



An objective electrophysiological marker of face individualisation impairment in acquired prosopagnosia with fast periodic visual stimulation

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ABSTRACT

One of the most striking pieces of evidence for a specialised face processing system in humans is acquired prosopagnosia, i.e. the inability to individualise faces following brain damage. However, a sensitive and objective non-behavioural marker for this deficit is difficult to provide with standard event-related potentials (ERPs), such as the well-known face-related N170 component reported and investigated in-depth by our late distinguished colleague Shlomo Bentin. Here we demonstrate that fast periodic visual stimulation (FPVS) in electrophysiology can quantify face individualisation impairment in acquired prosopagnosia. In Experiment 1 (Liu-Shuang et al., 2014), identical faces were presented at a rate of 5.88 Hz (i.e., ≈ 6 images/s, SOA = 170 ms, 1 fixation per image), with different faces appearing every 5th face (5.88 Hz/5 = 1.18 Hz). Responses of interest were identified at these predetermined frequencies (i.e., objectively) in the EEG frequency-domain data. A well-studied case of acquired prosopagnosia (PS) and a group of age- and gender-matched controls completed only 4 $\times$ 1-min stimulation sequences, with an orthogonal fixation cross task. Contrarily to controls, PS did not show face individualisation responses at 1.18 Hz, in line with her prosopagnosia. However, her response at 5.88 Hz, reflecting general visual processing, was within the normal range. In Experiment 2 (Rossion et al., 2015), we presented natural (i.e., unsegmented) images of objects at 5.88 Hz, with face images shown every 5th image (1.18 Hz). In accordance with her preserved ability to categorise a face as a face, and despite extensive brain lesions potentially affecting the overall EEG signal-to-noise ratio, PS showed 1.18 Hz face-selective responses within the normal range. Collectively, these findings show that fast periodic visual stimulation provides objective and sensitive electrophysiological markers of preserved and impaired face processing abilities in the neuropsychological population.

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1. Introduction

Shlomo Bentin was a man of multiple talents and wide interests. In his scientific career, he made numerous contributions to vastly different fields of research: face perception of course, but also visual word perception, semantic processing or memory among many others. The name of his laboratory at the Department of Psychology of Hebrew University (the *Cognitive Electrophysiology Lab*: <http://cel.huji.ac.il>) serves as a testimony of his varied research interests, centred on understanding high level brain functions in general, with electrophysiology (scalp electroencephalography, i.e. scalp EEG) as a primary tool of investigation. Bentin is best known for his outstanding contributions to the topic

of human cognition in the normal population. Yet he had an early interest for studying single cases and patient populations in cognitive neuropsychology (e.g., Bentin and Gordon, 1979) that persisted throughout his career.

Bentin's most renowned scientific contribution is undoubtedly his key paper published in the *Journal of Cognitive Neuroscience* two decades ago. In this paper, he and his co-authors reported the first systematic investigation, with no less than 5 experiments, of an early event-related potential (ERP) of particularly large amplitude elicited by face stimuli, an ERP component that they termed the N170 (Bentin et al., 1996). At the time, there were only a handful of published ERP studies about face perception, most of them using a few electrodes and focusing on what is largely believed to be the positive counterpart of the N170 located on central electrode sites, the vertex positive potential (VPP; Jeffreys, 1989; Bötzel and Grüsser, 1989; Joyce and Rossion, 2005 for a discussion of the VPP-N170 relationship and historical context; see also Bötzel et al., 1995; George et al., 1996 for early investigations of

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negative posterior ERPs evoked by faces). Today, there are hundreds, probably more than a thousand of studies that have been published on the N170 evoked by faces, as well as a substantial number of studies focusing on the N170 evoked by letterstrings, which was also described and characterised early on thanks to Shlomo Bentin's original work (Bentin et al., 1999a). Since the publication of this seminal study on the face-related N170, Bentin proposed that this component indexed the categorisation of a visual stimulus as a face by the human brain, this initial “face-specific” response “*reflecting an early mechanism operating at the early stages of face processing for the extraction of face-specific visual invariants and forming a sensory representation of a human face*” (Bentin et al., 1996, 1999b; Sagiv and Bentin, 2001; Carmel and Bentin, 2002). In this context of functional specificity of face perception, he and other authors enrolled in a research programme aiming at characterising the response properties of the N170 in order to understand the nature of this early face categorisation stage. For instance, Bentin and his colleagues carried out a series of elegant experiments demonstrating that the N170 was evoked depending on whether an exact same stimulus was consciously perceived as a face or not (Bentin et al., 2002; Bentin and Golland, 2002; see Navajas et al., 2013 for a recent contribution to this issue).

In parallel to this research on the normal brain, another of Bentin's scientific goals was to combine his research interests in electrophysiology and cognitive neuropsychology by using ERP components to study the functional origins of neuropsychological impairments. Hence, in this endeavour, Bentin was the first to use the N170 as a marker of face recognition impairment (Bentin et al., 1999b; see also Eimer and McCarthy, 1999). In Bentin et al.'s study (1999b), a person (YT) impaired at face recognition with no known history of brain injury (i.e., “congenital/developmental prosopagnosia”; Duchaine and Nakayama, 2006; Behrmann and Avidan, 2005) was presented with images of faces and houses and his N170 component was measured. Although YT did show a large N170, its face-specificity (i.e. difference of N170 amplitude between faces and houses) was significantly reduced relative to controls. This result led the authors to propose that YT's behavioural face recognition impairments could arise from a lack of selective processing of faces at the category-level, leading to weakened fine-grained processing of face identity (Bentin et al., 1999b; see also Bentin et al., 2007). Though recent studies have reported similar findings in a few cases (Németh et al., 2014), this pattern of results is not systematically found and reports of abnormal N170 components in congenital prosopagnosia are quite heterogeneous (e.g., Kress and Daum, 2003; Harris et al., 2005; Minnebusch et al., 2007; Towler et al., 2012; 2014; see also Feuerriegel et al., 2015).

