Color Vision Deficits

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Abstract

Purpose of Review Color provides important information about the identity of the objects we encounter. After early processing stages in the retinal cones, thalamus and occipital cortex, retinal signals reach the ventral temporal cortex for high-level color and object processing, which links color perception with top-down expectations and knowledge. In the language-dominant hemisphere, some of these regions communicate with the language systems; by assigning verbal labels to percepts, these circuits speed up stimulus categorization, and permit fast and accurate inter-individual communication. This paper provides a review of color processing deficits, from dysfunction of wavelength discrimination in the retinal photoreceptors to deficits of high-level processing in the ventral temporal cortex. *Recent Findings* Neuroimaging evidence defined the existence and localization of color-preferring domains in the ventral occipito-temporal cortex. Evidence from the performance of a brain-damaged patient with color anomia but preserved color categorization demonstrated the independence of color categorization from color naming in the adult brain. *Summary* Evidence from patients with brain damage suggests that high-level color processing may be divided into at least three functional domains: perceptual color experience, color naming, and color knowledge.

Keywords

Retinal cones, Ventral temporal cortex, Cerebral achromatopsia, Color anomia

Introduction

Color vision allows us to distinguish surfaces or sources of light, based on the wavelength of light received by the eye [1]. Color processing is likely to confer an evolutionary advantage by supporting object identification and social exchanges [2]. In particular, colors help increase object salience by separating them from background, and contribute to capture attention for object detection and discrimination [3]. For example, the yellow or green color of a banana tells us about its degree of ripeness and therefore palatability. In addition, colors serve non-verbal communication: the color of the face can signal a person's state of health, as well as certain emotions (e.g., reddening of the cheeks indicates embarrassment or irritation).

Retinal color processing and its anomalies

In humans, the light reflected by objects excites in different ways three types of retinal photoreceptors, called S cones (sensitive to short wavelengths, around 430 nm), M cones (sensitive to medium wavelengths, around 530 nm) and L cones (sensitive to long wavelengths, around 560 nm) (Fig. 1). There are 8 million cones, mostly concentrated in the fovea, the central portion of the retina, with a diameter of 0.4 mm. The rest of the retina mostly contains another type of receptor, the rods. The rods are much more numerous (120 million) than the cones, and respond to very low intensities of light (night vision), but are blind to colors.

The cones communicate with the bipolar cells in the retina, which in turn send a signal to the ganglion cells (Fig. 2). The axons of the ganglion cells form the optic nerve, which carries visual information to the lateral geniculate nucleus of the thalamus. Ganglion cells respond to both increased activity of one type of cone and decreased activity of another type of cone. This activity, together with that of the lateral geniculate nucleus, translates the activity of the

cones into three information channels: the luminance channel which sums the signals of the L and M cones (L + M), the red-green channel which renders the difference between the L and M cones (L - M) and the blue-green channel which corresponds to the (L + M) signal subtracted from the signal of the S cones: (S - (L + M)). This happens because the spectral tuning functions of the S-, M-, and L-sensitive cone photopigments in the retina are largely overlapping (see Fig. 1). Therefore, color signals are computed not as absolute values, but as differences between the responses of the different cone types (opponent process: L vs. M and S vs. L + M) [1].

Some congenital anomalies of color vision (dyschromatopsia) result from malfunction of the cones. These anomalies are often referred to as "color blindness", after John Dalton, an English chemist described them from his own case in the late 18th century. The genes which code for the proteins (pigments) specific to the L and M cones are located on the X chromosome; the gene for pigment S is on chromosome 7. The more frequent forms of abnormalities lead to a defect in discrimination between red and green, caused by a mutation of the genes for pigments L and M. These abnormalities occur in 12 men out of 100 (8%), but only in 1 woman out of 200 (0.5%), because a single healthy X chromosome is sufficient to provide the genetic information for normal cone function.

