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The Koniocellular Pathway and Its Role in Blindsight: Evidence Across Species

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Introduction

The **lateral geniculate nucleus (LGN)**, located in the thalamus, serves as the brain's primary relay for visual information from the retina to the cortex [1,2]. It is organized into three main cellular subsystems: **magnocellular** (M), **parvocellular** (P), and **koniocellular** (K) layers, each processing different visual features [3]. **Magnocellular neurons** (M cells) are large, fast-conducting cells specialized for detecting motion, luminance contrast, and low spatial frequency information—critical for tracking moving objects [4]. **Parvocellular neurons** (P cells) are smaller, slower-conducting, and tuned to fine detail, high spatial frequency, and red-green color discrimination—key for object recognition and reading [3]. **Koniocellular neurons** (K cells) are small, diverse relay cells located between the M and P layers. They process multiple visual modalities, including short-wavelength ("blue") color information, spatial and motion cues, and possibly brain-state–modulating rhythms [5, 6]. K cells have unique projections to both **primary visual cortex (V1)** and extrastriate visual areas, including MT/V5, making them a prime candidate for alternative visual processing routes [7].

One of the most striking phenomena linked to these alternate pathways is **blindsight**—the ability of individuals with damage to V1 to detect, localize, or respond to visual stimuli without conscious awareness [8, 9]. For example, a patient with a visual field scotoma might still correctly guess the direction of a moving object or orient toward a flash of light in their blind field despite reporting no visual perception. This suggests that subcortical–extrastriate pathways can bypass V1, preserving some aspects of visual function [1, 8].

Anatomical and functional studies have shown that a **direct LGN–MT/hMT+ projection dominated by K cells** exists in both primates and humans [11, 12]. Here we will refer to this as the **koniocellular geniculo–MT/hMT+ pathway (KGM pathway)**. Unlike the **classical LGN–V1–MT pathway** (primary cortical visual pathway, PCV), which mediates conscious vision [1, 4]. The KGM pathway can transmit motion and spatial information to MT/hMT+ without V1 involvement, potentially supporting blindsight. Another candidate route is the **superior colliculus–pulvinar–MT/hMT+ pathway (SC–Pulvinar pathway)**, which may complement the KGM pathway in residual vision [13].

The Koniocellular Pathway: Structure and Roles

K cells in the LGN receive input from specialized retinal ganglion cells, including small bistratified cells sensitive to blue-yellow color contrast and broad-band cells responsive to luminance and motion [5, 14]. They project diffusely—not only to layer 1 and 3 of V1 but also directly to MT/V5, V2 thin stripes, and other dorsal stream areas [7, 11]. Functionally, the KGM pathway is optimized for integrating motion and spatial cues under conditions where cortical processing is impaired or absent. Loss of K cell function or interruption of the KGM pathway disrupts residual motion detection and spatial orientation in the absence of V1[12] (Figure 1).

Evidence from Animal Models

Macaques are highly valuable in medical research due to their close biological similarities to humans, including comparable brain structure, immune and reproductive systems, and ocular anatomy. These similarities make findings from macaque studies highly translatable to human medicine, particularly in complex diseases spanning neurology, ophthalmology, vaccine development, infectious diseases, cardiovascular conditions, aging, and metabolic disorders [22].

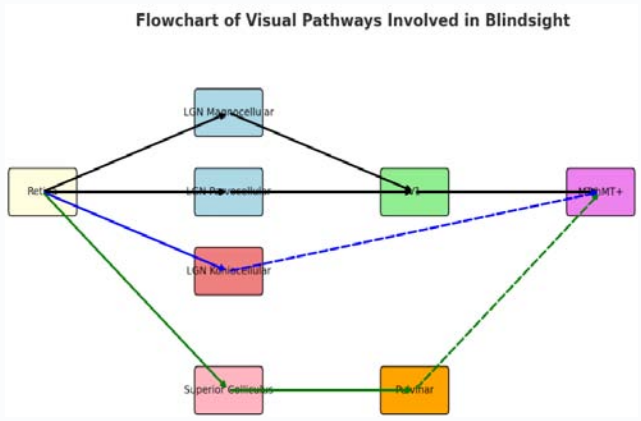

Figure 1: Flowchart of Visual Pathways:

- **Normal vision pathway** (black) – Retina → LGN Magnocellular/ Parvocellular → V1 → MT/hMT+
- **Koniocellular blindsight pathway** (blue) – Retina → LGN Koniocellular → MT/hMT+
- **SC–Pulvinar alternative pathway** (green) – Retina → Superior Colliculus → Pulvinar → MT/hMT+

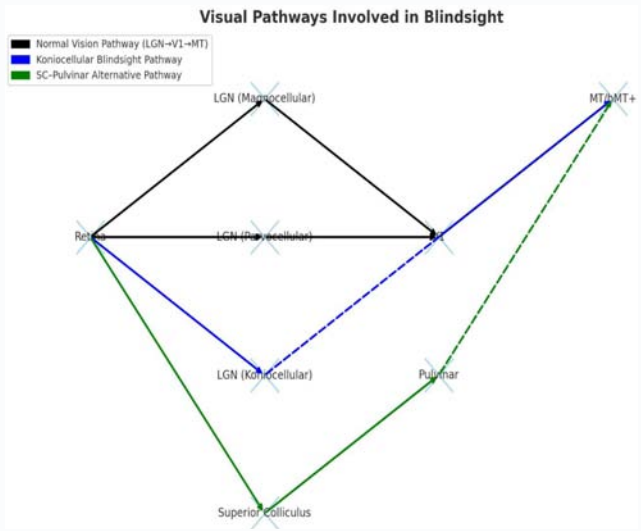

Figure 2: Diagram comparing the three main visual pathways:

- **Black** → Normal vision pathway (LGN → V1 → MT)
- **Blue** → Koniocellular blindsight pathway (LGN koniocellular → MT/ hMT+)
- **Green** → SC–Pulvinar alternative pathway

Their eye structure, especially the macula, closely mirrors that of humans, making them indispensable for vision science and modeling ocular diseases such as refractive errors, macular degeneration, glaucoma, and photoreceptor biology. Research involving macaques has contributed to significant clinical breakthroughs, including the discovery of the Rh blood factor, insights into HIV/AIDS mechanisms, and understanding vision impairments like amblyopia. Because many diseases, especially ocular and neurological, manifest similarly in macaques and humans, these studies form a critical foundation for current clinical approaches and therapeutic development [23].

Direct LGN–MT/hMT+ Projections in Macaques (KGM Pathway)

Sincich et al. (2004) investigated whether macaques possess a **direct LGN–MT/V5 projection** bypassing V1 [11]. MT/V5 is a dorsal stream area essential for processing motion direction and speed [15]. Using retrograde tracers—**cholera toxin B subunit (CTB)** injected into MT and **wheat germ agglutinin–horseradish peroxidase (WGA-HRP)** into V1—they found that ~70% of MT-projecting LGN neurons were located in the koniocellular layers. Minimal overlap between MT- and V1-projecting cells confirmed these are distinct populations. The estimated ~8,000 MT-projecting K cells per LGN were deemed sufficient to activate MT without V1 input, establishing strong anatomical evidence for the **KGM pathway** as a V1-independent motion route.

Functional Role in V1-Lesioned Macaques

Schmid et al. (2010) tested the functionality of the KGM pathway in macaques with chronic unilateral V1 lesions [12]. Visual stimuli in the scotoma activated extrastriate regions, including MT, and elicited accurate **saccadic eye movements** toward high-contrast targets. When the LGN was inactivated with a GABA_A agonist, both MT activity and blindsight-like behaviors vanished. This confirmed that LGN input—predominantly via K cells—is essential for residual motion perception and spatial localization when the PCV pathway is disrupted.

Evidence from Human Studies

Functional MRI Evidence for the KGM Pathway

Gaglianese et al. (2012) used fMRI and conditional Granger causality analysis in healthy humans to test for direct LGN–hMT+ connectivity [16]. Moving-dot stimuli activated hMT+ and showed significant direct effective connectivity from LGN to hMT+, with shorter latency than LGN–V1 connections. This timing advantage aligns with the KGM pathway's role in processing rapid, unconscious motion.

Structural Integrity Predicts Blindsight

Ajina et al. (2015) examined patients with unilateral V1 damage using diffusion MRI and tractography [17]. Blindsight-positive patients had intact LGN–hMT+ tracts with preserved microstructural integrity, while blindsight-negative patients had abnormal axonal properties—reduced fractional anisotropy and increased mean diffusivity. No behavioral link was found for SC–Pulvinar or interhemispheric hMT+ connections, underscoring the KGM pathway's specific importance.

Competing and Complementary Pathways

The SC–Pulvinar Pathway

Kinoshita et al. (2019) explored the **SC–Pulvinar–MT/hMT+ pathway** in macaques with unilateral V1 lesions [13]. Silencing this pathway impaired visually guided saccades and increased reaction times, suggesting it contributes to blindsight. However, K cells in the LGN remained intact, and staining methods used did not label all koniocellular neurons, meaning the KGM pathway likely remained functional. This indicates blindsight may result from parallel processing in both KGM and SC–Pulvinar pathways, with KGM playing a dominant role.

Synthesis, Clinical Implications, and Future Directions

Across primates and humans, converging anatomical, physiological, and neuroimaging evidence confirms the existence of the **koniocellular geniculo–MT/hMT+ pathway (KGM pathway)**—a direct LGN-to-MT/hMT+ projection dominated by K

cells that transmits motion and spatial information independently of V1 [11, 12, 16, 17]. The KGM pathway likely underpins blindsight by enabling dorsal stream processing when the PCV pathway is damaged [8, 9].

Clinically, these findings suggest new rehabilitation approaches could target dorsal stream activity through K cell stimulation, potentially improving functional outcomes in cortical blindness [18, 19]. In consciousness research, blindsight offers a natural model for dissociating perception from awareness [8, 20]. Neuroplasticity studies may further reveal how preserved subcortical–extrastriate connections support adaptive compensation [19, 21].

Future priorities include subtype-specific analyses of K cells to clarify their individual roles in blindsight, longitudinal imaging of KGM pathway integrity after V1 injury, comparative mapping of KGM and SC–Pulvinar contributions to residual vision, and neuromodulation trials targeting KGM circuits to test enhancement of unconscious and possibly conscious vision [19] (Figure 2).

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