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Damasio's error – Prosopagnosia with intact within-category object recognition

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The sudden inability to recognize individual faces following brain damage was first reported in a scientific journal 150 years ago and termed 'prosopagnosia' 70 years ago. While the term originally identified a face-selective neurological condition, it is now obscured by a sequence of imprecisions. First, prosopagnosia is routinely used to define symptoms of individual face recognition (IFR) difficulties in the context of visual object agnosia or other neurological conditions, or even in the normal population. Second, this over-expansive definition has lent support to a long-standing within-category recognition account of prosopagnosia, that is, that the impairment of IFR reflects a general impairment in recognizing within-category objects. However, stringent experimental studies of classical cases of prosopagnosia following brain damage show that their core impairment is not in recognizing physically similar exemplars within non-face object categories. Instead, the impairment presents specifically for recognizing exemplars of the category of faces. Moreover, compared to typical observers, the impairment appears even more severe for recognizing individual faces against physically dissimilar than similar distractors. Here, I argue that we need to limit accordingly our definition of prosopagnosia to a clinical (i.e., neurological) condition in which there is no basic-level object recognition impairment. Other criteria for prosopagnosia are proposed, with the hope that this conservative definition enables the study of human IFR processes in isolation, and supports progress in understanding the nature of these processes.

The year 2017 marked a double anniversary in the history of prosopagnosia. First, 150 years ago, Antonio Quaglino, professor of ophthalmology at the university of Pavia, reported the first patient with a sudden inability to recognize a person by his or her face in a scientific journal (Quaglino & Borelli, 1867; partial English translation by Della Salla & Young, 2003).[1] This patient, LL, had suffered a right hemisphere stroke obliterating his ability to recognize familiar individuals from their faces, yet was able to read even the smallest characters very well, with excellent near and far vision. Second, seventy years

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[1] *Perhaps the first description of difficulty with faces after suspected brain injury can be found as far back as in the writings of the Greek general Thucydides (Thucydides II, 47–50), where he describes the behaviour of plague survivors (De Haan, 1999): 'For the disorder which had originally settled in the head passed gradually through the whole body, and, if a person got over the worst, would often seize the extremities and leave its mark . . . Some again had no sooner recovered than they were seized with a forgetfulness of all things and knew neither themselves nor their friends'.*

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ago, in an extensive paper describing three neurological patients, the German neurologist Joachim Bodamer (1947) coined the term *prosopagnosia* (from the Greek 'prosopon', *face*, and 'a-gnosia', *without knowledge*) as a *selective* disorder of individual face recognition (IFR) following brain damage. However, the existence not only of prosopagnosia but also of visual agnosia in general remained heavily contested for decades after this definition. Eberhard Bay (1950, 1953) in particular, but also others (Bender & Feldman, 1972; Critchley, 1964), argued strongly against visual agnosia, namely a defect of recognition that could be specific to a given sensory modality (i.e., vision) and yet not accounted for by a deficiency of the sensory organs themselves or of the pathways conveying sensory information to the brain, considered to be the seat of amodal higher cognitive functions. This issue was progressively resolved only in the second half of the 20th century by showing that low-level visual impairments alone could not account for object recognition deficits in visual agnosia (De Haan, Heywood, Young, Edelstyn, & Newcombe, 1995; Ettlinger, 1956).

The time it took to accept visual agnosia and prosopagnosia in neuropsychology was related to the extreme rarity of the condition: relatively few patients could be studied, diagnosed, and understood carefully. Today, with prosopagnosia at least, neuropsychologists seem to have the opposite problem to deal with: the term *prosopagnosia* is routinely used to refer to a symptom rather than a neurological condition, that is, to virtually any complaint or difficulty at IFR in a given individual, with or without neurological history (i.e., the so-called developmental or congenital form of prosopagnosia; since McConachie, 1976; e.g., Behrmann & Avidan, 2005; Duchaine & Nakayama, 2006a, Geskin & Behrmann, 2018; Towler, Fisher, & Eimer, 2017). That is, while prosopagnosia implies a deficit at IFR, the scientific community now seems to accept the converse that a deficit at IFR necessarily implies prosopagnosia. As a result, the term prosopagnosia, which has now entered the layman's lexicon and even appeared in the latest James Bond movie, *Spectre*, has become largely unspecific, with many so-called cases of prosopagnosia reported and studied in scientific papers. For some researchers, it seems that the wider the definition of prosopagnosia the better, as if including as many cases as possible could ameliorate our understanding of the condition and of the nature of IFR (e.g., Geskin & Behrmann, 2018; Zhao *et al.*, 2016; see Rossion, 2018a for a brief critical view of this approach).

Individual recognition, as a term, refers to a subset of recognition that occurs when one organism identifies another according to its individually distinctive characteristics (Tibbetts & Dale, 2007). It is the most precise form of recognition, involving and requiring unique (combinations of) cues. In humans, the face is the visible body part which carries the richest, that is, most diverse, signals for identity across individuals, at both a genetic and morphological level (Sheehan & Nachman, 2014). In our species at least, IFR is a key brain function, considered to be at the interface of the most complex processes of visual perception and memory. It requires many subprocesses, including: the extraction and combination of low-level visual information, the discrimination of an individual's face as a unique visual pattern, concerning its shape, texture, and colour, from competitive similar patterns (i.e., faces of other individuals), the generalization of this face across substantial changes of appearance (i.e., invariance), the memory encoding, updating and implicit/explicit recollection of this face, as well as its association with specific semantic, lexical (i.e., names) and emotional information which cannot be extracted or constructed directly from the visual stimulus.

Although many animal species have a face – a part of the body which developed originally for food prehension and sensory interactions with the physical world (McNeill,

2000) – and live in social groups, very few animal species may actually rely on the face to individually recognize conspecifics (e.g., the queens in one species of wasps for a few individuals, see Tibbetts, 2002). In fact, no species appears to rely to such an extent on the face for individual recognition with such a high level of performance in terms of accuracy, speed, and automaticity as the human species. In humans, IFR is absolutely essential for social interactions,[2] and it has even been suggested that the face, in particular, has evolved throughout human evolution to carry face identity signals (Sheehan & Nachman, 2014). Indeed, humans often recognize a familiar face at a glance, encode a seemingly unlimited number of new faces throughout life without explicit instructions to do so, and recognize them in a wide variety of viewing conditions. For these reasons, some researchers have suggested that typical human adults are *experts* at IFR (Carey, 1992; Tanaka, 2001; Young & Burton, 2018) even if the definition of expertise and its operationalization remain controversial (see Rossion, 2018b; Young & Burton, 2018).

Although it can be painful in terms of social consequences for the patient, prosopagnosia is therefore a gift of nature for researchers because it potentially offers a unique window to understand how the human brain achieves IFR by comparing an *expert* recognition system (i.e., a typically developed human adult) to a system missing *only* this expertise. One of the goals of the present critical review is to try to convince the scientific community that adopting a conservative definition of prosopagnosia – as a specific neurological condition, not a symptom – is critical in this endeavour. In contrast, referring to any deficit at IFR, with or without brain damage, as 'prosopagnosia' has not only made the condition largely opaque, but is detrimental to our understanding of the nature of IFR. More specifically, I will consider only IFR deficits following brain damage here and will attempt to demonstrate that within this neurological population a conservative definition of prosopagnosia, that is, in the absence of basic-level object recognition impairment, is not only helpful but necessary to resolve one of the most contentious and enduring issues in the study of prosopagnosia: whether prosopagnosia can be accounted for as a general defect at recognizing items belonging to visually homogenous categories.

I chose to call this review 'Damasio's error' because Antonio Damasio, early in his career, made a number of contributions on prosopagnosia, including an influential review in 1982 in which he and his colleagues claimed that prosopagnosia was due to a general (i.e., not face-specific) deficit at recognizing items belonging to visually homogenous categories (Damasio, Damasio, & Van Hoesen, 1982; see also Damasio, Damasio, & Tranel, 1986; Damasio, Tranel, & Damasio, 1990). The initial paper of these authors, published 35 years ago, that is, exactly in between Bodamer's account in 1947 and the present review, is the most referred to review in the field[3] and is still cited today as an up-to-date review of prosopagnosia (e.g., Keller *et al.*, 2017; Ungerleider & Bell, 2011[4]). Of course, since Damasio *et al.*'s (1982) article, there have been a number of papers, including reviews, which have addressed the issue of domain-specificity of prosopagnosia (e.g., Barton, 2008; Barton & Corrow, 2016; Davies-Thompson, Pancaroglu, & Barton, 2014; De

[2] *It could be argued that those individuals who are bad at IFR or lose it after brain damage, despite obvious difficulties, still survive and can deal with this impaired function in their social environment relatively well, toning down its importance. However, the importance of the function should be judged at the level of the (human) population; not the affected individual alone, who could still be easily recognized by others from his/her face. If, suddenly,* all *humans were unable to recognize individual faces, their society would be profoundly affected.*

[3] *691 citations as of March 2018 in the Web of Science, 1,196 in Google Scholar.*

[4] *Somewhat ironically, Damasio* et al. *(1982)'s review is often cited to support the view of domain-specificity of prosopagnosia, for example, Ungerleider & Bell (2011, p. 783): 'In cases of pure prosopagnosia, the ability to classify and categorize nonface stimuli is unimpaired (see Damasio* et al.*, 1982, for review)'.*

Renzi, 1986; Ellis & Young, 1989; Farah, 1990/2004; Farah, Wilson, Drain, & Tanaka, 1998; Gauthier, Behrmann, & Tarr, 1999; Henke, Schweinberger, Grigo, Klos, & Sommer, 1998; McNeil & Warrington, 1993; Sergent & Signoret, 1992; Tovée, 1998). Some of these authors have argued against Damasio *et al.* (1982)'s position, that is, in favour of domain-specificity (e.g., De Renzi, 1986; Ellis & Young, 1989; Farah *et al.*, 1998; McNeil & Warrington, 1993), in particular because some patients with prosopagnosia were still able to recognize exemplars of non-face object categories (e.g., Bruyer *et al.*, 1983; De Renzi, 1986; Henke *et al.*, 1998; McNeil & Warrington, 1993; Rezlescu, Pitcher, & Duchaine, 2012; Sergent & Signoret, 1992). However, for many researchers, including those most active in this field, the within-category account remains completely open today (Barton & Corrow, 2016; Geskin & Behrmann, 2018). For example, to quote one of the latest reviews on prosopagnosia (Barton & Corrow, 2016, p. 78): 'This is the enduring question: is prosopagnosia truly about faces, or are faces only the most obvious stimulus type affected by a problem in discriminating between highly similar exemplars of any object category?'.

