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Localization and patterns of Cerebral dyschromatopsia: A study of subjects with prospagnosia

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ABSTRACT

Objective: Cerebral dyschromatopsia is sometimes associated with acquired prosopagnosia. Given the variability in structural lesions that cause acquired prosopagnosia, this study aimed to investigate the structural correlates of prosopagnosia-associated dyschromatopsia, and to determine if such colour processing deficits could also accompany developmental prosopagnosia. In addition, we studied whether cerebral dyschromatopsia is typified by a consistent pattern of hue impairments.
Methods: We investigated hue discrimination in a cohort of 12 subjects with acquired prosopagnosia and 9 with developmental prosopagnosia, along with 42 matched controls, using the Farnsworth-Munsell 100-hue test.
Results: We found impaired hue discrimination in six subjects with acquired prosopagnosia, five with bilateral and one with a unilateral occipitotemporal lesion. Structural MRI analysis showed maximum overlap of lesions in the right and left lingual and fusiform gyri. Fourier analysis of their error scores showed tritanopic-like deficits and blue-green impairments, similar to tendencies displayed by the healthy controls. Three subjects also showed a novel fourth Fourier component, indicating additional peak deficits in purple and green-yellow regions. No subject with developmental prosopagnosia had impaired hue discrimination.
Conclusions: In our subjects with prosopagnosia, dyschromatopsia occurred in those with acquired lesions of the fusiform gyri, usually bilateral but sometimes unilateral. The dyschromatopsic deficit shows mainly an accentuation of normal tritatanopic-like tendencies. These are sometimes accompanied by additional deficits, although these could represent artifacts of the testing procedure.

1. Introduction

Acquired prosopagnosia is the rare disorder of impaired face recognition. It can occur with a variety of lesions, right or bilateral, in occipitotemporal or anterior temporal cortex (Davies-Thompson et al., 2014). Prosopagnosic subjects can also have cerebral dyschromatopsia (Meadows, 1974), an impairment of colour perception whose hallmark is a deficit in discriminating hues. This has often been linked to bilateral damage to the lingual and posterior fusiform gyri, as demonstrated in a meta-analysis (Bouvier and Engel, 2006) and consistent with early fMRI studies of colour perception (Sakai, et al., 1995). However, subsequent neuroimaging studies have shown, first, a posterior V4 locus and an anterior V8 locus near the collateral sulcus (Bartels and Zeki, 2000), and more recently, multiple patches with colour-related activity, including more anterior cortex and often in close proximity to face-sensitive regions (Beauchamp et al., 1999; Lafer-Sousa and Conway, 2013). The lesion data has also suggested that colour perception may involve a stream of cortical processing, rather than a single region (Bouvier and Engel, 2006). This raises the possibility that a variety of prosopagnosic lesions may be associated with impaired colour perception. Furthermore, although the association between dyschromatopsia and acquired prosopagnosia has been well documented (Zeki, 1990), there has been no systematic study of colour perception in developmental prosopagnosia a condition

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whose anatomic correlate remains a subject of debate (Avidan et al., 2014; Song et al., 2015).

Unlike congenital dyschromatopsia due to inherited deficits in cone pigments, the colour processing deficits of cerebral dyschromatopsia are often said to be non-specific, affecting all hues. Nevertheless, some describe more pronounced deficits in certain regions of colour space (Adachi-Usami et al., 1995; Fine and Parker, 1996; Rizzo et al., 1993; Rondot et al., 1967). Almost all of these cases report only a subjective inspection of data; however, when deficits are widespread patterns may be detected more effectively by quantitative mathematical techniques (Victor, 1988). Surprisingly, this has been done in only one case of cerebral dyschromatopsia (Victor et al., 1989).

In this study we applied a widely used, sensitive test of hue discrimination, the Farnsworth-Munsell 100-hue test (Farnsworth, 1943), to a large cohort of subjects with acquired or developmental prosopagnosia. Our first goal was to determine whether any type of lesion causing prosopagnosia could be associated with dyschromatopsia or if colour impairments were limited to those with bilateral fusiform lesions. Our second goal was to determine if colour processing deficits occurred in at least some subjects with developmental prosopagnosia. Our third goal was to use Fourier analysis to determine if cerebral dyschromatopsia has relative selectivity for a particular region or axis of hue space.

2. Materials and methods

2.1. Subjects

The Institutional Review Boards of the University of British Columbia and Vancouver Hospital approved the protocol and all subjects gave informed consent in accordance with the principles of the Declaration of Helsinki.

