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Perceptual and anatomic patterns of selective deficits in facial identity and expression processing

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ABSTRACT

Whether a single perceptual process or separate and possibly independent processes support facial identity and expression recognition is unclear. We used a morphed-face discrimination test to examine sensitivity to facial expression and identity information in patients with occipital or temporal lobe damage, and structural and functional MRI to correlate behavioral deficits with damage to the core regions of the face-processing network. We found selective impairments of identity perception in two patients with right inferotemporal lesions and two prosopagnosic patients with damage limited to the anterior temporal lobes. Of these four patients one exhibited damage to the right fusiform and occipital face areas, while the remaining three showed sparing of these regions. Thus impaired identity perception can occur with damage not only to the fusiform and occipital face areas, but also to other medial occipitotemporal structures that likely form part of a face recognition network. Impaired expression perception was seen in the fifth patient with damage affecting the face-related portion of the posterior superior temporal sulcus. This subject also had difficulty in discriminating identity when irrelevant variations in expression needed to be discounted. These neuropsychological and neuroimaging data provide evidence to complement models which address the separation of expression and identity perception within the face-processing network.

The face is a source of multiple types of visual information, including identity of the person, expression, gaze direction, age, and gender, among others (Barton, 2003; Palermo & Rhodes, 2007; Posamentier & Abdi, 2003). Deriving these different forms of facial information may involve different types of analyses (Gosselin & Schyns, 2001; Joyce, Schyns, Gosselin, Cottrell, & Rossion, 2006), and these in turn may rely on different anatomic substrates. In particular, the perception of facial identity and facial expression are considered strong candidates for independent processing (Bruce & Young, 1986; Haxby, Hoffman, & Gobbini, 2000). Identity recognition may require analysis of temporally invariant properties of the face, so that it can be recognized regardless of short term variations in expression and long term variations from aging, whereas expression recognition may require analysis of the dynamic properties of the face, ignoring static structural properties so that expression judgments can be generalized across different individuals (Haxby et al., 2000). Independence of expression and identity processing is a prominent aspect of leading cognitive (Bruce & Young, 1986) and anatomic (Haxby et al., 2000) models, although others question the degree of independence for processing of these facial dimensions (Calder & Young, 2005).

Evidence from neuroimaging and neuropsychological studies has been used both to support and question this proposed independence of identity and expression processing. Regarding identity, the fusiform face area (FFA), the first region identified as showing preferential activation for faces (Kanwisher, McDermott, & Chun, 1997; Puce, Allison, Gore, & McCarthy, 1995; Sergent, Ohta, & MacDonald, 1992) shows release from adaptation in functional magnetic resonance imaging (fMRI) studies when the identity of the face changes (Andrews & Ewbank, 2004; Gauthier et al., 2000; Grill-Spector & Malach, 2001; Rotshtein, Henson, Treves, Driver, & Dolan, 2005; Winston, Henson, Fine-Goulden, & Dolan, 2004), suggesting that the FFA is encoding information related to identity. Whether the FFA is also involved in expression perception is less clear. While some studies fail to show release from adaptation in the FFA with changes in expression (Winston et al., 2004), others suggest that the presence of (Vuilleumier & Pourtois, 2007) or attention to (Ganel, Valyear, Goshen-Gottstein, & Goodale, 2005) facial

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expression modulates activation in the FFA. In the neuropsychological literature, prosopagnosia, a family of disorders that may affect different stages necessary for face identification, has been associated with expression deficits in some patients (Humphreys, Avidan, & Behrmann, 2007; Stephan, Breen, & Caine, 2006; Sergent & Signoret, 1992), but not others (Duchaine, Parker, & Nakayama, 2003; McNeil & Warrington, 1991; Takahashi, Kawamura, Hirayama, Shiota, & Isono, 1995; Tranel, Damasio, & Damasio, 1988; Young, Newcombe, de Haan, Small, & Hay, 1993). While at least some of these studies have linked fusiform damage to deficits in encoding facial structural properties relevant to identity recognition (Barton, Press, Keenan, & O'Connor, 2002), the anatomic correlates of impaired or spared expression processing in prosopagnosia are not known, particularly since many reports predate the use of functional or even structural MRI analysis.

Even less information is available regarding the substrate of independent expression processing in individuals with normal face processing capabilities. Current models attribute expression perception to the superior temporal sulcus (STS) (Haxby et al., 2000). Adaptation studies have shown that the middle STS exhibits a release from adaptation for changes in expression but not identity, while the posterior STS exhibits a release from adaptation for both identity and expression changes (Winston et al., 2004). Little is known about the effects of STS damage in humans. One case study demonstrated deficits in the perception of gaze direction after damage to the superior temporal sulcus, though neither identity nor expression perception was formally tested (Akiyama et al., 2006). Nevertheless, gaze direction, like expression, is a dynamic property of faces and both have been modeled as important functions of the posterior STS (Haxby et al., 2000). In other patient populations, deficits in expression perception have been linked to a myriad of lesions, including diffuse cortical atrophy (Kurucz, Soni, Feldmar, & Slade, 1980), right posterior hemispheric lesions (Adolphs, Damasio, Tranel, & Damasio, 1996), left posterior hemispheric lesions (Young et al., 1993), and bilateral (Adolphs, Tranel, Damasio, & Damasio, 1994) or unilateral (Brierley, Medford, Shaw, & David, 2004) lesions of the amygdala. Whether these deficits have spared identity processing is not always clear: Young et al. (1993) suggested that right-sided lesions impair both identity and expression processing, while left-sided lesions can lead to selective deficits in expression processing. However, more detailed anatomic analysis was lacking in this report.

One criticism leveled at previous attempts to contrast identity and expression processing is that the different tests used varied in the level of difficulty and in the resources demanded for performance. In particular, tests that require naming of identity versus expression (Barton, 2003; Kurucz, Feldmar, & Werner, 1979) are highly asymmetric in their requirements, given the potentially infinite number of unique identities versus the limited palette of expressions usually tested. Some suggest that there are only six universal emotions, from which all others are derived (Ekman & Friesen, 1971; Ekman, Sorenson, & Friesen, 1969), although others have created tests with more subtle variations of expression (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). Tests that require matching rather than naming of identities (Benton & van Allen, 1972) or facial expressions (Bowers, Blonder, & Heilman, 1991) are subject to a similar criticism. To address these concerns, we designed a test of identity and expression perception that was equated for the level of difficulty, which incorporated similar task demands and experimental design in the assessment of both perceptual functions, and which required only report of perceptual differences, without any naming or semantic associations required for the response.

With this tool we planned to directly test the independence of identity and expression processing proposed by current models of face perception (Bruce & Young, 1986; Haxby et al., 2000), in a series of patients with occipital or temporal damage. Our first aim was to determine whether selective deficits in identity and expression perception could be found in such patients. Our second aim was to correlate these perceptual deficits with the locus of their damage. We used structural MRI to determine the anatomic extent of the lesions, and fMRI to determine the impact of these lesions on specific face processing regions. fMRI is a powerful tool for relating functional damage to behavioral deficits, and for testing hypotheses generated from anatomic models, yet only a few cases of impaired identity perception following brain-damage have been studied with fMRI (Rossion et al., 2003; Steeves et al., 2006). We used fMRI to localize the core components of the face processing network, the FFA, posterior STS, and occipital face area (OFA; a region in the lateral occipital lobe also shown to preferentially respond to faces, Haxby et al., 2000; Kanwisher et al., 1997), to test the hypotheses that (i) selective damage to the FFA would be associated with a selective deficit of identity processing, and (ii) selective damage to the STS would be associated with a selective deficit of expression processing.

1. Methods

1.1. Participants

Five brain-damaged patients were included in the study (none of these patients has been included in previous reports). Two were selected because they presented with right occipitotemporal damage suggesting they may have damage to regions commonly associated with identity perception (FFA, OFA; Haxby et al., 2000). Two were selected because they complained of problems with face recognition although they presented with damage restricted to the anterior temporal lobes. Finally, one patient was selected because he presented with damage to the right superior temporal lobe, an area thought to be involved in the perception of facial expression (STS; Haxby et al., 2000). All testing was performed within a one week period for three of the patients with the remaining two patients (B-AT1 and R-AT1; see below for the description of naming) returning approximately 6–9 months after neuropsychological testing to perform the behavioral and imaging portions of the present study.

Sixteen right-handed healthy participants (8 females; mean age ± SD: 25.7 ± 5.9 years; range: 18–38 years) with normal or corrected-to-normal vision and no history of neurological disorders participated in standardization of the morphed-face discrimination test. Additionally, six older controls (2 females; mean age ± SD: 62.7 ± 7.5 years; range: 49–69 years), performed the morphed-face discrimination test to ensure impairments in older brain-damaged patients could not be attributed to an age-related decline in performance. The protocol was approved by the institutional review boards of the University of British Columbia and Vancouver General Hospital, and informed consent was obtained in accordance with The Code of Ethics of the World Medical Association, Declaration of Helsinki (Rickham, 1964).

1.1.1. Neuropsychological testing

All five patients had a neurological and neuro-ophthalmological exam, and participated in a neuropsychological assessment for the evaluation of general cognitive skills (Table 1). This assessment included tests of visuo-perceptual functioning/object recognition – Hooper's Visual Organization Test (Hooper, 1957), mental imagery – mental rotation test (Grossi, 1991), and visuospatial attention – star cancellation task (Wilson, Cockburn, & Halligan, 1987) and a visual search task (Spinnler & Tognoni, 1987). Memory was assessed with the Digit Span forward, Spatial Span forward, and Word List immediate recall taken from the Wechsler test (Wechsler, 1999), and with the Words portion of the Warrington Recognition Memory Test (Warrington, 1984). General intelligence, executive functioning and basic perceptual processing were assessed with the Full Scale IQ from the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999) and with the Trail Making Test, A and B (Reitan, 1958). The purpose of the neuropsychological assessment was to control for more general cognitive impairments that might impact performance on our experimental test.

A series of standard neuropsychological tests were then administered to assess the status of face perception in these five patients. The perception of facial identity was assessed with the Benton Facial Recognition Test (Benton & van Allen, 1972), and with the Identity Discrimination portion of the Florida Affect Battery (Bowers et al., 1991). The perception of facial expression was assessed with the Affect Discrimination, Affect Naming, Affect Selection, and Affect Matching portions of the Florida Affect Battery (Bowers et al., 1991) and with the revised version of the Reading the Mind in the Eyes Test (Baron-Cohen et al., 2001). Finally, facial memory and recognition were assessed using the Faces portion of the Warrington Recognition Memory Test (Warrington, 1984), a famous face recognition test (Barton, Cherkasova, & O'Connor, 2001), and a facial imagery test (Barton & Cherkasova, 2003). Importantly the famous face recognition test included a series of

Table 1
Neuropsychological assessment. Raw scores are reported. (WRMT = Warrington Recognition Memory Test; WASI = Wechsler Abbreviated Scale of Intelligence; FAB = Florida Affect Battery.)