I believe that the reason why this issue is still considered to be largely unresolved by many authors is due to a major problem: the loose definition and diagnosis of prosopagnosia. This issue will be the main focus of the present critical review, which will be divided into four further sections. Following the present introduction, the next section (The within-category recognition account of prosopagnosia) will summarize the evidence and arguments in favour of the within-category account, and their limitations. The following section (Ruling out the within-category recognition account) will summarize detailed evidence against this account, in particular focusing on studies performed with the patient PS (Rossion *et al.*, 2003). In the fourth section (Why does the visual similarity account persist?), I will discuss the reasons why the within-category/visual similarity account persists and show that these reasons are not valid. In particular, I will try to illustrate how the inclusion of patients with basic-level object recognition impairments as cases of 'prosopagnosia' leads to spurious supportive elements in favour of the within-category account. In this fourth section, I will also more directly address the issue of visual expertise, which has sometimes been conflated with the within-category account of prosopagnosia at the expense of clarity. I will argue in favour of a *domain-specific* (i.e., *face-specific*) *expertise* rather than a generic expertise account of prosopagnosia and IFR. Finally, the conclusion will offer a summary of the position held here and propose other criteria for a highly conservative definition of prosopagnosia, to isolate the IFR impairment and support progress in our understanding of the nature of IFR in the healthy adult human brain.

The within-category recognition account of prosopagnosia

Faust, a contemporary of Bodamer, initially proposed that prosopagnosia reflects a general inability to *grasp the detailed features of a stimulus so that its individuality is not appreciated*, a claim supported by his observations of neurological patients who had difficulties distinguishing objects belonging to the same category, such as a chair versus an armchair (Faust, 1947, 1955). Supporting this view, other reported prosopagnosic patients presented with difficulties recognizing specific foods and animals (Pallis, 1955), car brands (Macrae & Trolle, 1956), fruits (De Renzi, Faglioni, & Spinnler, 1968), horses (Newcombe, 1979), similar birds (Bornstein, 1963), or individual cows (Bornstein, Sroka, & Munitz, 1969) (see Barton & Corrow, 2016 for a longer list of such cases, and the discussion below). Subsequently, Damasio *et al.* (1982) described two patients who, in addition to their IFR impairment, were unable to recognize their own cars among other cars in a parking lot or 'were able to recognize visual items such as "owl," "elephant" or "horse", but failed at recognizing different instances of visually similar cats, with some being named "tiger" or "panther"'. Therefore, these authors adopted and clarified Faust's view that prosopagnosia was not a deficit specific to faces *per se*.

Damasio *et al.* (1982) argued that the patient often emphasized the IFR impairment because (1) faces are highly visually similar to each other; that is, they form a visually homogenous category and (2) their individualization is mandatory for social reasons. In contrast, objects may be usefully recognized following a coarser level of analysis ('generic', or 'basic-level' recognition; Rosch, Mervis, Gray, Johnson, & Boyes-Braem, 1976). According to these authors, prosopagnosic patients have no difficulty with individual recognition of visual stimuli belonging to groups in which different members have a different visual structure. However, they cannot recognize visual stimuli belonging to classes in which numerous members are physically similar and yet individually different, of which faces are only one example (Damasio *et al.*, 1982, 1986). This is the *within-category recognition* hypothesis, also called the *visual similarity account* of prosopagnosia. Yet, remarkably, despite continuous presentation and argumentation about this theoretical view in several review papers, Damasio and his colleagues never went beyond anecdotal reports to support it.

Aiming to provide experimental support for the within-category (visually homogenous) recognition hypothesis, Gauthier *et al.* (1999) tested two reported prosopagnosic patients, SM and CR, with matching tasks in which the similarity of a distractor to the target item was variable. For instance, the same picture of a duck ('Duck1') was used in a same/different decision task with three levels of difficulty for the 'different' trials: the target could differ from the distractor object at (1) the basic level (e.g., *Duck1* vs. *Chair*); (2) the subordinate level (e.g., *Duck1* vs. *Pelican*); or (3) the exemplar level (e.g., *Duck1* vs. *Duck2*) (Figure 1A). Following with eight similar experiments, often with 2-alternative forced-choice paradigms and various object shapes, including novel objects termed 'Greebles' (Figure 1B), the authors concluded that the two neurological patients showed a disproportionate decrease in performance relative to normal controls, as manifested in particular by abnormally increased response times (RTs) with increasing levels of visual similarity between the target and the distractor. Hence, they took these observations as supporting the within-category recognition account of prosopagnosia.

In essence, these authors' intentions were correct: a stringent test of the visual similarity hypothesis requires experimental manipulation of the degree of visual similarity between stimuli to discriminate. Moreover, and importantly, one needs to take into account not only accuracy rates (or sensitivity) but the speed of processing of the patients, as was assessed with the RT variable in that study. However, the results of this study are largely inconclusive and in fact contradict the conclusions of the authors (see also Busigny, Graf, Mayer, & Rossion, 2010). First, in the vast majority of cases, the results did not support the authors' hypotheses; that is, there was no disproportionate increase in error rates or RTs for the patients relative to normal controls (Figure 1A–C; see also figures and tables of results in Gauthier *et al.*, 1999). Second, there was no *parametric* manipulation of physical similarity between the different levels of discrimination tested. Consequently, the slopes of sensitivity and RTs were not always linear, and not even systematically ascending, in normal controls (Figure 1A,B), making it difficult to test the authors' hypotheses in several of their experiments.

Finally, and critically for our point here, the two patients tested, CR and SM, had large impairments of basic-level object recognition to start with, as indicated by their failures to

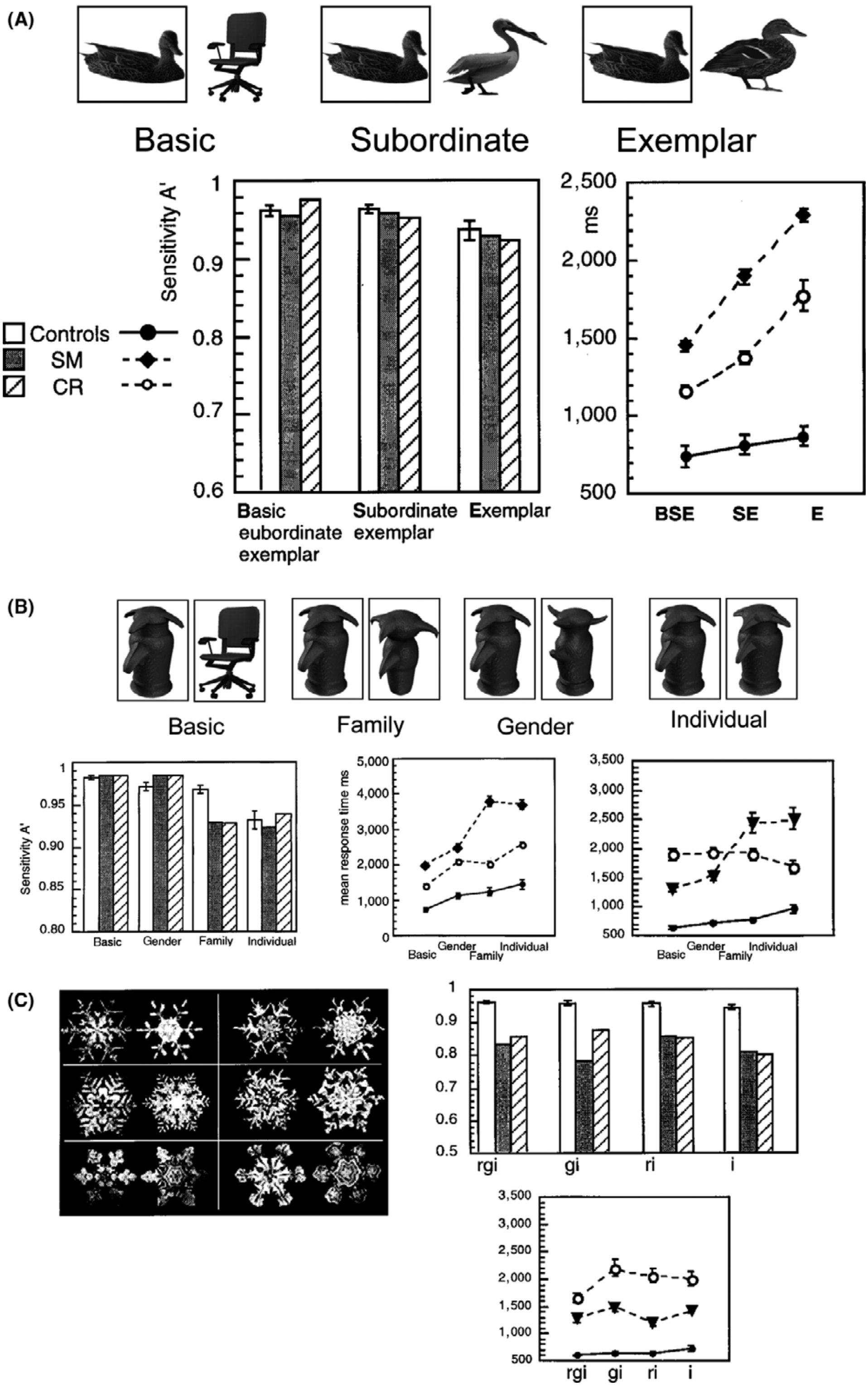
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Figure 1. Examples of stimuli and results from the study of Gauthier *et al.* (1999), testing the within-category recognition hypothesis with brain-damaged patients, SM and CR. (A) In the first experiment, the patients had to judge whether two simultaneously presented pictures were identical or different. Note that even for the discrimination of a chair from a duck, the two patients were largely slowed down relative to normal controls. (B) Similar experiments (forced-choice matching tasks) of that study performed with four levels of discrimination of novel shapes, the Greebles, failing to show disproportionate increases of errors and response times (RTs) for the two patients relative to controls. The two graphs with RTs are from two separate experiments with the same stimuli. (C) Results obtained at discriminating four types of snowflakes, organized by their level of physical similarity. Note the lack of decrease in performance of the patients (and controls) with increasing levels of physical similarity, as well as the significantly lower performance of the patients at all levels of discrimination.

