MISCELLANEOUS

The Functional Role of PING: mapping the space

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Abstract

This article is a *corpus*, not a review. It is an attempt at an exhaustive, auditable paper list for one narrow question, leaving the synthesis, the open debates, and the gaps to a later pass. The question:

> In which papers is the PING mechanism explicitly the vehicle for a functional claim?

PING here means gamma generated by the reciprocal pyramidal↔interneuron loop with its characteristic excitatory–inhibitory timing. A paper is in the corpus only if it both invokes that mechanism as the generator of the rhythm *and* argues that a cognitive or computational function follows from it: attention, stimulus selection, communication-through-coherence, gain control and coincidence windows, phase coding, or binding. Papers where gamma merely *correlates* with a function, or where the mechanism is studied with no functional claim, are out; so are critiques that attack a function without naming the mechanism, though the major ones are collected in the appendix so the eventual review can engage them. The scope was frozen and git tagged (`freeze-ar016-v1`) before any measured run, so the recall figure below is valid: the completeness estimate compares samples of one fixed population, which a scope that drifted mid-run would destroy.

Papers in the corpus	160, every DOI verified
Estimated true size	≈ 180–250 (two-source Chapman 246, 95% CI 175–316)
Estimated recall	≈ 65–90 %
Channels	memory · keyword-search · citation-graph · embedding
Membership authority	one two-stage judge (wide-net → confirm), consistent across channels
Coverage caveats	keyless (no Semantic Scholar / PubMed); single judge per paper; OpenAlex abstract gaps on seminal papers

The estimate is a range, not a point. Completeness is measured by *capture–recapture* (the Lincoln–Petersen / Chapman estimator): when two channels independently sample one fixed population, the size of their overlap implies how many papers both missed, and so the true size $\hat{N}$. Computed over the three channel-pairs that overlap, it yields three estimates, and they disagree: 246 (memory × search), 219 (search × citation), and 142 (memory × citation). The disagreement is expected rather than anomalous: the channels have heterogeneous catchability (memory reaches the old and seminal, search the recent and well-indexed), which

violates the independence the estimator assumes. No single $\hat{N}$ is reliable, so we report the band 180–250 and a recall of 65–90 % rather than a point value.

Methods

The model can estimate the size of a literature (it can tell hundreds from thousands) and judge whether a specific paper is in scope. Its memory is also a recall channel: unreliable in a single readout, where it is lossy and confabulates at the margin, but usable once multiplexed across many decorrelated passes and filtered through DOI verification. Recall is gathered from four channels (memory among them), membership is decided by judgment, and completeness is estimated statistically. The procedure, in order:

1. **Refine and scout the question, before anything is frozen.** The raw interest ("the big questions about PING") is too broad to search, so it is narrowed conversationally: the model reflects the corpus size back from memory (gamma broadly is thousands of papers; the mechanism-carries-function slice is hundreds), weighs narrowing and widening axes by their size, and converges on 4 anchors with explicit in/out boundaries, targeting 100–400. A cheap scout pass then probes live database counts to confirm the slice is exhaustible and expose traps: the mechanism core returns 100–510 hits against the "communication through coherence" balloon (5878 hits) the scope must exclude, and the coincidence/gain axis is nearly invisible to keywords (6 hits), needing concept-level queries later. It also flags collisions like "PING" naming a network tool. The result is the scope proposal, estimated at 100–250, which is then frozen.
2. **Freeze the scope.** The inclusion rubric is fixed and git tagged (`freeze-ar016-v1`) before any measured run, so both capture samples target one fixed population and the recall figure is meaningful.
3. **Gather recall from four orthogonal channels,** chosen to fail on different papers so their union covers what any one misses:
   - **Memory / weights:** 20 decorrelated elicitation passes (per-lab, per-function-axis, per-decade), each in its own context, unioned. This channel is the knowledge baked into the model's trained weights. Covers the old, seminal, abstract-less stratum.
   - **Keyword search:** a deep OpenAlex full-text sweep across the frozen synonym ring (the fixed set of the concept's synonyms and near-terms, agreed at scope-freeze), deliberately high-recall. Covers the recent, well-indexed stratum.
   - **Citation graph:** the backward references and forward citers of the found set, scored by how many of the corpus each candidate connects to. Covers the well-connected stratum, independent of words and of memory.
   - **Embedding:** nearest neighbours of the strongest seeds in SPECTER (a document-embedding model, served by Semantic Scholar, that places papers by learned content similarity), independent of shared words, citations, and authorship.
4. **Verify every candidate DOI against Crossref.** An unresolvable DOI is treated as a hallucination and discarded, so nothing enters the corpus that cannot be resolved to a real paper.
5. **Judge membership with one consistent two-stage judge:** a wide-net first pass, then a strict confirming pass against the frozen rubric, so the in/out boundary is one authority

applied uniformly. Membership is *not* decided by any keyword filter. An earlier attempt to do so failed, because a filter strict enough to exclude the broad "gamma and attention" literature also excludes the function-forward canonical papers whose abstracts never say "interneuron".

6. **Estimate completeness by capture–recapture.** Lincoln–Petersen / Chapman over the single judged population, treating each channel as an independent capture, gives the true-size band and the recall figure.

Hallucination and verification

Because a language model generates the memory channel, fabricated references are the obvious failure mode. Every model-proposed reference passes a two-layer DOI check: each generating pass self-verifies against Crossref and drops what will not resolve, then an independent re-check resolves the deduplicated union again. In a controlled pilot where the model gave author, year, and title (not DOIs) and the resolver did the lookup, all 44 of 44 recalled papers were real, resolvable works. In the full harvest the 20 memory passes returned 420 candidates, deduplicated to 180 references, of which 0 failed to resolve at the re-check.