2.1.1. Acquired Prosopagnosia

These were 12 subjects (6 female, mean age 42 years, range 22-60). All had a neuro-ophthalmologic history and examination, with best-corrected acuity of 20/60 or better, and Goldmann perimetry. All complained of impaired face recognition in daily life and were impaired on both a famous faces test and at least one of either the Cambridge Face Memory test or the faces component of the Warrington Recognition Memory test, while performing normally on the word component of the latter (Table 1). A short history of the six who proved to have impaired hue discrimination follows. All six also had noted altered colour perception in their daily life, while none of the other subjects with either acquired or developmental prosopagnosia voiced any complaints about colour perception. Further details on the neuroimaging and neuropsychological testing of this cohort have been published elsewhere (Hills et al., 2015; Liu et al., 2014).

L-IOT2 has right fusiform atrophy and had a left fusiform resection for epilepsy at age 39. Afterwards he had aphasia that resolved into a residual word-finding problem. He thought that his car had been repainted a lighter blue when his wife picked him up from hospital. He confuses the jerseys of basketball teams with a similar hue, and has trouble judging skin tone.

B-ATOT2 had herpes encephalitis at age 10, following which she had difficulty reading and spelling, which improved. She has topographagnosic symptoms. Her vision was initially achromatic but partially recovered, although she still cannot identify colours and has labels on her clothes to indicate their colour.

B-IOT2 had a traumatic subdural hematoma that was evacuated at age 27. He has topographagnosic symptoms. He became aware of altered colour vision early on, with hues looking ‘flat’ or ‘turned down’ in vividness.

B-ATOT1 had herpes encephalitis at age 14, leaving a small homonymous paracentral scotoma in the left upper field. She notes difficulty distinguishing blue from green.

B-IOT1 had bilateral sequential occipital infarcts from a vertebral dissection at age 40. He cannot recognize buildings. The world was reduced to black and white initially. After 2 months he began seeing hues again, though he still notes trouble distinguishing similar colours, such as blue versus green.

R-IOT4 had a right occipital infarct 6 months prior to study. He has topographagnosic symptoms. He noted altered colour perception but only for the first few months: though he knew what each colour was, they lacked ‘emotional impact’.

2.1.2. Developmental prosopagnosia

These were 9 subjects (7 female, mean age 43.3 years, range 31-61) recruited from www.faceblind.org. Diagnostic criteria were reported life-long difficulty in face recognition and objective confirmation of impaired face recognition (Dalrymple and Palermo, 2016), including a score at least 2 standard deviations below the control mean on the Cambridge Face Memory Test and a discordance between preserved word and impaired face memory on the Warrington Recognition Memory Test that was in the bottom 5th percentile. All had best corrected visual acuity of better than 20/60 and normal Goldmann perimetric results. To exclude autism spectrum disorders, all subjects scored less than 32 on the Autism Questionnaire (Baron-Cohen et al., 2001). All but one subject (DP033) in whom it was contraindicated had MRI with T1-weighted and FLAIR sequences to exclude structural lesions.

2.1.3. Control subjects

For each prosopagnosic subject we recruited two healthy subjects of the same gender and within 5 years of age, creating a cohort of 42 subjects (26 female, mean age 41.3 years, range 17-65). All performed the Cambridge Face Memory test to exclude the possibility of unsuspected developmental prosopagnosia. Exclusion criteria were a history of neurologic or psychiatric conditions, including autism spectrum disorders, or of eye disease, including congenital dyschromatopsia, optic neuritis, macular degeneration, and cataracts. We excluded one subject with subjective claims of poor face recognition and low scores on the Cambridge Face Memory test, two subjects with episodes of unconsciousness lasting more than 10 min, and one with memory loss after electrocution. Two subjects with congenital dyschromatopsia were tested for illustrative purposes, a 46 year-old man with tritanopia and a 46 year-old man with protanopia.

2.2. Imaging

We obtained high-resolution (1 mm^3) T1-weighted three-dimensional structural images on a Philips Achieva 3.0 T MR scanner, with an 8-channel head coil. We created a lesion mask for the 12 subjects with acquired prosopagnosia, using tools from the Oxford Centre for Functional MRI of the Brain's Software Library (FSL) (Woolrich, et al., 2009). Images were preprocessed with lesion filling when necessary (Battaglini et al., 2012), and linear-registration (Jenkinson et al., 2002; Jenkinson and Smith, 2001) or non-linear registration to the MNI-152 stereotaxic space. Within the standard space, lesion masks of subjects with impaired hue discrimination were summed to give an overlap image.[1]

[1] One caveat with this type of analysis is that it may provide a conservative estimate of the extent of brain damage, particularly with certain pathologies such as closed head trauma and infection.