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The Relation Between the Optic Nerve and its Visual Link to the Brain

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The optical nerve, a string – suchlike grouping of nerve filaments, connects and transmits visual information from the eye to the brain. The optical nerve is substantially composed of retinal ganglion cell (RGC) axons [1]. In the mortal eye, the optical nerve receives light signals from about 125 million photoreceptor cells (known as rods and cones) via two intermediate neuron types, bipolar and amacrine cells. In the brain, the optical nerve transmits vision signals to the side geniculate nexus (LGN), where visual information is bear to the visual cortex of the brain that converts the image impulses into objects that we see.

In the retinal apkins of the eye, further than 23 types of RGCs vary significantly in terms of their morphology, connections, and responses to visual stimulation [2]. Those visual transmitting RGCs are the neuronal cells. They all partake the defining parcels of

1. Enjoying a cell body (soma) at the inner face of the retina

2. Having a long axon that extends into the brain via the optical chiasm and the optical tract

3. Synapsing with the LGN. The RGCs form multiple functional pathways within the optical nerve to intervene the visual signal

Mortal beings can see three primary colors red, green, and blue [3]. This is due to our having three different kinds of color sensitive cone cells red cones, green cones, and blue cones.

The RGCs connecting to the red and green cones are runt RGCs. They're substantially located at the center of the retina (known as fovea). A single runt RGC communicates with as many as five photoreceptors. They transmit red-green color signals to the parvo cellular subcaste in the LGN [4]. The runt-parvocellular pathway responds to color changes, but has little or no response to discrepancy change. This pathway has center- compass open fields, and slow conduction rapidity. Because of this pathway, we can see objects precisely in detail and in full color.

The bistratified RGCs are likely involved in blue color vision. Bistratified cells admit visual information input firstly from an intermediate figures of cones and rods. The bistratified RGCs connect to the koniocellular layers in the LGN. The koniocellular neurons form robust layers throughout the visual hemi field and have moderate spatial resolution, moderate conduction rapidity, and can respond to moderate- discrepancy stimulants [5]. They've veritably large open fields that only retain on- center regions (no out- compass regions).

Objects can be seen in the dark with stir and coarse outlines accentuated due to the parasol RGCs. At the fringe of the retina, a single parasol RGC connects to numerous thousands of photoreceptors (numerous rods and many cones). The parasol RGCs project their axons to the magnocellular layers of the LGN and are primarily concerned with visual perception [6]. They've presto conduction rapidity, can respond to low- discrepancy stimulants, but aren't veritably sensitive to changes in color.

Eventually, humans can see objects in three- dimension courtesy of the crossing over of optical nerve filaments at the optical chiasm. This anatomic structure allows for the mortal visual cortex to admit the same hemispheric visual field from both eyes, therefore making it possible for the visual cortex to induce binocular and stereoscopic vision.

Lately, a new type of RGC, called photosensitive RGCs, was discovered. The photosensitive RGCs contribute minimally to our vision, but play a crucial part in vision regulation [7]. Photosensitive RGCs axons don't have connections to the LGN, but form the retino-hypothalamic tract, and synapse to three other locales in the brain for specific vision regulation functions

1. Pretectal nexus involved in reflexive eye movements, thereby helping to target what we want to see

2. Midbrain capitals involved in controlling the size of the pupil, therefore helping to acclimate the brilliance of objects; and coordinating movement of the eye for fastening

3. Suprachiasmatic nexus involved in regulating the sleep-wake cycle

A completely functional optical nerve is essential for vision. Obviously, any damage of the optical nerve will ramify the precise transmission of visual information between the retina and brain, directly leading to vision deformation and/ or vision loss [8]. Damage to the optical nerve can affect from

Direct/ circular physical damage (e.g. optical trauma)

Acute/sub-acute physiological lesion (e.g. infection or inflammation, or malice (cancer))

Habitual neuronal degeneration (e.g. glaucoma, a most common cause of optical nerve damage)

Also, the optical nerve is also a veritably important vivo model for studying central nervous protection and rejuvenescence [9]. At the cell biology position, the RGC axons are covered with myelin produced by oligodendrocytes (rather than Schwann cells of the supplemental nervous system) after exiting the eye on their way to the LGN and therefore part of the central nervous system. Scientists have lately acquired more and more substantiation that certain types of damage to the optical nerve may be reversible in the future [10]. Thus, the optical nerve provides a implicit window to explore more complicated neuronal degenerative conditions, similar as Alzheimer's complaint and Huntington complaint.

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