Modality	Test	Max	R-IOT1	R-IOT2	B-AT1	R-AT1	R-ST1
Visuo-perceptual	Hooper visual organization	30	27	19	20	25	25.5
Imagery	Mental rotation	10	10	9	10	10	10
Attention	Star cancellation	54	54	52	54	54	54
	Visual search	60	54	54	59	59	53
Memory	Digit span – forward	16	12	12	12	10	9
	Spatial span – forward	16	9	9	10	8	8
	Word list	48	28	30	27[a]	37	32
	Words, WRMT	50	41	49	45	41	47
Intelligence	Full scale IQ, WASI	160	132	100	101	104	114
	Trails A (s)	–	39	45	18	33	37
	Trails B (s)	–	61	107	25	59[a]	99
Faces – identity	Benton Facial Recognition	54	45	38[a]	45	41	50
	Identity discrimination, FAB	20	19	20	20	17[a]	18
Faces – expression	Affect discrimination, FAB	20	19	19	17	20	17
	Affect naming, FAB	20	17	17	18	20	15
	Affect selection, FAB	20	19	18	20	19	18
	Affect matching, FAB	20	18	15	17	20	15
	Reading the mind in the eyes	36	26	28	24	19[a]	21
Faces – memory	Faces, WRMT	50	33[a]	31[a]	27[a]	17[a]	33[a]
	Famous face recognition (d')	3.92	1.96	2.03	1.52[a,b]	1.22[a]	1.96
	Face imagery (%)	100	82	86	N/A	71[a]	88

[a] Impaired performance as determined by standardized norms published with individual tests..
[b] Due to poor knowledge of celebrities, a version of this test using personally familiar faces was given to AT1.

semantic controls in which subjects identified familiar names, matched familiar names to occupations and provided semantic information about famous names (Barton et al., 2001). This ensured that any impaired performance on famous face recognition could not be attributed to deficits in semantic memory. All results from patient testing were compared to published norms for each of the neuropsychological tests to determine impaired performance.

1.1.2. Patient descriptions

R-IOT1 (R = right; IOT = inferior occipitotemporal) is a 49 year-old left-handed male who, twelve years prior to testing, had an occipital cerebral hemorrhage from rupture of an arteriovenous malformation. Immediately following this event he complained of trouble recognizing hospital workers and needed to rely on hairstyle, facial hair, or voice for person recognition, a problem that persists. He also has a left superior quadrantanopia (with 20/20 vision in the remaining visual field) and mild topographagnosia (difficulty navigating in new locations). R-IOT1's self report also suggested letter-by-letter reading immediately following the cerebral hemorrhage, although this had resolved by time of testing. R-IOT1 showed normal performance on all neuropsychological tests, including the Benton Facial Recognition Test; famous face recognition and facial imagery, with the exception of the Faces portion of the Warrington Recognition Memory Test (33/50; Table 1).

R-IOT2 is a 65-year-old right-handed male who, two and a half years prior to testing, had a right posterior cerebral artery infarction. This stroke resulted in a left superior quadrantanopia not affecting the central 10°, but 20/20 acuity. He does not complain of problems with face recognition or topographical orientation, and neurological examination did not show any difficulties in language or color perception. R-IOT2 exhibited normal performance on most neuropsychological tests, but showed impaired performance on the Benton Facial Recognition Test (38/54), a test of facial identity perception, and on the Faces portion of the Warrington Recognition Memory Test (31/50; Table 1).

B-AT1 (B = bilateral; AT = anterior temporal) is a 24-year-old right-handed male. Three years prior to testing, he had herpes simplex encephalitis and was comatose. Since recovery, he has noted extreme difficulty in recognizing faces and learning new faces, though he can recognize some family members. General memory and mental functioning is unaffected, allowing him to attend college and hold full-time employment. Visual fields are normal and acuity is 20/20 in both eyes. He has mild topographagnosia and mild anomia for low-frequency items (although semantic knowledge of these items is evident). He performed normally on most neuropsychological tests (Table 1) but was severely impaired on the Faces portion of the Warrington Recognition Memory Test (27/50). B-AT1 performed poorly on the semantic control task for famous faces indicating problems with access to semantic information and as a result, this invalidated the famous face recognition and facial imagery tests. However on a modified version of the recognition task he showed poor facial recognition for family members of whom he could provide reliable semantic information (d' = 1.52). Impaired performance on the Word List immediate recall was also observed (27/48), while performance on all other memory tests, including the Word portion of the Warrington Recognition Memory Test was normal.

R-AT1 is a 24-year-old right-handed female. One year prior to testing she had a selective right amygdalohippocampectomy for epilepsy. The surgery was successful, with only one reported seizure in the following year, but she has since noted difficulty recognizing faces, needing to rely on voice or other means to recognize familiar individuals. General mental functioning is intact: she is currently attending university, although she reports problems with visual memory, relying on verbal strategies to study. She has normal visual fields with 20/20 visual acuity. On tests of intelligence, performance was mildly impaired on Trails – Test B (59 s), but was normal on Trails – Test A and the more comprehensive Full Scale IQ test. She was impaired on the Identity Discrimination portion of the Florida Affect Battery (17/20) but showed normal performance on the more difficult items with changes in lighting and viewpoint on the Benton Facial Recognition Test. She was impaired on the Faces portion of the Warrington Recognition Memory Test (17/50), the famous face recognition test (d' = 1.22) and the facial imagery test (71% accuracy). For expression, she showed normal performance on all Affect portions of the Florida Affect Battery, but was impaired on the Reading the Mind in the Eyes Test (19/36; Table 1).

R-ST1 (ST = superior temporal) is a 57-year-old right-handed male. Four years prior to testing he had a right middle cerebral artery infarction, causing immediate left-sided loss of sensation and paralysis that persisted for only a few days. He still notes clumsiness of the left hand and tripping over the toes of the left foot. He had left hemineglect for 4 months after the stroke. At present he does not have any problems with language, color perception, topographical orientation, or face recognition, although he does note trouble recognizing voices over the phone. Visual fields are unaffected, and acuity is 20/20 in both eyes. He was normal on all neuropsychological tests except for the Faces portion of the Warrington Recognition Memory Test (33/50; Table 1).

1.2. The morphed-face discrimination test

1.2.1. Stimuli

Angry and fearful images of two male identities (BM01, BM28) were selected from the Karolinska Database of Emotional Faces (Lundqvist & Litton, 1998). Background, hair, ears, and neck were removed, while external jaw contour was preserved, using Adobe Photoshop CS2 9.0.2 (http://www.adobe.com). Distinguishing marks such as moles were removed using the Spot Healing Brush Tool. Images were cropped to ensure that faces were centrally located within the image frame, and resized to a standard width of 400 pixels. A morph series of 21 images in 5% morph steps was created between the two angry faces using Abrosoft Fantamorph 3.0 (http://www.fantamorph.com). This process was repeated for the two fearful faces. Twenty-one morph series were then created between corresponding images from these angry and fearful morph series (i.e. – Angry1-Fearful1, . . ., Angry21-Fearful21), to create a 21 × 21 morph matrix (441 images total) with orthogonal axes representing identity and expression. Images included in all versions of the morphed-face discrimination test were selected from this morph matrix (see Section 1.2.2).

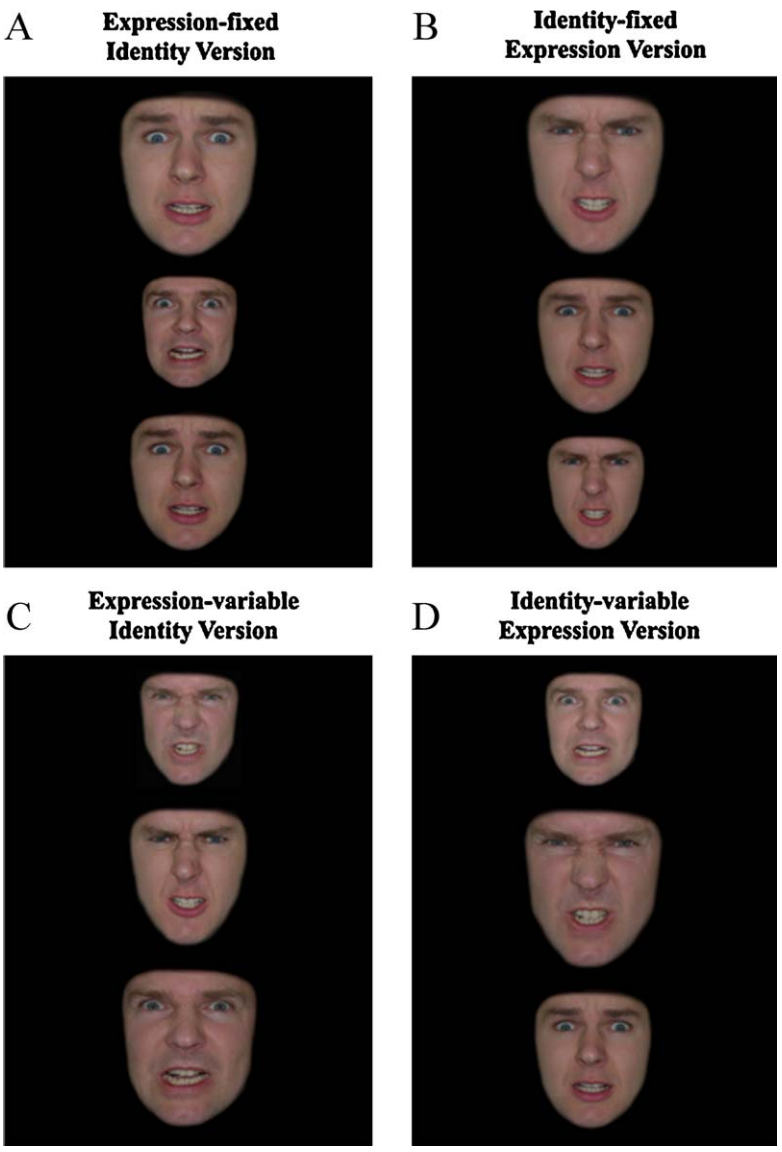

Fig. 1. Sample trials from each of the four versions of the morphed-face discrimination test. (A) *Expression-fixed Identity Version*; 70% Morph Distance. (B) *Identity-fixed Expression Version*; 50% Morph Distance. (C) *Expression-variable Identity Version*; 90% Morph Distance. (D) *Identity-variable Expression Version*; 70% Morph Distance. The middle face is the correct choice in all examples. Test images were selected from the Karolinska Database of Emotional Faces (Lundqvist & Litton, 1998) the images presented in Fig. 1 were selected from the author collection for presentation purposes only.

1.2.2. Experimental design

The three faces in each trial were presented in a vertical arrangement at the midline of a black screen (Fig. 1) to minimize the impact of any hemifield visual defects or horizontal hemineglect that may be present in brain-damaged patients. Size was varied across the three faces ($3.8^\circ \times 4.9^\circ$, $3.3^\circ \times 4.2^\circ$, $2.8^\circ \times 3.5^\circ$; 56 cm viewing distance) to minimize the contribution of a strategy wherein correct responses were generated by matching low-level image properties. The arrangement of facial sizes was randomized across trials. The screen location of the target face was balanced, resulting in an equal number of target faces in each of the three possible locations (top, middle, bottom), for each level of morph distance.