The caveat is that DOI-resolution catches an *invented identifier* but not a *valid DOI attached to the wrong paper*. That residual is real: a later audit removed 1 reference whose DOI resolved only to a journal, and corrected 4 records with valid DOIs but corrupt third-party metadata (a prostate-cancer note, a Japanese architecture paper). Resolution is necessary but not sufficient; a Crossref-versus-OpenAlex cross-check catches the rest. One such entry reached the list and was removed.

Results

Scope

Scouting produced the frozen scope (tag `freeze-ar016-v1`), estimated at 100–250 papers. A paper is in only if it names the PING / E–I loop as the gamma generator *and* draws a functional claim from it; correlation-only phenomenology, mechanism with no functional claim, non-gamma work, and mechanism-agnostic critiques are out. Four boundary questions were resolved during scouting: communication-through-coherence counts only when the E–I/PING generator is named (not for generic gamma); the coincidence-detection and gain axis is included, reached by concept-level rather than keyword queries; reviews that argue mechanism→function are kept as members; and there is no era floor. The scope was seeded by 4 anchor papers:

1. **Borgers, Kopell (2008)**. Gamma Oscillations and Stimulus Selection. doi:10.1162/neco.2007.07-06-289
2. **Borgers, Epstein, Kopell (2008)**. Gamma oscillations mediate stimulus competition and attentional selection in a cortical network model. doi:10.1073/pnas.0809511105
3. **Tiesinga, Sejnowski (2009)**. Cortical Enlightenment: Are Attentional Gamma Oscillations Driven by ING or PING?. doi:10.1016/j.neuron.2009.09.009
4. **Fries (2015)**. Rhythms for Cognition: Communication through Coherence. doi:10.1016/j.neuron.2015.09.034

Selection

The four channels surfaced 3111 distinct candidate papers, every one put through the same membership judge. 160 were accepted and 2951 rejected, an acceptance rate of 5.1 %. Most rejected candidates are gamma-and-cognition papers that do not make the mechanism-*carries*-function argument.

Candidates judged (search + memory union)	1694
Candidates judged (citation graph)	1154
Candidates judged (embedding)	263
Total distinct papers judged	3111
Accepted into the corpus	160 (5.1 %)
Rejected	2951

Provenance

The corpus is the union of complementary channels; no single one would have produced it:

Found by memory only	59
Found by search only	45
Found by memory and search	21
Found by citation graph only	35
Total	160

Only 21 of the 160 papers were caught by both memory and keyword search. The low overlap reflects the channels' complementarity, and is why four were needed rather than one.

Saturation

The stopping point rests on the marginal-yield curve rather than a target count. Each successive channel returned fewer new in-scope papers:

Channel	New in-scope	Candidates screened
memory	80	n/a
keyword search	+45	1552
citation graph	+36	1154
embedding (SPECTER)	+0	263

The yield fell from 80 to +45 to +36 to +0. The final channel (orthogonal, abstract-rich, centred on the hundred strongest seeds) screened 263 semantic neighbours and added nothing. This is a saturation signal rather than a proof: falling returns together with a null from an orthogonal channel. It should be discounted somewhat, since SPECTER is recency-biased and its null is strongest for recent work; but the citation channel already covered the older literature, so the remaining un-caught set is probably small.

Distribution by function axis

Every paper is tagged with the one function its mechanism-to-function argument chiefly serves, from a fixed set of eight. The distribution is the first-order map of the field:

Communication-through-coherence, routing & gating	39
Attention & stimulus selection	17
Stimulus & biased competition	15
Coincidence detection, gain & the temporal window	16
Phase coding & spike timing	26
Binding by synchrony	12
Causal / optogenetic PV–gamma & circuit performance	17
Reviews & theory (mechanism → function)	18
Total	160

Communication-through-coherence and routing is the largest axis (39): the recurring claim is that the E–I gamma rhythm gates which signals pass between areas. The causal / optogenetic cluster (17) is a sizeable group of its own, reflecting a shift from correlating gamma with function to manipulating PV interneurons and measuring the effect. The full annotated list, grouped by axis with a one-line rationale per paper, is the bibliography below.

Discussion

This is a DOI-verified corpus of 160 papers, with a *measured* recall of roughly 65–90 %, and a recorded reason for each paper's inclusion. It is not complete. The tail of a semantic corpus is expensive (papers in no index, mis-tagged, or obscure cost more per paper than the bulk did), and the capture–recapture band (180–250) is itself uncertain because the channels are heterogeneous. The most-cited references of the corpus are foundational papers that are purely mechanism (how gamma is generated) or purely function (attention modulates gamma), each satisfying only half the "mechanism carries function" test; this is why the citation graph returned many candidates but few members. The scope is restrictive, and the set it defines is small.

The strict scope excludes one class of paper by construction: the *critique*. Papers that question a functional claim (does synchrony bind? does gamma carry a usable code?) argue at the level of spike timing or coding in general, without naming the pyramidal↔interneuron loop as the generator, so they fail the mechanism-as-vehicle test. The judge rejected the canonical skeptics it saw (Shadlen & Movshon's *Synchrony Unbound* and both Ray & Maunsell papers), and the recall channels did not reach the others (Thiele & Stoner, Palanca & DeAngelis, Merker), since the scope does not target them. This is consistent for a corpus but a limitation for a review, which should not omit the critical literature. The major critiques are listed in the appendix, outside the corpus, for that later pass.

The run did not support the assumption that a language model is a poor recall channel. Multiplexed across 20 decorrelated passes and filtered through DOI verification, memory was the largest single channel: 80 of the 160 in-scope papers, 59 of them found by memory alone, with low fabrication (44 of 44 real in the controlled pilot, 0 of 180 unresolvable in the full harvest). What made memory usable was decorrelation and verification, not restricting its use.

Next steps

The corpus is the input to the review, not the review. The obvious continuations: (1) a second, independent judge pass to turn the single-judge boundary into a voted one and tighten precision; (2) one more orthogonal recall channel where a key exists (PubMed for the biomedical silo, or author-complete feeds for the core labs), expected, from the yield curve, to add only single digits; (3) the synthesis itself, reading down each axis below to map which mechanistic claims are contested and where the gaps are. Scope reopens only under a new freeze tag; questions that surfaced during the build are logged, not silently acted on.

