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Response to Susilo and Duchaine: beyond neuropsychological dissociations in understanding face and word representations

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We welcome the opportunity to clarify our theoretical position in light of comments by Susilo and Duchaine. Our central claim is that face and word processing are carried out by a distributed network of partially specialized cortical regions, with the degree of specialization varying across individuals (partly as a function of language lateralization). Thus, and perhaps not surprisingly, Susilo and Duchaine are adopting an overly all-or-none perspective when they mischaracterize our views as implying that cortical regions are 'not specialized for particular categories' or that 'individuals with prosopagnosia will *always* have some deficits in word recognition while individuals with alexia will *always* have some deficits in face recognition' (emphasis added). Rather, on our view, cortical regions are not dedicated to categories, and patients with severe face or word impairments will, as a population, tend to be more moderately impaired in the other domain, as well.

To be clear, Susilo and Duchaine are absolutely correct to emphasize the importance of considering neuropsychological dissociations of face and word processing in evaluating our theory. It is worth pointing out that our claims concern the representations that support visual recognition of faces and words; we certainly do not claim that there are no mechanisms of any sort that are relevant to the processing of words, but not of faces (or vice versa). Thus, depending on the nature of the testing, performance on faces and words might dissociate for reasons outside the scope of our theory.

More generally, however, as theories confront the complexities of individual differences among pre- and post-morbid populations, traditional neuropsychological inference based on single-case studies does not suffice [1,2] and a broader consideration of the full distribution of effects caused by brain damage is needed (see, e.g., [3]). Thus, it is difficult to interpret Farah's [4] report of cases of prosopagnosia without pure alexia and vice versa, not only because such cases were not always subject to sufficiently and equally rigorous testing in both domains, but also because we have little information about the distribution of patients with comparable brain damage who are impaired at both (most of whom would not even be reported in the literature). Specifically regarding patient CK, although he may simply fall in the tail of the expected distribution, he is also atypical in the nature of his lesion. Whereas most patients with higher-level recognition impairments have a lesion to occipitotemporal regions, often including the fusiform gyrus [5,6], CK has more posterior bilateral occipital thinning with no frank lesion apparent on MRI.

For all their other flaws, modular theories do have the virtue that their implications are relatively straightforward. Distributed theories, by contrast, may require more subtle reasoning, but, we believe, are closer to the actual nature of the system.

References

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