The amount by which the target face differed from the other two faces varied from 10% to 100% morph distance, with both target and non-target faces centered around the 50:50 midpoint of the matrix: thus, the 10% morph distance required discriminations between the 55/45% and 45/55% morph faces, while the 100% morph distance required discriminations between the 100/0% and 0/100% morph faces.

The irrelevant facial dimension (i.e. expression during *Identity* versions of the test, and identity during *Expression* versions of the test) was held constant within each trial of the '*-fixed*' test versions. However, to ensure that participants did not become familiar with any particular face, the level of this irrelevant dimension was randomly varied between trials. During the '*-variable*' test versions, the irrelevant dimension randomly varied, both between the three stimuli on any given trial, and also between trials.

1.2.3. Procedure

Each participant performed all four test versions. In the *Identity* versions participants were instructed to find the face with the different identity in each set of three faces; in the *Expression* versions they were asked to find the face with the different expression. They were told to ignore any changes in the size or the irrelevant dimension of the face, and to indicate their selection with a key press as quickly as possible. The four test versions were presented in random order. Short rest breaks were provided between test versions and appropriate instructions were given prior to each version. Experimental trials were displayed until the participant made his or her response and were followed by a black screen for 500 ms, which served as the inter-trial-interval. Trials were presented on a 17″ widescreen Compaq nx9600 notebook using SuperLab Pro 2.0.4 (http://www.cedrus.com).

Our strategy was to use a full version of the test in the 16 younger controls, with 12 trials at each of the 10 morph-difference levels, for a total of 120 trials per version, and 480 trials in total. From these results we would select a set of morph-difference levels for each of the four tests that would create a shorter test for the patients, in which level of perceptual difficulty was equated across all four tests, with performance not at ceiling but at a high level of accuracy and with low variance across the control group.

1.2.4. Analysis and results of younger control data

For the data from the 16 younger controls, we performed a general linear model (GLM) with test version (Expression-fixed Identity Version, Identity-fixed Expression Version, Expression-variable Identity Version, Identity-variable

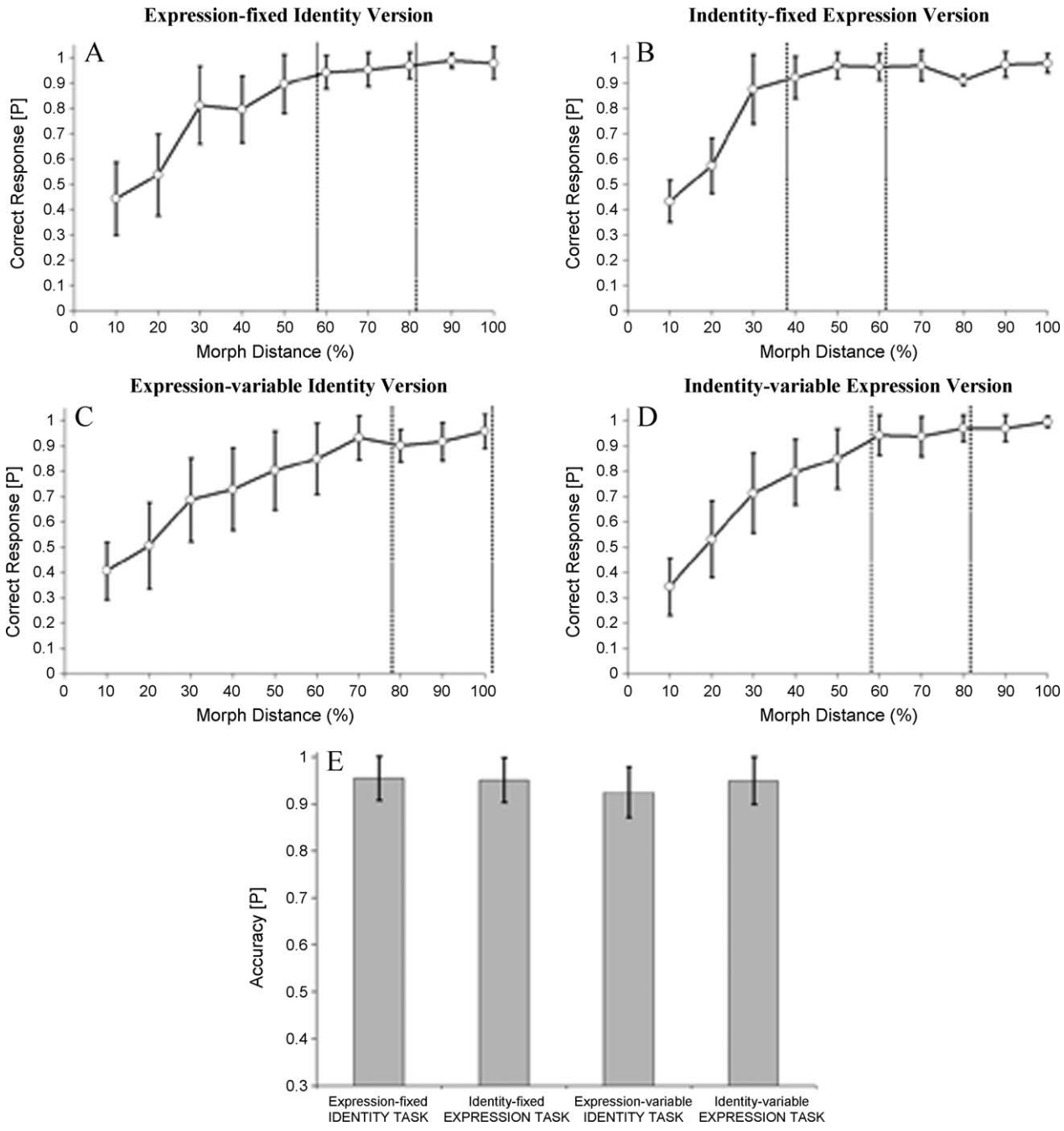

Fig. 2. Control data from the full presentation of the four tasks; Mean correct ± SD. (A) Expression-fixed Identity Version. (B) Identity-fixed Expression Version. (C) Expression-variable Identity Version. (D) Identity-variable Expression Version. A portion of each version (3 morph distances; within dotted lines) was chosen for patient testing in order to equate difficulty across all four versions of the test. (E) Three morph distances (within dotted lines) were chosen to create balanced versions of the morphed-face discrimination test. Collapsed accuracy ± standard deviation is presented for the four balanced versions of the test.

Expression Version) and morph-difference (10%, 20%, . . ., 100%) as fixed factors, subject as a random factor and proportion correct as the dependent measure. A significant effect of test version or an interaction between test version and morph distance would indicate a differing level of difficulty across the different test versions. Linear contrasts were used to examine all significant interactions. Significance was set at $\alpha < 0.05$ on all statistical tests. The general linear model (GLM) based on the full set of behavioral results revealed a significant main effect of test version [$F(3,45) = 11.77$; $p < 0.001$]. All four test versions differed significantly from each other ($p < 0.05$, all comparisons). Overall, the Identity-fixed Expression Version was the easiest (Mean proportion correct ± SEM; 0.87 ± 0.02), followed by the Expression-fixed Identity Version (0.83 ± 0.02), the Identity-variable Expression Version (0.80 ± 0.02), and the Expression-variable Identity Version (0.77 ± 0.02), which was the most difficult. A significant main effect of morph distance was also observed [$F(9,135) = 224.21$, $p < 0.001$], with difficulty decreasing as morph distance was increased. Finally, there was a significant interaction between test version and morph distance [$F(27,405) = 2.78$, $p < 0.001$]. Significant differences between two or more test versions were seen at each morph distance except the 20% ($p > 0.20$, all tests) and 70% ($p > 0.10$, all tests) morph distances (Fig. 2A–D). Thus, the initial results from our morphed-face discrimination test showed that Identity versions of the test were more difficult than Expression versions [Expression-fixed Identity version v. Identity-fixed Expression version – $t(159) = -2.49$, $p < 0.05$; Expression-variable Identity version v. Identity-variable Expression version – $t(159) = -3.00$, $p < 0.01$]. Interference effects (Ganel, Goshen-Gottstein, & Ganel, 2004) were also demonstrated by an increased difficulty when the irrelevant dimension varied [Expression-fixed Identity version v. Expression-variable Identity version – $t(159) = 5.38$, $p < 0.001$; Identity-fixed Expression version v. Identity-variable Expression version – $t(159) = 4.71$, $p < 0.001$].

To balance the level of difficulty for the test to be administered to patients, the three morph distances located just before the curves reached asymptotic 'ceiling' performance were selected for each test version separately (Fig. 2A–D, within

dashed lines). This ensured that: (1) the test was not so easy that controls performed with 100% accuracy, in which case some patients with subtle deficits might also appear normal, and (2) the test was not too difficult with control performance either too poor or too variable, in which case 95% prediction intervals would approach the chance accuracy of 33%, making it impossible to demonstrate a deficit in patients. As an illustration of this point, two recent studies of prosopagnosic patients performing a morphed face test showed that peak difference between control and patient accuracy was not the 100% morph level but rather at the 80% morph level, similar to what we use, and the ability to discriminate between the performance of patients and controls deteriorated at more difficult levels (Busigny, Graf, Mayer, & Rossion, 2010, see Fig. 7/Table 5; Busigny, Joubert, Felician, Ceccaldi, & Rossion, 2010, see Fig. 8/Table 4). Thus, for the *Expression-fixed Identity Version* we chose the data for the 60–80% morph distance levels; for the *Identity-fixed Expression Version* the 40–60% morph distance levels; for the *Expression-variable Identity Version* the 80–100% morph distance levels; and for the *Identity-variable Expression Version* the 60–80% morph distance levels). The scores on these three points for each of the test versions in each subject were then compared in a second GLM, with test version (*Expression-fixed Identity Version*, *Identity-fixed Expression Version*, *Expression-variable Identity Version*, *Identity-variable Expression Version*) and morph distance (1, 2, 3) as fixed factors, subject as a random factor and proportion correct as the dependent measure. A significant main effect of morph distance was observed [$F(2,30) = 12.00$; $p < 0.001$], with the upper morph distance (0.96 ± 0.01) slightly easier than the middle (0.94 ± 0.01) or lower (0.93 ± 0.01) morph distances. Importantly, there was now no significant main effect of test version [$F(3,45) = 1.73$; $p > 0.15$] or an interaction between test version and morph distance [$F(6,90) = 0.92$; $p > 0.40$] indicating equivalent level of difficulty across all versions of the test (*Expression-fixed Identity Version* – 0.95 ± 0.01; *Identity-fixed Expression Version* – 0.95 ± 0.01; *Expression-variable Identity Version* – 0.93 ± 0.01; *Identity-variable Expression Version* – 0.95 ± 0.01). By using these portions of the curve, we selected items to create an assessment in which all four versions were (a) equal in the degree of perceptual difficulty, so that any dissociation in performance could not be attributed to variations in task difficulty (Fig. 2E) and (b) generated results with low variance (SD = 0.05) and non-ceiling performance (0.93–0.95) in healthy subjects, increasing our chances of detecting subtle perceptual deficits.