High-level color processing in the ventral temporal lobe

Visual input from the retina reaches the primary visual cortex in the occipital lobe (V1) via the lateral geniculate nucleus of the thalamus. Traditionally, it is considered that two main visual cortical pathways move rostrally from V1: a ventral (occipito-temporal) pathway, and a dorsal (occipito-parietal) pathway [4]. The ventral pathway reaches the antero-inferior portions of the temporal lobe, especially through the inferior longitudinal fascicle [5], and subserves the

perceptual identification of visual objects (so-called "what" stream). The dorsal pathway is instead important for localizing visual objects in space ("where" stream), and to program the hand movements necessary to reach and grasp them ("how" stream) [6].

The ventral cortical visual stream contains several regions that preferentially process visual information related to faces, object shape, object color, written material, body parts, etc. [7, 8]. These domain-preferring areas represent nodes in large-scale brain systems [8, 9], where each area reaches a widespread network of regions that share processing of information in the same domain [10]. Thus, the appropriate level of analysis in anatomo-clinical correlations is not focal damage to 'critical' cortical sites, but lesion-induced dysfunctions of large-scale cortical circuits [11]. Such network dysfunctions can result from direct cortical lesion to the nodes, or from white matter disconnections (e.g., inducing deafferentation from occipital visual areas).

High-level color processing

Perception and categorization of colors contribute in important ways to object identification [2]. High-level color-preferring areas in the ventral occipito-temporal cortex [12**] allow us to perceive colors in relative independence of variations of luminance (color constancy). Thanks to these mechanisms, we see the "same" red poppies at sunrise and at noon, despite major differences in the reflected wavelengths. These regions in the ventral cortical visual stream transform the cone-opponent signals, which only depend on the wavelengths hitting the retina, into a stable perceptual color space [13]. Retinotopic mapping identified two extrastriate clusters showing a preference for color: a ventral area adjacent to V3, dubbed hV4 (human homolog of V4) [14], and VO, anterior to hV4 and divided into two subsections, VO1 and VO2 [15]. Region hV4 is apparently color-selective, while VO seems to show both color and object selectivity. Regions lateral to VO1 exhibit some degree of lateralization in

color responsiveness, favoring the right hemisphere [15]. Task fMRI demonstrated three distinct color-biased regions, labeled as posterior, central and anterior color regions [12**] (Fig. 3).

The posterior and central color regions correspond to hV4 and VO1, respectively, and respond selectively to color, with little or no sensitivity to other visual domains such as faces or objects. The anterior color region responds instead to both colors and objects. These regions appear to be organized in a caudo-rostral hierarchy [16*]. In this hierarchy, the posterior color-preferring regions encode single hues and compute the perceptual color space. The central color-preferring regions group continuously varying hues into the discrete, behaviorally relevant ensembles of color categories. In the left hemisphere, the posterior [17] and central color regions may be important for color naming. Finally, the anterior color regions subserve memory of diagnostic (canonical) colors for given objects, such as banana/yellow (object-color knowledge).

A primary function of color perception is to facilitate object recognition. While shapes are relatively invariant in time and space, colors may provide information about the behavioral relevance of an object at a given moment of time; e.g., the ripeness of the fruit or the emotional state of the face [16*, 18]. Thus, shapes tell us what the objects are, and colors add details on whether and how we may want to act upon them [13]. To this end, information about color and shape is likely to be integrated in the anterior color region [12**], located in the rostral portions of the ventral cortical visual stream (see Fig. 3).

This hierarchical model of high-level color processing seems consistent with the occurrence of different patterns of impairment after brain damage. These patterns of deficits suggest the existence of at least three functionally segregated abilities: impaired perceptual color experience in cerebral achromatopsia, impaired color naming in color anomia, and

disconnection between perceived colors and color-related knowledge in color agnosia [13]. High-level color processing deficits rarely occur in isolation from other perceptual deficits, such as object agnosia or pure alexia. Causes include strokes in the territory of the posterior cerebral artery, craniocerebral trauma, or neurodegenerative conditions. In some cases, unilateral lesions can induce perceptual deficits in the opposite visual hemifield, such as unilateral hemiachromatopsia [19].

