figure 4d displays the modified t scores for the upright items for the siblings and their parents, which range from −1.4 to −2.5 and averaged −1.9. Their average was 60.7 upright errors. The family members made an average of 76 inverted errors (average modified t score = −1.0). Both of these scores were significantly worse than those of controls, $F(1, 28) = 72.6$, $p < .001$, with a greater impairment for upright items than for inverted items, $F(1, 28) = 6.4$, $p = .017$. Hence, the CFPT results demonstrate that the family has difficulties with both facial identity recognition and facial identity perception.

Facial emotion recognition

Despite severe impairments with facial identity recognition, many DPs recognize facial expressions normally (Bentin et al., 1999; Duchaine, Parker, & Nakayama, 2003b; Humphreys, Minshew, Leonard, & Behrmann, in press; Nunn et al., 2001). To assess the extent of the family members' face processing impairments, we tested them with the Eyes Test (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001) Participants were presented with 36 items consisting of an eye region and four emotion state words (See Figure 4c). Participants had to choose which word best described the eyes. This is a challenging test requiring subtle discriminations, and it appears to require face-specific processing because inversion dramatically decreases performance. DPs with facial identity impairments comparable to those of the family members have scored out of the normal range on it (Duchaine et al., 2003a; Duchaine et al., 2006).

Baron-Cohen et al. (2001) tested 122 members of the general population, and they averaged 26.2/36 ($SD = 3.6$). Figure 4d shows that family members' z scores cluster around the control mean; their average score of 26.3 was nearly identical to the control mean. This result accords with their belief that they have normal facial emotion recognition abilities.

Within-category object recognition

Of the approximately 100 DPs tested in our laboratory, all can recognize objects at the basic level (e.g., car, dog, stapler). However, some individuals with face recognition deficits have within-class object recognition deficits (Behrmann et al., 2005; de Haan & Campbell, 1991; Duchaine & Nakayama, 2005; Duchaine et al., 2003a) whereas others perform normally (Bentin et al., 1999; Duchaine & Nakayama, 2005; Duchaine et al., 2006; Nunn et al., 2001). To assess the family members' within-class object recognition abilities, we tested them with tasks used in a number of previous papers (Duchaine & Nakayama, 2006; Duchaine et al., 2003a; Harris, Duchaine, & Nakayama, 2005). We did not have the opportunity to test the parents or maternal uncle with these tests. The tests require recognition of individual items from within two object categories—cars and guns. Although the family members clearly have face memory deficits, we also ran a parallel face test so that we could compare their performance with faces and objects in the same paradigm.

During the study phase of each test, participants were presented with 10 target items for 3 s each, and each item was shown twice. Participants were then presented with 50 items one at a time; each target was shown twice along with 30 nontargets. Study and test items were identical images. Participants decided whether items were targets (old) or nontargets (new) as quickly as possible. Figure 5 shows an item from each test.

Controls for the three experiments were graduate students in their 20s and 30s, who were comparable in age to the siblings. A', an unbiased measure of discrimination, which varies between .5 (chance) and 1.0 (perfect), was used as the accuracy measure. With faces, the average A' for the 21 controls was .96 ($SD = .023$) while the average response time (RT) was 988 ms ($SD = 217$). Figure 5 presents modified t scores calculated by averaging each sibling's A' modified t score and RT modified t score. Modified t scores for the face test are shown in black, and as expected the siblings were again impaired with faces, $t(26) = -9.9$, $p < .001$.

With cars, 22 controls averaged .95 ($SD = .037$) for A' and 1,190 ms ($SD = 389$) for RTs. The modified t scores for cars are shown in dark grey. Although the scores are not as low as the face scores, 5 of 7 showed a significant deficit (Crawford & Howell, 1998), and the siblings' average was significantly worse than that of controls, $t(27) = -6.3$, $p < .001$. On the guns test, the average A' for 20 controls was .92 ($SD = .036$), and the average RT was 1,239 ms ($SD = 319$). Again, the average scores for the siblings were worse than those for controls, $t(25) = -5.9$, $p < .001$. Of the siblings, 3 were impaired using the modified t test, 1 narrowly failed to reach significance, and all were negative. On the 42 measures that contributed to the 21 t scores in Figure 5, all but 3 were negative so poor composite scores resulted from both low A's and slow RTs (see Appendix for A' and RT values). These tests demonstrate that the siblings' visual recognition deficits are not restricted to facial identity but affect within-class object recognition as well.