1.2.5. Analysis of patient data

The five patients and six older controls were administered the four shorter, balanced versions of the morphed-face discrimination test. Test versions were administered in random order, with short rest breaks separating each version. Results from each test version were analyzed separately but, due to the lack of interaction between test version and morph distance (see above), accuracy was collapsed across morph distance within each test version. This resulted in a single accuracy score for each of the four versions of the test in each participant. The 95% prediction interval (PI) for each test version was calculated from control data using the following formula:

$$\mathrm{PI}_{95} = X - t_{.05}\left(SD\sqrt{\frac{n+1}{n}}\right)$$

where X represents the mean performance, $t_{.05}$ the one-tailed t value with a significance of $p < 0.05$, SD the standard deviation, and n the number of participants. Patient scores that fell below the 95% PI indicated impaired processing. To provide a measure of the magnitude of impairment, the 99%, 99.9%, and 99.99% PIs were also calculated. Averaged data from the older controls is reported as a separate data point.

1.3. Functional and structural MRI

1.3.1. Apparatus

Structural and functional MRIs were performed on the five patients. All scans were acquired in a 3.0 Tesla Philips scanner. Stimuli were presented using Presentation 9.81 software and were rear-projected onto a mirror mounted on the head coil. Whole brain anatomical scans were acquired using a T1-weighted echoplanar imaging (EPI) sequence, consisting of 170 axial slices of 1 mm thickness (1 mm gap) with an in-plane resolution of 1 mm × 1 mm (FOV = 256). T2-weighted functional scans (TR = 2 s; TE = 30 ms) were acquired using an interleaved ascending EPI sequence, consisting of 36 axial slices of 3 mm thickness (1 mm gap) with an in-plane resolution of 1.875 mm × 1.875 mm (FOV = 240).

1.3.2. Procedure

Two functional localizers were used to identify the six regions comprising the core system of face processing, namely the OFA, FFA, and posterior STS in both right and left hemispheres (Haxby et al., 2000). The first, a standard static face localizer, presented static photographs of objects (e.g. television, basketball) and faces (neutral and expressive) in separate blocks (Kanwisher et al., 1997; Saxe, Brett, & Kanwisher, 2006). Patients performed an irrelevant 'one-back task': that is, to press a button if an image was identical to the previous one. The localizer began and ended with a fixation block showing a cross in the center of an otherwise blank screen. Additional fixation blocks were alternated with image blocks, all blocks lasting 12 s. Six blocks of each image category (object, neutral face, expressive face) were presented in a counterbalanced order. Each image block consisted of 15 images (12 novel and 3 repeated), all sized to a standard width of 400 pixels and presented at screen center for 500 ms, with an inter-stimulus-interval of 300 ms. The second, a dynamic localizer (UBC-HVEM protocol), presented dynamic videos of objects and faces (available for download, email: cjfox@interchange.ubc.ca). The UBC-HVEM protocol was developed in our laboratory and was subsequently shown to increase the ability of localizers to identify all core face-selective regions at the level of individual subjects (Fox, Iaria, & Barton, 2009). Video-clips of faces all displayed dynamic changes in facial expression (i.e. from neutral to happy). So that dynamic changes in objects were comparable to those seen in faces, all video-clips of objects displayed types of motion that did not create large translations in position (i.e. rotating basketball). Patients again performed a one-back task. Fixation blocks began and ended the session and were alternated with image blocks, all blocks lasting 12 s. Eight blocks of each image category (object, face) were presented in a counterbalanced order. Each image block consisted of 6 video-clips (5 novel and 1 repeated) presented centrally for 2000 ms each. The UBC-HVEM protocol consisted of video-clips of objects which were gathered from the internet and video-clips of faces which were provided by Chris Benton (Department of Experimental Psychology, University of Bristol, UK) (Benton et al., 2007). All video-clips were resized to a width of 400 pixels.

1.3.3. Analysis

The first volume of each functional scan was discarded to allow for scanner equilibration. All MRI data were analyzed using BrainVoyager QX Version 1.8 (http://www.brainvoyager.com). Anatomical scans were not preprocessed, but were standardized to Talairach space (Talairach & Tournoux, 1988). Preprocessing of functional scans consisted of corrections for slice scan time acquisition, head motion (trilinear interpolation), and temporal filtering with a high pass filter to remove frequencies less than 3 cycles/time course. Functional scans were individually co-registered to their respective anatomical scan using the first retained functional volume to generate the co-registration matrix.

The static localizer time course was analyzed with a single subject GLM, with object (O), neutral (NF) and expressive (EF) faces as predictors, and a NF + EF > 2*O contrast was overlaid on the whole brain. A similar procedure was adopted for the dynamic localizer, the time course of which was analyzed via a single subject GLM with objects (O) and faces (F) as predictors, and a F > O contrast was overlaid on the whole brain. Within each patient we attempted to define, bilaterally, each of the three face-related regions comprising the core system of face perception (Haxby et al., 2000). Contiguous clusters of face-related voxels located on the lateral temporal portion of the fusiform gyrus were designated as the fusiform face area (FFA), while clusters located on the lateral surface of the inferior occipital gyrus were designated as the occipital face area (OFA). Face-related clusters located on the posterior segment of the superior temporal sulcus were designated as the posterior STS. For the present study, we did not perform a similar analysis of components of the extended face network (e.g. middle STS, inferior frontal gyrus, precuneus, amygdala) because the sensitivity of the dynamic face localizer for detecting these regions is on average only around 69%, compared to 98% for the core network (Fox, Iaria, et al., 2009). With only moderate sensitivity, it is difficult to make firm deductions about the status of a region from the BOLD signal of a single subject.

To avoid false negatives in the localization of regions-of-interest we employed multiple statistical thresholds within each patient's analysis (all with a minimum cluster size of 50 voxels). First, a threshold of $p < 0.05$ (1-tailed Bonferroni, corrected for multiple comparisons) was applied to the static localizer. Failure to localize all possible regions-of-interest (excluding regions located in areas of lesion) resulted in lowering this threshold to a more liberal False-Discovery-Rate of $q < 0.05$ corrected for multiple comparisons. If localization was still unsuccessful this process was repeated, using data from the more robust dynamic localizer (Fox, Iaria, et al., 2009). In our prior study the static localizer using the Bonferroni threshold, failed to detect 28% of core face-selective regions in healthy subjects, whereas the dynamic localizer found 98% of regions with the same Bonferroni threshold, dramatically reducing the likelihood of a false negative (Fox, Iaria, et al., 2009). The most conservative threshold that identified all possible regions-of-interest was used to report cluster values in that particular patient.

2. Results

2.1. Behavioral findings

Using the morphed-face discrimination test we found selective deficits in identity perception in four of the five patients (R-IOT1, R-IOT2, B-AT1, R-AT1; Fig. 3). All four patients were impaired on both *Identity* versions of the test – i.e. regardless of whether expression was kept constant or allowed to vary across the stimuli. In contrast all four patients performed within the normal range on both *Expression* versions of the test. Thus these four patients demonstrated selective deficits of identity perception with spared expression perception on a morphed-face discrimination test which was balanced for difficulty across the different perceptual tasks.

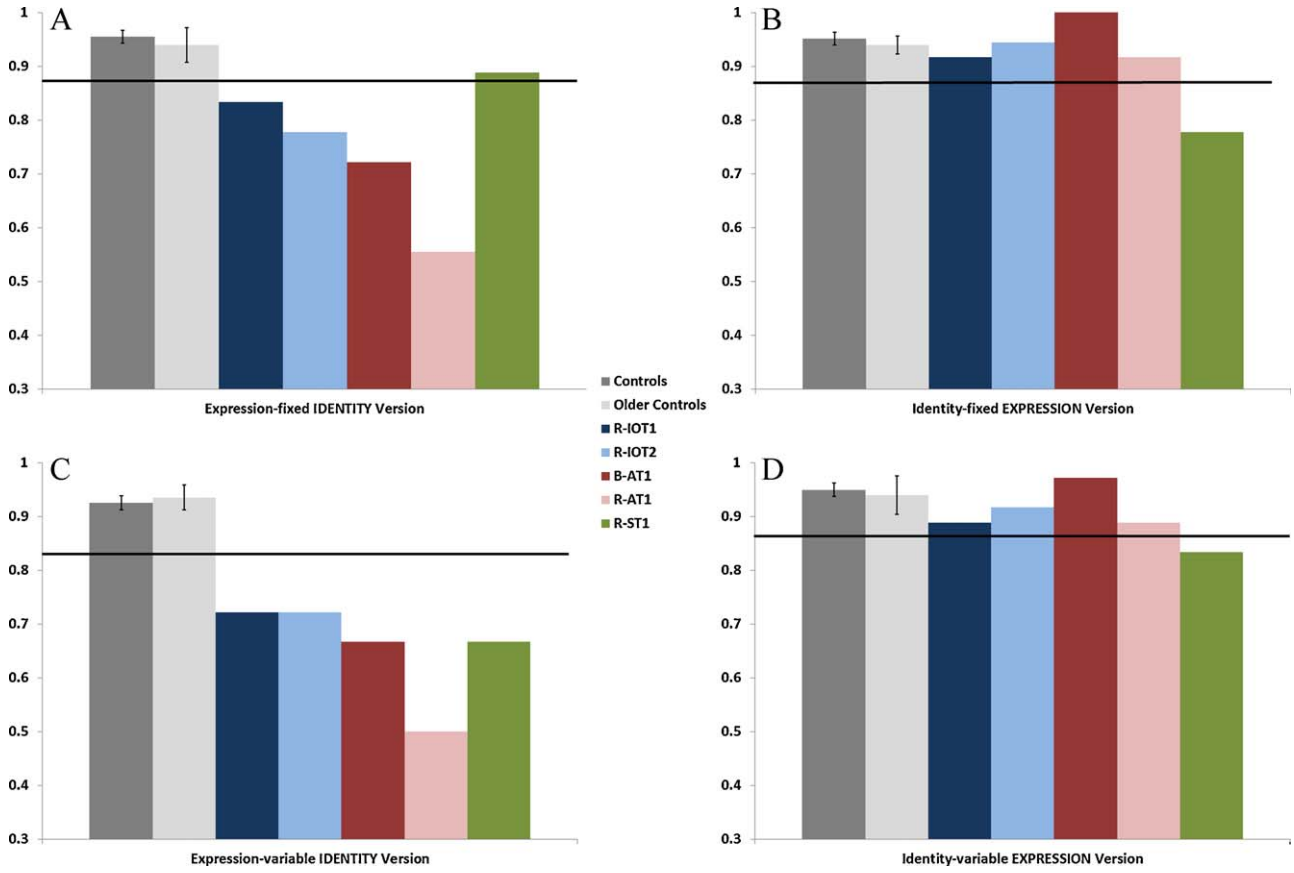

Fig. 3. Results of the morphed-face discrimination test. In all figures the black bar represents the younger control data (mean ± SEM), the gray bar the older controls (mean ± SEM), the dark blue bar R-IOT1, the light blue bar R-IOT2, the dark red bar B-AT1, the light red bar R-AT1, and the green bar R-ST1. Solid lines represent 95% prediction intervals for the respective versions of the test. (A) Performance on the *Expression-fixed Identity Version* of the morphed-face discrimination test. R-IOT1, R-IOT2, B-AT1, and R-AT1 are all impaired on this version of the test. (B) Performance on the *Identity-fixed Expression Version* of the morphed-face discrimination test. R-ST1 is the only patient impaired on this version of the test. (C) Performance on the *Expression-variable Identity Version* of the morphed-face discrimination test. All patients are impaired on this version of the test. (D) Performance on the *Identity-variable Expression Version* of the morphed-face discrimination test. R-ST1 is the only patient impaired on this version of the test. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

The fifth patient displayed a different pattern of results (Fig. 3). R-ST1 demonstrated impairments on both *Expression* versions of the test. However, when performing the identity versions of the test, R-ST1's performance was within the normal range when expression was held constant (*Expression-fixed Identity Version*), yet was markedly impaired when performing the *Expression-variable Identity Version*, a test which required recognition of identity changes across random variations in expression (Fig. 3C). Thus R-ST1 demonstrates a deficit in expression perception, but also has some difficulty with perceiving changes in facial identity when this requires him to simultaneously discount irrelevant variations in the stimuli resulting from a changed expression.

