

COMPUTATIONAL considerations suggest that efficient face identification requires the categorization and exclusive streaming of previously encoded face visual primitives into a dedicated face recognition system. Unique evidence supporting this claim is provided by a rare case of developmental pure prosopagnosia with otherwise normal visual and cognitive functions. Despite his normal visual memory and ability to describe faces, he is extremely impaired in face recognition. An early event related brain potential (N170) that is normally elicited exclusively by human faces, showed no specificity in this person. MRI revealed a smaller than normal right temporal lobe. These data emphasize the indispensability of the early streaming process for face recognition. *NeuroReport* 10:823–827 © 1999 Lippincott Williams & Wilkins.

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Selective visual streaming in face recognition: evidence from developmental prosopagnosia

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Introduction

The human ability to elicit an instantaneous feeling of familiarity while recognizing faces is remarkable in view of the complex computational demands of the task. Different sets of data converged suggesting that this outstanding perceptual ability relies on specific neural mechanisms. Single-unit recordings in monkeys revealed cells in the inferotemporal cortex that respond to monkey and human faces [1–4] and face components [5] but not to other complex visual stimuli such as snakes, spiders or food [6], or to low-level perceptual characteristics of the stimuli [7,8]. In humans, face specificity is suggested by neuropsychological observations and supported by results of electrophysiological and neuroimaging studies.

Neuropsychological data distinguishing face recognition from other perceptual abilities was provided by the double dissociation between visual agnosic patients who cannot recognize objects although their ability to recognize faces is spared [9], and patients who suffer from prosopagnosia, a deficit characterized by a deficiency in identifying familiar faces without marked impairment in recognizing other visual stimuli following brain damage [10]. These patients, however, usually suffer from additional perceptual and cognitive lesion-related deficiencies [11,12], which makes it difficult to assess the nature of this specificity [13].

Event-related potentials (ERPs) recorded directly

from the surface of the occipitotemporal cortex revealed distinct regions where faces, but not other stimuli, evoked a negative component with a mean latency of 192 ms (N200) [14,15]. Scalp recordings also revealed a face-specific negative potential (N170), which was recorded from a relatively circumscribed region at the posterior–inferior aspects of the temporal lobes and was bigger at right than at left hemisphere sites [16]. Extensive studies of the stimulus-related characteristics of the N170 demonstrated that it is not sensitive to the familiarity of the face [17], and it is not affected by the spatial organization of the inner components of the face or by face inversion [16], factors known to influence face recognition. These findings strongly suggest that the N170 is related to an early mechanism operating at the early stages of face processing, at which the structural encoding of face (in terms of Bruce and Young's functional model of face perception [18]) occur. Moreover, because it is not affected by face familiarity, it is conceivable that N170 is not associated with later stages of processing where faces are identified and the face-related semantic information is retrieved. Consequently, Bentin *et al.* [16] suggested that the N170 can index the integrity of a visual processor which is responsible for the extraction of the face-specific visual invariants and forming a sensory representation of a human face.

The above review suggests that a better understanding of the specificity in face processing could be gained by combining neuropsychological and phy-

biological data. Studies of prosopagnosic patients revealed that their impairment is frequently confined to the identification of faces (i.e. relating between a face and a person) whereas their ability to distinguish between faces and other visual stimuli is intact. Moreover, most patients with prosopagnosia are able to determine the sex and the relative age of the person's face and the emotion that it is expressing [10], many can match between two identical photographs of the same individual, and some of them can do that even if the faces are seen from different angles [19]. This pattern of neuropsychological evidence suggests that in many patients with acquired prosopagnosia, at least when there are no symptoms of a broader visual agnosia, the structural encoding and the formation of a sensory representation of the face are preserved. On the other hand, while attempting to identify a face, they need to rely on distinctive non-facial visual information, such as a person's hair-style or a particular piece of clothing, or on non-visual information such as tone of the voice [10]. It is conceivable, therefore, that the face recognition impediment in the majority of (more or less) pure forms of acquired prosopagnosia stem from deficient processing at stages that follow structural encoding, (i.e. matching of the sensory representation of the face to its stored figural description, accessing the semantic information associated with the stored representation of the face, or both). According to this view, the N170 in such patients should be normal. This hypothesis, however, has never been tested. In fact, with one exception [20] there are no previous reports of any electrophysiological studies in prosopagnosic patients. Part of the problem in studying ERPs in brain damaged patients is that brain lesions may have a general effect on the brain electrophysiological activity. This difficulty, together with the diversity of cognitive deficiencies which is usually found in patients with acquired prosopagnosia, makes the study of developmental prosopagnosia particularly valuable. In the absence of a structural brain lesion and especially when other types of perceptual and cognitive processing are intact, these individuals can unveil important aspects of the normal mechanism for face perception. Pure developmental prosopagnosia, however, is a very rarely encountered syndrome.

