



Research report

The processing of voice identity in developmental prosopagnosia



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ABSTRACT

Background: Developmental prosopagnosia is a disorder of face recognition that is believed to reflect impairments of visual mechanisms. However, voice recognition has rarely been evaluated in developmental prosopagnosia to clarify if it is modality-specific or part of a multi-modal person recognition syndrome.

Objective: Our goal was to examine whether voice discrimination and/or recognition are impaired in subjects with developmental prosopagnosia.

Design/methods: 73 healthy controls and 12 subjects with developmental prosopagnosia performed a match-to-sample test of voice discrimination and a test of short-term voice familiarity, as well as a questionnaire about face and voice identification in daily life.

Results: Eleven subjects with developmental prosopagnosia scored within the normal range for voice discrimination and voice recognition. One was impaired on discrimination and borderline for recognition, with equivalent scores for face and voice recognition, despite being unaware of voice processing problems.

Conclusions: Most subjects with developmental prosopagnosia are not impaired in short-term voice familiarity, providing evidence that developmental prosopagnosia is usually a modality-specific disorder of face recognition. However, there may be heterogeneity, with a minority having additional voice processing deficits. Objective tests of voice recognition should be integrated into the diagnostic evaluation of this disorder to distinguish it from a multi-modal person recognition syndrome.

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1. Introduction

Prosopagnosia is the inability to recognize faces. In line with cognitive models of face recognition as a hierarchy of operations (Bruce & Young, 1986), neuropsychological studies of acquired prosopagnosia suggest at least two functional variants, an apperceptive form in which subjects have difficulty perceiving facial structure, and an associative/amnesic form in which perception is relatively spared, but the ability to access memories of previously seen faces is disrupted (Barton, 2008; Davies-Thompson, Pancaroglu, & Barton, 2014). In acquired prosopagnosia, the apperceptive variant has been linked to right or bilateral occipitotemporal lesions, and the associative variant to right or bilateral anterior temporal lesions (Barton, 2008; Damasio, Tranel, & Damasio, 1990).

The fact that voice recognition also activates the right anterior temporal lobes (Belin & Zatorre, 2003) has prompted questions as to whether some cases of associative prosopagnosia may instead have a multi-modal person recognition disorder, since voice recognition is seldom tested (Gainotti, 2013). This is not only a question with theoretical implications, but also potential clinical relevance: as there is some evidence that simultaneous voice processing can enhance face learning, (Bulthoff & Newell, 2015), impaired voice processing could accentuate prosopagnosic deficits while intact voice processing might offer rehabilitative avenues. To address this, a recent study of acquired prosopagnosia found that voice recognition was unaffected after right anterior temporal lesions but impaired by bilateral anterior temporal lesions (Liu, Pancaroglu, Hills, Duchaine, & Barton, 2014). These data tentatively suggest that impaired face recognition can be divided into an apperceptive form linked to occipitotemporal lesions, an associative form linked to right anterior temporal lesions, and a multi-modal person recognition syndrome, with parallel associative deficits in face and voice processing, typically after bilateral anterior temporal lesions. These preliminary function-structure correlations require replication.

These observations are also relevant to the developmental form of prosopagnosia, whose functional and structural basis remains uncertain. Recent studies suggest the possibility of similar apperceptive, associative and other variants in developmental prosopagnosia (Dalrymple, Garrido, & Duchaine, 2014; Stollhoff, Jost, Elze, & Kennerknecht, 2011; Susilo & Duchaine, 2013). Less known is whether it is a modality-specific disorder or part of a multi-modal syndrome: currently there is only one report that studied voice recognition in one developmental prosopagnosic subject, which found some impairment (von Kriegstein, Kleinschmidt, & Giraud, 2006), and brief mention of inferior voice recognition in another who also had Asperger syndrome (Kracke, 1994). Addressing these functional questions in developmental prosopagnosia may help guide and refine studies of this condition.

The goal of this study was to apply the same two tests of voice discrimination and recognition used in the study of acquired prosopagnosia (Liu et al., 2014) to a cohort with developmental prosopagnosia. We asked a) whether this condition is a modality-specific disorder of face recognition or part of a

multi-modal person recognition syndrome, and b) whether the results were homogenous or heterogeneous in the cohort.

2. Material and methods

2.1. Subjects

We studied 12 subjects with developmental prosopagnosia (9 females, age range 27–67 years, mean 41.82 years, SD 12.91 years). These were local residents and native English speakers recruited from www.faceblind.org. Diagnostic criteria included an in-person clinical interview that revealed a subjective report of life-long difficulty in face recognition, and objective confirmation of impaired face recognition that included a score at least two standard deviations below the control mean on the Cambridge Face Memory Task (Duchaine & Nakayama, 2006) and a discordance between preserved word memory and impaired face memory on the Warrington Recognition Memory Test for Faces and Words (Warrington, 1984) that was in the bottom 5th percentile (Table 1). One subject had a unilateral retinal detachment, since corrected and now with normal acuity and fields; no other subject reported perceptual problems. Apart from mild concussions years prior in four subjects, none had other neurologic problems. None of these subjects reported any significant changes in face perception associated with their mild concussions or other medical events. All subjects had best corrected visual acuity of better than 20/60, normal visual fields on Goldmann perimetry, and normal color vision on the Farnsworth-Munsell hue discrimination test. To exclude autism spectrum disorders, all subjects had a score less than 32 on the Autism Questionnaire (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001).

Although it is not part of current diagnostic criteria (Behrmann & Avidan, 2005; Bate et al., 2014; Susilo & Duchaine, 2013), eight subjects had magnetic resonance brain imaging with T1-weighted and FLAIR sequences to exclude structural lesions that would have indicated early acquired rather than developmental prosopagnosia (Barton, Cherkasova, Press, Intriligator, & O'Connor, 2003); in one subject MRI was contraindicated and in the remaining three it was declined because of time limitations (Table 1). To evaluate face discrimination, subjects also completed the Cambridge Face Perception Test (CFPT; Duchaine, Yovel, & Nakayama, 2007).

Seventy-three control subjects completed the voice discrimination test (50 females, age range 19–70 years, mean 33.6 years, SD 15.5 years), 54 of whom also completed the voice recognition test (41 females, age range 19–70 years, mean age 37.2, SD 16.4 years). All subjects were born in North America, lived in North America for at least five years, and spoke English as their first language. All control subjects reported normal or corrected-to-normal vision, normal hearing, and no history of brain damage. All control subjects were remunerated ten dollars per hour for their participation.

All subjects gave informed consent to a protocol approved by the University of British Columbia and Vancouver General Hospital Ethics Review Boards.

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