2.2. Neuroimaging correlates

The four patients with selective impairments of identity perception have widely varying lesions (Fig. 4). R-IOT1 has a unilateral right lesion primarily involving the occipital lobe and the posterior portion of the right inferior temporal lobe. The functional localizer identified the left OFA, left FFA, and bilateral pSTS in R-IOT1, but not the right OFA or right FFA, suggesting a possible correlation between functional damage to the core face processing network and selective deficits in identity perception (Table 2, Fig. 5). However, R-IOT2 had a similar medial occipitotemporal lesion, stretching from the pole and medial surface of the occipital lobe to the middle inferotemporal cortex, yet fMRI revealed that the FFA and OFA were intact in this patient (Table 2, Fig. 5). B-AT1 has extensive bilateral temporal damage extending from the anterior poles to the middle and inferior temporal lobes, while R-AT1 has a small lesion in the anterior right temporal lobe, affecting the temporal cortex, hippocampus and amygdala. As with R-IOT2, all six regions of the core face processing network were preserved in these two prosopagnosic patients (Table 2, Fig. 5).

Next, we examined the lesion in R-ST1, who demonstrated a primary deficit in expression perception and an associated deficit in perceiving facial identity across variations in facial expression. R-ST1 has a large right hemispheric lesion extending from the right anterior temporal pole to the posterior temporal lobe, along the superior temporal sulcus, but sparing the right amygdala (Fig. 4 – 0 mm). fMRI showed activation in the right FFA and right OFA but not the right posterior STS; all core regions were identified in the left hemisphere (Table 2, Fig. 5).

3. Discussion

The first aim of this study was to determine if dissociations in impairments of identity and expression perception occurred in brain-damaged patients. To do this we designed and administered tests of identity and expression perception that (a) were equivalent in perceptual difficulty, so that any dissociation in performance could not be attributed to variations in task difficulty (Fig. 2), (b) generated results with low variance and not at ceiling, so that the test was sensitive to subtle deficits, and (c) did not require verbal identification, thus minimizing the contribution of

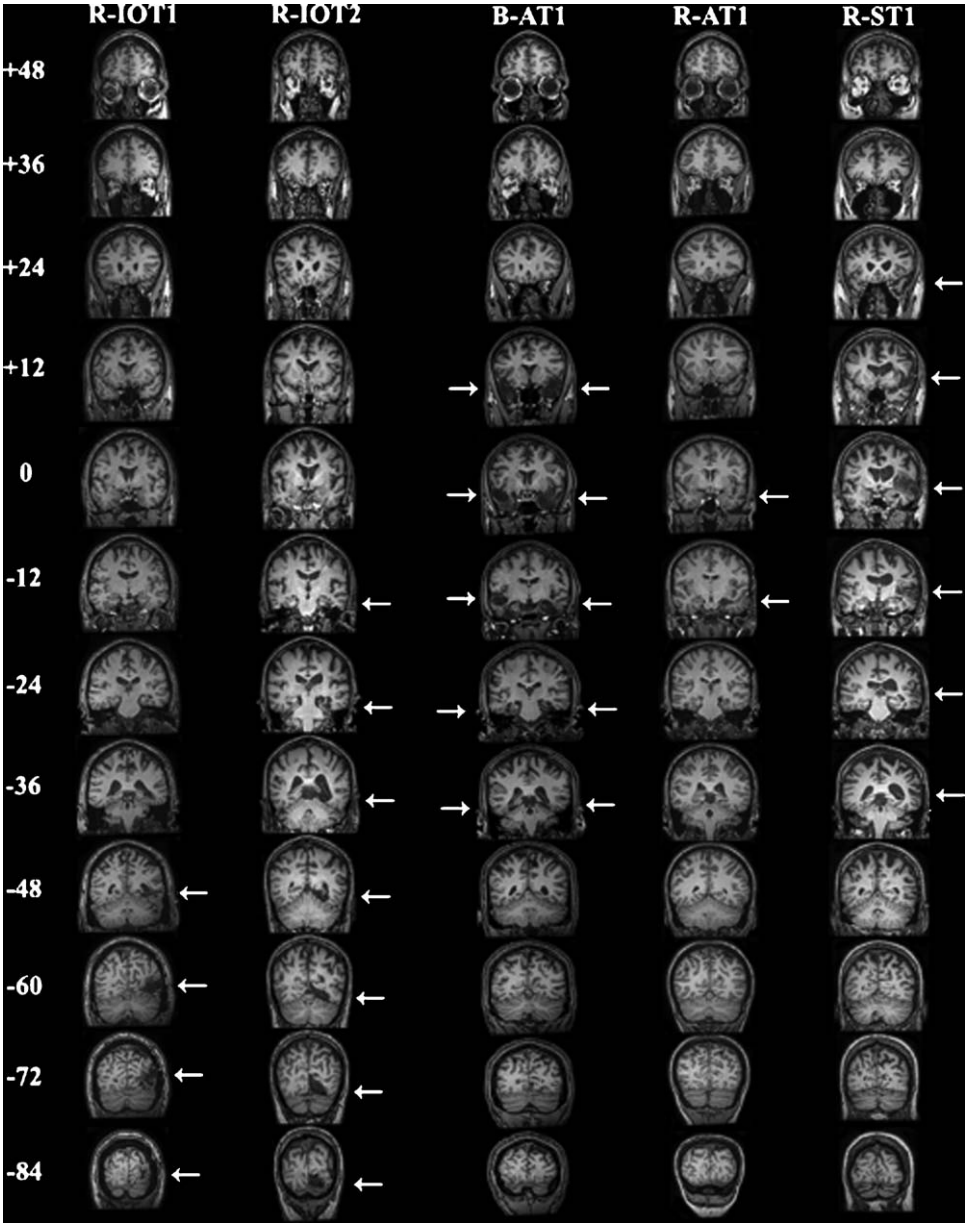

Fig. 4. Coronal slices of the five patients included in this study (standardized to Talairach space). Slices were taken in 12 mm increments from $y = +48$ mm to -84 mm. In R-IOT1, a single right hemispheric infarct stretches from the occipital pole (-84 mm) to the posterior temporal lobe (-48 mm). In R-IOT2, a single right hemispheric infarct stretches from the occipital pole (-84 mm) to the medial aspect of the temporal lobe (-12 mm). In B-AT1, bilateral temporal lesions can be seen stretching from the temporal poles (+12 mm) to the posterior temporal lobe (-36 mm). In R-AT1, a small surgical lesion affecting the right anterior temporal lobe, hippocampus and amygdala can be seen (0 mm, -12 mm). In R-ST1, a single right hemispheric infarct stretches from the temporal pole (+24 mm) along the superior temporal sulcus to the posterior temporal lobe (-36 mm).

semantic associations to the behavioral response and ensuring the test was primarily perceptual in nature. The second aim was to reveal the link between perceptual deficits and the survival or loss of specific components of the core face network following brain damage (Table 3), by using an fMRI-based functional localizer in each patient. This technique of combining behavioral data with functional imaging data in patients has proven to be a powerful tool for refining current cognitive-anatomic models of face perception (Rossion et al., 2003; Steeves et al., 2006).

3.1. Impaired identity perception with posterior damage

We first examined two patients with damage restricted to the right inferior occipitotemporal cortex (R-IOT1 and R-IOT2). Although R-IOT2 does not have prosopagnosic complaints, both had some difficulty on standard neuropsychological tests of identity processing, but not on tests of expression perception. On the morphed-face discrimination test, both patients showed impaired identity perception and intact expression perception (Table 3). This dissociation in perceptual deficits is consistent with prior reports in acquired (McNeil & Warrington, 1991; Takahashi et al., 1995; Tranel et al., 1988; Young et al., 1993) and congenital (Duchaine et al., 2003) prosopagnosia.

Older reports on acquired prosopagnosia are understandably limited in anatomic detail, and the data from congenital prosopagnosia is of limited use for structure–function correlations because of the lack of visible structural lesions. More recently damage to the fusiform gyrus has been identified as a correlate of apperceptive prosopagnosia (Barton et al., 2002; de Gelder, Frissen, Barton, & Hadjikhani, 2003). fMRI has demonstrated prosopagnosia in a

Table 2
Results of the functional localizers, in brains standardized to Talairach space (FDR – False Discovery Rate, $q < 0.05$; BF – 1-tailed Bonferroni, $p < 0.05$). Differences of the peak *t*-value, as compared to a separate control sample (Fox, Iaria, et al., 2009), are presented in units of standard deviation (SD). In all cases the peak *t*-value of ROIs localized in the 5 patients fall within the normal range of variation (i.e. <2SD), excluding the L-OFA in R-AT1 which is significantly larger than the control sample. Right sided regions are highlighted in boldface.