Cerebral achromatopsia

Bilateral occipito-temporal lesions can provoke c*erebral achromatopsia*, a rare condition characterized by impaired perceptual color experience [20, 21]. Patients typically report seeing the world in shades of grey, white or brown (sepia). For example, Madame D, an amateur painter who became achromatopsic after bilateral occipito-temporal strokes (Fig. 4), spontaneously complained of seeing the world in shades of grey [22]. Other patients are unaware of their deficit (anosognosia), and simply refrain from using color terms to describe their visual experience [23]. These patients may report that the world looks dull and drab, that their food is unappealing, that people around them look pallid and unwell. Anosognosia can also occur in hemi-achromatopsia, the loss of color vision in half of the visual fields. Madame R, who had developed right-sided hemi-achromatopsia after a lesion in the posterior parts of the left lingual and fusiform gyri (see Fig. 4), was unaware of her color processing deficit, and had sought medical help for concomitant difficulties in reading [24] (English translation in Ref. [21]). When tested with time-limited, lateralized color processing tasks, several patients with ventral lesions, who often had no complaints or clinical signs of achromatopsia, demonstrated contralesional deficits [25].

Patients with full-field achromatopsia are typically unable to rank a series of colored tablets in different shades (Farnsworth test), or to discriminate the stimuli of the Ishihara

plates, which are identifiable only by color. They are also unable to match equal hues (for example in the Holmgren skein test, consisting of skeins of wool of various colors). However, a meta-analysis [20] reported that up to 29% of achromatopsic patients could read normally the Ishihara plates; most patients performed better than chance on the Farnsworth Munsell 100 Hue test (with some even performing in the normal range); furthermore, 49% of the patients were able to name visually presented colors (although some of them were only clinically tested with colored objects, and might have used object shapes to retrieve the relevant color name from memory). The dissociation between subjective reports of color vision loss with better than chance color discrimination may suggest a deficit in conscious color experience, with relatively unimpaired unconscious color processing [13]. Alternatively, in analogy with apperceptive agnosia [8], patients might have become unable to establish a global representation of surface colors, with preserved discrimination of local color contrasts [23]. Mental visualization of colors can be spared in cerebral achromatopsia [26]. For example, some of these patients can correctly state from memory which red is darker, between the red of cherries and that of strawberries [22]. This dissociation may be explained by considering that brain damage may deafferent color-preferring regions from bottom-up input coming from low-level perceptual areas, but leave intact top-down activity in more anterior temporal regions, which are likely to sustain visual mental imagery [27-30].

Lesions that lead to achromatopsia tend to overlap with the location of the posterior color center detected in fMRI [12**, 20]. As mentioned before, unilateral lesions can lead to achromatopsia restricted to the contralateral visual hemifield [19, 21, 24]. In one patient, full-field achromatopsia occurred after a unilateral lesion in the right hemisphere [31]. However, this patient only received a CT scan, thus concomitant left hemisphere impairment cannot be completely excluded (see also Ref. [32], for discussion of a patient with object agnosia and

unilateral anatomical damage to the right hemisphere, but bilateral reduction of visual responses in functional neuroimaging).

Color anomia

Lesions affecting the left lingual, fusiform and parahippocampal gyri in the left, language-dominant hemisphere [33] can selectively impair the patient's ability to name visually presented colors, or to designate the color named by the examiner among various colored cards presented to the patient. This disorder is often associated with right homonymous hemianopia. Color perception and object-color knowledge are often, but not always, spared [13]. For example, the patient of the seminal 1890 study by Heinrich Lissauer [34] (English translation in Ref. [35]), with associative agnosia and alexia, also had problems in naming colors. He was also impaired in associating colors to color-diagnostic objects, labeling for example a pale pink or even a deep blue a "real blood color". When asked to indicate the color of a canary he hesitated between green, grey, and yellow. Color perception was apparently normal: when tested with Holmgren skeins, he would immediately match identical hues, and was able to differentiate between subtle hues of grey, green, and yellow.