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recognize many of the Snodgrass and Vanderwart drawings, or items from the Boston naming test (table 5 in Gauthier *et al.*, 1999). That is, the two patients had general *visual object agnosia*, a condition for which they have been studied in other experiments (e.g., Behrmann & Kimchi, 2003; Behrmann & Williams, 2007). Hence, there was already a difference at the basic-level discrimination (i.e., baseline) between the patients and controls; that is, they performed worse than controls even to differentiate the images of a duck and a chair (Figure 1A).

This is especially problematic with real objects, because while typical participants can discriminate objects based both on visual and semantic/lexical processes, the visual agnosic patients have to rely on visual processes exclusively. This issue was taken care of using novel stimuli such as Greebles (Figure 1B), or requiring within-category discriminations, as in other experiments of that study, for example, with images of snowflakes (Figure 1C). However, regardless of this precaution, and most importantly, basic-level object recognition difficulties cause a baseline deficit between the patients and controls' performance (Figure 1). Note that this baseline basic-object recognition problem is not unique to the patients tested by Gauthier *et al.* (1999), but appears also in the anecdotal reports of Damasio *et al.* (1982) on their patients' recognition difficulties, or other reports as listed by Barton and Corrow (2016). Exactly how this deficit needs to be taken into account is unclear, but one could argue that to estimate the cost of increasing similarity between a target and a distractor, *proportional* increases of error rates and RTs should be considered with increasing levels of visual similarity rather than stable levels as in Gauthier *et al.* (1999).[5]

In short, beyond anecdotal reports, which could be interpreted in different ways, the proponents of the within-category account of prosopagnosia have not developed solid arguments: there is no empirical evidence that the severe IFR difficulty in a number of neurological patients is *due* to individual faces being more similar to one another than individual exemplars of other object categories. Yet, surprisingly, as mentioned at the beginning of the present review, this view is still largely considered to be a valid account of prosopagnosia (Barton & Corrow, 2016; Geskin & Behrmann, 2018). This is unfortunate because it leads to a decreased specificity in understanding and studying the nature of the

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[5] *This point could be illustrated by the following example: if the patient takes 1,000 ms at level 1 of discrimination and 2,000 ms at level 2, while normal controls, on average, take 500 and 1,000 ms, respectively, they show the same proportional RT increase, by a factor of 2, between the two levels of discrimination. However, the slope will appear steeper in the patient's data, and the analysis of Gauthier* et al. *(1999) would conclude in favour of an abnormally increased impairment in the patient's data with increasing levels of discrimination.*

key process impaired in prosopagnosia, that is, IFR. To demonstrate that, let me first provide decisive evidence against the within-category recognition account of prosopagnosia.

Ruling out the within-category recognition account

PS, a case of prosopagnosia with preserved basic-level object recognition

Patient PS sustained a severe closed head injury in 1992, just before her 42nd birthday. Before the dramatic accident (she was hit by the mirror of a bus), she had no neurological history and was working as a kindergarten teacher. My colleagues and I started testing PS about 7.5 years after her accident, in early 2000. Structural scans of PS's brain show extensive lesions of the right inferior occipital gyrus, the left mid-ventral occipito-temporal cortex (i.e., midfusiform gyrus), together with smaller lesions to the left posterior cerebellum and the right middle temporal gyrus (Figures 1,2; Rossion *et al.*, 2003; see Sorger, Goebel, Schiltz, & Rossion, 2007 for detailed anatomical data).

Since her accident and to this date, PS's only continuing complaint concerns her profound difficulty at recognizing individuals by their face, including those of family members and herself. To determine a person's identity, she usually relies on contextual information, her excellent memory, and non-facial cues such as the person's voice, posture, and gait. However, she may also use suboptimal facial cues such as the mouth (Caldara *et al.*, 2005; Ramon, Busigny, Gosselin, & Rossion, 2016). Despite her excellent knowledge of celebrities, PS's recognition of pictures of famous faces is close to zero, as is her discrimination between personally familiar and unfamiliar faces (Busigny, Prairial, *et al.*, 2014; Busigny & Rossion, 2010a; Rossion *et al.*, 2003). She is also severely impaired and slowed down at individual face matching tasks such as the Benton Face Recognition Test (Benton & Van Allen, 1972; see Busigny & Rossion, 2010a, 2010b; Rossion & Michel, 2018), and impaired at explicit encoding and recognition of individual faces among distractors as in the Cambridge Face Memory Test (Duchaine & Nakayama, 2006b; see Ramon *et al.*, 2016).

PS's case has been described extensively in previous publications spanning from 2003 to near the present date, that is, about 30 papers in total, reflecting over 18 years of testing in various laboratories. This makes her case of prosopagnosia, to my knowledge, by far the most documented in the scientific literature (see Rossion, 2014 for a previous review; and Ramon *et al.*, 2016 for updated references). Her performance at standard clinical and neuropsychological tests of visual perception and recognition was initially reported in table 1 of Rossion *et al.* (2003) and Sorger *et al.* (2007). PS's colour vision is in the low normal range (Sorger *et al.*, 2007). Her visual field is almost full (with an exception of a small left paracentral scotoma of about 2–3° by 3°, see Sorger *et al.*, 2007), and her visual acuity is in the lower normal range.

Despite this, PS neither complains nor presents any difficulty at recognizing non-face objects in real life, to the point that she is surprised that we tested her so much at recognizing non-face object stimuli. For instance, we asked her to name out loud, as accurately and rapidly as possible, the full set of the 260 coloured drawings of Snodgrass and Vanderwart (Rossion & Pourtois, 2004) presented one by one. Although some of these objects are not easy to recognize from the drawings and it is a difficult task for which she cannot correct herself, PS named each object in turn without hesitation. She made only a

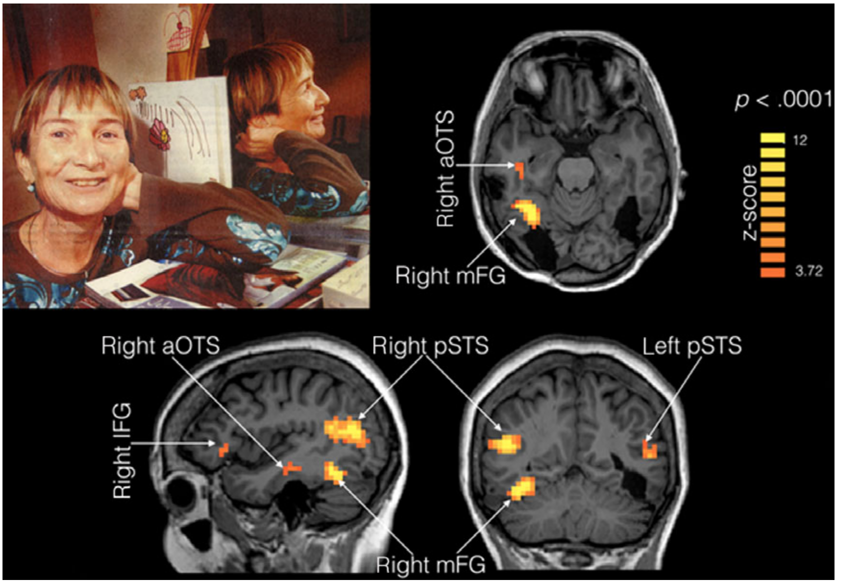

Figure 2. The prosopagnosic patient PS, here photographed around 2005. The brain images are from a recent neuroimaging examination localizing face-selective regions with high specificity and sensitivity in typical participants (Gao, Gentile, & Rossion, 2018). PS underwent severe brain damage mainly in the right inferior occipital gyrus and the left middle fusiform gyrus. Note that the third cortical lesion, in the right hemisphere, concerns the lateral section of the temporal lobe, that is, the right middle temporal gyrus, not the ventral anterior temporal lobe, which is shown to be intact here on the sagittal, coronal, and transverse slices. Despite extensive brain damage, face-selective responses are observed in the right middle fusiform gyrus ('Fusiform Face Area'), but also in the right and left posterior superior temporal sulcus (pSTS), and the right anterior occipito-temporal sulcus (aOTS).

few mistakes or failures to recognize, that is, on 8 stimuli of 260, with no systematic difficulties: she did not recognize a poor drawing of a pepper (which she named correctly and rapidly on another drawing), a lobster drawn from the top, an ant, a cloud (poorly drawn); a bee was named as a fly with hesitation, but there also the poor drawing and ambiguous colour make it understandable. A violin was too rapidly named a guitar. She also could not recognize the raccoon and black-footed ferret, animals that she simply did not know about and never encountered before in her environment. Excluding these last two, her score on rapid naming of animal pictures was of 49/52 and of 23/24 for fruits and vegetables.[6] As also shown in videos, PS is able to name another set of fruits and vegetables pictures, which often have similar shapes,[7] without any mistakes, and quickly, and whose recognition is often impaired in other reported cases of prosopagnosia (e.g., 9 of 10 patients reported in Barton 2008). On this basis, it is clear that PS does not suffer from a basic-level visual object recognition impairment.

[6] *See here http://face-categorization-lab.webnode.com/pictures/ for a full video of PS naming the objects one by one (in French). The video was filmed a few a few years after the original report of PS's performance at naming the Snodgrass and Vanderwart stimuli (Rossion* et al.*, 2003).*

[7] *See here (http://face-categorization-lab.webnode.com/pictures/) for a full video of PS naming the fruits and vegetables one by one (in French).*

A systematic deconstruction of the within-category recognition account

Computer games and within-category discrimination

One anecdotal piece of evidence against the visual similarity account was offered to us when PS spontaneously started to match arbitrary shapes in a video memory game that she found in a bookstore[8]: she had absolutely no difficulty doing that. At the experimental level, we tested her in a task requiring to match one of two exemplars of a given category (birds, boats, cars, houses, and faces) to a previously shown target (Figure 3). For non-face objects, PS was able to do this task at the same level of performance as age-matched controls, and even equally rapidly as controls (which is impressive, regarding her slight low-level visual defects, in particular the scotoma). However, for faces, PS made many more mistakes than age-matched controls and, importantly, was also much slower.

