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Microscopic Phase-Locked Vision: Cortical Columns as Oscillatory Observers of Photoreceptor Discs

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Abstract

Classical visual models struggle to explain how humans achieve extremely rapid and high-fidelity perception. For example, simple visual decisions can occur in tens of milliseconds while cortical visual processing seems to take over 100 ms, and ~10^8 retinal photoreceptors feed into only ~10^6 ganglion channels without apparent loss of detail. We propose a novel model in which individual photoreceptor outer segments act as multilayered optical encoders and primary visual cortex (V1) columns act as phase-locked observers of these micro-images. In this view, each stack of hundreds (cones) to thousands (rods) of photoreceptor discs can create a series of slightly phase-shifted images, and cortical columns oscillate in the gamma band (30–80 Hz) to "watch" the resulting layered patterns. This phase-locked coupling allows the brain to reconstruct rich spatial detail without slow, hierarchical transmission. The model resolves the latency and compression paradoxes by leveraging parallel microcircuits: ensemble coding of millions of "mini-eye" images provides both rapid onset and lossless detail. We outline specific predictions for retinal optics and cortical synchrony that can be tested with adaptive optics imaging and neurophysiology. If validated, this framework would have broad implications for vision neuroscience, consciousness models, and biophotonic information processing in the brain.

Keywords

Microscopic Phase-Locked; compression paradox; Multilayered Focal Encoders; Neurons spanning layers.

Introduction

Paradoxes in Vision Science

Human vision is astonishingly fast and detailed, yet classical theories struggle to account for two persistent paradoxes. First, the **latency paradox**: humans can reflexively respond to visual stimuli in tens of milliseconds (sometimes reported ~13–25 ms), even though cortical processing of vision typically requires at least 100–150 ms. This gap between perceived immediacy and neural response latency suggests that some mechanisms of vision must occur much faster than standard feedforward pathways. Second, the **compression paradox**: the human retina contains on the order of 120 million rod photoreceptors and 6 million, but only ~1.2 million retinal ganglion cells transmit visual information to the brain. If each ganglion cell simply pooled signals linearly, enormous compression would degrade spatial detail. Yet perception remains essentially photorealistic over the entire visual field. No mainstream model fully explains how the brain maintains such precision despite massive thinning of information.

Classic accounts liken the eye to a camera that projects an image onto photoreceptors, which then stream data through layered pathways to the cortex. However, recent evidence indicates that the retina itself performs complex processing. For example, bipolars, amacrines, and ganglion cells form parallel circuits even before reaching the brain. This suggests that we must reconsider where and how visual features are encoded. In this article, we introduce a radical hypothesis: each photoreceptor's outer segment acts as a series of optical encoders, and each cortical column in V1 acts as a synchronized observer of those encodings. We show that this "microscopic phase-locked vision" framework naturally resolves the speed and resolution paradoxes and makes distinct experimental predictions.

Photoreceptor Discs as Multilayered Focal Encoders

Photoreceptors have a unique structure: their light-sensitive outer segments consist of stacks of membranous discs. In rods, these stacks can number over 1000 discs per cell, each ~10–15 nm thick. Cones typically have hundreds of discs. These densely packed layers contain the photopigment (rhodopsin or cone opsins) that captures photons. Structurally, the discs lie nearly parallel to the incident light path and are tightly spaced (20–30 µm total length for rods

We propose that this multilayered arrangement effectively creates a multi-plane optical encoder. As a photon wavefront traverses the outer segment, each successive disc can shift the phase and amplitude of the light slightly. In other words, each photoreceptor may form a series of very similar images at incrementally different optical depths. This is analogous to having multiple micro-lenses in series, each imprinting a subtly shifted image on the photopigment sheets. In traditional optics, a stack of semi-transparent plates can produce interference and phase shifts; here, the aqueous disc membranes may play a similar role. Because the discs are so thin and numerous, even small refractive index variations could cause distinct phase delays.