Case report: Y.T., a 36-year-old man, is a business administrator who approached us following a public lecture on face perception that one of us (SB) delivered. He reported having severe problems in recognizing faces since early childhood. By his account, Y.T. does not usually experience any sense of familiarity or previous encounter with a face, and is unable to evoke a visual mental image of a familiar face except, perhaps, of his closest family members. This inability stands in contrast with his normal

ability to recollect face details such as eye color, the wearing of eyeglasses, etc. His ability to recognize some familiar faces is based primarily on hair-style or distinctive features such as a moustache of a particular form, or on non-visual cues. In contrast to face identification, Y.T. can easily determine the sex, age and emotional state of a person by looking at his/her face. Except for face recognition, Y.T. did not report any additional perceptual problems. He easily identifies car models, familiar locations and animals.

When formally tested, Y.T. was able to recognize only 24 of 670 famous faces (mixed with 580 faces of unknown individuals), without any false alarms. He did not recognize the faces of politicians such as the Israeli president, the American president, or famous movie stars with whose names he was very familiar. In contrast, a control group of 24 subjects, matched with Y.T. for age and education, recognized 391 faces (s.d. = 80) from this set. The possibility that although having no explicit sense of familiarity Y.T. is implicitly sensitive to the identity of the face was tested using an adapted version of De Haan *et al.*'s procedure [21]. His speed and accuracy of categorizing a printed name as a politician or a media celebrity was similar regardless of whether the name was accompanied by the matching face or by another face (hit rate >99% for both, RT = 1356 and 1354, respectively). The absence of the name/face congruity effect in Y.T. contrasted with the performance of control subjects who, although being instructed to ignore the face, were faster and more accurate when the name and the face were of the same person (970 ms), than when the name and the face were from the opposite categories (1163 ms), ($t(8) = 6.929$, $p < 0.0001$; hit rate = 98% *vs* 91%). Thus, unlike patients with acquired prosopagnosia who showed implicit recognition of faces [20,21], Y.T. has a very impaired ability to recognize famous faces implicitly as well as explicitly.

The developmental and medical history of Y.T. was uneventful with the exception of childhood strabismus treated with alternating monocular blindfolding followed by corrective surgery at age 7. Complete neurological examination revealed no deviation from normality except for a residual mild right eye esotropia with normal visual acuity.

The visual and cognitive abilities of Y.T. were tested by a set of standardized and specially designed neuropsychological tests (in which a group of undergraduate students served as controls). His full scale IQ was 124 (verbal scale 127, performance scale 113). His verbal and visual memory were above average as tested by the Wechsler Memory Scale, Revised, Benton's Visual Retention Test, Rey-Osterrieth Complex Figure test, and the verbal part of the Warrington's Visual Memory Test. He

judged perfectly the orientation of lines (Benton's Line Orientation Test, 30/30), and was above average fast and accurate in his ability to replicate the location of a dot placed in a 18×9 cm rectangle. Y.T.'s ability to identify and name colors was perfect (20/20). Most importantly, he had no problems to identify and name line drawings of objects (50/50 items from the Boston Naming Test), could perfectly identify real objects, overlapping line drawings of objects, and pictures of common objects photographed from prototypic and non prototypic angles from the Loewenstein Occupational Therapy Cognitive Assessment. In addition, his speed and accuracy in identifying photographs of everyday objects and English words that were masked by overlapping oblique white stripes which concealed 50% of the display, or by white noise, was within the range of the control group. His ability to recognize the Gollin's incomplete drawings, and fragmented objects from the Hooper's visual organization test was normal. In summary, Y.T. did not show any sign of a general agnosic problem in the visual modality. His holistic and analytic visual and general cognitive functions seem to be normal or above normal.

