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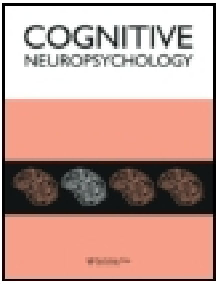

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EARLY COMMITMENT OF NEURAL SUBSTRATES FOR FACE RECOGNITION

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We present evidence of a striking failure of plasticity in the neural substrates of face recognition, which suggests that the distinction between faces and other objects, and the localisation of faces relative to other objects, is fully determined prior to any postnatal experience. A boy who sustained brain damage at 1 day of age has the classic lesions and behavioural profile of adult-acquired prosopagnosia. He has profoundly impaired face recognition, whereas his recognition of objects is much less impaired. This implies that the human genome contains sufficiently explicit information about faces and nonface objects, or visual features by which they can be distinguished, that experience with these categories is not necessary for their functional delineation and differential brain localisation.

INTRODUCTION

Localisation of function as a long history of controversy in neuropsychology. Since the 19th century neuropsychologists have debated whether the neural substrates of psychological functions are specialised and segregated or multipurpose and shared (Feinberg & Farah, 1997). Recent advances in theory and methods have helped to resolve the issue in favour of a high degree of localisation. Information-processing theories from cognitive psychology have guided task analyses, which are crucial for testing localisation because they allow the relevant individual psychological functions, rather than whole tasks, to be localised. Functional neuroimaging has expanded the domain of evidence that can be used to test localisation, from behavioural impairments in brain-damaged patients to regional patterns of activity in normal brains. As a result of these developments, we now have strong evidence that many higher functions are carried out in localised neural substrates.

Face recognition constitutes a particularly interesting case of a localised brain function. It might seem implausible that face recognition would be segregated in the brain from other forms of object recognition, given the apparent similarity of the two processes. This intuition calls our attention to the distinct functional and anatomical aspects of the issue. The hypothesis that face recognition is a localised function of the brain can be thought of as two hypotheses bundled together. One of these concerns the functional organisation of the visual system, and is the hypothesis that face and object

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recognition are distinct in that they are carried out by separate systems. The other concerns the localisation per se of face recognition, and is the hypothesis that face and object recognition are functions of physically segregated populations of neurons.

A growing body of evidence supports the hypothesis that face recognition is functionally distinct and segregated from object recognition in the brain. Prosopagnosia, the impairment of face recognition after brain damage, can leave object recognition relatively intact (Farah, Levinson, & Klein, 1995; McNeill & Warrington, 1994). Conversely, some object agnosics have relatively spared face recognition (Feinberg, Schindler, Ochoa, Kwan, & Farah, 1994; Moscovitch, Winocur, & Behrmann, 1997). This double dissociation between face and object recognition implies that the two abilities are functionally distinct, in that either one can proceed without the other, and also implies that they are anatomically segregated, in that focal brain damage can selectively impair either one. Although inferences from brain-damaged patients concerning localisation are constrained by the size and systematic placement of the lesions, it would appear that object recognition is most dependent on left ventromedial temporo-occipital cortex (Feinberg et al., 1994), whereas ventral temporo-occipital areas of both hemispheres are necessary for face recognition, with some degree of greater specialisation on the right (DeRenzi, Perani, Carlesimo, Silveri, & Fazio, 1994).

Converging evidence for the anatomical segregation of face and object recognition comes from recent neuroimaging studies. A number of PET and fMRI studies have found distinct areas of activation during face and object recognition (e.g. Kanwisher, McDonald, & Chun, 1997; Sergent, Ohta, & McDonald, 1992). Although the precise localisation for face recognition is not consistent across these studies, the ventral temporo-occipital region is almost universally implicated.

As the issue of the localisation of face recognition reaches a resolution, another issue is raised: How might this localisation come about? Is the functional distinction between faces and other objects, and their segregation in neural tissue, fully determined at birth by genetic encoding, such that visual experience with faces and other objects is not necessary? Or does our genetic endowment for face and object recognition require interaction with a visual environment containing stimuli from these categories in order for the normal organisation of face and nonface object recognition to be expressed?

Studies of an earlier stage of visual perception suggest that experience plays a significant role in determining localisation of function. Research with cats, monkeys, and humans has shown that lesions of primary visual cortex have relatively little effect when sustained early in life (see Payne, Lomber, MacNeil, & Cornwell, 1996, for a review). This implies that the localised functions of a mature primary visual cortex are not entirely specified genetically; in an immature brain other systems have at least a partial capacity to carry out these functions. Is the same true of the ventral temporo-occipital regions that normally take on the function of face recognition in the mature brain? Or is the unique commitment of this area for face recognition present at birth?