The corpus: annotated bibliography by function axis

All 160 papers, grouped by primary function axis and sorted by year, each with a one-sentence statement of how the E–I/PING gamma mechanism carries its functional claim and a tag for the channel that found it (m memory · s search · $m{+}s$ both · c citation).

Communication-through-coherence, routing & gating (39)

1. **Kopell et al. (2000)**. Gamma rhythms and beta rhythms have different synchronization properties. *Proceedings of the National Academy of Sciences*. doi:10.1073/pnas.97.4.1867
   E-I gamma synchronizes only over short delays while beta tolerates long ones, assigning gamma the role of local computation and beta of long-range interaction — the mechanism sets the spatial scale of communication. [m]
2. **Tiesinga et al. (2001)**. Optimal information transfer in synchronized neocortical neurons. *Neurocomputing*. doi:10.1016/s0925-2312(01)00464-7
   Argues synchronization among neocortical neurons optimizes information transfer, with inhibition-set synchronous windows serving as the vehicle for efficient signal transmission between populations. [m]
3. **Salinas & Sejnowski (2001)**. Correlated neuronal activity and the flow of neural information. *Nature Reviews Neuroscience*. doi:10.1038/35086012
   Argues correlated/synchronous activity gates the flow of neural information, so inhibition-generated synchrony acts as the vehicle controlling which signals are transmitted downstream. [m]
4. **Fries (2005)**. A mechanism for cognitive dynamics: neuronal communication through neuronal coherence. *Trends in Cognitive Sciences*. doi:10.1016/j.tics.2005.08.011
   The founding communication-through-coherence hypothesis: rhythmic (gamma) inhibitory windows must be phase-aligned between groups for effective interaction, making E-I gamma coherence the vehicle for flexible inter-areal communication. [m]
5. **Middleton et al. (2008)**. NMDA receptor-dependent switching between different gamma rhythm-generating microcircuits in entorhinal cortex. *Proceedings of the National Academy of Sciences*. doi:10.1073/pnas.0809302105
   NMDA-dependent switching of interneuron-mediated gamma frequencies in entorhinal cortex matches distinct hippocampal subfield frequencies, so the E-I gamma mechanism sets the temporal channel for information transfer between regions. [c]
6. **Gielen, Krupa & Zeitler (2010)**. Gamma oscillations as a mechanism for selective information transmission. *Biological Cybernetics*. doi:10.1007/s00422-010-0390-x
   Shows PING-type gamma-modulated input makes a downstream neuron phase-lock to

and selectively transmit the larger of competing gamma inputs, implementing selective information routing. [m+s]

7. **Tiesinga & Sejnowski (2010)**. Mechanisms for Phase Shifting in Cortical Networks and their Role in Communication through Coherence. *Frontiers in Human Neuroscience*. doi:10.3389/fnhum.2010.00196
   Reciprocal E-I network models phase-shift their gamma output in response to depolarization or I-cell pulses, providing the phase-adjustment mechanism that implements communication-through-coherence and stimulus selection. [m+s]
8. **Ainsworth et al. (2011)**. Dual Gamma Rhythm Generators Control Interlaminar Synchrony in Auditory Cortex. *The Journal of Neuroscience*. doi:10.1523/jneurosci.2209-11.2011
   Distinct laminar gamma generators (including a granular-layer PING) must frequency-match to provide temporal coupling of co-active regions, making E-I gamma the vehicle for interlaminar/inter-regional synchronization and signal control. [m+s]
9. **Bastos et al. (2012)**. Canonical Microcircuits for Predictive Coding. *Neuron*. doi:10.1016/j.neuron.2012.10.038
   Maps predictive-coding computations onto the canonical cortical microcircuit, assigning gamma from superficial E-I populations to feedforward message passing between hierarchical areas. [m]
10. **Hahn et al. (2013)**. Synfire chains and gamma oscillations: two complementary modes of information transmission in cortical networks. *BMC Neuroscience*. doi:10.1186/1471-2202-14-s1-p226
    Flexible routing of activity across specialized networks is achieved by consistent phase relations between E-I-generated population gamma oscillations, so PING-type coherence is the vehicle for high-fidelity inter-network communication. [s]
11. **Srinivasan, Thorpe & Nunez (2013)**. Top-Down Influences on Local Networks: Basic Theory with Experimental Implications. *Frontiers in Computational Neuroscience*. doi:10.3389/fncom.2013.00029
    A Wilson-Cowan E-I gamma local network is modulated by slow (alpha/theta) rhythms carrying attention/arousal biases, making cross-frequency control of E-I gamma the vehicle for top-down/bottom-up interaction between spatial scales. [s]
12. **Roberts et al. (2013)**. Robust Gamma Coherence between Macaque V1 and V2 by Dynamic Frequency Matching. *Neuron*. doi:10.1016/j.neuron.2013.03.003
    Despite the garbled title, the abstract shows V1-V2 gamma coherence is maintained across stimulus-induced frequency changes, supporting communication-through-coherence via correlated E-I gamma between areas. [m+s]
13. **Pietersen et al. (2014)**. Transition between fast and slow gamma modes in rat hippocampus area CA1 in vitro is modulated by slow CA3 gamma oscillations. *The Journal of Physiology*. doi:10.1113/jphysiol.2013.263889
    Feedback-inhibition-generated fast CA1 gamma can be forced to the slower CA3 gamma via feed-forward inhibition, letting CA1 switch its effective communication between entorhinal cortex and CA3, with gamma synchrony determining the effectiveness of neuronal communication for memory encoding vs recall. [s]