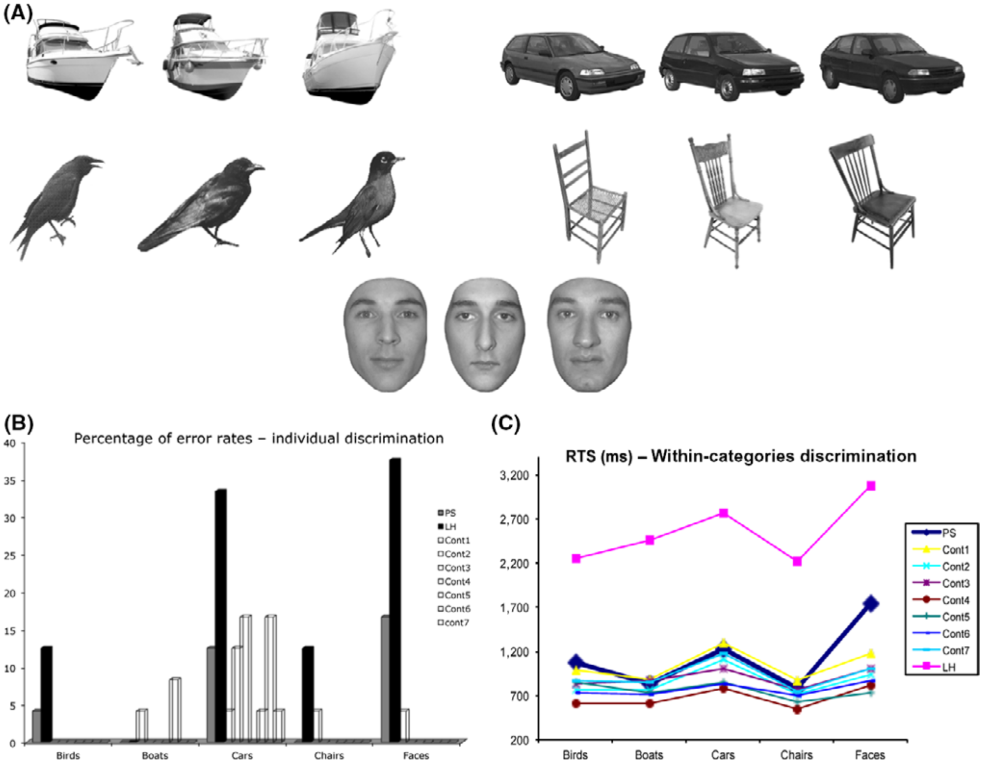

Figure 3. Within-category discrimination task as reported in Schiltz *et al.* (2006) for testing the patient PS. (A) Exemplars of the five categories of stimuli used, in which a target is shown first, followed by the same target and a distractor. (B) PS made more mistakes than controls only for faces, while the visual agnosic patient LH, often referred to as a case of prosopagnosia, made mistakes also for non-face object categories (cars, birds, chairs). (C) Relative to normal controls, PS was slowed down only for faces, while LH was slowed down for all categories. LH, who suffered a car accident when he was 18-year old, was tested in this task in 2004, at the age of 54, and is matched in age to PS and the control participants.

[8] *See here (http://face-categorization-lab.webnode.com/pictures/) for a full video of PS matching similar shapes in the 'memory' game.*

Contrast that with the performance at the exact same task of the patient LH, defined by many researchers as a case of prosopagnosia (e.g., Barton, 2008; Etcoff, Freeman, & Cave, 1991; Farah, Levinson, & Klein, 1995; Farah, Wilson, Drain, & Tanaka, 1995; Levine, Calvanio, & Wolf, 1980) in the context of his visual object agnosia (Farah, McMullen, & Meyer, 1991; Levine *et al.*, 1980): LH was most severely impaired with faces, but also had clear difficulties at within-category recognition, in particular making more mistakes than controls with pictures of birds, cars, and chairs (Figure 3B). He was also slowed down for all within-category discriminations (Figure 3C). It is clear that the two patients do not show the same profile of responses across conditions.

Parametric manipulations with non-face shapes

To address the visual similarity account of prosopagnosia more directly, PS was tested with delayed forced-choice matching tasks in which the similarity of the distractor to a target was parametrically manipulated (Figure 4; Busigny, Graf, *et al.*, 2010). In three separate behavioural experiments, PS was tested with single shapes ('geons'), non-face artificial objects from both living and non-living categories, and photographs of a well-known category (cars). In these tasks, PS was shown with a single stimulus for 500 ms (geon shapes, objects) or 2,000 ms (cars), which was replaced, after a brief blank screen, by the same object appearing next to a distractor, until the response was made. This is a difficult task, in which typical observers make mistakes. In the worst case, we thought that PS might be a little bit delayed at the easiest discrimination level if low-level visual defects had an effect in the task. However, the most important thing was to assess whether any slight decrement of performance at baseline level for PS would increase *disproportionally* with increasing visual similarity between a target and its distractors, as postulated by the visual similarity account of prosopagnosia (Damasio *et al.*, 1982; Faust, 1955; Gauthier *et al.*, 1999).

Strikingly, whether PS was tested with single geon shapes, morphed non-face artificial objects or morphed pictures of cars, she did not show abnormally high error rates or (correct) RTs with increasing levels of visual similarity relative to controls. In fact, her performance was undistinguishable from controls' at *any* level of physical similarity between the target and its distractors (Busigny, Graf, *et al.*, 2010; Figure 4). These observations were made despite the tasks being quite difficult for typical observers, as judged by their error rates and RTs, with some of the discriminations truly requiring fine-grained analysis of the stimuli (Figure 4). Hence, they do not support the view that prosopagnosia – when there is no basic-level object recognition impairment – is due to a difficulty at recognizing visually similarly objects.

Parametric manipulations with faces

Before going further, let me address two potential limitations of these observations. First, as there is only minimal short-term memory load in the paradigm described above, one could argue that these experiments evaluate only 'matching' or 'discrimination' of images, and not really 'recognition', thought to be the primary impairment in prosopagnosia. In the jargon of experimental psychology, a 'recognition task' is a terminology often used to refer to tasks in which an item is encoded in long-term memory and then later recognized; while a 'matching task' involves two or more of those signals that have to be associated (i.e., matched), sometimes with a brief delay, sometimes when the two signals are presented simultaneously. Such tasks are also sometimes referred to as a 'memory task'

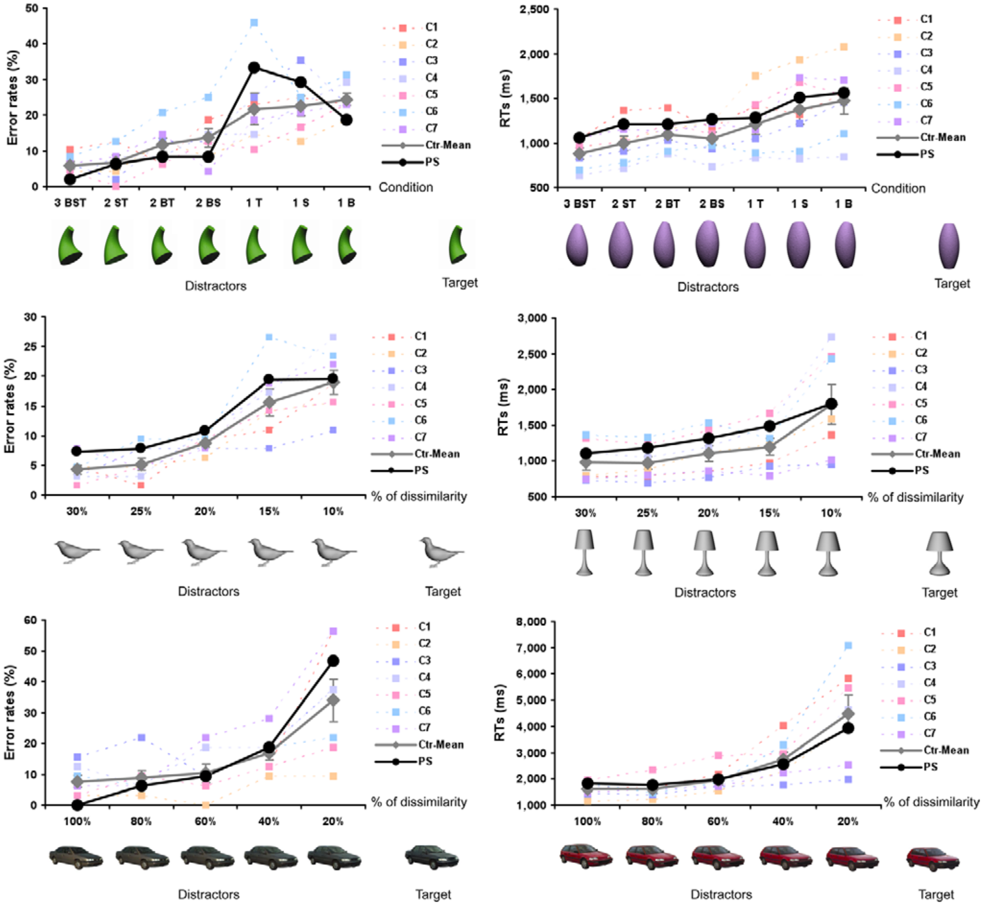

Figure 4. PS's performance compared to normal controls at three separate experiments testing the matching of single geon shapes (top row; 12 shapes in total in the experiment), morphed living and non-living objects (eight of each category), and morphed photographs of cars (20). Parametric increases in visual similarity between the target and a distractor leads to increases in error rates and response times, with no difference in slope between PS and age- and gender-matched typical controls (from Busigny, Graf, *et al.*, 2010).

versus a 'perceptual task', respectively, but this is misleading, if only because behavioural performance at (individual face) matching tasks reflects much more than the contribution of a perceptual process, and is never memory-free. In fact, fundamentally, these tasks measure the same process, that is, *recognition*: in order to be matched to one another efficiently, these two signals (i.e., two pictures of individual cars) have to be associated with the same signal in the brain, that is, 'recognized'. In any case, it is worth a reminder that the patient PS is not only impaired at tasks requiring the recognition of a face stored in memory for a long time, but she also scores well below normal range, and is abnormally slowed down, at matching two individual pictures of unfamiliar faces (e.g., Figure 3), even when these images are presented simultaneously (Busigny & Rossion, 2010a, 2010b; Rossion *et al.*, 2003). Hence, the use of a delayed matching task rather than a long-term old/new recognition paradigm cannot be the issue here.