Color naming deficits can occur in two main subtypes [36]: (1) *color-name anomia*, whereby patients cannot name visually presented colors, retrieve color names from memory, or verbally associate objects with their typical color (e.g. banana-yellow); and (2) *color visuo-verbal disconnection* or optic aphasia for colors, whereby patients remain able to recall color names and to verbally associate colors with objects, but they cannot name visually presented colors, or point to colors on verbal cues. In this case, language cannot access the visual percept. All known cases of color anomia also had right homonymous hemianopia and alexia without agraphia (review in Ref. [13]). Naming deficits for non-color items such as objects or faces can co-occur, albeit color naming remains (by definition) disproportionally more

affected. Color anomic patient RDS [37*] (Fig. 5) was able to decide whether an object was appropriately colored or not, but could not choose the color patch appropriate to a greyscale object. Thus, he could access his color knowledge when looking at naturalistically colored objects, but was not able to do so with abstract color patches. This pattern of performance suggests that the ability to abstract colors from visual objects may require the integration of color perception with language processes, which was impossible in this patient. However, patient RDS could categorize colors despite his inability to name them [38**]. Rather than resulting from the interaction between color perception and language (as maintained by many authors [39]), color categorization may thus constitute a separate module of color processing, which can perhaps also exploit the contribution of right-hemisphere-based processes [38**]. This conclusion does not exclude the possibility that color terms do contribute to color categorization; verbal labels can speed up and disambiguate the categorization process, and facilitate its learning and communication.

Color agnosia

Color agnosia is an umbrella term which designates the selective loss of associations between objects and their typical (canonical) colors. Color agnosia would thus represent the equivalent deficit for colors of associative object agnosia. This disorder impairs the knowledge about color as a semantic property of the object (just like shape, texture, functional context, etc.). Recognition and naming of the color may be intact (indicate a blue card, say what color a crayon is), but the patient does not know how to name the canonical color of objects from memory ("What color is a banana?" "It is blue"), or how to choose the appropriate pencil to color a black and white drawing (use a brown pencil to color an apple). Color agnosia can be understood as a selective deficit of object-color knowledge, which in most patients affects both visual color knowledge (the ability to visually differentiate between typical and atypical

colors of objects) and verbal color knowledge (the ability to associate color names with object names) [13, 40, 41]. However, dissociations between verbal and visual color knowledge may also occur in some patients [42]. Individual differences in the hemispheric organization of color-related knowledge might contribute to the rarity of color agnosia. Similar to knowledge of object form [43], color-related knowledge requires left hemisphere semantics; however, in some individuals the effects of left hemisphere strokes can be partially compensated by right hemisphere circuits. in these patients, unilateral strokes in the left hemisphere would not produce full-fledged color agnosia, but optic aphasia for colors, characterized by color anomia with some preserved access to color-related knowledge [37*]. In addition, some cases of color agnosia might remain undetected, because this condition requires specific tests to be revealed, whereas deficits of color perception or color naming are often clinically evident.

Lesions causing color agnosia usually affect the left occipito-temporal cortex. Case reports mention damage to the left anterior temporal lobe (including the rostral portions of the lingual gyrus), to the isthmus and to the parahippocampal areas [40, 41, 44]. Left parietal damage is also occasionally reported [41, 44].