14. **Akam & Kullmann (2014)**. Oscillatory multiplexing of population codes for selective communication in the mammalian brain. *Nature Reviews Neuroscience*. doi:10.1038/nrn3668
Review proposes oscillatory (gamma) multiplexing of firing-rate population codes enables selective inter-areal communication by reconfiguring effective connectivity, with periodic inhibitory modulation as the routing substrate. [m]
15. **Bastos et al. (2015)**. Visual Areas Exert Feedforward and Feedback Influences through Distinct Frequency Channels. *Neuron*. doi:10.1016/j.neuron.2014.12.018
Shows visual areas route feedforward influences through gamma and feedback through lower frequencies, so E-I gamma coherence implements directional inter-areal communication. [m]
16. **Fries (2015)**. Rhythms for Cognition: Communication through Coherence. *Neuron*. doi:10.1016/j.neuron.2015.09.034
Fries's canonical statement that flexible neuronal communication is mechanistically implemented by coherence of E-I gamma rhythms establishing open windows between sending and receiving groups. [m]
17. **Lee, Whittington & Kopell (2015)**. Potential Mechanisms Underlying Intercortical Signal Regulation via Cholinergic Neuromodulators. *The Journal of Neuroscience*. doi:10.1523/jneurosci.0629-15.2015
Cholinergic modulation lets top-down beta/gamma interact with a bottom-up E-I gamma rhythm to gate signal flow between primary sensory and association cortex, making the PING gamma the vehicle for flexible inter-areal routing. [s]
18. **Harnack, Ernst & Pawelzik (2015)**. A model for attentional information routing through coherence predicts biased competition and multistable perception. *Journal of Neurophysiology*. doi:10.1152/jn.01038.2014
A two-layer spiking network with lateral inhibition uses phase shifts between sending and receiving layers to route the attended stimulus, reproducing both biased competition and selective information routing via gamma coherence. [c]
19. **McLelland & VanRullen (2016)**. Theta-Gamma Coding Meets Communication-through-Coherence: Neuronal Oscillatory Multiplexing Theories Reconciled. *PLOS Computational Biology*. doi:10.1371/journal.pcbi.1005162
Feedback-inhibition (PING) gamma phase-matching selectively routes single items via communication-through-coherence while theta-gamma multiplexing supports a phase code, with the E-I gamma rhythm as the vehicle for both. [m+s]
20. **Veit et al. (2017)**. Cortical gamma band synchronization through somatostatin interneurons. *Nature Neuroscience*. doi:10.1038/nn.4562
Optogenetics shows dendrite-targeting SOM interneurons are required for visually induced gamma and long-distance coherence across visual cortex, casting the inhibitory gamma mechanism as the vehicle for synchronizing distributed assemblies for information transfer. [m]
21. **Palmigiano et al. (2017)**. Flexible information routing by transient synchrony. *Nature Neuroscience*. doi:10.1038/nn.4569
Modeling shows transient gamma synchrony among E-I networks flexibly routes

information by dynamically reconfiguring effective connectivity, the core communication-through-coherence claim. [m]

22. **Wal & Tiesinga (2017)**. Phase Difference between Model Cortical Areas Determines Level of Information Transfer. *Frontiers in Computational Neuroscience*. doi:10.3389/fncom.2017.00006
    Two PING circuits transfer information maximally only when their frequency-set phase difference aligns their susceptibility windows, so the E-I gamma loop's phase relationship is the vehicle for flexible inter-areal routing (communication-through-coherence). [m]
23. **Strüber et al. (2017)**. Distance-dependent inhibition facilitates focality of gamma oscillations in the dentate gyrus. *Nature Communications*. doi:10.1038/s41467-017-00936-3
    Distance-dependent inhibition among perisomatic interneurons fragments dentate gamma into multiple focal synchronous centers, letting the interneuron-based PING mechanism route and process complex inputs in parallel across flexibly organized assemblies. [c]
24. **Sherfey et al. (2018)**. Flexible resonance in prefrontal networks with strong feedback inhibition. *PLOS Computational Biology*. doi:10.1371/journal.pcbi.1006357
    Strong feedback inhibition makes PFC networks resonant relays that preferentially transmit inputs near their gamma frequency, so the PING E-I loop is the vehicle for frequency-selective routing/gain of oscillatory signals. [m]
25. **Adesnik (2018)**. Layer-specific excitation/inhibition balances during neuronal synchronization in the visual cortex. *The Journal of Physiology*. doi:10.1113/jp274986
    Layer-specific optogenetic drive shows excitatory neurons in any layer entrain gamma while balanced inhibition promotes propagation of activity to downstream layers, making layer-specific E-I gamma the vehicle for inter-laminar information flow. [c]
26. **Sherfey et al. (2019)**. Prefrontal oscillations modulate the propagation of neuronal activity required for working memory. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/531574
    Biophysical PFC modeling shows local inhibition-based gamma/beta oscillations set the inter-burst period that selectively gates which working-memory item propagates to downstream effectors, making the E-I rhythm the vehicle for flexible signal routing. [s]
27. **Dumont & Gutkin (2019)**. Macroscopic phase resetting-curves determine oscillatory coherence and signal transfer in inter-coupled neural circuits. *PLOS Computational Biology*. doi:10.1371/journal.pcbi.1007019
    Macroscopic phase-resetting curves of PING/ING gamma circuits determine the phase-locking states that govern signal transfer between coupled areas, making the E-I gamma rhythm the vehicle for inter-areal communication. [s]
28. **Sherfey et al. (2020)**. Prefrontal oscillations modulate the propagation of neuronal activity required for working memory. *Neurobiology of Learning and Memory*. doi:10.1016/j.nlm.2020.107228
    Biophysical PFC model argues local interneuron-generated oscillations selectively gate which working-memory item propagates to downstream effectors, making the inhibition-based rhythm the vehicle for flexible signal routing. [s]
29. **Lewis et al. (2020)**. Cortical resonance selects coherent input. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/2020.12.09.417782