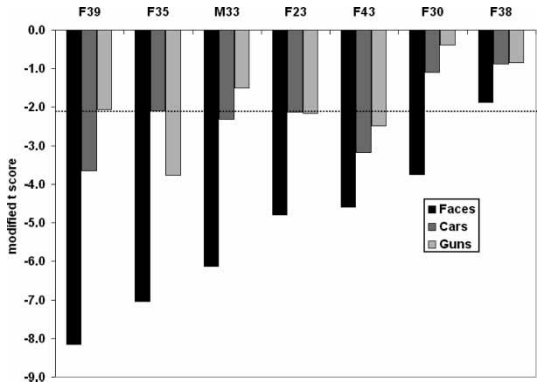

Figure 5. *Old–new recognition memory for faces (a), cars (b), and guns (c). Each column displays a modified t score computed by averaging each sibling's t score for accuracy (A′) and t score for reaction time. For both measures, performance worse than the control mean was negative. All 21 averaged t scores displayed are worse than the control mean, and of the 42 measures that contributed to the 21 scores, all but 3 were worse than the control mean. An item from each test is displayed in the plot.*

The scores for the family members on the three old–new tests were strongly correlated, with correlations of .76 for faces and cars, $t(5) = 2.61$, $p = .05$, .53 for cars and guns, $t(5) = 1.42$, $p = .11$, and .62 for faces and guns, $t(5) = 1.78$, $p = .07$. Although two of the correlations did not reach significance, there certainly appears to be a relationship between performance on the old–new tests.

Global–local task

Global–local tasks are visual cognition tests that require rapid letter identification at different scales (Navon, 1977). We examined the siblings' performance with a global–local task for three reasons. First, it allows us to determine whether the family's visual recognition deficits affect this well-researched aspect of visual cognition. Second, because RTs are critical in this task, normal performance would rule out a general psychomotor slowness as a factor contributing to the family members' poor performance in the speeded tests discussed above. Lastly, it has been suggested that DP results from a general impairment in global processing for visual stimuli (Behrmann et al., 2005). This predicts that DPs will show slowed global responses relative to local responses. It also predicts that they will

show weaker global interference on local trials and greater local interference on global trials.

On each trial, participants were presented with a compound stimulus consisting of a global letter formed by the configuration of local letters in one of four vertically aligned positions (See Figure 6a). The global and local letters were either consistent (e.g., global S, local Ss) or inconsistent (e.g., global H, local Ss). Participants responded to either the local aspect or the global aspect in blocks of 48 trials. The order of the blocks for all participants was local, global, global, and local. Each participant was tested twice for a total of 384 trials. The vertical position of the compound letter was varied so that participants could not focus on an area of the screen during the local trials. The letter remained visible until participants made a key press indicating which letter was presented at the attended level. The fixation point was presented for 600 ms after each response. Five practice trials preceded the initial global block and the initial local block in each set of four blocks. Response times for incorrect trials were not included when response time averages were computed.