To conclude, the patterns of performance and lesion location in patients with deficits of high-level color processing are consistent with the hypothesis of a caudo-rostral hierarchy along the ventral cortical visual stream, from more perceptual to more cognitive stages of color processing [8, 13, 16*]. Lesions affecting or deafferenting the posterior color-preferring regions can impair perceptual color experience, up to the dramatic condition of complete achromatopsia. Lesions damaging or disconnecting the anterior color-biased regions in the left hemisphere may determine impaired object color knowledge. Left-hemisphere lesions to the mesial portions of the ventral cortical visual stream may result in selective naming deficits for visually presented colors. The observed patterns of lesion lateralization suggest that the

right hemisphere may be preferentially (although not exclusively) engaged in perceptual color experience, while left-hemisphere circuits are important for object color knowledge and crucial for color naming.

General conclusions

A key role of high-level visual regions in the ventral temporal cortex is to integrate sensory and semantic information, in order to guide object-related behavior in a fast and efficient way. Specialized circuits of the occipito-temporal visual pathway are thus dedicated to the complex tasks of processing object shape and color for object identification. Shape- and color-preferring processing stages in the ventral visual stream appear to follow a caudo-rostral gradient, from more perceptual to more cognitive levels of processing, including the integration of shape and color information in the anterior color region. In the language-dominant hemisphere, connections with the language circuits integrate perceptual processing with linguistic abilities, in order to speed up stimulus categorization and enable effective communication between individuals. These high-level visual regions also receive top-down signals to build visual mental images, thus granting us some freedom from the external world in order to simulate possible or future perceptual settings.

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objects may be processed in a holistical way, perhaps by right hemisphere circuits in this patient.

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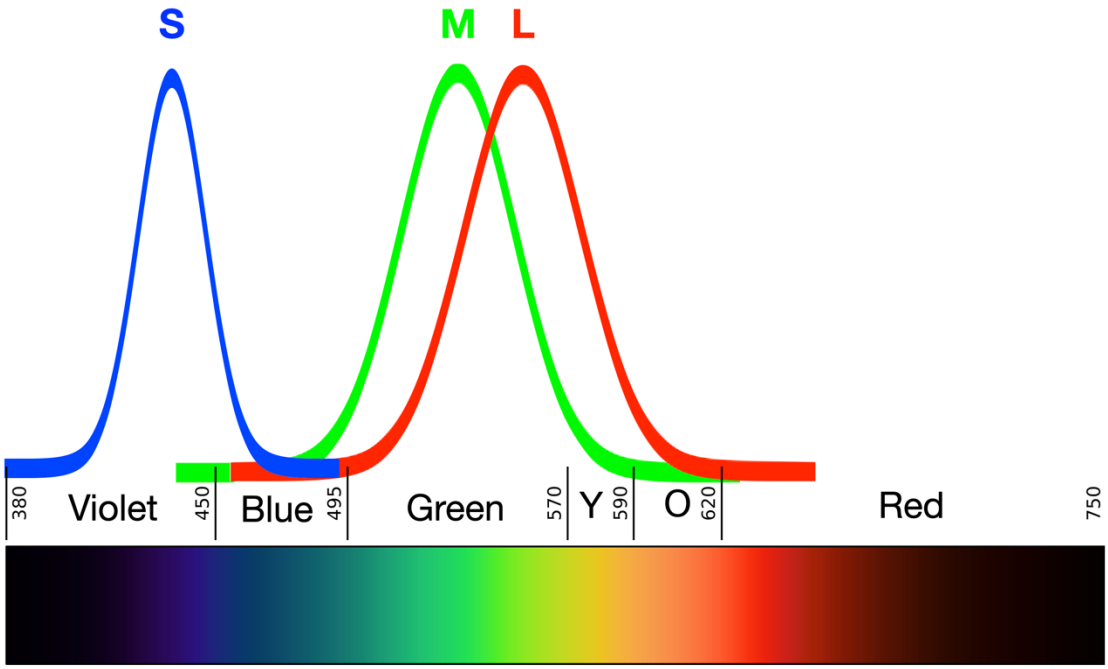

Fig. 1. Top, schematic representation of absorption peaks of the three types of retinal cones. Bottom, the visible spectrum. Numbers indicate wavelengths in nanometers. Y, yellow; O, orange (image modified from wikimedia commons, CC BY 4.0).