Optogenetically injecting white-noise excitation into pyramidal cells reveals that feedback-inhibition-generated cortical gamma resonance selectively transmits coherent input components, giving causal support for communication-through-coherence. [c]

30. **Reyner-Parra & Huguet (2021)**. Phase-locking patterns underlying effective communication in exact firing rate models of neural networks. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/2021.08.13.456218
    Exact mean-field PING models show excitatory-inhibitory populations must phase-lock so input volleys arrive at excitability peaks, implementing communication-through-coherence between gamma-oscillating circuits. [s]
31. **Lucas, Klaus & Christoph (2021)**. Synchronization through uncorrelated noise in excitatory-inhibitory networks. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/2021.10.29.466430
    Uncorrelated synaptic noise induces synchronization between PING excitatory-inhibitory networks and thereby facilitates inter-regional gamma communication via a mechanism distinct from strong coupling. [s]
32. **Lewis et al. (2021)**. Cortical gamma-band resonance preferentially transmits coherent input. *Cell Reports*. doi:10.1016/j.celrep.2021.109083
    Optogenetically probing pyramidal cells shows the recurrent E-I gamma resonance preferentially transmits coherent input, providing causal evidence for communication-through-coherence as the function of cortical gamma. [c]
33. **Reyner-Parra & Huguet (2022)**. Phase-locking patterns underlying effective communication in exact firing rate models of neural networks. *PLOS Computational Biology*. doi:10.1371/journal.pcbi.1009342
    Exact mean-field PING networks are analyzed to find the phase-locked states that make input volleys arrive at excitability peaks, so the E-I gamma rhythm is the vehicle for effective communication-through-coherence. [s]
34. **Rebscher, Obermayer & Metzner (2022)**. Synchronization Through Uncorrelated Noise in Excitatory-Inhibitory Networks. *Frontiers in Computational Neuroscience*. doi:10.3389/fncom.2022.825865
    Uncorrelated synaptic noise enhances between-region synchronization of PING networks, so the E-I gamma mechanism supports inter-regional communication even under noisy conditions. [s]
35. **Veit et al. (2023)**. Cortical VIP neurons locally control the gain but globally control the coherence of gamma band rhythms. *Neuron*. doi:10.1016/j.neuron.2022.10.036
    Shows VIP disinhibition (via VIP->SST) globally tunes long-range gamma coherence for stimulus-matched routing while locally scaling gamma gain, making the E-I gamma loop the vehicle for coherence-based inter-areal communication. [m+s]
36. **Katsanevaki et al. (2023)**. Attentional effects on local V1 microcircuits explain selective V1-V4 communication. *NeuroImage*. doi:10.1016/j.neuroimage.2023.120375
    Dynamic causal modeling attributes selective V1-V4 routing to attentional changes in intrinsic V1 inhibition that let the attended stimulus's gamma entrain V4, making the local E-I gamma circuit the vehicle for selective communication. [c]
37. **Gonzalez-Burgos et al. (2023)**. Mechanisms regulating the properties of inhibition-based gamma oscillations in primate prefrontal and parietal cortices. *Cerebral Cortex*.

doi:10.1093/cercor/bhad077
Recurrent-excitation and GABAAR-mediated synchrony set distinct inhibition-based gamma frequencies in primate DLPFC versus PPC, and these matched frequencies are argued to enable information transfer between the two areas of the working-memory network. [c]

38. **Phensy et al. (2024)**. Prefrontal gamma oscillations engage dynamic cell type-specific configurations to support flexible behavior. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/2024.03.08.584173
PV-interneuron-entrained ~40Hz gamma is shown (via optogenetics/voltage imaging) to organize distinct circuit-specific synchronization motifs that route PFC->mediodorsal-thalamus communication in a behaviorally selective way to support flexible behavior. [s]
39. **Phensy et al. (2026)**. Prefrontal gamma oscillations engage dynamic cell-type-specific configurations to support flexible behavior. *Neuron*. doi:10.1016/j.neuron.2026.05.002
Uses voltage imaging and optogenetics to show PV-interneuron gamma synchronization with MD-projecting PFC neurons forms circuit-specific motifs that route PFC->MD communication in support of flexible behavior. [s]

Attention & stimulus selection (17)

1. **Niebur, Koch & Rosin (1993)**. An oscillation-based model for the neuronal basis of attention. *Vision Research*. doi:10.1016/0042-6989(93)90236-p
Proposes an oscillation-based model where interneuron-network gamma synchronization among attended neurons implements the neuronal basis of selective attention. [m]
2. **Niebur & Koch (1994)**. A model for the neuronal implementation of selective visual attention based on temporal correlation among neurons. *Journal of Computational Neuroscience*. doi:10.1007/bf00962722
Models selective visual attention via temporal correlation among neurons, using inhibitory-network-generated synchrony to tag and select the attended stimulus's assembly. [m]
3. **Börgers, Epstein & Kopell (2005)**. Background gamma rhythmicity and attention in cortical local circuits: A computational study. *Proceedings of the National Academy of Sciences*. doi:10.1073/pnas.0502366102
Computational model links cholinergic neuromodulation to a background PING gamma rhythm that amplifies stimulus-specific responses and enhances competition, proposing gamma as the substrate of preparatory attention. [m+s]
4. **Tiesinga et al. (2005)**. Inhibitory synchrony as a mechanism for attentional gain modulation. *arXiv (Cornell University)*. doi:10.48550/arxiv.q-bio/0503019
Top-down control of local interneuron-network synchrony modulates a target neuron's firing rate and spike-LFP gamma coherence, so attentional gain in V4 is the vehicle-function of synchronized inhibitory (E-I) gamma. [s]
5. **Buia & Tiesinga (2006)**. Attentional modulation of firing rate and synchrony in a model cortical network. *Journal of Computational Neuroscience*. doi:10.1007/s10827-006-6358-0
Models attention as reduced drive to interneurons in a cortical E-I network, so that gamma synchrony set by the interneuron network shifts the contrast-response (gain) curve as seen in V4. [m]

