

We're getting warmer—characterizing the mechanisms of face recognition with acquired prosopagnosia: A comment on Riddoch et al. (2008)

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Prosopagnosia is a striking neuropsychological condition characterized by severe deficits in facial identity recognition despite normal early visual processing and high-level cognition. Individuals experiencing prosopagnosia have great difficulty recognizing friends, family, and even their own face. Reports of prosopagnosia in the neurological literature date back to the mid-19th century (Wigan, 1844), but Hoff and Potzl (1937) and then Bodamer (in 1947; see Ellis & Florence, 1990) were the first to suggest that selective face recognition deficits indicate that face recognition involves different mechanisms from those used for other types of visual recognition. These early studies of prosopagnosia inspired further investigations of this issue using a wide range of methods (Gross, Rocha-Miranda, & Bender, 1972; Sergent, Ohta, & MacDonald, 1992; Yin, 1969), but despite hundreds of relevant studies, debate about the mechanisms of face recognition continues.

Neuropsychological support for separate processing for faces and objects comes from double dissociations between face recognition and object recognition. Several cases have shown preserved or relatively spared face recognition with impaired object recognition (Feinberg, Schindler, Ochoa, & Kwan, 1994; McMullen, Fisk, & Phillips, 2000;

Moscovitch, Winocur, & Behrmann, 1997). Mr. C.K., the most well documented of these cases, repeatedly demonstrated normal recognition of upright faces coupled with very severe deficits with objects (Moscovitch & Moscovitch, 2000; Moscovitch et al., 1997). Conversely, a number of acquired prosopagnosics have exhibited normal within-class recognition of objects (McNeill & Warrington, 1993; Sergent & Signoret, 1992), and Riddoch et al.'s paper (Riddoch, Johnston, Bracewell, Boutsen, & Humphreys, 2008) adds another piece to the puzzle by documenting one of the best examples of pure prosopagnosia in the literature.

F.B., the case tested by Riddoch and colleagues (2008), has a right hemisphere lesion affecting the inferior occipital, fusiform, inferior temporal, and middle temporal gyri. Two of these areas, the inferior occipital gyrus and the fusiform gyrus, normally contain regions showing much stronger blood-oxygen-level-dependent (BOLD) responses to faces than to objects (Gauthier et al., 2000; Kanwisher, McDermott, & Chun, 1997; McCarthy, Puce, Gore, & Allison, 1997). On tests of face memory and face matching, F.B. showed very severe deficits. She was unable to name a single famous face, was at chance on an unfamiliar face memory test, and could not

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match sequentially presented faces in different views. Consistent with leading models that suggest parallel processing of different aspects of face processing, F.B.'s impairment with faces appears to be restricted to identity as she was normal on tests involving judgements of age, sex, and expression. To examine F.B.'s object recognition, she was asked to name objects from a wide range of categories (animals, fruits, vegetables, artefacts, tools, birds, flowers), and she did so normally. In a more rigorous test, her ability to associate names to a set of unfamiliar faces and a set of computer-generated multipart objects was compared. Controls found it more difficult to learn the object names than the face names. F.B. showed the reverse pattern: She learned the object names in fewer sessions and with shorter RTs than the control averages while she required more sessions than any of the controls to master the face names, and she made her responses more slowly than controls. F.B.'s dissociation between faces and objects suggests that recognition of faces and objects depends on separate mechanisms, but there are issues—one empirical and one theoretical—that limit the conclusions that can be drawn from the results of this interesting study.

Gauthier and her colleagues (Gauthier, Behrmann, & Tarr, 1999) pointed out that many neuropsychological results demonstrating dissociations between face and object recognition did not measure response time (RT). The absence of RT measures weakens evidence of dissociation by leaving open the possibility that normal object accuracy was achieved through slow responses. F.B. showed normal RTs with multipart objects, but RTs were not measured in other object tasks. Her normal performance with the multipart objects is suggestive, but to demonstrate an unequivocal dissociation between face and object recognition it will be necessary to show normal accuracy and RTs with several tasks requiring recognition of individual items from within an object category.

Riddoch and colleagues (2008) suggest that F.B.'s face and object dissociation are consistent with the existence of domain-specific cortical

regions specialized for face processing. We agree that her results are consistent with face-specific processes, but they are also consistent with other accounts of face recognition. Cases showing dissociations between impaired face recognition and normal object recognition are extremely valuable, but additional testing is needed in such cases to characterize the mechanisms carrying out face recognition. This is because a number of accounts of prosopagnosia acknowledge that face recognition and object recognition are dissociable, but do not propose that deficits to face-specific mechanisms explain the dissociation. For example, Martha Farah (1990) proposed a two-process theory of visual recognition, which consisted of a holistic system and a parts-based system. Faces were the prototypical stimulus for the holistic system, object recognition depended on one or both systems depending on the properties of the object, and word recognition depended on the parts-based system. Farah's scheme predicts dissociations between different categories but does not involve face-specific mechanisms. Similarly, other accounts of face-specific deficits that do not propose face-specific mechanisms suggest that the impaired mechanisms in such cases are specialized for something other than faces. These other proposed specializations include expert object processing (Gauthier et al., 1999), individual item recognition (Damasio, Damasio, & Van Hoesen, 1982), configural processing (Behrmann, Avidan, Marotta, & Kimchi, 2005; Levine & Calvanio, 1989), and curvature processing (Laeng & Caviness, 2001). To characterize the impaired mechanisms precisely in a prosopagnosic showing what appears to be a face-selective deficit, it is necessary to test the predictions of each account. Otherwise, what appears to be a face-specific deficit could be an impairment to holistic processing, expert object processing, and so on. Our laboratory carried out extensive testing with a developmental prosopagnosic and ruled out all of the existing alternatives to the face-specific account (Duchaine, Yovel, Butterworth, & Nakayama, 2006), but such testing has not been done with an acquired prosopagnosic. The selectivity of F.B.'s deficit

makes her an ideal candidate for such an approach.

Her case also may shed light on the neural basis of face processing. A network of areas is involved in face processing, but the role of these areas and their interactions remain poorly understood. Valuable insight into these issues has come from functional magnetic resonance imaging (fMRI) studies of prosopagnosic patients P.S. and D.F. that measured face-selective activations and the response of these areas when facial identity was repeated (Rossion et al., 2003; Schiltz et al., 2006; Steeves et al., 2006). Similar experiments with F.B., especially given the selectivity of her deficits, would be valuable. To take one example, if F.B. shows no right occipital face area (Gauthier et al., 2000) or right fusiform face area (Kanwisher et al., 1997) as her lesion location suggests, what effect does their absence have on the face-selective activation normally seen in the superior temporal sulcus in the right hemisphere or the face-selective activations in the left hemisphere? Similar testing done with substantial numbers of prosopagnosics using a battery of well-designed behavioural tests and structural and functional magnetic resonance imaging (MRI) should allow considerable progress into the neural basis of face processing and might lead to the development of a taxonomy of prosopagnosia. Such studies would be a nice human complement to recent monkey work combining fMRI, single-cell recording, and microstimulation (Tsao, Freiwald, Tootell, & Livingstone, 2006), and convergence between human and monkey results would be an encouraging sign.

In summary, F.B. is an exciting case who presents what appears to be a very selective impairment for facial identity. Dissociations like F.B.'s provide strong support for the dissociability of face and object recognition, but future testing involving theoretically motivated tasks may allow a more precise characterization of the mechanisms whose impairment has left her unable to recognize faces. We agree with the authors that face-selective mechanisms are the most likely culprit, but acquired prosopagnosia has yet to provide unequivocal evidence for this possibility.

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