A second potential limitation is that the target images to match in the tasks depicted above (Figure 3) are strictly identical, except for a change of position. One could therefore argue that PS succeeds at this task only because she would rely on so-called pictorial codes (Riddoch, Johnston, Bracewell, Boutsen, & Humphreys, 2008) or stimulus recognition rather than face recognition (Hay & Young, 1982), with the latter requiring a true invariant representation of the stimuli (i.e., a 'structural code'; Bruce, 1982; Bruce & Young, 1986). This type of criticism has generally been made by authors who argue that matching performance for pictures of unfamiliar faces in the typical population is low, unless the exact same images are used and matching is based on 'pictorial cues' (e.g., Hancock, Bruce, & Burton, 2000; Young & Burton, 2018). Although I fully agree with the importance of testing the matching of object and face representations across different viewing conditions in general (see Busigny & Rossion, 2010a, 2010b; Rossion *et al.*, 2003; Schiltz *et al.*, 2006 for such tests in PS), there is no reason to expect that typical human observers primarily match identical images of objects or faces based on pictorial cues, whatever these may be (see Sergent, 1989 for a discussion of this issue). In any case, should PS be the only one to have to rely on such pictorial cues, she should at least be largely slowed down relative to normal controls in these tasks.

Rather than a long(er) argumentation, there is a very direct way to rule out these potential criticisms and show that the paradigm is appropriate to reveal the presence of visual recognition impairments: testing PS in the exact same paradigm with pictures of *faces*. This is in fact a key manipulation, which had been missing in previous attempts to dismiss the within-category account of prosopagnosia. For instance, following Damasio *et al.* (1982)'s emphasis on the within-category account of prosopagnosia, De Renzi (1986) reported a case of prosopagnosia (his fourth patient), who could still identify his own belongings mixed with 6–10 similar items from the same category (i.e., razor, wallet, ties, glasses). The patient could also identify his own handwriting from others, pick out a Siamese cat from photographs of different cats and sort out Italian coins from foreign coins. Although De Renzi (1986) used this evidence to conclude against the within-category account of prosopagnosia (see also Ellis & Young, 1989), the author did not report any objective data, neither in terms of accuracy rates nor speed of recognition. Moreover, such tasks may not truly reflect individualization of many exemplars from visually homogenous categories, as required for IFR. In fact, as Sergent and Signoret (1992) rightly noted and demonstrated for two cases of prosopagnosia, these tasks could be succeeded even for faces (e.g., finding his own face or a specific face among 6–10 exemplars, sorting out Asian from Caucasian faces, etc.). In the same vein, Henke *et al.* (1998) reported a case of prosopagnosia, MT, who was able to recognize car brands as well as fruits and vegetables, but without testing exemplar recognition, providing RT measures or comparing with (individual) face recognition.

In contrast, the paradigms described above and tested with the patient PS required exemplar recognition, the stimuli belonged to increasingly homogenous categories and the patient did not differ from normal participants on both accuracy rates and RTs. To make the final step, we therefore tested the same paradigm with faces in which we parametrically manipulated the physical similarity of the individual face distractors. This key experiment, using morphed pictures of faces, showed that PS clearly performs below controls, both in accuracy and RTs, even *when the exact same images* of faces have to be matched in this paradigm (Figure 5). Moreover, for the same normal controls as tested with cars, the task was even slightly easier than the exact same task performed with pictures of cars (compare Figures 4 and 5, see also Busigny, Graf, *et al.*, 2010), leading to a double dissociation between PS's and typical participants' performance.

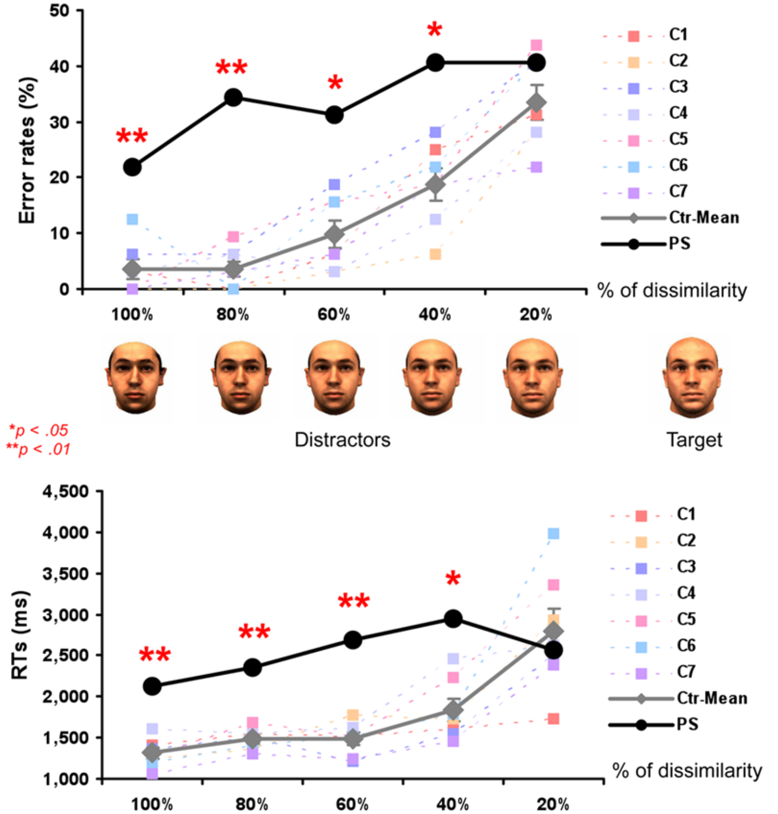

Figure 5. Performance of the prosopagnosic patient PS at the same delayed matching task as tested with non-face objects (Figure 4), here with morphed face stimuli. Despite the task being, if anything, relatively easier than with pictures of cars for normal controls (compare Figures 4 and 5), here PS showed clear difficulties, which were apparent even at the easiest individual face discrimination level (i.e., 100% difference on morph continua). Thirty-two individual faces were used in the experiment (see Busigny, Graf, *et al.*, 2010 for details about the stimuli and procedure).

Strikingly, for faces, PS was impaired even at the *lowest* level of similarity between exemplars, that is, when faces were the most different from each other. In fact, relative to normal controls, PS was impaired the most significantly when the faces were clearly different (100–80%), and was not impaired when the faces were as similar as twins (20% morph difference; despite performance being above chance level in the normal population in this condition, see Figure 5). Overall, the pattern of preserved and impaired performance is shown in Figures 4 and 5 rules out an account of PS's prosopagnosia as a defect in discriminating similarly looking visual patterns.

Why does the visual similarity account persist?

It seems to me that the empirical observations summarized above should have put to bed the view of prosopagnosia as a defect in recognizing items belonging to visually homogenous categories. Why then, as noted in the Introduction, do recent reviews on prosopagnosia present this issue as still completely open to debate (Barton & Corrow, 2016; Geskin & Behrmann, 2018)? If we leave aside reasons belonging to the sociology of science (e.g., the difficulty to identify signal among noise in the enormous amount of scientific publications these days, and the fact that scientists do not only like to create debates but are also encouraged to raise debates, reopen them, and extend them indefinitely by the commercial publication system), let me discuss a few reasons why the within-category/visual similarity account persists.

The burden of the proof

It has been stated that proving that a patient does not have any problems at object recognition requires one to accept proof of the null hypothesis (Barton & Corrow, 2016; Ellis & Young, 1989). As I understand this, one would have to *prove* the domain-specificity of prosopagnosia, and doing so would require testing an infinite number of categories in the visual world. To quote Barton and Corrow (2016, p. 79): 'some have devised batteries testing within-category recognition of a diversity of object types (. . .) but this still begs the question of how many and which types of objects one needs to test to prove that a deficit is unique to faces'. In all due respect, I do not think that this is the correct way of reasoning about this issue. In my view, there is no need to prove the domain-specificity of prosopagnosia by testing the recognition of an infinite, or even a large (e.g., Rezlescu *et al.*, 2012) number of visual categories. In most cases of reported prosopagnosia, there is not even a need to test for within-category recognition because the patients *do* have problems at basic-level object recognition that are readily apparent (e.g., 9 of 10 patients reported in Barton, 2008 fail at a simple and untimed recognition test of fruits and vegetables; see also the section The meaning of visual underspecification errors in cases of visual agnosia below). Moreover, the visual similarity account (Damasio *et al.*, 1982; Faust, 1955; Gauthier *et al.*, 1999) is by definition a *non-category-specific* account: it states that there should be a problem in discriminating between exemplars of *any* visually homogenous object category (as acknowledged by Barton & Corrow, 2016, p. 78, see the first quotation of these authors, above). Hence, it can be ruled out with the kind of evidence provided above, with a few categories tested stringently (i.e., parametric manipulations including correct RT measures), including faces. To put it differently, if a patient has difficulties recognizing only faces and animal forms but not other visually homogenous categories, providing that visual homogeneity/difficulty is matched between the preserved and impaired categories, then the visual similarity account is already highly questionable.