6. **Mishra, Fellous & Sejnowski (2006)**. Selective attention through phase relationship of excitatory and inhibitory input synchrony in a model cortical neuron. *Neural Networks*. doi:10.1016/j.neunet.2006.08.005
   Models selective attention as controlled by the phase relationship between synchronous excitatory and inhibitory (interneuron) inputs, gating a cortical neuron's response by gamma-phase alignment. [m]
7. **Tiesinga & Buia (2006)**. Attentional modulation in layer 4 of the visual cortex could be mediated by interneurons with complex receptive field characteristics. *arXiv (Cornell University)*. doi:10.48550/arxiv.q-bio/0611030
   Increasing interneuron excitability in a layer-4 orientation-tuned circuit raises spike-LFP gamma coherence without changing rate, reproducing attentional modulation through control of the E-I gamma network. [s]
8. **Buia & Tiesinga (2008)**. Role of Interneuron Diversity in the Cortical Microcircuit for Attention. *Journal of Neurophysiology*. doi:10.1152/jn.01004.2007
   A V4 microcircuit model with PV-like feedforward interneurons shows feature-based attention shifts assembly synchrony into the gamma band and resolves stimulus competition, arguing interneuron-diversity-based gamma mediates attentional selection among competing stimuli. [m+s]
9. **Deco & Thiele (2009)**. Attention – oscillations and neuropharmacology. *European Journal of Neuroscience*. doi:10.1111/j.1460-9568.2009.06833.x
   Review of attention arguing that NMDA/AMPA- (and ACh-) tuned gamma-range rhythmic synchronization in local circuits enables enhanced processing of attended stimuli and efficient communication between ensembles. [m+s]
10. **Ardid et al. (2010)**. Reconciling Coherent Oscillation with Modulationof Irregular Spiking Activity in Selective Attention:Gamma-Range Synchronization between Sensoryand Executive Cortical Areas. *The Journal of Neuroscience*. doi:10.1523/jneurosci.4222-09.2010
   A reciprocal MT<->PFC spiking loop reproduces attention-driven interareal gamma coherence that is feature-selective and enhances sensory gain, making sparsely-synchronized E-I gamma the vehicle for selective attention. [m+s]
11. **Vinck et al. (2013)**. Attentional Modulation of Cell-Class-Specific Gamma-Band Synchronization in Awake Monkey Area V4. *Neuron*. doi:10.1016/j.neuron.2013.08.019
   Dissects attention's effect on gamma synchronization of narrow- vs broad-spiking (interneuron vs pyramidal) cells in V4, showing attention reshapes the E-I gamma cycle that selects attended stimuli. [m+s]
12. **Blaes & Burwick (2015)**. Attentional Bias Through Oscillatory Coherence Between Excitatory Activity and Inhibitory Minima. *Neural Computation*. doi:10.1162/neco_a_00742
   A coupled excitatory-inhibitory gamma network implements attentional bias as oscillatory (spike-field) coherence between target-encoding excitatory units and the inhibitory pool, selecting the attended target while suppressing distracters. [s]
13. **Boroujeni, Tiesinga & Womelsdorf (2020)**. Interneuron Specific Gamma Synchronization Indexes Cue Uncertainty and Prediction Errors in Lateral Prefrontal and Anterior Cingulate Cortex. *bioRxiv (Cold Spring Harbor Laboratory)*.

doi:10.1101/2020.07.24.220319
A specific interneuron subclass's 35-45 Hz gamma-synchronous spiking indexes cue uncertainty and prediction errors in PFC/ACC and is modeled as a soft winner-take-all gate — interneuron gamma as the selection/gating vehicle. [s]

14. **Lu et al. (2020)**. Phasic cholinergic signaling promotes emergence of local gamma rhythms in excitatory–inhibitory networks. *European Journal of Neuroscience*. doi:10.1111/ejn.14744
Computational E-I modeling shows brief acetylcholine pulses (via M-current suppression) trigger transient PING gamma, arguing the E-I loop's cholinergically driven gamma underlies cue detection in behavioral attention tasks. [s]
15. **Wagatsuma, Nobukawa & Fukai (2023)**. A microcircuit model involving parvalbumin, somatostatin, and vasoactive intestinal polypeptide inhibitory interneurons for the modulation of neuronal oscillation during visual processing. *Cerebral Cortex*. doi:10.1093/cercor/bhac355
A PV/SOM/VIP microcircuit model in which PV-generated gamma (vs SOM beta) regulates the integration of feedforward sensory and feedback attentional signals during visual processing. [s]
16. **Zheng et al. (2024)**. Analyzing top-down visual attention in the context of gamma oscillations: a layer- dependent network-of- networks approach. *Frontiers in Computational Neuroscience*. doi:10.3389/fncom.2024.1439632
A layer-dependent network-of-PING-networks reproduces top-down attentional enhancement of neuronal responses, using the E-I gamma loop across cortical layers as the vehicle for selective attention. [s]
17. **Sajedin, FallahTaherpazir & Menhaj (2025)**. Cholinergic modulation mediates attentional mechanism to enhance coherence between cortical layers in macaque V1 and V4. *Scientific Reports*. doi:10.1038/s41598-025-24109-1
Biophysical V1-V4 model shows cholinergic drive enhances gamma synchrony/coherence via increased inhibitory drive to reproduce attentional modulation of inter-areal communication in visual cortex. [s]

Stimulus & biased competition (15)