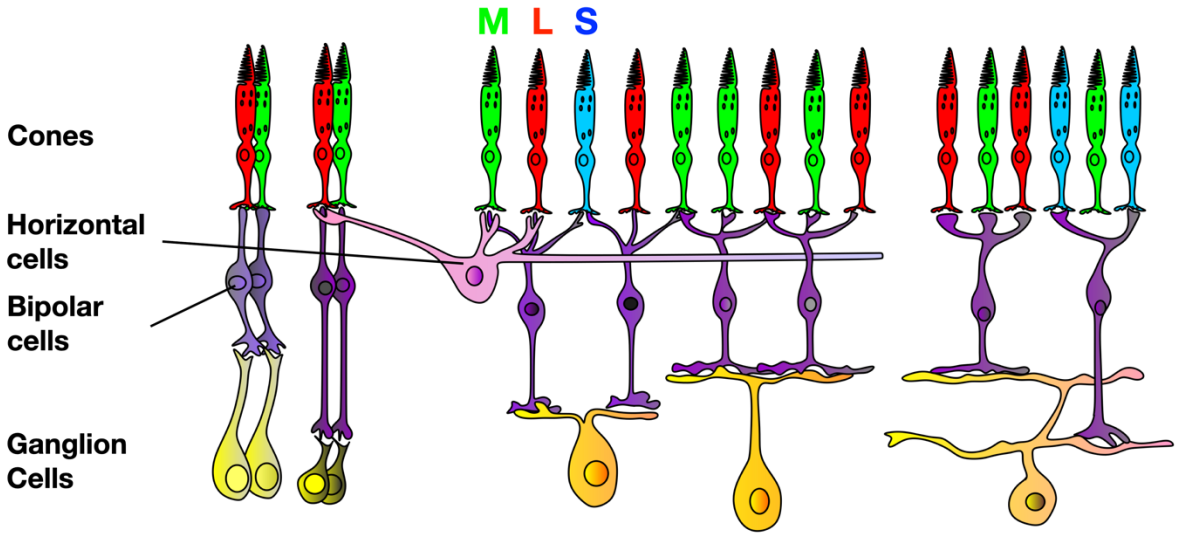

Fig. 2. The main cell types of the retinal fovea, with the different cone subtypes (image modified from https://commons.wikimedia.org/wiki/File:Ganglion_cell.svg, CC BY-SA 3.0).

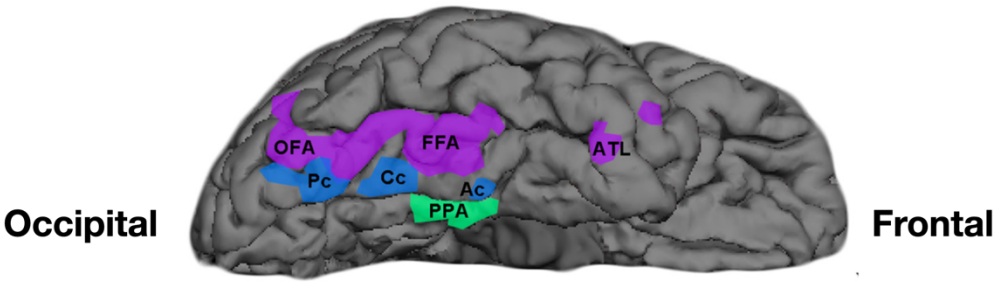

Fig. 3. Left hemisphere of the human brain seen from below. Functional MRI showed that the color-preferring regions (in blue: Posterior, Central and Anterior color-preferring regions) are placed between those important for the treatment of places (in green) and those which are important for the perception of faces (in purple). Figure reproduced from Ref. [12**] (CC BY 4.0).