The weight of single-case evidence

Another reason why the within-category account persists is that, to assess the visual similarity hypothesis, Barton and Corrow (2016), as well as other review papers discussing this issue (e.g., Ellis & Young, 1989; Geskin & Behrmann, 2018), seem to adopt the logic that *all* reported cases of prosopagnosia should be considered. Thus, to be more specific, they consider for instance Bornstein's (1963) patients who had difficulties recognizing birds, Barton (2008)'s and De Renzi *et al.* (1968)'s at recognizing fruits, Damasio *et al.* (1982)'s at findings their car or recognizing tigers and panthers, or Clarke, Lindemann, Maeder, Borruat, and Assal (1997)'s at recognizing plants, fish, or mountains, as well as cases who could recognize objects within some visually homogenous categories, in search of a *global* explanation of all reported cases of prosopagnosia. I believe that this is also misdirected: assessing the validity of the within-category recognition hypothesis does not require explaining *all kinds* of visual recognition deficits in the neurological population. Indeed, these IFR impairments can be due to various aetiologies, a variety of brain lesions and associated impairments. Rather, the goal of this research is to put constraints on our understanding of the organization of the normal visual recognition system using the pathology. To do this, studying the most selective impairment, which can be well defined and isolated in a single case, is not only sufficient but is even recommended as a methodology (Caramazza & McCloskey, 1988; Ellis & Young, 1988; Shallice, 1979; Zihl & Heywood, 2016). In this context, providing that the observations are solid and replicated, a *single* case of prosopagnosia such as the patient PS, can be sufficient to disprove the within-category recognition hypothesis.

Still, to lend further support, it is worth mentioning that the experiments described above were replicated in two other cases of prosopagnosia, LR and GG, who differ at several levels from PS (e.g., age, gender, aetiology, lesion localization, and associated impairments). Patient GG suffered a stroke of the right posterior cerebral artery in 2002, at the age of 60 years old. Besides his prosopagnosia, he had a left hemianopia and a topographical orientation impairment. The case of LR was reported originally by Bukach, Bub, Gauthier, and Tarr (2006) as a patient who had received a head wound in a motor vehicle accident at 19 years of age, affecting primarily his right anterior temporal lobe and causing his inability to recognize individual faces, including highly familiar individuals, such as his daughter. Both GG and LR have preserved basic-level object recognition and reading abilities (Busigny, Joubert, Felician, Ceccaldi, & Rossion, 2010; Busigny, Van Belle, *et al.*, 2014). Importantly, when tested in the very same experiments as PS, neither LR nor GG showed abnormally increased difficulty relative to normal controls in matching increasingly similarly looking non-facial shapes, such as cars (Busigny, Joubert, *et al.*, 2010; Busigny, Van Belle, *et al.*, 2014). Moreover, as PS, both patients were impaired relative to controls even more at the lowest level of physical similarity for faces, showing similar profiles of responses, differing from healthy participants (i.e., a less steep increase in error rates and RTs; Busigny, Joubert, *et al.*, 2010; Busigny, Van Belle, *et al.*, 2014). Note that the overall performance of the patients in these tasks varied from PS, and that this performance had to be compared to the patients' own controls. Ideally, they should also have been tested with their own parameters of stimulation. For instance, while LR, the youngest of these patients, has an intact visual field and no low-level visual defects (Bukach *et al.*, 2006), GG has a complete left hemianopia. It is remarkable that despite these differences, these three prosopagnosic patients show a similar *profile* of performance in these tasks (Busigny *et al.*, 2010b).

The meaning of visual underspecification errors in cases of visual agnosia

The third reason why the within-category account persists is the inclusion of patients with clear visual agnosia as cases of prosopagnosia. Let me illustrate this in this section and suggest an alternative account of the difficulty for identifying items from physically similar distractors by cases defined as prosopagnosic who have basic-level object recognition defects. First, it seems to me that patients who have difficulties at basic-level object recognition simply complain particularly about recognizing objects that matter to them in their environment. If they have a hobby with plants, fish, or mountains for instance, they complain about recognizing plants, fish, or mountains (e.g., Clarke *et al.*, 1997). However, it is not because the kinds of objects that they find difficult to recognize belong to visually homogenous categories that the *root* of their impairment (with faces and non-face objects) lies in a specific visual recognition process that deals with visually similar objects.

The problem can be well illustrated by the patient LH (mentioned above, and included in Figure 3), who has been defined as a case of prosopagnosia by many researchers (Barton, 2008; Etcoff *et al.*, 1991; Farah, Levinson, *et al.*, 1995; Farah, Wilson, *et al.*, 1995). As described by Farah, Wilson, *et al.* (1995, p. 663) 'The prosopagnosic subject was LH, a 40 year old man who has been prosopagnosic since an automobile accident 20 years earlier . . . His main residual impairment is in face recognition. He is profoundly prosopagnosic, unable to recognize friends, neighbors or even his wife and children without extra-facial cues. His recognition of real objects and pictures is only mildly impaired' (text underlined by the present author). In fact, this case was even used to support the view that IFR is supported by a domain-specific system (Farah, Levinson, *et al.*, 1995), and LH has subsequently been cited as a patient having a selective face recognition deficit by other authors (e.g., Henke *et al.*, 1998).

However, LH's 'mild impairment at object recognition' or 'selective impairment for faces' is at odds with the report of the very same patient described a few years earlier by Levine and Calvanio (1989, pp. 153–154): 'In addition to his extraordinary difficulties in recognizing faces, LH is impaired in identifying other visual forms. Animals are especially difficult. In one session, he was shown 12 realistic color drawings of animals (3 different dogs, 3 bears, 3 cats and 3 horses). He named only 4 correctly, making errors of visual underspecification, such as calling a panda an owl, and a horse a giraffe'. The octopus was called 'I have no idea. It's not mammalian'. For this reason, LH was also studied as a case of visual object agnosia, showing dissociation between the recognition of living (impaired) and non-living (preserved) things (Farah *et al.*, 1991) (see also the patient RM in Sergent & Signoret, 1992; and Herschel in Rezlescu *et al.*, 2012; who had particular difficulties with animal shapes). Moreover, Levine and Calvanio reported that the patient LH was particularly slowed down at object recognition: 'LH identified 21 of the 25 pictures of common objects, each exposed on an illuminated screen in a dark room for 10 seconds. He named 15 of them before the exposure was complete, but his identification time was prolonged (3.7 seconds on average). Six were named after 10 seconds. . .' (Levine & Calvanio, 1989, p. 154). On this basis, LH can hardly be defined as a case of selective impairment in face recognition, contradicting the statement of Farah, Levinson, *et al.* (1995) and Farah, Wilson, *et al.* (1995): he has basic-level object recognition deficits, that is, visual object agnosia (additionally, see Figure 3).

Most importantly for our purpose, this description of LH by Levine and Calvanio (1989) serves as a good illustration of a patient with visual object agnosia who has enhanced difficulties at recognizing object shapes when they have physically similar competitors ('visual underspecification errors'). Yet, there is no evidence that such errors are due to a specific visual process also used to individualize exemplars of faces. As reported by Levine and Calvanio (1989, p. 154), LH confused a horse for a giraffe, animals that are not particularly similar in shape, let alone a panda and an owl. This could be because in the first case, they both have four legs and a large body well above the ground. Faced with the picture of an octopus, LH could presumably tell it was not mammalian from the number of tentacles of the animal. In the same vein, the picture of an anvil was called 'maybe a briefcase with string coming out'. What these examples serve to illustrate is that recognition mistakes may simply arise because visual agnosic patients can guess single parts of the objects, one by one, and then attempt to make semantic inferences about what the object could be.

This behaviour is typical of cases of *integrative visual agnosia*, such as the patient HJA (Riddoch & Humphreys, 1987), NS (Delvenne, Seron, Coyette, & Rossion, 2004), or in fact, SM and CR (Behrmann, Peterson, Moscovitch, & Suzuki, 2006; Behrmann & Williams, 2007), who are impaired at recognizing objects, although they can still guess a number of these objects correctly. Although, semantically, these part-by-part deductions have more chance to land on objects that are visually similar, it does not imply that the *core* of the impairment lies in a specific process shared with faces to individualize exemplars. Rather, I would argue these patients with visual agnosia cannot recognize objects because they cannot match a visual input to an object representation. This is true whether the objects have many physically similar distractors in real life or not: their visual recognition process is as impaired when they are shown fruits, vegetables, animals, or common objects. And, with animals, their visual recognition system does not work better when faced with the picture of a penguin or a platypus (i.e., atypical animals) than a cat or a dog (i.e., typical animals). However, by analysing an object part-by-part, they can make semantic guesses, which are more likely to be successful in the case of man-made objects, which have distinct shapes often related to their function, or animals with atypical shapes than animals sharing the same physical properties (four legs, etc.).

Weak definition and diagnosis of prosopagnosia

The above example shows that without a careful selection of patients, that is, excluding patients with basic-level object recognition impairments to start with, these anecdotal reports of patients having problems at recognizing items that appear visually similar may be misleading regarding the source of the impairment in prosopagnosia. In this context, it is worth remembering that Bodamer (1947) defined *prosopagnosia* as: 'The selective disturbance in grasping physionomies, both of one's own face as well as of those of others, which are seen, but not as the physionomy of a particular individual'.[9] He next claimed that: 'It appears in varying strengths and together with the various forms of agnosia, but can be separated from these from the outset' (Bodamer, 1947, p. 10; text underlined here; see Ellis & Florence, 1990 for a partial translation of the original paper). Hence, as noted before by Ellis and Young (1989), the very term 'prosopagnosia' *implies* specificity.[10] Along Bodamer's initial proposal, I would indeed argue that one of the criteria for diagnosis of prosopagnosia should be that the subject does not have visual object agnosia, that is, deficits at basic-level object recognition.

In truth, one of Bodamer (1947)'s patients had basic-level object recognition difficulties, as he could not recognize a rabbit, a dog or a chicken; his second patient was not tested in depth, and his third patient did not suffer from prosopagnosia but from metamorphopsia, a deformation of face percepts usually with preserved recognition (Hécaen & Angelergues, 1962; Nijboer, Ruis, van der Worp, & De Haan, 2008; see discussion in Jonas *et al.*, 2018). Hence, Bodamer was in fact unable to prove that prosopagnosia existed, such that his proposal at the time was particularly audacious.