Critically, this means that a single photoreceptor cell could encode a stack of layered images representing the local visual scene. Each disc may act like a separate "slice" of image data. For instance, the first few discs might capture the most focused image plane, while deeper discs capture slightly defocused or phase-shifted versions. Downstream retinal circuitry (bipolar and ganglion cells) could then sample different combinations of these phase-shifted signals. In effect, each photoreceptor becomes a multilayered focal encoder that carries more information than a single binary response would imply. We anticipate that adaptive optics microscopy or

optical coherence tomography might one day reveal subtle back-scatter patterns from these disc layers under live imaging, although such experiments are challenging.

(**Figure 2**: Photoreceptor Discs as Multilayered Encoders. Each outer-segment disc (OS) in a rod (or cone) contributes to a stacked, phase-shifted image. Light entering a photoreceptor is successively modulated by each disc, effectively creating multiple sub-images at slightly different phases.)

Cortical columns as phase-locked observers

Primary visual cortex is organized into cortical columns – microcircuits of neurons spanning layers with shared functional properties. These columns are well known for feature selectivity (orientation, eye dominance, etc.), but here we suggest they also serve as phase-locked observers of the photoreceptor disc images. Neurons in V1 exhibit prominent gamma-band oscillations (30–80 Hz) during visual stimulation. Such oscillations provide a temporal reference frame: spikes become locked to particular phases of the gamma rhythm. We hypothesize that each V1 column can lock its local oscillatory network to the phase of incoming signals from specific photoreceptors or small groups thereof.

In practical terms, one cortical column could "listen" to one photoreceptor's micro-images by aligning its gamma rhythm to the timing of that receptor's output. Within a gamma cycle, the column's neurons might sample the input repeatedly, effectively reconstructing the layered image over time. The rapid oscillation acts like a clock, and the photoreceptor stack provides a temporally distributed input across the cycle. In this way, a column observes not just a single intensity value, but a time-encoded sequence of values corresponding to the various disc layers. Once these inputs are phase-aligned, standard intracolumn processing can integrate them into a coherent representation.

Evidence supports the plausibility of this scheme. Gray and Singer (1989) showed that cortical neurons in cat V1 fire in synchrony at ~40 Hz for coherent stimuli. Fries (2009) argues that gamma synchronization is a fundamental mechanism by which visual inputs are segmented and selected in cortexp. Moreover, recent recordings in primate V1 reveal multiple distinct gamma sub-bands across layers, indicating rich phase-coding potential within columns. In a given column, neurons can thus form a dynamic assembly that is relatively independent from neighboring columns, allowing many parallel observers across the cortex. We suggest that this ensemble of oscillatory observers collectively samples the mosaic of photoreceptor outputs in a phase-locked fashion, analogous to having millions of tiny "eyes" whose images are read out rhythmically.

Ensemble precision from millions of mini-eyes

By treating each photoreceptor as a layered encoder and each cortical column as a local observer, the visual system effectively comprises millions of mini-eyes working in concert. Each micro-eye (photoreceptor) generates multiple phase-coded measurements of a small part of the visual field, and the cortex contains as many observing units as there are photoreceptors (on the order of 10^8) in principle. Even though the ganglion cell layer compresses these signals to ~10^6 fibers, the compression is offset by the massively parallel cortical sampling. In other words, the combinatorial ensemble of many phase-locked circuits can preserve the rich detail of the original input. This resembles population coding principles in neural systems: by averaging across many similarly tuned units, noise is reduced and resolution increases. Here, the resolution gain comes from the redundancy and slight variation of the stacked disc inputs.

Practically, this means that two adjacent photoreceptors with overlapping receptive fields still carry complementary shifted images. A V1 column observing the first receptor's outputs will reconstruct one micro-image, while a neighboring column observing the second receptor reconstructs another. The brain can then stitch these micro-images seamlessly through the known retinotopic maps and thalamocortical connectivity. Importantly, because the columns operate in parallel and in synchrony, this stitching happens without temporal lag. The vast number of parallel pathways – millions of discs times thousands of columns – provides a built-in error-correction: random fluctuations (thermal or synaptic) average out, while the coherent phase signal remains. Thus, the ensemble approach explains how fine spatial detail can survive massive information bottlenecks: each bit of visual space is effectively encoded and transmitted by a large collective of oscillators rather than a single channel.