1. **Olufsen et al. (2003)**. New Roles for the Gamma Rhythm: Population Tuning and Preprocessing for the Beta Rhythm. *Journal of Computational Neuroscience*. doi:10.1023/a:1021124317706
Proposes a new role for gamma in population tuning, where the E-I gamma cycle sharpens which neurons fire (winner selection) and preprocesses activity for the beta rhythm. [m]
2. **Doiron et al. (2003)**. Inhibitory feedback required for network oscillatory responses to communication but not prey stimuli. *Nature*. doi:10.1038/nature01360
Shows inhibitory feedback is required to generate oscillatory network responses selectively to global communication stimuli but not prey stimuli, making the E-I feedback loop the vehicle for stimulus-specific selection. [m]
3. **Tiesinga & Sejnowski (2004)**. Rapid Temporal Modulation of Synchrony by Competition in Cortical Interneuron Networks. *Neural Computation*. doi:10.1162/089976604322742029

Hodgkin-Huxley interneuron-network models show selectively activating a fraction of GABAergic interneurons rapidly synchronizes them and suppresses competitors, so 'synchrony by competition' modulates gamma synchrony (with minimal rate change) to enhance attended stimulus impact on downstream cortex. [m]

4. **Tiesinga (2004)**. Stimulus competition by inhibitory interference. *arXiv (Cornell University)*. doi:10.48550/arxiv.q-bio/0410019
   A V4 model where two interneuron networks deliver synchronous inhibitory volleys shows stimulus competition set by inter-volley delay, with attention biasing the competition, making interference between inhibitory gamma volleys the vehicle for biased competition. [c]
5. **Tiesinga (2005)**. Stimulus Competition by Inhibitory Interference. *Neural Computation*. doi:10.1162/0899766054796905
   A V4 model driven by synchronous inhibition from competing local interneuron networks reproduces biased-competition firing-rate and spike-field-coherence effects, arguing attention resolves stimulus competition by modulating gamma-generating interneuron synchrony. [m]
6. **Tiesinga (2006)**. Stimulus competition by inhibitory interference. *Neurocomputing*. doi:10.1016/j.neucom.2005.12.089
   Proposes that interference between competing inhibitory (interneuron) gamma rhythms lets one stimulus assembly suppress another, implementing stimulus competition through inhibition. [m]
7. **Börgers & Kopell (2008)**. Gamma Oscillations and Stimulus Selection. *Neural Computation*. doi:10.1162/neco.2007.07-06-289
   Borgers and Kopell show a coherent gamma-frequency input gains a large competitive advantage over less coherent inputs when the target circuit includes GABA-A interneurons, providing the PING-based mechanism for attentional biasing of stimulus competition. [m+s]
8. **Börgers, Epstein & Kopell (2008)**. Gamma oscillations mediate stimulus competition and attentional selection in a cortical network model. *Proceedings of the National Academy of Sciences*. doi:10.1073/pnas.0809511105
   Cortical network model shows PING gamma oscillations mediate stimulus competition and attentional selection via oscillatory selection, where interneuron coherence regulates competing assemblies' firing rates. [m+s]
9. **Almeida, Idiart & Lisman (2009)**. A Second Function of Gamma Frequency Oscillations: An E%-Max Winner-Take-All Mechanism Selects Which Cells Fire. *Journal of Neuroscience*. doi:10.1523/jneurosci.6044-08.2009
   Gamma-frequency feedback inhibition implements an E%-max winner-take-all that selects which principal cells fire and sharpens V1 orientation tuning, making the E-I gamma loop the vehicle for competitive stimulus selection. [m]
10. **Wildie (2012)**. Establishing communication between neuronal populations through competitive entrainment. *Frontiers in Computational Neuroscience*. doi:10.3389/fncom.2011.00062
    Multiple stimuli compete to entrain a target population by gamma coherence and only

the winner is transmitted via communication-through-coherence, making the PING E-I loop the vehicle for competitive stimulus selection and routing. [m+s]

11. **Börgers & Walker (2013)**. Toggling between gamma-frequency activity and suppression of cell assemblies. *Frontiers in Computational Neuroscience*. doi:10.3389/fncom.2013.00033
    The PING suppression boundary lets strongly-driven I-cells suppress subsets of E-cells so that strong assembly volleys are followed by weaker ones, making the reciprocal E-I loop a mechanism for competitive gating/winner-selection among cell assemblies. [m]
12. **Mostafa, Müller & Indiveri (2015)**. Rhythmic Inhibition Allows Neural Networks to Search for Maximally Consistent States. *Neural Computation*. doi:10.1162/neco_a_00785
    Gamma-band rhythmic inhibition drives coupled cortical motifs to search for maximally consistent states, implementing constraint-satisfaction and perceptual multistability so the E-I gamma rhythm mediates competition among candidate network configurations. [c]
13. **Rennó-Costa, Teixeira & Soltesz (2019)**. Regulation of gamma-frequency oscillation by feedforward inhibition: A computational modeling study. *Hippocampus*. doi:10.1002/hipo.23093
    Argues the reciprocal E-I (PING) gamma rhythm selects which subset of cells fires each cycle (level of selection/winner-take-all), and that feedforward inhibition stabilizes this selection against input magnitude. [s]
14. **Onorato et al. (2020)**. A Distinct Class of Bursting Neurons with Strong Gamma Synchronization and Stimulus Selectivity in Monkey V1. *Neuron*. doi:10.1016/j.neuron.2019.09.039
    Argues a distinct bursting cell class in V1 that strongly gamma-synchronizes carries the strongest stimulus selectivity, tying E-I/PING gamma synchronization to which stimulus-tuned population dominates the local representation. [m]
15. **Boroujeni, Tiesinga & Womelsdorf (2021)**. Interneuron-specific gamma synchronization indexes cue uncertainty and prediction errors in lateral prefrontal and anterior cingulate cortex. *eLife*. doi:10.7554/elife.69111
    An identified interneuron subclass shows uncertainty-linked gamma-synchronous spiking that circuit modeling interprets as soft winner-take-all gating, making interneuron gamma the vehicle for competitive selection of high-uncertainty information. [m+s]

Coincidence detection, gain & the temporal window (16)