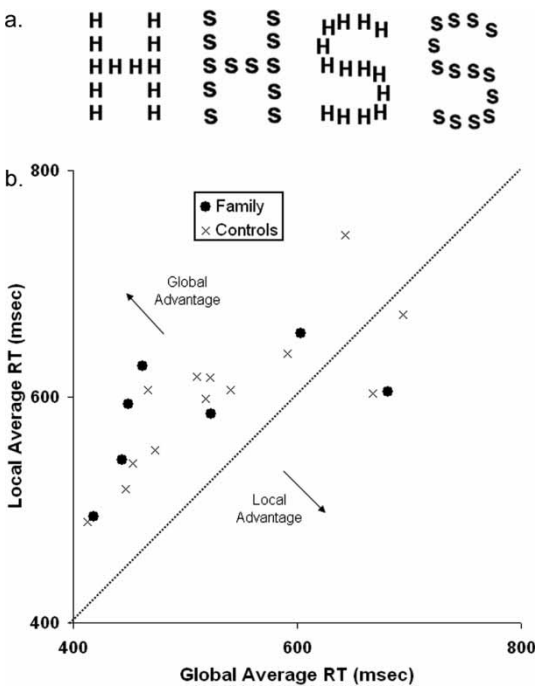

Figure 6. *Global–local task. (a) Examples of compound stimuli used in the global–local task. (b) Average response times for individual controls and siblings. Individuals with symbols above the dotted line were faster with global discriminations while those below it were faster with local discriminations.*

Figure 6b shows average response times for controls and the 7 siblings. Controls were 14 participants between the ages of 24 and 45 (average age = 32.9 years). As expected, global RTs were faster than local RTs, $F(1, 18) = 22.5$, $p < .001$. The figure shows that the siblings' RTs for the task were very similar to those of the controls, $F(1, 18) = 0.28$, *ns*. The siblings showed a normal global advantage and typical global interference on local discriminations. Percentage correct was high for controls and siblings. These results provide further confirmation that the family's deficits do not affect all aspects of visual cognition. In addition, they show that a general psychomotor slowness does not account for their longer RTs in previous tasks and that general global deficits do not account for their prosopagnosia.

To examine whether the family members' performance on the global–local test predicted their face scores, we computed each participant's global–local bias. Average global RT, which was (global consistent RT + global inconsistent RT)/2, was divided by average local RT, which was (local consistent RT + local inconsistent RT)/2. Values below 1 indicate a global bias while values above 1 indicate a local bias. Correlations with the CFMT (.02) and the CFPT upright (−.41) provided no support for the hypothesis that the family members' prosopagnosia results from a local bias.

Discussion

Our results demonstrate a familial aggregation of facial identity perception and recognition deficits in individuals with normal acuity and normal cognitive abilities. Their deficits strongly point to the existence of a genetic condition that selectively affects the development of the neurocognitive mechanisms necessary for visual recognition. This condition even has selective effects within

higher level vision, with facial emotion recognition and global–local processing unaffected. Investigations into the neural basis of visual recognition suggest that the likely locus of the family's deficit lies in ventral visual areas (Grill-Spector et al., 1999; Kanwisher, McDermott, & Chun, 1997; Perrett, Rolls, & Caan, 1982), though not those involved with emotion recognition (Haxby, Hoffman, & Gobbini, 2000).

Two studies based on self-report data have suggested that an autosomal dominant gene may lead to prosopagnosia (Grueter et al., in press; Kennerknect et al., 2006). The distribution of recognition deficits in this family can be accommodated by such an account, but because both parents are prosopagnosic our results are consistent with a variety of possibilities. Some siblings scored better (F23, F38, F43) than other siblings on a number of face and object tests. This variability hints that the genetic basis is not the same in all siblings, possibly due to distinct contributions of the deficit from affected parents.

The results from the object memory tests demonstrate that the siblings' prosopagnosia is also accompanied by within-class object recognition deficits. Our evidence does not allow us to determine the basis of this association. Deficits to a mechanism necessary for face and object recognition could account for the results. However, areas showing face-selective activations (Kanwisher et al., 1997; McCarthy, Puce, Gore, & Allison, 1997) are close to object-selective areas (Grill-Spector et al., 1999; Reddy & Kanwisher, 2006) so events leading to developmental problems in one area are likely to often affect the other area. That genetic prosopagnosia can occur without object recognition problems is suggested by unpublished data from members from two families with fewer affected members (two and three). Despite face perception and recognition deficits comparable to those of the more severely affected members of this family, their scores on nearly all of our object recognition tests are normal.