Resolving the Latency and Compression Paradoxes

With this framework in place, the visual speed and detail paradoxes find natural resolutions. First, the latency paradox: if each cortical column is phase-locked to its photoreceptor input, then the arrival of new visual information can be detected and integrated almost instantaneously relative to the gamma cycle. For example, a sudden change in the image reaching a given photoreceptor would shift the phase or amplitude of the oscillatory input, which the column would immediately sense on the next gamma cycle. In effect, cortical neurons do not wait for a slow serial relay of information; instead they continuously observe the photoreceptor outputs in real time. The result is that perceptual detection can occur on the order of one or two gamma cycles (tens of milliseconds), consistent with the fastest visual reaction times. This also aligns with observations that subcortical pathways (e.g. via the superior colliculus) can trigger early responses, since the retina's layered output would be broadcast widely.

Second, the compression paradox: because each photoreceptor's output is sampled by a dedicated local circuit, the apparent thinning of information is illusory. It is not that all 120+ million photoreceptor signals are funneled serially through 1.2 million fibers; rather, the retina's local cells distribute portions of each photoreceptor's multi-phase signal to multiple downstream neurons. The cortical columns then recombine these portions. In other words, the parallel microcircuits essentially invert the retinal compression. Each cortical area reconstructs a micro-image from its local photoreceptors independently. When all columns do this simultaneously, the brain regains the full spatial resolution. In this picture, compression only occurs in time (within each gamma cycle) rather than in space. Thus, no visual detail is truly lost – it is spread across time and across the ensemble of columns. This design elegantly explains how human vision achieves photorealistic detail with a massively under sampled optic nerve.

Predictions and experimental tests

Our model makes several testable predictions. First, adaptive optics retinal imaging should reveal subtle interference or phase effects in photoreceptor outer segments. For instance, one might observe temperature- or voltage-dependent scattering changes corresponding to disc-level activity, as hinted by Li and Dai's detection of biophotonic emissions in dark-adapted retinav. Second, neurophysiological recordings should detect gamma-phase correlations between single photoreceptor signals and local V1 column activity. For example, simultaneous patch recordings from retinal cells and laminar V1 electrodes (under anesthesia or in vitro retina-cortex preparations) should show phase-locked spike timing at 30–80 Hz (as in invertebrate and cortico-cortical studies). Disrupting gamma oscillations (pharmacologically or via optogenetics) ought to impair

the resolution or speed of visual detection more severely than predicted by classical models.

Third, behavioral experiments could probe differential ON/OFF processing predicted by this scheme. Because each photoreceptor generates layered images, abrupt light–dark transitions (which engage ON vs OFF bipolar pathways might produce distinct temporal signatures in cortical oscillations. For example, an ON-dominant stimulus might align the early phase of gamma differently than an OFF stimulus. Advanced techniques like two-photon imaging or calcium imaging in primate V1 might reveal a mosaic of mini-responses reflecting the underlying photoreceptor array. Finally, artificial retina prostheses and cortical implants should consider phase coding: stimulators that deliver pulses synchronized to cortical gamma may restore higher acuity than static stimulation. These and other empirical tests (see Box 1) could validate or refute the phase-locked micro-eye hypothesis.

Quantum and biophoton implications

Our hypothesis also intersects with emerging ideas in quantum biology and biophotonics. Photons at the level of individual discs and microcircuits may exhibit quantum coherence or entanglement effects that classical models ignore. Some theories, propose that biological oscillators can achieve quantum coherence. In the retina, ultraweak photon emissions (biophotons) have been measured and linked to neural processing. Li and Dai (2016) showed that temperature-dependent biophotonic activity contributes to the discrete “dark noise” of photoreceptors, suggesting that thermal energy can induce coherent photon signals. In our model, such biophotons could carry high-precision timing information between layers.

At the systems level, this view dovetails with the Default Space Theory (DST) of consciousness. DST posits that the brain (with body) creates a unified 3D “default space” of experience via synchronized oscillations in thalamocortical loops. Our phase-locked vision model fits neatly into DST: the thalamus could serve as a hub coordinating each column’s timing, while whole-body oscillations (including in the eyes and retina) contribute to a shared frame of reference. In other words, sensory organs themselves become part of the oscillatory network that generates conscious imagery. If true, the fundamental unit of visual experience is not a single pixel or spike, but a temporally synchronized assembly of many cells – a perspective compatible with quantum information frameworks.