In contrast to his high-normal general visual perception, his performance with faces, although within normal range, was relatively low. For example, whereas scoring 48/50 in Warrington Visual Memory Test for words, his score with faces in that test was only 32/50. Similarly, in the Benton & Van Allen's Facial Recognition Test his corrected score was 41/54, a score which placed him in the lower normal range (39–40 is considered borderline). Since performance on the latter test is based on perceptual factors rather than on face recognition, Y.T.'s low performance on this test suggests a deficiency already at the level of structural encoding of faces. This suggestion was further tested using ERP recordings.

Subjects: The control subjects in the ERP experiment were 12 undergraduate students aged 21–30 years, paid for their participation.

Covert recognition: In our version of De Haan *et al.*'s task, the subjects were presented with 32 names of politicians and 32 names of movie stars and instructed to press one button if the name was the name of a politician, and another button if the name was the name of a movie star. Each name was presented in a 'balloon' coming out of the mouth of a face. Within each group of names there were eight congruent trials in which the name and the face were of the same person, eight related trials in which the name was presented with the face of a different person from the same semantic category, eight

incongruent trials in which the name was presented with the face of a person from the other semantic category, and eight neutral trials in which the name was presented with an unfamiliar face. The subjects were instructed to read the names and ignore the faces.

EEG recording: The EEG was recorded continuously via 48 electrodes mounted on a custom-made cap (ECI), sampled at a rate of 250 Hz, and amplified $\times 20\,000$ with an analog band-pass filter of 0.01–30 Hz, and stored for off-line analysis. Impedance was kept below 5 k Ω . The EOG was recorded with two electrodes, one located at the outer cantus of the right eye and the other at the infraorbital region of the same eye. ERPs resulted from averaging epochs starting 100 ms before and ending 900 ms after stimulus onset. Epochs with EEG or EOG exceeding $\pm 100 \mu\text{V}$ were excluded from the averaging. The epochs were averaged separately for each stimulus type. The baseline was adjusted by subtracting the mean amplitude of the prestimulus period of each ERP from all the data points in the epoch.

Procedure: The subjects were exposed to pictures of faces, cars, furniture and nonsense stimuli. The nonsense stimuli were constructed by scrambling the phase of the spatial frequencies of the faces, hence preserving their luminance and spatial frequencies. The stimuli were randomly presented one after another while the subjects monitored the occasional occurrence of pictures of butterflies.

MRI volumetric measurements: The temporal lobe volumes were measured in a control group of 15 male subjects (32 ± 7 years old), by manual tracing the perimeter of the temporal lobe (each hemisphere separately) in a series of contiguous 3 mm coronal slices. Tracing commenced posteriorly in the slice 3 mm anterior to the colliculi and encompassed the entire temporal lobe anteriorly. Areas were determined summing the enclosed pixels. Since each screen pixel represents a three dimensional voxel, multiplying that area by the voxel's dimension resulted in the structure's volume. The measurements were performed on a reformed series corrected for head rotation and oriented perpendicular to the long axis of the hippocampus. This latter interpolation was calculated by defining the hippocampal plane with three points: two at the center of each hippocampus in the image 3 mm anterior to the colliculi, and one at the center of either hippocampus in the image 15 mm anterior to the colliculi.

Y.T.'s brain was scanned using a high-resolution series of coronal sections of the whole brain using a 2.1 Gyrex (Elcint) magnet (TR, 25 ms; TE, 8.6 ms;

$\alpha = 45^\circ$; NEX, 2; field of view, 20×25 cm; slice thickness, 1.5 mm; skip factor, 0; imaging matrix, 176×176). The calculation of the volume was performed as described above for the control subjects. The raw volume of his temporal lobes were transformed in z-score deviations from the control group means.