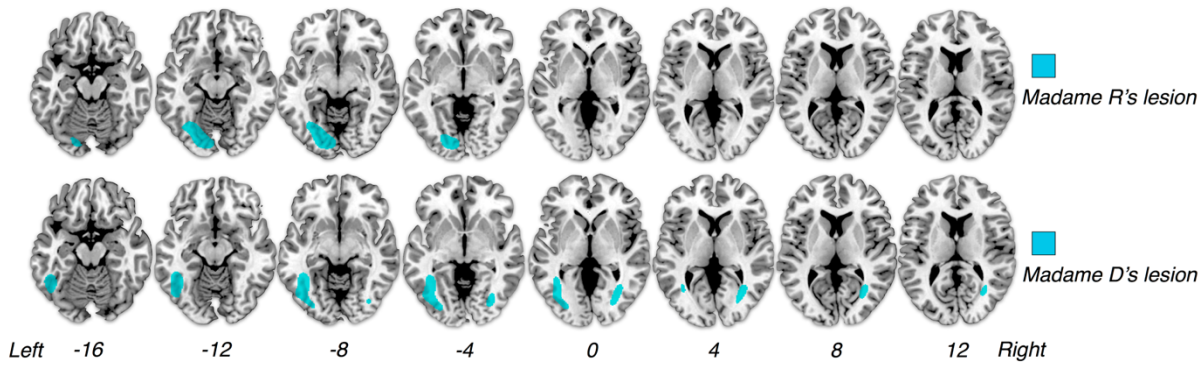

Fig 4. Reconstruction of the lesions of Madame R, with right hemiachromatopsia [24], and of Madame D, with full-field achromatopsia [22]. Image modified from Ref. [21], with permission of Elsevier.

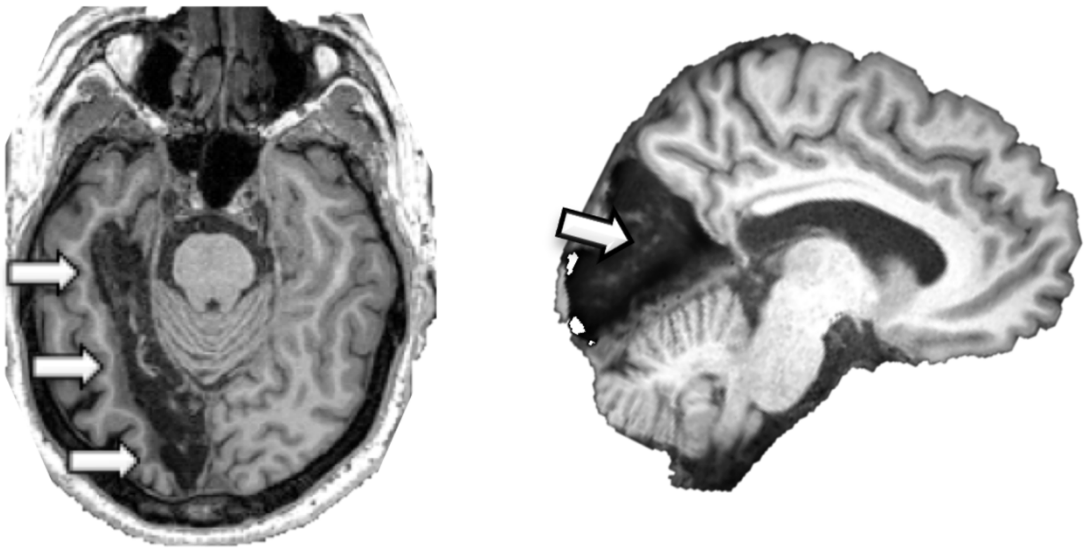

Fig. 5. Lesional correlates of color anomia in patient RDS. Axial and sagittal views of a T1-weighted structural MRI. The ischemic lesion (arrows) encompassed the calcarine sulcus, the lingual, fusiform and parahippocampal gyri in the left hemisphere, as well as the callosal splenium. L, left; R, right. Figure from Ref. [38**] (CC BY-NC-ND 4.0).