In truth, relating a behavioural deficit in face recognition to the N170, or to another electrophysiological marker, is complicated for several reasons. First, the functional interpretation of the N170 is the subject of a longstanding debate. On the one hand, for some researchers, including Bentin himself, the N170 reflects face categorisation but not face individualisation, which would take place at a later stage (Bentin et al., 1996; 1999b; Amihai et al., 2011; see also e.g., Schweinberger et al., 2002). On the other hand, other authors argue for an early sensitivity to face identity as early as in the N170 time-window (e.g., Itier and Taylor, 2002; Heisz et al., 2006; Jacques and Rossion, 2006; Jacques et al., 2007; Caharel et al., 2009a). This topic constituted a source of scientific disagreement between Shlomo Bentin and the senior author of this paper for many years (see e.g., Amihai et al., 2011; Rossion and Jacques, 2011). Given the ambiguity regarding the functional specificity of this component, an abnormal N170 in a prosopagnosic patient can be related to either a deficit in face categorisation or to a deficit in face individualisation. However, it is face

individualisation, rather than categorisation, that is predominantly impaired in prosopagnosia. Hence, in cases of face recognition impairment following brain damage, i.e. “acquired” prosopagnosia (Bodamer, 1947), patients complain of important difficulties at recognising *specific people* by their face, regardless of whether faces belong to known or unknown individuals (e.g., Quaglino and Borelli, 1867; Hecan and Angelergues, 1962; De Renzi, 1986; McNeil and Warrington, 1993; Sergent and Signoret, 1992; Henke et al., 1998; Barton et al., 2002; Riddoch et al., 2008; Busigny et al., 2010a, 2010b; Rossion, 2014a for review). Unless the patients suffer from a general form of visual agnosia also affecting the category of faces (e.g., Farah et al., 1995; Boutsen and Humphreys, 2002; Delvenne et al., 2004; Gauthier et al., 1999; Xu and Biederman, 2014), they do not complain of problems at categorising a face as a face and this function appears to be preserved (e.g., Schiltz et al., 2006; Rossion et al., 2011; Bobes et al., 2003). This is also largely the case in congenital/developmental prosopagnosia: the impairment concerns the individualisation of faces rather than the categorisation of a face as a face (Behrmann and Avidan, 2005; Duchaine et al., 2007; see Garrido et al., 2008; Dalrymple and Duchaine, 2015 for evidence of impairment at difficult face categorisation tasks in some cases). As a consequence, depending on the theoretical framework, the N170 component may or may not be an appropriate marker to examine face recognition deficits in prosopagnosia.

A second difficulty in using neural markers to measure face recognition in prosopagnosia arises due to the limited sensitivity to face identity during the N170 time-window. More precisely, whether an effect of face identity is found on this component depends heavily on the stimulation parameters. Hence, the N170 is reduced in amplitude by the second presentation of a specific individual face stimulus only when this repetition is immediate, occurs with a short interstimulus interval, and particularly when the first face is presented for a long duration of several seconds (e.g., Jacques et al., 2007; Caharel et al., 2009a, 2009b; 2015). However, even when these specific parameters are used, the reduction of the N170 following face identity repetition remains a relatively small effect compared to the overall amplitude of the N170 (Jacques et al., 2007). Therefore, this effect requires many trials to reach statistical significance and is not found in every single subject. This is also true for the subsequent N250r, which is more negative following repeated exposures of familiar than unfamiliar faces (Schweinberger et al., 1995; Pfützte et al., 2002), or experimentally learned faces (Tanaka et al., 2006). The presence and the modulation of this later deflection are similarly difficult to objectively quantify in single participants. Given this low sensitivity, the standard ERP approach is rather inadequate for a fast and reliable diagnosis of face individualisation impairments in individual patients, whether they suffer from acquired or congenital prosopagnosia.

Third and finally, attempts to combine electrophysiology and cognitive neuropsychology are hindered by the presence of brain lesions. The effect of brain damage is particularly problematic for the study of patients with acquired prosopagnosia as these lesions affect current flows inside the brain and through the skull, reducing signal-to-noise ratio (SNR), and potentially deforming visual ERP components. As a result, the N170 can be modified in shape, polarity and scalp topography in such patients (e.g., Eimer and McCarthy, 1999; Alonso-Prieto et al., 2011; Dalrymple et al., 2011; Bobes et al., 2003). Moreover, earlier component such as the P1 can also be affected by brain damage in some patients (Eimer and McCarthy, 1999; Alonso-Prieto et al., 2011), affecting baseline measures of the N170. Altogether, these effects of brain damage may prevent the objective definition of electrophysiological responses such as the N170 (or other components), the quantification of its amplitude or its amplitude modulation by stimulus

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