To answer these questions, we must observe the effects of early damage to the cortical areas necessary for face recognition. The most relevant case reported to date is that of a prosopagnosic girl, who sustained brain damage at the age of 14 months (Young & Ellis, 1989). From this case we can conclude that by 14 months of age, the delineation of faces as a separate category of visual patterns, and the localisation of face recognition, are effectively complete. However, 14 months' worth of experience with faces allows for considerable learning-based changes in brain organisation.

How early would brain damage need to occur, in order for there to be reasonable certainty that brain organisation and localisation had been only minimally affected by experience? Morton and Johnson (1991) have put forward a two-process theory of infant face perception, according to which early face perception is driven by innate factors, and later face perception results from the interaction of these innate factors with a learning-based system. Their research suggests that the learning-based system begins functioning at about 2 months of age. From the vantage point of Morton and Johnson's work, then, the onset of brain damage at 14 months is too late to disentangle many of the factors in question. In order to evaluate the role of experience versus innate factors in the functional delineation and localisation of face recognition, a subject is needed whose brain damage occurred well before 2 months of age, ideally as a newborn. In the present paper we describe such an individual.

SUBJECT

Adam is a 16-year-old boy who, after a normal gestation and delivery, developed Group B streptococcal meningitis at 1 day of age. Following this diagnosis, the only developmental deficit noted was a lack of visual interest. This was presumed to be the result of infarction of the posterior cerebral arteries, a common occurrence in such cases. Later visual field testing suggested bilateral homonymous hemianopia, denser on the right than on the left, and denser inferiorly than superiorly.

Further confirmation of damage in the territories of the posterior cerebral arteries came from a CT scan performed at age 6 years, shown in Fig. 1, which reveals bilateral occipital and occipitotemporal lesions. These lesions, typical of prosopagnosia acquired in adulthood, clearly encompass more brain tissue than just the small "face area" identified in neuroimaging studies (e.g. Kanwisher et al., 1997). The present case study will therefore leave open the possibility of plasticity on a very local scale, with the function of one small part of the fusiform gyrus being taken over by another small neighbouring region of the same gyrus. However, Adam's relatively preserved object recognition

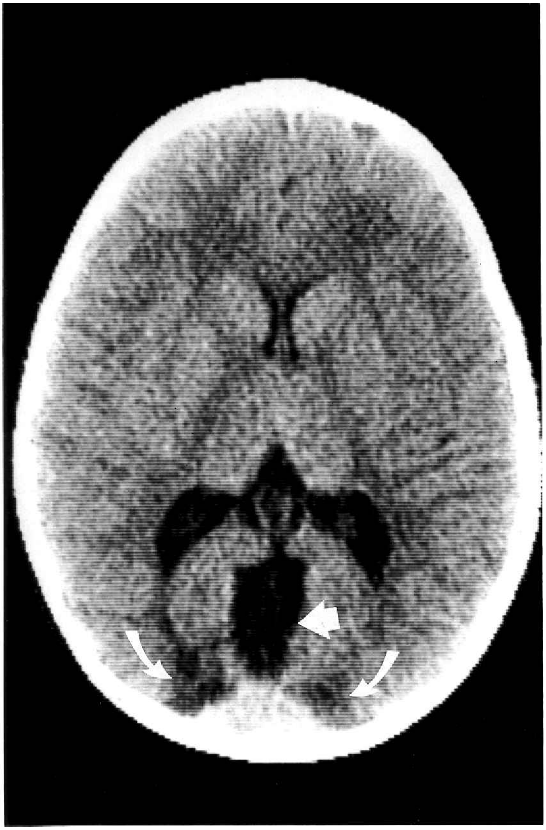

Fig. 1. *Axial CT scan of the brain, taken when the subject was 6 years of age, showing bilateral occipital (wide arrow) and occipitotemporal (curved arrow) infarctions.*

provides evidence against the form of plasticity most relevant to the issue of how face and object recognition come to be implemented separately in the brain, as the parts of visual association cortex that are able to support object recognition could not do the same for face recognition.

Adam's neurologic development has been normal except for problems with vision and visual recognition. At age 9 he was referred to a neuro-ophthalmologist because of his difficulty with face recognition. His most recent ophthalmologic exam showed esotropia, amblyopia of the left eye, and visual field abnormalities. Best corrected acuity is 20/80 for distance and 20/40 for near vision in the right eye and counting fingers vision in the left. Goldman kinetic perimetry performed at age 12 demonstrated a left central homonymous hemianopia and an incomplete right homonymous hemianopia with macular sparing. Although there were areas in the right visual field that appeared defective only for the left eye, this seemed attributable to the patient's concentration and cooperation with the procedure. Aside from some degree of incongruity in the right visual field, the results of perimetry were consistent with bilateral posterior lesions.