Subject	Localizer	Threshold	Region	Peak *t*-value (ΔSD)	Difference from control peak *t*-value (SD)	Cluster size (voxels)	*X*	*Y*	*Z*
R-IOT1	Dynamic	FDR	**ROFA**	**LESION**					
			RFFA	**LESION**					
			RpSTS	**5.52**	**−1.6**	**146**	**57**	**−40**	**13**
			LOFA	4.98	−1.1	51	−36	−79	−14
			LFFA	6.71	−0.9	281	−33	−67	−23
			LpSTS	6.32	−0.5	785	−57	−28	−2
R-IOT2	Dynamic	FDR	**ROFA**	**4.80**	**−1.3**	**182**	**30**	**−85**	**−17**
			RFFA	**6.33**	**−1.2**	**606**	**33**	**−40**	**−23**
			RpSTS	**9.37**	**−0.4**	**1074**	**45**	**−25**	**−5**
			LOFA	3.92	−1.4	204	−33	−64	−20
			LFFA	5.95	−1.0	168	−42	−49	−32
			LpSTS	5.64	−0.8	517	−51	−25	−5
B-AT1	Dynamic	BF	**ROFA**	**12.37**	**+1.0**	**3956**	**30**	**−88**	**−5**
			RFFA	**13.09**	**+0.9**	**1064**	**39**	**−52**	**−20**
			RpSTS	**9.67**	**−0.3**	**329**	**46**	**−49**	**−2**
			LOFA	9.43	+0.2	1543	−30	−85	−8
			LFFA	5.96	−1.0	57	−39	−55	−26
			LpSTS	5.90	−0.7	50	−60	−46	4
R-AT1	Static	BF	**ROFA**	**10.32**	**+1.1**	**470**	**30**	**−67**	**−17**
			RFFA	**10.42**	**+0.7**	**227**	**36**	**−58**	**−14**
			RpSTS	**8.14**	**+1.1**	**240**	**42**	**−40**	**4**
			LOFA	12.48	+3.0	648	−39	−70	−8
			LFFA	12.35	+1.7	574	−36	−49	−14
			LpSTS	6.27	+0.7	149	−60	−55	1
R-ST1	Dynamic	FDR	**ROFA**	**7.49**	**−0.5**	**1001**	**27**	**−82**	**−11**
			RFFA	**9.36**	**−0.3**	**738**	**33**	**−49**	**−17**
			RpSTS	**LESION**					
			LOFA	6.5	−0.7	828	−39	−85	−2
			LFFA	4.69	−1.3	144	−42	−64	−14
			LpSTS	8.1	−0.8	1497	−48	−46	1

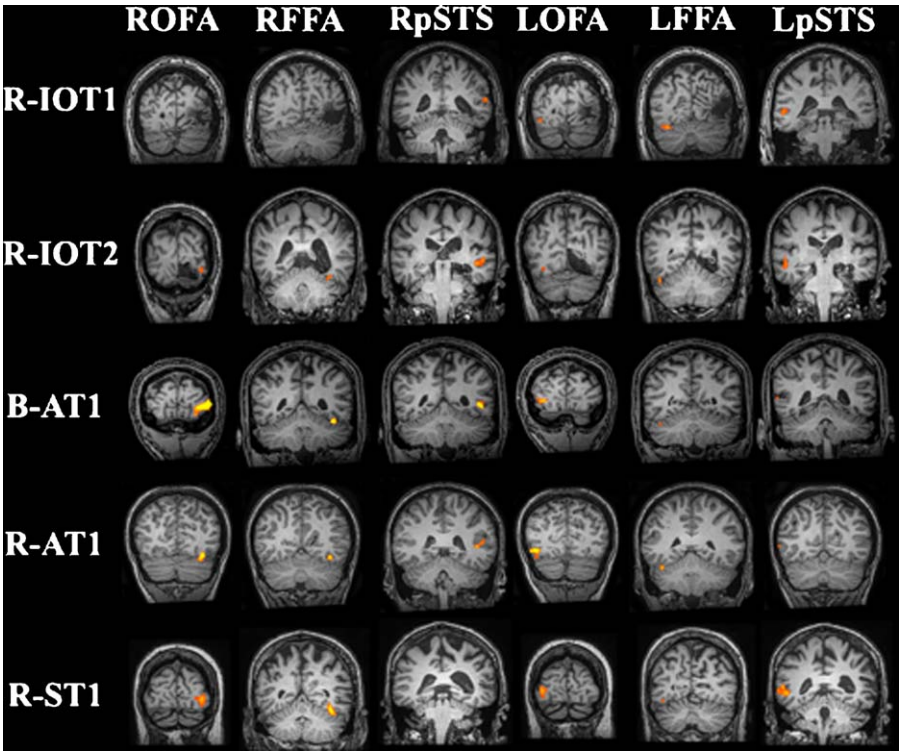

Fig. 5. Core system regions-of-interest identified with the functional localizers (all brains standardized to Talairach space). Due to the location of the lesion, R-IOT1 does not display a right OFA or right FFA. However a right posterior STS (pSTS) was identified along with all three core regions in the left hemisphere. All six core regions were identified in R-IOT2, with the right OFA and FFA located just lateral to the lesion. All six regions of the core system were identified in B-AT1 and R-AT1. R-ST1 showed all regions of the core system except the right posterior STS which would have been located within the region of damage.

Table 3
Summary of results from the morphed-face discrimination test and the functional localizer in the right hemisphere. The left OFA, FFA and posterior STS were identified in all patients. Successful localization and normal performance is indicated with an 'N'. Failure to localize regions is indicated with 'Lesion'. The magnitude of functional impairment is indicated by the prediction intervals, outside of which, each individual performance falls.

Patient	Right OFA	Right FFA	Right posterior STS	Expression-fixed Identity Version	Expression-variable Identity Version	Identity-fixed Expression Version	Identity-variable Expression Version
R-IOT1	Lesion	Lesion	N	<95%	<99%	N	N
R-IOT2	N	N	N	<99%	<99%	N	N
B-AT1	N	N	N	<99.9%	<99.9%	N	N
R-AT1	N	N	N	<99.99%	<99.99%	N	N
R-ST1	N	N	Lesion	N	<99.9%	<99%	<95%

patient with damage to the right OFA and left FFA but preservation of the right FFA (Rossion et al., 2003) which has led some to conclude that normal perceptual processing of faces requires a network of face areas beyond simply the fusiform gyrus and FFA (Rossion et al., 2003).

Our fMRI study showed a large lesion of the lateral inferior occipitotemporal cortex in R-IOT1 that affected both the right OFA and FFA and spared the right posterior STS (Fig. 5). In contrast, fMRI of R-IOT2 demonstrated a large lesion of the right medial inferior occipitotemporal cortex which spared all three core face regions, with the right OFA and FFA located just lateral to the lesion (Fig. 5). While R-IOT1's data support the hypothesis that a lesion that damages the OFA/FFA but spares the STS will impair identity processing but spare expression processing, R-IOT2's data make an equally important and novel point, that integrity of the FFA and/or OFA is not sufficient for normal identity processing.

It is of interest to consider the anatomic basis of R-IOT2's deficit. One possibility is that his lesion affects white matter tracts important for communication between core face processing regions in the right hemisphere, between these regions and their left hemispheric counterparts, or between these regions and more anterior temporal regions involved in face recognition, via the inferior longitudinal fasciculus (Catani, Jones, Donato, & Ffytche, 2003; Fox, Iaria, & Barton, 2008; Habib, 1986; Takahashi et al., 1995), a possibility that may underlie the deficits seen in congenital prosopagnosia (Thomas et al., 2009). Another possibility is that the processing of face identity may involve both highly selective face regions (FFA, OFA) as well as other regions in the inferior occipitotemporal cortex that respond to faces without being selective for faces, and which therefore are not isolated by a localizer that contrasts faces with other objects (Haxby et al., 2001). If so, damage to these inferotemporal regions that are not face-selective could also impair face perception to a degree, as seen in R-IOT2. Indeed, recent studies of a prosopagnosic patient with damage to the OFA suggest that some of her residual face-processing abilities are mediated at least partly by other non-face-selective cortex in the occipitotemporal region (Dricot, Sorger, Schiltz, Goebel, & Rossion, 2008). Alternatively, while the preserved FFA/OFA in R-IOT2 does demonstrate normal sensitivity to faces, these regions may be impaired in discriminating different facial identities, a function attributed to the FFA (Haxby et al., 2000; Winston et al., 2004). Further studies examining adaptation effects within these spared regions will help to resolve this issue (Fox, Iaria, Duchaine, & Barton, 2011).

In summary, there are two important points to be learned from these first two patients. First, inferotemporal damage can selectively affect face identity processing, and leave expression processing intact. This may appear to conflict with recent fMRI adaptation studies in healthy controls (Fox, Moon, Iaria, & Barton, 2009; Xu & Biederman, 2010) that found sensitivity to changes in both identity and expression processing within the right FFA. However, the fMRI-adaptation technique is designed to demonstrate regional sensitivity to specific properties of a stimulus: by their nature, such studies cannot determine whether those regions perform operations *necessary* for the perception of those properties. Hence in our case, one cannot infer from sensitivity of the right FFA to identity and expression signals that it makes critical contributions to the perception of not only face identity but also face expression. One can speculate that the expression sensitivity may reflect information being used for operations other than expression recognition, such as encoding of identity in an expression-invariant manner, for example. Furthermore, fMRI adaptation effects may reflect patterns of information inherited from other areas in communication with the region of interest (Krekelberg, Boynton, & Van Wezel, 2006). Hence expression-related signals could be encoded in other areas and then reflected in the information these regions transfer to the right FFA, without necessarily implying that the FFA is performing any expression-related operations of its own. These limits, to what one can logically infer from fMRI adaptation studies, illustrate the continuing importance of performing lesion studies. Only by studying inactivation or damage to various regions of cortex are we able to verify whether that region is in fact necessary for performing a perceptual operation hypothesized in fMRI studies of healthy controls.

Second, although both R-IOT1 and R-IOT2 have inferotemporal damage, and both are impaired in the perceptual processing of identity, only R-IOT1 has damage to the right FFA and OFA. Interestingly, only R-IOT1 has prosopagnosic complaints, whereas R-IOT2, whose FFA and OFA have survived, does not. This suggests that there are additional elements of face processing related to FFA/OFA function that are not captured by our perceptual test, and which contribute to the experience of prosopagnosia. This is not necessarily surprising, given the complexity of the high-level task of face recognition.

3.2. Impaired identity perception with anterior damage

Besides R-IOT1 and R-IOT2, we studied two cases of prosopagnosia resulting from anterior temporal damage. One had extensive bilateral lesions (B-AT1) and another had a right amygdalohippocampectomy (R-AT1). Like the findings in R-IOT2, the FFA, OFA and posterior STS were present bilaterally in both patients. Behaviorally, B-AT1 and R-AT1 showed impaired performance on tests that involve facial memory, such as the Warrington Recognition Memory Test, Famous Face Recognition Test and the Face Imagery Test, but did well on a perceptual test of face matching (Benton Face Recognition Test; Table 1), consistent with the anterior temporal loci of their damage (Barton, 2008). Despite their good performance on the Benton Face Recognition Test, though, the morphed-face discrimination test did show perceptual impairments in the discrimination of facial identity but not facial expression (Table 3).

These results carry three implications. First, the morphed-face discrimination test may be more sensitive than standard neuropsychological instruments to subtle failures in perceiving facial structure, consistent with suggestions (Farah, 1990) and studies showing that normal scores on the Benton Face Recognition Test do not necessarily indicate normal identity perception (Duchaine

& Nakayama, 2004; Duchaine & Weidenfeld, 2003; Davidoff & Landis, 1990; Delvenne, Seron, Coyette, & Rossion, 2004; Levine & Calvanio, 1989). To improve the ability of the Benton Face Recognition Test to identify impaired identity perception some have suggested analyzing reaction times as well, as these are significantly longer in prosopagnosic individuals even when accuracy is maintained (Delvenne et al., 2004). Our morphed faces may proved an alternative approach to detect impairments in perceiving face identity with greater sensitivity with the added benefit of showing whether deficits are selective for identity or also affect expression perception. Second, perceptual deficits related to anterior temporal damage remain selective for identity and not expression, similar to the findings in patients with posterior occipitotemporal damage. Third, although the anterior temporal lobes have usually been assigned roles in determining familiarity or linking faces with names or semantic associations (Douville et al., 2005; Glosser, Salvucci, & Chiaravalloti, 2003; Gobbini & Haxby, 2007; Haxby et al., 2000; Snowden, Thompson, & Neary, 2004; Tsukiura et al., 2002; Tsukiura, Mochizuki-Kawai, & Fujii, 2006), they may also contribute to perceptual processing. Using multivariate fMRI analyses Kriegeskorte, Formisano, Sorger, and Goebel (2007) found a region in the anterior temporal cortex which is able to discriminate between individual faces, and in fact did so more robustly than the FFA, though this study used only two faces and these differed in both identity and gender. Furthermore, discrimination of faces in an oddity paradigm has been shown to bilaterally activate the medial temporal lobes, including the amygdala, anterior hippocampus and fusiform cortex, suggesting these regions may play a role in facial discrimination (Lee, Scahill, & Graham, 2008). Likewise, while patients with prosopagnosia following anterior temporal damage lack the severe deficits in perceiving facial configuration found in prosopagnosic patients with fusiform damage, they can still have subtler problems in integrating this perceptual information (Barton, Zhao, & Keenan, 2003; Bukach, Bub, Gauthier, & Tarr, 2006).

