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COMMENTARY

Prosopdysgnosia? What could it tell us about the neural organization of face and object recognition?

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Geskin and Behrmann (2017) analyzed more than 700 scientific reports of people suffering from long-term developmental difficulties at individual face recognition (IFR) published over four decades (1976–2017). The goal of their review, an invaluable contribution for the field, is to enlighten the long-standing issue of whether faces are handled by a specific neural visual recognition system or if face and non-face objects share the same system. Here, I focus mainly on the parallel made by the authors, both at the theoretical and methodological levels, between long-term developmental difficulties for IFR—what they call *congenital prosopagnosia* (commonly named *developmental prosopagnosia*)—and the classic form of prosopagnosia, which follows brain damage in a typically developed individual.

It is worth reminding that Bodamer (1947) coined the term *prosopagnosia* (from the Greek "prosopon", *face*, and "a-gnosia", *without knowledge*) as a *selective* neurological disorder of IFR, stressing that the recognition deficit could "*appear in varying strengths and together with the various forms of agnosia, but could be separated from these from the outset*" (Bodamer, 1947, p. 10). In reality, his first patient had basic-level object recognition difficulties, the second was not tested sufficiently, and the third suffered from a deformation of face percepts without IFR impairment. Hence, Bodamer could not prove the very existence of prosopagnosia, making his a particularly audacious proposal at the time. According to the first review of the condition, the IFR impairment could be observed in some neurological patients "*in the absence of deficit in the recognition of objects*" (Hecaen & Angelergues, 1962, p. 98). However, systematic experiments to ensure the absence of object recognition impairment, i.e., with quantitative measures and statistical analyses, started only in the mid-1960s, with the first experimental studies on IFR in neurological patients (e.g., De Renzi & Spinnler, 1966).

Given that prosopagnosia was initially defined as a form of visual (object) agnosia, the recognition deficit has to be modality-specific (i.e., visual), yet unaccountable by low-level visual defects and/or intellectual impairments. Critically, the prosopagnosic deficit concerns both the faces known before and encountered after brain damage. This is valid whether prosopagnosia is of the apperceptive or associative type (if one believes in such a distinction), and allows prosopagnosia to be distinguished from patients with anterograde amnesia, who can still recognize faces learned before medial temporal lobe damage. Finally, in this classical sense, prosopagnosia is a *massive* disorder of IFR, not to be confused with mere difficulties at IFR as found in a fair proportion of patients with right posterior brain damage (Benton & Van Allen, 1972).

Given the rarity of this classic, domain-specific, neurological *syndrome* (i.e., with inclusive and exclusive clinical signs), the term prosopagnosia became used to refer to the *symptom*, i.e., the IFR impairment in visual object agnosia. Even so, on the basis of such criteria, it is difficult to understand how the term came to be applied to a neurodevelopmental disorder. Indeed, in the so-called congenital/developmental form of prosopagnosia, a retrograde deficit is by definition impossible, and the impairment can reflect mere difficulties, i.e., a score below 2 standard deviations at a given test (or even less, see Geskin & Behrmann, 2017). Moreover, cases have been reported with deficits in other modalities (e.g., name

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recognition: Dobel, Bölte, Aicher, & Schweinberger, 2007). In short, while prosopagnosia implies a deficit at individual face recognition, the scientific community seems to have now accepted the converse, that any difficulty at individual face recognition should be called prosopagnosia.

Is this a problem? For some researchers, the wider the definition of prosopagnosia, the better, as if including as many cases as possible in an analysis—such as performed by Geskin and Behrmann (2017)—could ameliorate our understanding of the condition and of IFR. I beg to differ. Rather, I believe that the most conservative definition of prosopagnosia, restricted to a neurological syndrome with stringent criteria, gives us the best chance as a scientific community to understand human IFR. Other researchers may argue the name is not important as long as the definition is clear, but I think the modern ambiguity of the term prosopagnosia leads to major difficulties in understanding the nature of IFR, its impairment, and neural basis. For this reason, and given the (lack of) current criteria, I suggest referring to the developmental disorder as *prosopdysgnosia* (i.e., "dys" for "difficulty" in Greek, as often used for neurodevelopmental learning disorders, e.g., *dys*lexia or *dys*calculia). But is the developmental deficit at IFR selective enough to faces to even deserve the "*prosop*" label? This is the question asked by Geskin and Behrmann (2017), who, after thorough analysis of 238 cases sufficiently described, conclude against face selectivity, or at least against convincing evidence for selectivity.

Although I largely agree with these conclusions, I do not follow a number of derived implications, especially in the authors' attempt to generalize their findings and methods to classical cases of prosopagnosia (i.e., the acquired disorder) and to constrain the neural organization of face and object recognition from studies of prosopdysgnosia.