Adam's recognition abilities for both objects and faces appear more impaired than one would expect on the basis of these elementary visual deficits, with face recognition disproportionately impaired relative to object recognition. His performance on object and face processing tasks, described in detail here, conforms to the pattern of a classic prosopagnosic.

Adam has had a normal childhood in most respects, attending a combination of special education and mainstream classes at his local public school. On recent neuropsychological testing his verbal IQ was 101 and his performance IQ was 68.

INVESTIGATIONS OF OBJECT AND FACE RECOGNITION

The goal of the following investigations is to assess Adam's ability to recognise objects and faces, and to compare his pattern of performance with that of typical adult-onset prosopagnosia.

Object Recognition

Adam shows no discernable object agnosia with real objects in everyday life, but he does have some difficulty with photographs of objects. Line drawings pose a considerably greater problem, and when they are misidentified it is often for a visually similar item. The detrimental effect of going from realistic to more impoverished depictions of objects is typical of associative visual agnosics, including prosopagnosics (see Farah, 1990, for a review). In contrast, this pattern is not seen in other syndromes affecting high-level vision, such as optic aphasia. Another feature of Adam's performance that is seen in most cases of adult prosopagnosia is a difference between recognition of living and nonliving things (Farah, 1990); Adam is worse at recognising pictures of animals and plants than pictures of nonliving things.

Recognition of Photographs of Objects. Thirty colour photographs of inanimate objects were cut out of magazines and mounted on index cards. They included common household objects (e.g. cooking pot, keys), vehicles (e.g. plane, bicycle), and toys (e.g. balloon, skateboard). With 4 seconds to view each picture, he named 26/30, or 87%, of them correctly. He made errors on the teapot (calling it a "watering can"), the wooden stool ("a painting easel"), the screws (calling them "drill bits") and the rolls of toilet paper (calling them "rolls of tape"). All of these errors involve a visual resemblance between the object and his answer.

Recognition of Line Drawings of Objects. Stimuli were selected from the Snodgrass and Vanderwart (1980) corpus of line drawings, depicting a variety of common objects, buildings and other large outdoor objects, body parts, animals, and plants. Each drawing in the corpus is rated for complexity, familiarity, name frequency, and a number of other properties. We used the same subset of 85 drawings selected by Funnell and Sheridan (1992) in their study of semantic memory impairment, 43 of which depicted living things and 42 of which depicted nonliving things. Living and nonliving things in this subset were roughly equated for complexity, familiarity, and name frequency. The drawings were mounted on index cards and presented to Adam for 4sec each, with instructions to name each picture. He was asked to name each picture a total of six times without feedback, twice on each of three occasions.

Averaging over the six trials with each picture, Adam correctly named an average of 31.3/42, or 75%, of nonliving things, and 17/43, or 40%, of living things. This suggests that Adam has a mild or moderate visual object agnosia for nonliving things. Although exceptions may exist (e.g., DeRenzi, 1986), this is generally the rule in prosopagnosia. For example, case LH, a prosopagnosic who has been studied by a number of research groups because of the highly selective nature of his face recognition impairment (e.g. Etcoff, Freeman & Cave, 1991; Farah et al., 1995; Levine & Calvanio, 1989) named 84% of the same subset of drawings on which Adam obtained 75% correct. Another similarity to many adult prosopagnosics, including case LH, is that Adam performs worse with pictures of living things. For example, when tested on the same pictures of living things used with Adam, LH named an average of only 52% correctly.

The nature of Adam's errors in drawing recognition, as in photograph recognition, was invariably visual. For example, he misidentified celery as rope, a cigar as a crayon, and a broom as a spatula.

Face Recognition

In everyday life, Adam is profoundly impaired at face recognition. This was the problem that originally caused his mother to seek the help of a neuro-ophthalmologist. In group situations, for example in the school cafeteria, Adam generally waits until he is addressed to speak, because he is able to recognise voices but not faces. His mother reports that he never greets his school friends if he sees them unexpectedly out of context, for example on a shopping trip. In short, his prosopagnosia is profound, and has an enormous effect on his everyday life.