3.3. Impaired expression perception

In contrast to the four patients with selective deficits of identity perception, R-ST1 has a large right lateral lesion involving the superior temporal sulcus and does not complain of problems in face recognition. Structural and functional MRI showed sparing of the inferior occipitotemporal cortex including the right OFA and FFA but damage to the superior temporal sulcus, with loss of the posterior STS in the right hemisphere (Fig. 5). R-ST1 was severely impaired on both the *Identity-variable* and *Identity-fixed Expression Versions*, but normal on the *Expression-fixed Identity Version* of the morphed-face discrimination test, a pattern opposite to the previous four patients (Table 3). However, his perception of identity changes was impaired when expression also varied between the stimuli.

Compared to reports on identity impairments, there are fewer studies of deficits in face expression processing. Expression deficits in prior reports have been attributed to diffuse bilateral damage (Kurucz et al., 1980), to right (Adolphs et al., 1996) or left (Young et al., 1993) hemisphere lesions, or selective amygdala damage (Adolphs et al., 1994; Brierley et al., 2004). In a lesion overlap study of patients with deficits in expression recognition a right hemisphere bias was demonstrated, with the most common site of lesion being the right temporoparietal junction, in the vicinity of, though not directly correlated with the posterior STS (Adolphs et al., 1996), though the same group also pointed to the importance of the right somatosensory cortex in a separate group of patients (Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000). In contrast to these studies, another large patient series demonstrated selective impairments of expression perception following left hemisphere damage only (Young et al., 1993). Selective expression impairments were defined as poor performance on expression naming and expression matching tasks but spared familiar face recognition and unfamiliar face matching (Young et al., 1993). Patients with right hemisphere damage also showed impairments in expression processing but these were usually associated with impairments in familiar face recognition or unfamiliar face matching (Young et al., 1993).

Also of note in that study (Young et al., 1993), deficits following right hemisphere damage primarily involved expression matching rather than expression naming, suggesting a problem with expression perception rather than recognition of, or memory for, facial expressions. Other studies have shown impaired performance on expression naming following amygdala damage (Adolphs et al., 1999) and one study even demonstrated a selective deficit in emotion memory but not emotion perception (Brierley et al., 2004). Like the right hemisphere patients of Young et al. (1993), R-ST1 has a problem with expression perception, in that he shows normal emotion naming and memory on our neuropsychological tests (Table 1) but impaired emotion-matching on the morphed-face discrimination test. A similar pattern can be observed in patients with problems in color vision (achromatopsia) who can name colors yet exhibit deficits in matching of colors on more sensitive tests (Spillmann, Laskowski, Lange, Kasper, & Schmidt, 2000). The key anatomic observation is that R-ST1's lesion does not damage the amygdala but involves the posterior STS, making R-ST1 the first patient with demonstrable pSTS lesions underlying deficits in facial expression perception (Figs. 4 and 5). Within our own sample, we observe an interesting anatomical contrast in R-AT1, who has unilateral right amygdala damage but a spared right posterior STS. She performed normally on both *Expression* versions of the morphed-face discrimination test (emotion perception), but was impaired on the Reading the Mind in the Eyes Test (emotion recognition or memory), bringing to mind the prior data on amygdala damage (Brierley et al., 2004).

R-ST1's impaired performance on the *Expression-variable Identity Task* also suggests that the STS region may make a contribution to expression-invariant identity processing. This contribution may be indirect, in that failure to recognize changes in a face as attributable to variations in expression may interfere with the ability to discount these when attempting to match faces for identity. Recent fMRI studies show that the right posterior STS is sensitive to changes in either facial identity or expression (Fox, Moon, et al., 2009; Winston et al., 2004), with one interpretation being that the posterior STS is required for tasks that integrate analyses of both facial identity and expression.

In conclusion, by using a non-verbal perceptual test of identity and expression discrimination, matched for the level of perceptual difficulty, we showed that impairments in these two functions are dissociable. As with most studies of rare neurological disorders our patient sample is small and their respective lesions are quite heterogenous (Fig. 4). This makes our results hard to generalize to the entire prosopagnosic population, but this is in fact what we are arguing. Current models generalize OFA/FFA damage as the root cause of identity impairments, yet we were able to demonstrate selective impairments in identity perception after right inferior occipitotemporal or anterior temporal lesions that affected, and in 3 cases spared, the OFA and FFA. Thus the important locus of damage may not be the peak regions of face selectivity in the occipitotemporal cortex (OFA/FFA), but rather may involve these regions as well as multiple connections between them and other regions of cortex. Our fifth patient showed us that impairments in discriminating expression can occur with damage to the right superior temporal sulcus that affects the posterior STS, a finding which is supported by current models of face perception. Thus these patients provide important lesion data to complement the functional neuroimaging work upon which current cognitive-anatomic models of

face processing are based. Future studies using both behavioral and functional imaging methods with larger patient samples will help to further characterize the nature of lesions that selectively damage identity and expression processing.

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References

Adolphs, R., Damasio, H., Tranel, D., Cooper, G., & Damasio, A. R. (2000). A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. *Journal of Neuroscience*, *20*, 2683–2690.

Adolphs, R., Damasio, H., Tranel, D., & Damasio, A. R. (1996). Cortical systems for the recognition of emotion in facial expressions. *Journal of Neuroscience*, *16*, 7678–7687.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*, *372*, 669–672.

Adolphs, R., Tranel, D., Hamann, S., Young, A. W., Calder, A. J., Phelps, E. A., et al. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia*, *37*, 1111–1117.

Akiyama, T., Kato, M., Muramatsu, T., Saito, F., Nakachi, R., & Kashima, H. (2006). A deficit in discriminating gaze direction in a case with right superior temporal gyrus lesion. *Neuropsychologia*, *44*, 161–170.

Andrews, T. J., & Ewbank, M. P. (2004). Distinct representations for facial identity and changeable aspects of faces in the human temporal lobe. *Neuroimage*, *23*, 905–913.

Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The 'reading the mind in the eyes' test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *42*, 241–252.

Barton, J. J., & Cherkasova, M. (2003). Face imagery and its relation to perception and covert recognition in prosopagnosia. *Neurology*, *61*, 220–225.

Barton, J. J. (2003). Disorders of face perception and recognition. *Neurologic Clinics*, *21*, 521–548.

Barton, J. J., Cherkasova, M., & O'Connor, M. (2001). Covert recognition in acquired and developmental prosopagnosia. *Neurology*, *57*, 1161–1168.

Barton, J. J., Zhao, J., & Keenan, J. P. (2003). Perception of global facial geometry in the inversion effect and prosopagnosia. *Neuropsychologia*, *41*, 1703–1711.

Barton, J. J. S. (2008). Structure and function in acquired prosopagnosia: Lessons from a series of ten patients with brain damage. *Journal of Neuropsychology*, *2*, 197–225.

Barton, J. J. S., Press, D. Z., Keenan, J. P., & O'Connor, M. (2002). Lesions of the fusiform face area impair perception of facial configuration in prosopagnosia. *Neurology*, *58*, 71–78.

Benton, A., & van Allen, M. (1972). Prosopagnosia and facial discrimination. *Journal of the Neurological Sciences*, *15*, 167–172.

Benton, C. P., Etchells, P. J., Porter, G., Clark, A. P., Penton-Voak, I. S., & Nikolov, S. G. (2007). Turning the other cheek: The viewpoint dependence of facial expression after-effects. *Proceedings. Biological Science*, *274*, 2131–2137.

Bowers, D., Blonder, L. X., & Heilman, K. M. (1991). *Florida affect battery*. University of Florida.

Brierley, B., Medford, N., Shaw, P., & David, A. S. (2004). Emotional memory and perception in temporal lobectomy patients with amygdala damage. *Journal of Neurology, Neurosurgery and Psychiatry*, *75*, 593–599.

Bruce, V., & Young, A. (1986). Understanding face recognition. *British Journal of Psychology*, *77*(Pt 3), 305–327.

Bukach, C. M., Bub, D. N., Gauthier, I., & Tarr, M. J. (2006). Perceptual expertise effects are not all or none: Spatially limited perceptual expertise for faces in a case of prosopagnosia. *Journal of Cognitive Neuroscience*, *18*, 48–63.

Busigny, T., Graf, M., Mayer, E., & Rossion, B. (2010). Acquired prosopagnosia as a face-specific disorder: Ruling out the general visual similarity account. *Neuropsychologia*, *48*, 2051–2067.

Busigny, T., Joubert, S., Felician, O., Ceccaldi, M., & Rossion, B. (2010). Holistic perception of the individual face is specific and necessary: Evidence from an extensive case study of acquired prosopagnosia. *Neuropsychologia*, *48*, 4057–4092.

Calder, A. J., & Young, A. W. (2005). Understanding the recognition of facial identity and facial expression. *Nature Reviews Neuroscience*, *6*, 641–651.

Catani, M., Jones, D. K., Donato, R., & Ffytche, D. H. (2003). Occipito-temporal connections in the human brain. *Brain*, *126*, 2093–2107.

Davidoff, J., & Landis, T. (1990). Recognition of unfamiliar faces in prosopagnosia. *Neuropsychologia*, *28*, 1143–1161.

de Gelder, B., Frissen, I., Barton, J., & Hadjikhani, N. (2003). A modulatory role for facial expressions in prosopagnosia. *Proceedings of the National Academy of Sciences of the United States of America*, *100*, 13105–13110.

Delvenne, J. F., Seron, X., Coyette, F., & Rossion, B. (2004). Evidence for perceptual deficits in associative visual (prosop)agnosia: A single-case study. *Neuropsychologia*, *42*, 597–612.

Douville, K., Woodard, J. L., Seidenberg, M., Miller, S. K., Leveroni, C. L., Nielson, K. A., et al. (2005). Medial temporal lobe activity for recognition of recent and remote famous names: an event-related fMRI study. *Neuropsychologia*, *43*, 696–703.

Dricot, L., Sorger, B., Schiltz, C., Goebel, R., & Rossion, B. (2008). The roles of "face" and "non-face" areas during individual face perception: Evidence by fMRI adaptation in a brain-damaged prosopagnosic patient. *Neuroimage*, *40*, 318–332.

Duchaine, B. C., & Nakayama, K. (2004). Developmental prosopagnosia and the Benton facial recognition test. *Neurology*, *62*, 1219–1220.