Biophoton-mediated communication could be especially relevant at high visual acuities and fast speeds. Sun et al. (2010) demonstrated that in situ biophoton autography (IBP) signals can travel along neural pathways, acting as communication signals. Tang and Dai (2014) showed spatiotemporal propagation of glutamate-induced biophotonic activity in cortical slices. These studies suggest that neurons can emit and transmit photons in synchrony with electrical activity. Our model implies that such photon signals from photoreceptor discs may entrain cortical rhythms. In summary, a range of non-classical phenomena – from biophotons to global coherence – may play roles in the phase-locked vision system.

Implications and outlook

If validated, this microscopic phase-locked vision model would shift our understanding of early vision and consciousness. It suggests that even the smallest sensory cells participate in large-scale neural codes, bridging quantum, cellular, and systems levels. Technologically, the model could inspire new imaging methods that capture phase information in living retina, or new prosthetic designs that emulate layered encoding. Clinically,

conditions that disrupt retinal disc structure (e.g. retinitis pigmentosa) might be reinterpreted as failures of phase coding, not just photopigment loss. In basic neuroscience, the model predicts new roles for cortical columns – not only as feature detectors but as active time observers – and demands that we consider oscillatory timing as a carrier of fine-grained sensory data.

The connection to consciousness theories is also profound: vision may be more than feedforward image construction; it may be a continual reconstruction of a 3D "mental movie" by a large oscillatory ensemble. In this light, classical 2D retinal images are just one layer of a multi-planar code. Future work should explore how this layered coding interacts with attention, learning, and multisensory integration, possibly via the thalamus as a central pacemaker. We also anticipate connections with other rhythmic systems (e.g. respiratory and cardiac rhythms) that modulate brain oscillations and could thereby influence perception. Overall, the phase-locked micro-eye theory opens a rich landscape of experiments and concepts in neuroscience.

Conclusion

We have proposed a unified model in which each photoreceptor's stacked discs generate a set of phase-shifted images, and each cortical column is an oscillatory observer that decodes those images in concert. This microscopic, parallel coding scheme naturally explains how humans see so quickly and precisely despite the eye's anatomical constraints. By invoking gamma-phase locking and ensemble averaging, the model resolves both the latency and compression paradoxes of vision. It makes concrete predictions – from adaptive optics imaging of photoreceptors to cortical synchronization patterns – that can be tested with current neuroscience tools. Ultimately, this framework suggests that perception arises from a dynamic, oscillatory interplay between "mini-eyes" and cortical networks, with potential implications for theories of consciousness, quantum brain dynamics, and visual prosthetics.

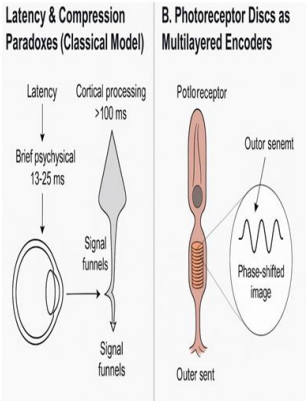

Figure 1: Letency & Compression Paradoxes (Classical Model), Photoreceptor Discs as Multilayered Encoders.

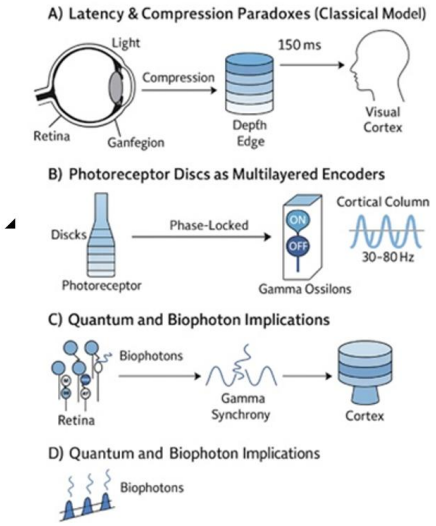

Figure 2: Cortical Columns as Oscillatory Observers of Photoreceptor Discs.

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