Results

Structural MRI revealed no parenchymal damage, thus confirming the developmental origin of Y.T.'s disorder. Volumetric MRI analysis revealed that Y.T. had a small brain (1051 cm^3) relative to a group of 15 normal male subjects (1155 cm^3) matched with Y.T. for age and height ($z = -1.39$). This difference, however, was not statistically significant except when comparing the right temporal lobes (54.2 cm^3 vs $69.3 \pm 5.4 \text{ cm}^3$, $z = -2.79$).

As evident in Fig. 1A, a conspicuous N170 was evident for Y.T. as well as for the control subjects, and its distribution was roughly similar (Fig. 1B). However, whereas for control subjects the N170 was elicited exclusively by faces, for Y.T. it was elicited by objects as well. In fact, as evident in Fig. 1C, Y.T.'s face selectivity effect (i.e. the difference in amplitude between the N170 elicited by faces and that elicited by objects) was significantly lower ($3.55 \mu\text{V}$ and $0.73 \mu\text{V}$ over the right and left hemispheres, respectively) than the normal range (right hemisphere: mean = $6.6 \mu\text{V}$, 99% confidence interval $4.5\text{--}8.7 \mu\text{V}$; left hemisphere: mean = $4.4 \mu\text{V}$, 99% confidence interval $2.8\text{--}5.9 \mu\text{V}$).

Discussion

Up to now there have been only two published reports of relatively pure developmental prosopagnosia, A.B. [22,23] and Dr S. [24]. Two additional cases, K.D. [25] and L.G. [26], suffered from more general visual agnosic symptoms and cannot, therefore, be easily compared with Y.T. It is interesting to note that, like both A.B. and Dr. S, Y.T. reported having a parent with face recognition problems. Although the formal testing of both Y.T.'s father and A.B.'s mother showed that their face recognition handicap is by far less severe than that of their children, one cannot ignore the coincidence of this claim. It raises the possibility that developmental prosopagnosia may indeed reflect a congenitally transmitted functional brain deficiency.

The comparison of the neuropsychological profiles of these previous cases to Y.T.'s profile leads to important insights. Compared with A.B., the prosopagnosia displayed by Y.T. is more specific. Indeed, his inability to recognize faces and his lack of mental