Duchaine, B. C., Parker, H., & Nakayama, K. (2003). Normal recognition of emotion in a prosopagnosic. *Perception*, *32*, 827–838.

Duchaine, B. C., & Weidenfeld, A. (2003). An evaluation of two commonly used tests of unfamiliar face recognition. *Neuropsychologia*, *41*, 713–720.

Ekman, P., & Friesen, W. V. (1971). Constants across cultures in the face and emotion. *Journal of Personality and Social Psychology*, *17*, 124–129.

Ekman, P., Sorenson, E. R., & Friesen, W. V. (1969). Pan-cultural elements in facial displays of emotion. *Science*, *164*, 86–88.

Farah, M. J. (1990). *Visual agnosia: Disorders of visual recognition and what they tell us about normal vision*. Cambridge: MIT Press.

Fox, C. J., Iaria, G., & Barton, J. J. (2008). Disconnection in prosopagnosia and face processing. *Cortex*, *44*, 996–1009.

Fox, C. J., Iaria, G., & Barton, J. J. (2009). Defining the face processing network: Optimization of the functional localizer in fMRI. *Human Brain Mapping*, *30*, 1637–1651.

Fox, C. J., Iaria, G., Duchaine, B. C., & Barton, J. J. (2011). Residual fMRI sensitivity for identity changes in acquired prosopagnosia. In preparation.

Fox, C. J., Moon, S. Y., Iaria, G., & Barton, J. J. (2009). The correlates of subjective perception of identity and expression in the face network: An fMRI adaptation study. *Neuroimage*, *44*, 569–580.

Ganel, T., Goshen-Gottstein, Y., & Ganel, T. (2004). Effects of familiarity on the perceptual integrality of the identity and expression of faces: The parallel-route hypothesis revisited. *Journal of Experimental Psychology: Human Perception and Performance*, *30*, 583–597.

Ganel, T., Valyear, K. F., Goshen-Gottstein, Y., & Goodale, M. A. (2005). The involvement of the "fusiform face area" in processing facial expression. *Neuropsychologia*, *43*, 1645–1654.

Gauthier, I., Tarr, M. J., Moylan, J., Skudlarski, P., Gore, J. C., & Anderson, A. W. (2000). The fusiform "face area" is part of a network that processes faces at the individual level. *Journal of Cognitive Neuroscience*, *12*, 495–504.

Glosser, G., Salvucci, A. E., & Chiaravalloti, N. D. (2003). Naming and recognizing famous faces in temporal lobe epilepsy. *Neurology*, *61*, 81–86.

Gobbini, M. I., & Haxby, J. V. (2007). Neural systems for recognition of familiar faces. *Neuropsychologia*, *45*, 32–41.

Gosselin, F., & Schyns, P. G. (2001). Bubbles: A technique to reveal the use of information in recognition tasks. *Vision Research*, *41*, 2261–2271.

Grill-Spector, K., & Malach, R. (2001). FMR-adaptation: A tool for studying the functional properties of human cortical neurons. *Acta Psychologica*, *107*, 293–321.

Grossi, D. (1991). *La riabilitazione dei disordini della cognizione spaziale*. Milano: Masson.

Habib, M. (1986). Visual hypoemotionality and prosopagnosia associated with right temporal lobe isolation. *Neuropsychologia*, *24*, 577–582.

Haxby, J. V., Gobbini, M. I., Furey, M. L., Ishai, A., Schouten, J. L., & Pietrini, P. (2001). Distributed and overlapping representations of faces and objects in ventral temporal cortex. *Science*, *293*, 2425–2430.

Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Sciences*, *4*, 223–233.

Hooper, H. E. (1957). *Hooper visual organization test*. Los Angeles: Western Psychological Services.

Humphreys, K., Avidan, G., & Behrmann, M. (2007). A detailed investigation of facial expression processing in congenital prosopagnosia as compared to acquired prosopagnosia. *Experimental Brain Research*, *176*, 356–373.

Joyce, C. A., Schyns, P. G., Gosselin, F., Cottrell, G. W., & Rossion, B. (2006). Early selection of diagnostic facial information in the human visual cortex. *Vision Research*, *46*, 800–813.

Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: A region in human extrastriate cortex specialized for face perception. *Journal of Neuroscience*, *17*, 4302–4311.

Krekelberg, B., Boynton, G. M., & Van Wezel, R. J. (2006). Adaptation: from single cells to BOLD signals. *Trends in Neurosciences*, *29*, 250–256.

Kriegeskorte, N., Formisano, E., Sorger, B., & Goebel, R. (2007). Individual faces elicit distinct response patterns in human anterior temporal cortex. *Proceedings of the National Academy of Sciences of the United States of America*, *104*, 20600–20605.

Kurucz, J., Feldmar, G., & Werner, W. (1979). Prosopo-affective agnosia associated with chronic organic brain syndrome. *Journal of the American Geriatrics Society*, *27*, 91–95.

Kurucz, J., Soni, A., Feldmar, G., & Slade, W. (1980). Prosopo-affective agnosia and computerized tomography findings in patients with cerebral disorders. *Journal of the American Geriatrics Society*, *28*, 475–478.

Lee, A. C., Scahill, V. L., & Graham, K. S. (2008). Activating the medial temporal lobe during oddity judgment for faces and scenes. *Cerebral Cortex*, *18*, 683–696.

Levine, D. N., & Calvanio, R. (1989). Prosopagnosia: A defect in visual configural processing. *Brain and Cognition*, *10*, 149–170.

Lundqvist, D., & Litton, J. E. (1998). *The averaged karolinska directed emotional faces-akdef*.

McNeil, J. E., & Warrington, E. K. (1991). Prosopagnosia: a reclassification. *The Quarterly Journal of Experimental Psychology. A, Human Experimental Psychology*, *43*, 267–287.

Palermo, R., & Rhodes, G. (2007). Are you always on my mind? A review of how face perception and attention interact. *Neuropsychologia*, *45*, 75–92.

Posamentier, M. T., & Abdi, H. (2003). Processing faces and facial expressions. *Neuropsychology Review*, *13*, 113–143.

Puce, A., Allison, T., Gore, J. C., & McCarthy, G. (1995). Face-sensitive regions in human extrastriate cortex studied by functional MRI. *Journal of Neurophysiology*, *74*, 1192–1199.

Reitan, R. M. (1958). Validity of the trail making test as an indicator of organic brain damage. *Perceptual and Motor Skills*, *8*.

Rickham, P. P. (1964). Human experimentation. Code of ethics of the world medical association. Declaration of Helsinki. *British Medical Journal*, *2*, 177.

Rossion, B., Caldara, R., Seghier, M., Schuller, A.-M., Lazeyras, F., & Mayer, E. (2003). A network of occipito-temporal face-sensitive areas besides the right middle fusiform gyrus is necessary for normal face processing. *Brain*, *126*, 2381–2395.

Rotshtein, P., Henson, R. N., Treves, A., Driver, J., & Dolan, R. J. (2005). Morphing Marilyn into Maggie dissociates physical and identity face representations in the brain. *Nature Neuroscience*, *8*, 107–113.

Saxe, R., Brett, M., & Kanwisher, N. (2006). Divide and conquer: a defense of functional localizers. *Neuroimage*, *30*, 1088–1096.

Sergent, J., & Signoret, J. L. (1992). Varieties of functional deficits in prosopagnosia. *Cerebral Cortex*, *2*, 375–388.

Sergent, J., Ohta, S., & MacDonald, B. (1992). Functional neuroanatomy of face and object processing. A positron emission tomography study. *Brain*, *115*(Pt 1), 15–36.

Snowden, J. S., Thompson, J. C., & Neary, D. (2004). Knowledge of famous faces and names in semantic dementia. *Brain*, *127*, 860–872.

Spillmann, L., Laskowski, W., Lange, K. W., Kasper, E., & Schmidt, D. D. (2000). Stroke-blind for colors, faces and locations: Partial recovery after three years. *Restorative Neurology and Neuroscience*, *17*, 89–103.

Spinnler, H., & Tognoni, G. (1987). Standardizzazione e taratura italiana di test neuropsicologici. *Italian Journal of Neurological Sciences*.

Steeves, J. K., Culham, J. C., Duchaine, B. C., Pratesi, C. C., Valyear, K. F., Schindler, I., et al. (2006). The fusiform face area is not sufficient for face recognition: evidence from a patient with dense prosopagnosia and no occipital face area. *Neuropsychologia*, *44*, 594–609.

Stephan, B. C., Breen, N., & Caine, D. (2006). The recognition of emotional expression in prosopagnosia: decoding whole and part faces. *Journal of the International Neuropsychological Society*, *12*, 884–895.

Takahashi, N., Kawamura, M., Hirayama, K., Shiota, J., & Isono, O. (1995). Prosopagnosia: A clinical and anatomical study of four patients. *Cortex*, *31*, 317–329.

Talairach, J., & Tournoux, P. (1988). *Co-planar stereotaxic atlas of the human brain*. New York: Thieme.

Thomas, C., Avidan, G., Humphreys, K., Jung, K. J., Gao, F., & Behrmann, M. (2009). Reduced structural connectivity in ventral visual cortex in congenital prosopagnosia. *Nature Neuroscience*, *12*, 29–31.

Tranel, D., Damasio, A. R., & Damasio, H. (1988). Intact recognition of facial expression, gender, and age in patients with impaired recognition of face identity. *Neurology*, *38*, 690–696.

Tsukiura, T., Fujii, T., Fukatsu, R., Otsuki, T., Okuda, J., Umetsu, A., et al. (2002). Neural basis of the retrieval of people's names: evidence from brain-damaged patients and fMRI. *Journal of Cognitive Neuroscience*, *14*, 922–937.

Tsukiura, T., Mochizuki-Kawai, H., & Fujii, T. (2006). Dissociable roles of the bilateral anterior temporal lobe in face-name associations: an event-related fMRI study. *Neuroimage*, *30*, 617–626.

Vuilleumier, P., & Pourtois, G. (2007). Distributed and interactive brain mechanisms during emotion face perception: Evidence from functional neuroimaging. *Neuropsychologia*, *45*, 174–194.

Warrington, E. (1984). *Warrington recognition memory test*. Los Angeles: Western Psychological Services.

Wechsler, D. (1999). *Wechsler abbreviated scale of intelligence—manual*. San Antonio: Psychological Corporation.

Wilson, B., Cockburn, J., & Halligan, P. W. (1987). *Behavioural inattention test*. Titchfield: Thames Valley Test Company.

Winston, J. S., Henson, R. N., Fine-Goulden, M. R., & Dolan, R. J. (2004). FMRI-adaptation reveals dissociable neural representations of identity and expression in face perception. *Journal of Neurophysiology*, *92*, 1830–1839.

Xu, X., & Biederman, I. (2010). Loci of the release from fMRI adaptation for changes in facial expression, identity, and viewpoint. *Journal of Vision*, *10*(14), 1–13, 36.

Young, A. W., Newcombe, F., de Haan, E. H., Small, M., & Hay, D. C. (1993). Face perception after brain injury. Selective impairments affecting identity and expression. *Brain*, *116*(Pt 4), 941–959.