[9] *'. . . ist die Prosopagnosie die elektive Storung im Erfassen von Physiognomien, . . .' (Bodamer, 1947, p. 10).*

[10] *Here the term 'selective' clearly means 'specific', that is,* only *for faces. In human face recognition research, the term 'selective' has become somewhat ambiguous, following its application to single-neurons in the monkey superior temporal sulcus responding only to faces, but with a quantitative criterion (i.e., a response to faces that is twice as much as for non-face objects; Desimone, 1991) and then its application to functional brain regions in neuroimaging. These regions, such as the 'Fusiform Face Area' (Kanwisher, McDermott, & Chun, 1997) are defined as being face-selective not because they respond only to faces but for their significantly lower response to non-face objects (see Jonas* et al., *2016 for face-exclusive responses in human intracerebral recordings).*

Similarly, Quaglino's patient also spontaneously complained of difficulties at recognizing familiar visual scenes and the facades of houses (Quaglino & Borelli, 1867). Nevertheless, Hécaen and colleagues reported in the first reviews on the condition that prosopagnosia could be observed in several cases 'in the absence of deficit in the recognition of objects' (Hécaen & Angelergues, 1962; Hécaen, Angelergues, Bernhardt, & Chiarelli, 1957). However, systematic experiments to ensure the absence of a clear visual object agnosia, that is, with the collection of quantitative measures and statistical analyses, were not carried out at the time on these patients and started only in the mid-1960s, in fact forming the first experimental studies on IFR in the field (e.g., Benton & Van Allen, 1968; De Renzi & Spinnler, 1966; Warrington & James, 1967).

Defining prosopagnosia as an impairment which exists only when isolated from visual object agnosia may be seen as extremely restrictive, confining its study to a relatively small number of cases. However, as illustrated in the present review, this restriction may be *essential* if we aim to study cases of prosopagnosia to provide critical information about the nature of IFR. Note that other authors have also included this criterion in their definition (Corrow, Dalrymple, & Barton, 2016). Yet, either they have not applied this criterion to their own selection of patients (e.g., Barton, 2008), or they seem to consider that patients with visual object agnosia are only those that are so severely impaired that they confuse their wives for hats – patients who only existed in Oliver Sacks' (admittedly very rich) imagination. In reality, patients such as LH (Levine *et al.*, 1980), SM and CR (Gauthier *et al.*, 1999), HJA (Riddoch & Humphreys, 1987), NS (Delvenne *et al.*, 2004), WF (Henke *et al.*, 1998), the two cases in Clarke *et al.* (1997), but also Herschel (Rezlescu *et al.*, 2012), as well as the patients reported by Barton (2008) are cases of *visual object agnosia*: these patients spontaneously complain of object recognition deficits and/or these deficits are easily found when they are required to recognize objects even at the basic level. They also show a typical profile of patients with visual agnosia, namely better recognition of real objects as compared to photographs, and photographs as compared to line drawings (see, e.g., Delvenne *et al.*, 2004), due to the availability of cues that can be used to guess the identity of the object (e.g., size for the real objects).

Hence, if we stick to the definition of prosopagnosia as a face-selective recognition disorder (Bodamer, 1947), these patients should not be defined as cases of prosopagnosia in the first place, but cases of visual (object) agnosia. In contrast, other cases such as PS, GG, LR, but also potentially the fourth patient of De Renzi (1986), patient MT (Henke *et al.*, 1998), and FB (Riddoch *et al.*, 2008) or the case of Wada and Yamamoto (2001) (providing that more extensive testing is performed and speed of recognition is normal in these latter four cases) do not have deficits at basic-level object recognition and could truly be defined as cases of prosopagnosia.

Domain-specific visual expertise

In this subsection, I would like to address another reason why the within-category account persists: its confusion with the notion of visual expertise, considered as generic expertise in fine-grained discrimination. Indeed, it has also been argued that the deficit in prosopagnosia may not be specific to faces but rather due to 'visual expertise' (Barton, Hanif, & Ashraf, 2009; Bukach *et al.*, 2012; Gauthier *et al.*, 1999). This is a major source of confusion in the field of human face recognition in my opinion. Obviously, the impairment in prosopagnosia is due to a defect in visual expertise: the expertise that typical adults have *with faces* in order to be able to recognize individual exemplars of that category accurately, rapidly and automatically. If typical adults reach this level of expertise *only* for faces, it may in fact be the very reason why this expert recognition process is only required for faces, explaining why sudden brain damage at adulthood can sometimes cause an impairment restricted to the category of faces. In other words, a visual expertise account of prosopagnosia is not an alternative to the domain-specificity account of prosopagnosia, it merely proposes *a reason* why a face-specific deficit can occur: because visual expertise is domain-specific, that is, restricted to the category of faces in typical human adults. This level of expertise in IFR shared by typical human adults could be due to biological constraints (e.g., Farah, Rabinowitz, Quinn, & Liu, 2000; Wilmer *et al.*, 2010; Morton & Johnson, 1991) or opportunities (e.g., the large amount of facial interindividual phenotypic variability in the human species, Sheehan & Nachman, 2014), as well as to our overwhelming experience with faces during development (e.g., Carey, 1992; Sugden, Mohamed-Ali, & Moulson, 2014) and social requirements to individuate numerous conspecifics in the human species.

A major problem with the term 'visual expertise' in the field of human face recognition is that it is usually associated with a *generic* expertise account: the view that there would be a generic 'expert' system in the brain, which could be recruited to individualize virtually any type of visual pattern at an expert level (e.g., dogs in Diamond & Carey, 1986; see Bilalic, 2017). According to a particular version of this view, this expert system would even be so flexible that it could become involved in adulthood for non-face object shapes, for example, after learning to individualize these shapes for only a few hours (Gauthier & Tarr, 1997; Wong, Palmeri, & Gauthier, 2009). As discussed extensively in previous reviews (McKone, Kanwisher, & Duchaine, 2007; Rossion, 2013), but also demonstrated with cases of reported prosopagnosia (Rezlescu, Barton, PItcher, & Duchaine, 2014), this latter view does not hold (see also Lochy *et al.*, 2018; Vuong *et al.*, 2017, for evidence that small learning effects attributed to visual expertise acquired in adulthood depend on stimulus facelikeness).

I am proposing that typical human adults have a specific visual expertise with the category of faces only. Now, whether this visual expertise level is restricted to faces in all individuals, or could be potentially approached or reached for other object categories if, say, one spends an enormous amount of time recognizing models of cars, or species of dogs, or birds, during development (i.e., 'natural expertise' (e.g., Diamond & Carey, 1986; Hagen, Vuong, Scott, Curran, & Tanaka, 2014; Tanaka & Curran, 2001) is also a different issue. It is in this context that Barton *et al.* (2009) have claimed to provide evidence in favour of a generic visual expertise account of prosopagnosia, by demonstrating that five reported cases of prosopagnosia had a low correlation between a measure of verbal semantic knowledge (finding the manufacturer from an index, e.g., '450SL?': 'Mercedes') with their ability to provide semantic information about cars (model, year, manufacturer) from their pictures, taken as a measure of visual recognition. That is, while the two measures were highly correlated in typical participants, these patients had high verbal semantic indexes and yet had low visual recognition scores (Figure 6). This is taken by Barton *et al.* (2009; Barton & Corrow, 2016) as evidence for a generic visual expertise account of prosopagnosia: according to these authors, in prosopagnosia, the recognition deficit would not concern faces *per se* but the objects for which there is a high level of visual expertise prior to brain damage. This is interesting from a methodological point of view; that is, it is important to take into account individuals' knowledge about objects or faces before testing their recognition of such stimuli. However, these authors' demonstration is not incompatible with the domain-specificity of prosopagnosia: most of us reach a high level of visual expertise, that is, the ability to automatically individualize exemplars accurately and rapidly, only with one category: faces.

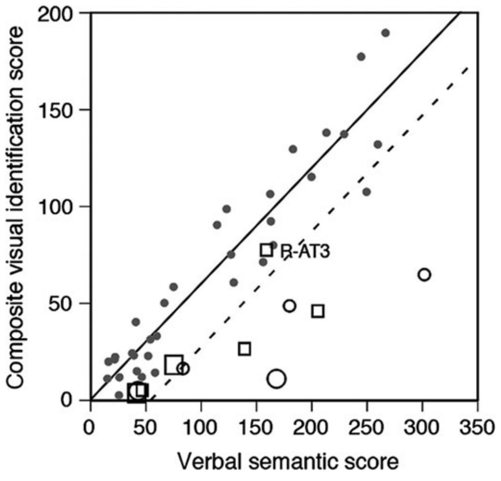

Figure 6. Original data from the study of Barton *et al.* (2009), completed by additional patients in Barton and Corrow (2016), showing the relationship between the score at recognizing pictures of cars (including model, manufacturer, and decade of make) in relation to the score at providing semantic information without the picture present. The data show that 5 of 11 patients should perform better at recognition of visual images of cars given their semantic knowledge of these cars predicts in the absence of any visual cue. This pattern of results is completely expected if these patients suffer from visual object agnosia, and cannot be taken as evidence for a generic visual expertise account of their impairment at individual face recognition.

In addition, Barton *et al.* (2009)'s empirical demonstration is not convincing for two reasons. First, once again, this argument is only valid if the patients tested do not suffer from visual object agnosia. Suppose instead that a patient suffering from visual object agnosia is a car enthusiast. She or he would show a high verbal semantic score but a relatively low visual recognition score, like five of the patients of Barton *et al.* (2009; Figure 6). This case would be taken as supporting the generic visual expertise account by these authors while, in reality, it would simply be due to the patient having visual object agnosia, which would appear relatively more severe for objects of interest. In contrast, if a patient without a basic-level object recognition deficit, such as PS, was a car enthusiast, it would then make sense to test this hypothesis with her.[11] As Barton and colleagues did not document basic-level object recognition for their patients, their observation cannot be taken as supporting a generic visual expertise account.