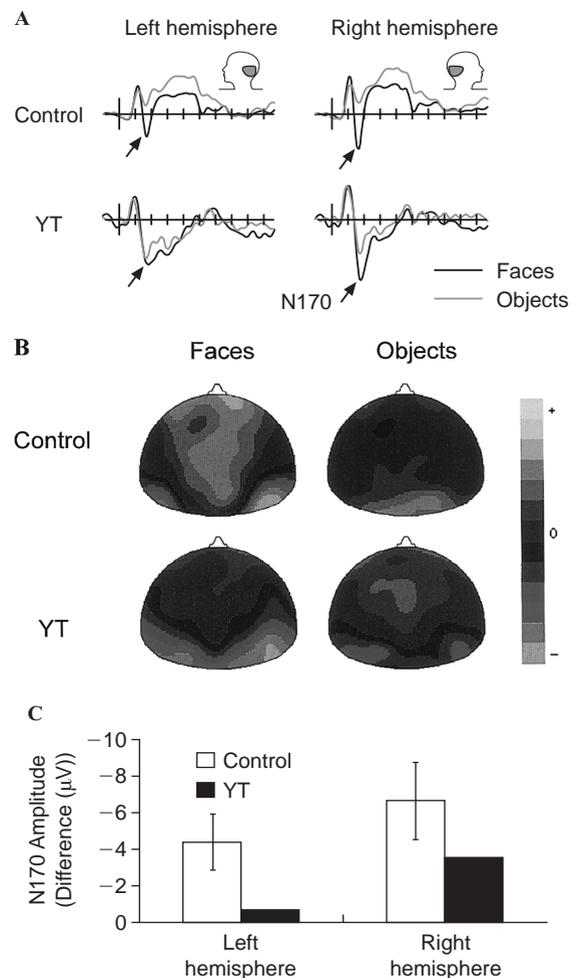


FIG. 1. (A) The N170 ERP elicited by faces and by objects in Y.T. and the control group. The ERPs are spatially averaged across the posterior parieto-temporal sites at each hemisphere (P7/8, PO7/8, IM1/2 and the mastoid; see embedded head). (B) The distribution of the N170 elicited by faces and objects in Y.T. and the control group. Note the significant posterior temporal negativity elicited by objects in Y.T. but not in control subjects. (C) The difference between the N170 amplitude elicited by faces and by objects (faces-objects). The confidence intervals denoted by the error bars were calculated for a type I error of $p = 0.01$.

imagery for familiar faces are at least as bad as those of A.B. and, like her, he is borderline on matching faces seen from different angles (Benton and Van Allen's Facial Recognition Test), and scores considerably lower in the faces than in the words part of the Warrington Recognition Memory Test. However, in contrast to A.B., he had no problems with object recognition, no spatial disorientation and no abnormality in motor coordination and fine movements. The feasibility of a selective disruption of face processing alone strongly supports the existence of a domain-specific functional organization of the human visual system, at least in regards to the processing of faces.

The prosopagnosia observed in Dr. S. seems to be as specific as that of Y.T. Nonetheless, whereas Dr. S. displayed normal memory for new faces in the

faces part of the Warrington Recognition Memory test, Y.T. was considerably impaired in this respect. This supports the possibility that the impairment of these two patients resides in different stages of face processing. Dr. S. was assumed to have a significant impairment in accessing person identity information, despite having intact structural encoding processes for faces. In contrast, the present ERP and neuropsychological data suggest that Y.T.'s impairment of face recognition has an earlier origin in the perceptual process. However, his ability to identify visually degraded objects and to verbally describe faces suggests that he is able to efficiently extract the visual structure of objects and faces is intact. Additional support for this hypothesis stems from the N170 data in a patient with acquired prosopagnosia reported by Eimer and McCarthy [27]. In contrast to Y.T., that patient suffers from a more general visual agnosia having problems in recognizing common animal, fruits and vegetables. Indeed, the N170 in that patient was reduced for faces to the level of that elicited by houses, suggesting that his visual impairment is at the level of extracting structural features from the stimulus. The non-selective (rather than missing) N170 response in Y.T. points to an impairment in a subsequent process of selecting and/or representing face-specific information in a way that is optimal for use by higher-level, dedicated face recognition units. This failure of distinctive encoding could account for his relative low score in the face memory test and may be the reason for his prosopagnosia. In light of the MRI volumetric analysis of Y.T.'s temporal lobes, this functional impairment seems to be associated with malfunction of the posterior right temporal lobe, or to an abnormal distribution of face-processing load between the hemispheres.

Conclusion

By way of negation, the combination of neuropsychological and electrophysiological data, obtained from this unique case of pure developmental prosopagnosia, highlights the importance of having separate streams of information processing. What could be the benefits of having a compartmentalized functional organization of the visual system? The consequences of the particular breakdown of this specificity, as demonstrated by Y.T., suggests that

the early extraction and discrimination of the invariant features of a visual stimulus may be crucial for efficiently streaming the visual information into higher-level recognition modules. The efficiency of recognizing faces is dependent, according to this view, on having a dedicated neural mechanism optimally structured to process and store face primitives. When the streaming of visual information from the structural encoding process is not sufficiently discriminative, the primitives of non facial objects may clutter the face recognition system, rendering it inefficient for its original purpose. Moreover, if the breakdown happens early in development, face recognition circuits in the brain will be less than optimally tuned for their task. As explicitly expressed by Ullman [28], from a computational perspective, "If the image can be classified, for example, as representing a face, without identifying the individual face, then subsequent processing stages can be directed to specific face models, rather than to models of other 3-D objects" (pp. 163–164).

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