First, the authors point to insufficient documentation of most reported cases of prosopdysgnosia, especially for the limited proportion (20%) with apparent preserved object recognition. Indeed, one cannot conclude on the basis of a few discrimination tasks with non-face objects that object recognition is preserved. For instance, the reported case of prosopagnosia LH performed normally in discriminating pictures of glasses (Farah, Levinson, & Klein, 1995), as the authors mention, yet he suffered from severe object recognition deficits, i.e., visual object agnosia (Levine & Calvanio, 1989).

Second, they call for response times (RTs) to be taken into consideration to evaluate integrity of the visual recognition function. I could not agree more on this point, which is emphasized in all of our case studies of prosopagnosia (e.g., Busigny, Graf, Mayer, & Rossion, 2010). Unless there is a general slowing down (e.g., in response execution), if object recognition is abnormally slow, then it must be impaired. This is particularly critical in matching tasks, which can often be successfully completed by prosopagnosic patients, even for faces, at the expense of prolonged RTs. However, I do not share Geskin and Behrmann (2017)'s proposal that brief exposure duration may replace RT measures so that a measure of accuracy alone would be sufficient in such conditions. Rather, I would suggest taking advantage of recent developments in human electrophysiology to apply severe processing time constraints (e.g., requiring single glance recognition) while measuring neural indexes of individual face discrimination during simple orthogonal tasks (see Liu-Shuang, Torfs, & Rossion, 2016, for the use of this approach in prosopagnosia).

Third, the authors tend to diminish the importance of single case reports, emphasizing analyses of large cohorts. I agree for prosopdysgnosia. In fact, I would even argue against a single case approach for a neurodevelopmental disorder occurring frequently in the population (even more so if the individuals studied represent the lower tail of a normal distribution of performance in the population). However, for classical cases of prosopagnosia, single case studies should be the methodology of choice. This is not because of brain damage *per se*: the provided example of the single exception in a hundred cases of semantic dementia is well taken, but there is no shortage of semantic dementia patients due to progressive brain degeneration of fronto-temporal regions, so that there is no reason to favor a single-case approach in this domain. However, a single case approach is favored when patients are extremely rare, but also when acute focal brain injuries and associated defects are vastly different across patients (Caramazza, 1986), which is the case in prosopagnosia. In this context, my colleagues and I have been studying the patient PS for 18 years, in hundreds of experiments reported in about 30 publications (Rossion, 2014, for review; Ramon, Busigny, Gosselin, & Rossion, 2016,

for updated references), and her single case of prosopagnosia has proved invaluable—at least for me—in understanding the nature of IFR. Admittedly, such an approach is only valid in the long run if the findings are replicated and strengthened with various methodologies, as well as in other single cases of prosopagnosia, properly defined, when they become available (Busigny, Joubert, Felician, Ceccaldi, & Rossion, 2010).

Fourth, Geskin and Behrmann (2017) play down the theoretical relevance of neuropsychological dissociations and emphasize the theoretical relevance of the frequent association between object and face recognition deficits. However, early dysfunction of general low-level and/or high-level processes (e.g., in the early visual pathway or the medial temporal lobe) is likely to affect the *organization* of visual categories as the system develops, necessarily leading to frequent associations of deficits. This does not imply that, during *typical* development, visual recognition mechanisms cannot specialize for faces, due to extensive experience with these signals and the specific requirements in their individualization for social communication. Brain damage occurring suddenly in such a typically developed face recognition system, in which categories have been carved out in the ventral occipito-temporal cortex, could then lead to selective individual face recognition impairments.

Finally, Geskin and Behrmann (2017) discuss the difficulties in recognizing exemplars of visually homogenous categories in reported cases of prosopagnosia. This long-standing within-category recognition account (Damasio, Damasio, & Van Hoesen, 1982; Faust, 1947) does not hold: prosopagnosic patients without basic-level object recognition impairments have been reported, such as PS, who are able to correctly and rapidly individualize visual exemplars of non-face categories (Busigny, Graf, et al., 2010; Busigny, Joubert, et al., 2010). Moreover, these patients are relatively less impaired with physically similar than dissimilar faces. Unfortunately, this within-category account of prosopagnosia persists because of its evaluation with visual object agnosic patients (Gauthier, Behrmann, & Tarr, 1999), who make visual underspecification errors when attempting to recognize non-face objects (Levine & Calvanio, 1989). This again supports the view that significant progress in understanding the nature of human face recognition requires restricting the definition of prosopagnosia to an acquired selective disorder of visual recognition, i.e., with no basic-level object recognition impairment, in line with its original definition.

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