Like most prosopagnosics, Adam's face recognition impairment seems disproportionate to his elementary visual abilities, even when those elementary abilities are tested with photographs of faces. He has no trouble identifying the individual features of a face, and given sufficient time, can match identical photographs of faces quite accurately. However, when the camera angle or lighting changes between two photographs of the same face, his most diligent attempts at matching fail. This pattern is typical of prosopagnosia (Farah, 1990; Shuttleworth, Syring, & Allen, 1982).

The goals of the tests we performed were to establish his face recognition impairment with a set of famous faces to which he had ample exposure, and to assess his face perception ability with a standardised face matching task.

Recognition of Photographs of Faces. The Famous Faces test that has been used with many adult amnesics and prosopagnosics (Albert, Butters, & Levin, 1979) is not appropriate for a person Adam's age because he will never have encountered pictures of many of the people depicted. One source of famous faces to which Adam has been exposed is the TV show *Baywatch*. Adam's mother told us this is his favourite show, and that for the past 1½ years Adam has watched it for 1 hour every day. Luckily, the show has seven stars, and the studio kindly provided us with a press packet containing numerous photographs. We assembled a set of 10 clear, portrait-style colour photographs of the *Baywatch* cast, which we intermixed with 30 other photographs. Ten of the other pictures were clear magazine photographs of famous individuals with whom Adam was familiar (Tim Allen, Jim Carrey, Connie Chung, Bill Clinton, Newt Gingrich, Whitney Houston, Mick Jagger, Farrah Fawcett-Majors, Cybill Shepherd, and John Travolta), and the remaining 20 were magazine photographs of nonfamous faces.

Adam was shown these faces for as long as he wished to view them, and was told that some faces were famous and some were not. For each face he was asked who the person was, and was encouraged to guess even if he was not sure. He was unable to identify a single face, and refused to guess. We acknowledge that his refusal to guess could merely reflect an extremely conservative response bias, rather than an impairment in face recognition. However, on other tasks he showed himself willing to make errors, including both object picture naming and the face matching task to be described next. On these grounds, as well as his failure to demonstrate face recognition in everyday life, we suggest that a response bias account of his performance in this task is unlikely to be correct.

Matching of Faces. We used the Benton Test of Facial Recognition (Benton & van Allen, 1972), a standardised test in which black-and-white photographs of unfamiliar faces must be matched. When matching identical photographs, Adam proceeded in a slow and careful manner, comparing individual features of the faces and frequently commenting on the difficulty of the task and the specific features that he was finding helpful for a given face. Despite his abnormal approach, he obtained a perfect score on these trials. The slow but successful feature-based strategy is a common finding in associative prosopagnosia (Farah, 1990; Newcombe, 1979), and suggests that his elementary visual deficits are not responsible for his face recognition impairment. Also like a typical associative prosopagnosic, he performed poorly on trials in which angle and lighting varied, obtaining only 11 out of 21 correct. His overall score of 36 falls in the severely impaired range on this test.

GENERAL DISCUSSION

In every way that we have been able to observe, Adam appears to be a typical prosopagnosic. Anatomically, his lesions affect ventral occipitotemporal cortex bilaterally, as is the case with most adult prosopagnosics, and resulted from the most common aetiology of prosopagnosia in adults, namely bilateral infarction of the posterior cerebral arteries. His visual recognition impairments show a degree of selectivity comparable to most adult prosopagnosics. Although his object recognition is far from perfect, it is considerably better than his face recognition. In everyday life he is unable to recognise faces, whereas his object recognition ability is fully adequate for activities of daily living. We have also reported four ways in which the qualitative nature of his object and face recognition is similar to typical adult prosopagnosics. First, the more impoverished the visual stimulus (as with line drawings relative to photographs and photographs relative to real objects), the worse his performance. Second, living things are more difficult for him than nonliving things. Third, when errors of recognition are made, the wrong answer tends to be visually similar to the correct answer. Fourth, when faces are successfully matched, it is by virtue of a laborious, feature-by-feature strategy.

Adam differs from the typical prosopagnosic in one major way: His brain damage was sustained as a newborn, long before he had ever recognised a face. The fact that face recognition ability in this individual could not be supported by other, intact parts of the brain (for example the parts of the brain that enable him to recognise objects), has direct implications for the mechanisms by which face recognition comes to be localised in the brain. Specifically, it suggests that prior to visual experience, we are destined to carry out face and object recognition with different neural substrates. This in turn implies that some distinction between face and object recognition, and the anatomical localisation of face recognition, are explicitly specified in the genome. Whatever role environmental factors play in the normal unfolding of separate face and object recognition systems, some factor distinguishing between faces and nonface objects and their separate brain localisations does not require experience with stimuli from these different categories.

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