The results also shed light on issues concerning the organization and development of face processing. First, the clear distinction between the family members' deficits with facial identity perception and normal performance with emotion recognition support models proposing that facial identity and facial emotion are processed by separate mechanisms (Bruce & Young, 1986; Haxby et al., 2000) and are inconsistent with unitary models (Calder & Young, 2005). Because these cases are developmental in origin, this dissociation indicates that different developmental processes are involved in the creation of these separable mechanisms. Second, the siblings' normal performance on the global–local task demonstrates that their prosopagnosia does not result from general deficits with global processing (Behrmann et al., 2005). Similar results were seen for another group of 14 DPs (Duchaine, Yovel, & Nakayama, in press), so it appears that deficits in nonface global processing is not a common cause of developmental prosopagnosia.

Given previous findings relevant to theories of the development of face recognition, it has seemed likely that familial aggregations of prosopagnosics exist. For example, infantile cataracts, which are heritable (Graw, 2004), lead to impaired face processing (Le Grand et al., 2001). Genetic conditions can also increase the likelihood of autism (American Psychiatric Association, 2000), a disorder often accompanied by face processing deficits (Bird, Catmur, Silani, Frith, & Frith, 2006; Hefter, Manoach, & Barton, 2005). However, none of our participants had low-level visual problems early in life, and all were social individuals who showed no signs of autistic traits. Instead, their impairments appear to result from a genetic condition primarily affecting high-level face and object recognition mechanisms.

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APPENDIX

	Control ave	*SD*	*n*	*F39*	*F23*	*F35*	*F38*	*F43*	*F30*	*M33*	*M66*	*F65*	*M64*
Facial identity memory, facial identity perception, and facial emotion recognition: Figure 2													
Famous faces identified	53.3	4.0	20	6	30	14	37	34	15	15	34	24	45
Famous faces exposure	58.6	1.9	20	47	43	53	56	53	41	43	41	35	56
CFMT	59.6	7.6	20	39	46	31	46	41	40	29	34	39	38
CFPT Up	36.7	12.2	21	66	54	68	64	58	60	58	58	60	
CFPT Inv	65.0	9.8	21	88	80	60	82	66	66	90	84	68	
Eyes Test	26.2	3.6	122	28	25	30	27	24	26	23	29	25	
Old New Discriminations: Figure 3													
Faces A′	0.96	0.02	21	0.66	0.97	0.79	0.93	0.95	0.89	0.79			
Faces RT	998	211	21	1611	3137	2352	1527	2871	1952	1981			
Cars A′	0.94	0.04	22	0.69	0.88	0.88	0.89	0.83	0.87	0.84			
Cars RT	1190	389	22	1400	2240	2111	1349	2547	1272	1933			
Guns A′	0.91	0.04	20	0.77	0.91	0.73	0.87	0.89	0.87	0.85			
Guns RT	1239	319	20	1287	2611	2051	1398	2630	1099	1643			
Global-local task: Figure 4													
Global consistent RT	507	77	13	444	435	452	395	494	622	574			
Global inconsistent RT	560	105	13	484	453	446	440	551	741	633			
Local consistent RT	565	67	13	573	500	544	436	519	551	638			
Local inconsistent RT	635	68	13	681	588	642	553	652	657	673			
Global consistent %	99.0	1.0	13	94.8	96.9	95.8	97.9	99.0	100	100			
Global inconsistent %	96.2	3.0	13	92.7	94.8	93.7	92.7	96.9	97.9	91.7			
Local consistent %	99.7	0.7	13	96.9	99.0	97.9	100	100	100	99.0			
Local inconsistent %	97.0	2.7	13	81.3	92.7	87.0	82.3	96.9	95.8	92.7			
Non-visual cognitive tests													
NART	38.8	15	182	45	40	45	47	42	16	38			
Digit Span – Forward	6.6	1.4		6	6	8	8	6	4	8			
Digit Span – Backward	4.9	1.4		3	3	5	6	4	4	5			
Advanced Matrices	≈9	≈1	84	11	12	8	11	9	12	12			