A second issue with these studies is that they rely on an inadequate measure of *visual* expertise. The ability to provide rich semantic information about cars from pictures does not necessarily mean that these patients have a visual expertise with cars. To make an analogy, there are people who know a lot about celebrities, but may still be less good at recognizing pictures of celebrity faces than people who are less interested in celebrities, or they may perform less well at recognizing personally familiar faces in real life. In other words, one does not establish visual expertise by asking for semantic knowledge from pictures. Hence, the level of *visual* expertise with faces should be evaluated by

[11] *As far as I know, the patient PS had not developed a specific interest in cars or any other non-face object category before her accident, but she collects swan figurines, having hundreds of them that she recognizes without difficulty.*

performance at learning/recognizing and matching pictures that no-one has seen before, that is, *unfamiliar* faces (Russell, Duchaine, & Nakayama, 2009), precisely because this measure is not contaminated by semantic knowledge. Similarly, for instance, car expertise should be established by performance at matching pictures of unknown cars (e.g., Rossion & Curran, 2010).[12]

Therefore, I argue that Barton *et al.* (2009; Barton & Corrow, 2016) should have used unfamiliar images of the domain of expertise in matching tasks to provide any support for their hypothesis. Nevertheless, it is important to note that these authors reported one patient with right anterior ventral occipito-temporal cortex damage ('R-AT3') with rich knowledge of cars who was able to generate this knowledge from car pictures very well, fitting perfectly with the normal population distribution (Figure 6). Presumably, if this patient had been tested in the same tasks with familiar people, he would have been way out of the distribution, at the bottom right corner. Hence, the contrasting pattern of performance for faces and cars in this patient alone – who could be studied in depth as a single case – is sufficient to reject the generic visual expertise account of prosopagnosia.

Conclusions: towards a conservative redefinition of prosopagnosia

In this review, I summarized the evidence and arguments against the long-standing view (Faust, 1947, 1955) that prosopagnosia is accounted for by a general impairment at recognizing individual exemplars of visually homogenous categories, a hypothesis championed by Damasio *et al.* (1982) and apparently still very much alive today (Barton & Corrow, 2016; Gauthier *et al.*, 1999; Geskin & Behrmann, 2018; Rezlescu *et al.*, 2012). Specifically, I suggested that restricting the definition of prosopagnosia to patients cleared of basic-level object recognition deficits is necessary, both to evaluate this within-category visual recognition account in particular, and for improving learning about the nature of IFR in general. Indeed, basic-level recognition deficits can cause semantic underspecification errors with non-face objects, which can be erroneously interpreted as evidence for a generic defect in fine-grained visual analysis processes in prosopagnosia.

When tested stringently for their ability to recognize visually similar objects, using parametric manipulations of physical similarity and taking into account both accuracy rates and RTs as variables of interest, there are cases of prosopagnosia, such as PS, that do not differ from typical observers in these measures (Figure 4). Critically, when they are tested in the exact same paradigms with faces, prosopagnosic patients show a different response profile than typical observers, being impaired and/or slowed down even when faces are highly discriminable. In fact, compared to these typical observers, the patients are also relatively less impaired at recognizing highly physically similar faces, showing that the nature of their difficulty at IFR does not lie in a generic fine-grained visual analysis

[12] *I am fully aware that this view clearly differs from the recent proposal that when it comes to the recognition of face exemplars, humans would only be experts with familiar but not unfamiliar faces (Young & Burton, 2018). This view is essentially based on the lower performance for matching pictures of unfamiliar faces as compared to pictures of familiar faces (celebrities or personally familiar people) (e.g., Bruce, Henderson, Newman, & Burton, 2001). However, here again, there is no evidence that this higher performance for familiar faces is based on visual representations rather than associations of semantic, affective or lexical (name) representations (see Rossion, 2018b). Moreover, when properly tested (i.e., considering speed of response in addition to accuracy rates), patients with prosopagnosia perform significantly worse for matching pictures of unfamiliar faces than typical human adults, who therefore appear to present a genuine expertise level with such stimuli. Other adequate points of reference to judge this level of expertise of typical human adults are on the one hand the significantly lower performance of children at unfamiliar face matching tasks (Carey, 1992; Mondloch, Geldart, Maurer, & Le Grand, 2003) and on the other hand the performance of other animal species such as monkeys, who struggle at matching the exact same images of unfamiliar faces of conspecifics even after extensive task training (Parr, Heintz, & Pradhan, 2008; see Rossion & Taubert, 2018).*

process (Figure 5). This latter observation questions the tendency to assess prosopagnosia and IFR abilities by artificially increasing physical similarity between unfamiliar individual faces, as is done in a number of tests. For example, in the prone-to-response bias same/different task of the Glasgow Face Matching Test (Burton, White, & McNeill, 2010), the distractors are systematically selected to physically resemble target faces using pairwise similarity measures. Or, in the Cambridge Face Perception Test (Duchaine, Yovel, & Nakayama, 2007), relying on morphed individual face stimuli, or else in the recently developed Caledonian Face Test (Logan, Wilkinson, Wilson, Gordon, & Loffler, 2016), which uses synthetic faces increasing in physical similarity.

I also argue that this selective deficit in IFR in prosopagnosia is due to a breakdown of a domain-specific, that is, face-specific, visual expertise, rather than a generic visual expertise that could be applied to other visual forms. Again, whether this domain-specific expertise originates from biological constraints/opportunities, or extensive visual experience with social requirements to individuate people by their face during development, or both, is a tangential issue. Importantly, this conclusion in favour of a face-specific neurological impairment does not imply that a typical human adult brain contains separate neural systems for non-face objects and faces, with their own specific visual recognition processes (i.e., a 'face module'). Although other authors have advocated this position (e.g., Kanwisher, 2000; see also Moscovitch, Winocur, & Behrmann, 1997; arguing for a part-based processing system for non-face objects and a holistic-based processing system for faces), it requires at least a clear functional 'double dissociation' between face and object recognition (see Young, 2011). However, the evidence for such a double dissociation remains weak so far and deviates from other (neural) sources of evidence. For instance, there are no brain regions associated with visual object agnosia, not topographic agnosia, that respond only to non-face objects in neuroimaging. Moreover, regions of the ventral occipito-temporal cortex in the right hemisphere respond to both faces and non-face objects, yet lesions of this region, or even focal electrical stimulation, can lead to face-selective impairments (e.g., Jonas *et al.*, 2012, 2015; Riddoch *et al.*, 2008). Rather, as I have argued elsewhere, IFR may share a number of fundamental processes with general visual object recognition, but also requires finer-grained *holistic* representations (Rossion, 2014). This extra process may depend on an increased pool of category-selective neural populations, in particular in the ventral occipito-temporal cortex of the right hemisphere, making IFR more susceptible to the effect of brain damage than object recognition.

Overall, the present demonstration points to the need to redefine prosopagnosia as a *selective* form of visual agnosia, that is, preserving basic-level object recognition, in line with the original definition of Bodamer (1947). As prosopagnosia implies a selective impairment for faces, there is no reason to use term 'pure prosopagnosia'. Although I risk stepping beyond the scope of the present paper and will need to provide more detailed argumentation in the future, I would like to conclude by mentioning what other criteria should be used for defining prosopagnosia, in an attempt to bring more clarity to the neuropsychological literature and human face recognition research in general.

First and foremost, as I emphasized in this review, *prosopagnosia* was originally defined as a subtype of visual agnosia, namely a neurological deficit of recognition limited to one modality (i.e., vision), which cannot be accounted for by low-level (visual) defects and intellectual impairments. Hence, neurological patients with severe intellectual limitations and/or low-level visual impairments directly causing their IFR impairment should not be defined as cases of prosopagnosia (including severe cases of 'apperceptive' visual agnosia following hypoxia, e.g., the famous patient DF; Whitwell, Milner, &

Goodale, 2014). Moreover, as discussed extensively by Guido Gainotti over recent years, patients with impairments at recognizing individuals from faces but also from other channels and modalities (e.g., voices, names, i.e., 'loss of memory for people or 'person agnosia', which usually extends beyond people, e.g., Ellis, Young, & Critchley, 1989) should be excluded (see Gainotti, 2010, 2013).

Next, it is important to emphasize that prosopagnosia always concerns the recognition of *both* the faces encoded before and after the accident, a fundamental criterion to distinguish prosopagnosia from learning acquisition deficits (i.e., anterograde amnesia), due to medial temporal lobe damage, for instance (e.g., Milner, 2005; see also, e.g., Hanley, Pearson, & Young, 1990; Tippett, Miller, & Farah, 2000). Without denying that there are individuals without neurological history who have major difficulties at IFR, this criterion would exclude *de facto* any neurodevelopmental disorder from being defined as prosopagnosia. This has been the case with 'developmental/congenital prosopagnosia', a misnomer in my opinion (instead, the term *prosopdysgnosia* may be more appropriate to refer to such cases, see Rossion, 2018a).

Along these lines, I also suggest that we restrict our definition of prosopagnosia to a *massive* and *sudden* impairment occurring in a mature face recognition system without neurological history. This would exclude mere IFR difficulties without complete impairment of the function, which are found in a large fraction of the population with right posterior brain damage (Benton & Van Allen, 1972; Valentine, Powell, Davidoff, Letson, & Greenwood, 2006; Young, Newcombe, de Haan, Small, & Hay, 1993), but also IFR impairments occurring during development, in a system that has not reached a high level of expertise at IFR (i.e., no 'childhood prosopagnosia', Young & Ellis, 1989). In the same vein, IFR difficulties in patients with a long history of temporal epilepsy (e.g., Drane *et al.*, 2013), neurodegenerative disorders such as Alzheimer's disease (e.g., Lavallée *et al.*, 2016), the right temporal pole variant of frontotemporal dementia (Rtv-FTLD (e.g., Busigny, Robaye, Dricot, & Rossion, 2009; Joubert *et al.*, 2003) or yet diffuse low-grade gliomas (e.g., Corrivetti, Herbet, Moritz-Gasser, & Duffau, 2016) should not be labelled (i.e., put in the same basket) as cases of prosopagnosia.

These criteria, in addition to the requirement of preserved basic-level object recognition and the need of stringent assessment of the IFR impairment with multiple tasks and variables, would severely restrict the number of cases considered to be prosopagnosia and increase the time needed for diagnosis. I argue that this is the price to pay if one aims at using prosopagnosia to isolate and understand the key visual recognition processes subtending human expertise at IFR.

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