1. **König, Engel & Singer (1996)**. Integrator or coincidence detector? The role of the cortical neuron revisited. *Trends in Neurosciences*. doi:10.1016/s0166-2236(96)80019-1
    Argues cortical neurons operate as coincidence detectors rather than integrators, so inhibition-set narrow integration windows enable synchrony-based signaling relevant to temporal binding. [m]
2. **Burchell, Faulkner & Whittington (1998)**. Gamma frequency oscillations gate temporally coded afferent inputs in the rat hippocampal slice. *Neuroscience Letters*. doi:10.1016/s0304-3940(98)00676-4
    Hippocampal gamma oscillations gate temporally coded afferent inputs, so the inhibition-defined gamma window of the E-I network determines which timed inputs are admitted (title-inferred, empty abstract). [c]

3. **Palva et al. (2000)**. Fast Network Oscillations in the Newborn Rat HippocampusIn Vitro. *The Journal of Neuroscience*. doi:10.1523/jneurosci.20-03-01170.2000
   GABAergic gamma-frequency modulation synchronizes newborn hippocampal pyramidal firing to millisecond precision, proposed as a substrate for selective coincidence detection, making the E-I gamma the vehicle for a precise integration window. [s]
4. **Tiesinga et al. (2002)**. Information transfer in entrained cortical neurons. *Network: Computation in Neural Systems*. doi:10.1080/net.13.1.41.66
   Synchronized gap-junction-coupled interneuron inhibition entrains pyramidal cells and, counterintuitively, increases the mutual information their spike phase carries about input — inhibitory gamma entrainment as the vehicle for information transfer. [m]
5. **Jonas et al. (2004)**. Interneuron Diversity series: Fast in, fast out – temporal and spatial signal processing in hippocampal interneurons. *Trends in Neurosciences*. doi:10.1016/j.tins.2003.10.010
   Reviews how fast-spiking hippocampal interneurons' rapid, precise signaling ('fast in, fast out') imposes narrow temporal integration windows, positioning interneuron speed as the basis for coincidence detection and gamma timing. [m]
6. **Tiesinga et al. (2004)**. Inhibitory synchrony as a mechanism for attentional gain modulation. *Journal of Physiology-Paris*. doi:10.1016/j.jphysparis.2005.09.002
   Shows that increasing synchrony of local interneuron (inhibitory) inputs modulates a neuron's f-I gain and spike-LFP gamma coherence, offering inhibitory synchrony as the mechanism of attentional gain modulation. [m]
7. **Tiesinga et al. (2004)**. Synchronization as a mechanism for attentional gain modulation. *Neurocomputing*. doi:10.1016/j.neucom.2004.01.108
   Companion modeling work arguing synchrony of inhibitory interneuron input controls neuronal gain and spike-input coherence, the substrate of attentional gain modulation in the gamma band. [m]
8. **Paik, Kumar & Glaser (2009)**. Spontaneous Local Gamma Oscillation Selectively Enhances Neural Network Responsiveness. *PLoS Computational Biology*. doi:10.1371/journal.pcbi.1000342
   In an E-I Hodgkin-Huxley model, reciprocal E-I/I-E gamma injects a periodic current that amplifies weak feedforward inputs, so the PING rhythm acts as an input-strength-dependent gain mechanism selectively boosting network responsiveness. [c]
9. **Knoblich et al. (2010)**. What do We Gain from Gamma? Local Dynamic Gain Modulation Drives Enhanced Efficacy and Efficiency of Signal Transmission. *Frontiers in Human Neuroscience*. doi:10.3389/fnhum.2010.00185
   Optogenetic 40 Hz FS-interneuron drive sharpens a post-stimulus temporal window that boosts spike precision and downstream gain, making inhibition-set coincidence windows of the E-I loop the vehicle for efficient signal transmission. [m+s]
10. **Otte, Hasenstaub & Callaway (2010)**. Cell Type-Specific Control of Neuronal Responsiveness by Gamma-Band Oscillatory Inhibition. *The Journal of Neuroscience*. doi:10.1523/jneurosci.4818-09.2010
    Using dynamic-clamp gamma-frequency perisomatic inhibition, the paper shows cell-type-specific control of neuronal responsiveness, so synchronized interneuron gamma sets the gain and integration window with which each cell type transforms input to output. [c]

11. **Li et al. (2011)**. Impact of gamma-oscillatory inhibition on the signal transmission of a cortical pyramidal neuron. *Cognitive Neurodynamics*. doi:10.1007/s11571-011-9169-6
    Modeling a pyramidal neuron under gamma-oscillatory inhibition, the paper shows this rhythmic perisomatic inhibition shapes signal transmission, so the E-I gamma cycle gates the neuron's gain and effective integration window. [c]
12. **Lozano-Soldevilla et al. (2014)**. GABAergic Modulation of Visual Gamma and Alpha Oscillations and Its Consequences for Working Memory Performance. *Current Biology*. doi:10.1016/j.cub.2014.10.017
    Pharmacological GABAergic modulation shifts visual gamma (and alpha), and these changes in inhibition-generated gamma track working-memory performance, casting the E-I gamma cycle as the inhibitory processing window supporting memory. [c]
13. **Cardin (2018)**. Inhibitory Interneurons Regulate Temporal Precision and Correlations in Cortical Circuits. *Trends in Neurosciences*. doi:10.1016/j.tins.2018.07.015
    Reviews how GABAergic interneurons in the E-I circuit regulate spike-timing precision and suppress slow correlations, making inhibition the vehicle for temporal windowing and coordinated cortical rhythms. [m]
14. **Orekhova et al. (2018)**. Input-dependent modulation of MEG gamma oscillations reflects gain control in the visual cortex. *Scientific Reports*. doi:10.1038/s41598-018-26779-6
    MEG visual gamma power follows a bell-shaped input-output curve interpreted as inhibitory gain control, using the E-I balance underlying gamma as a non-invasive index of the cortex's capacity to counterbalance excitation with inhibition. [c]
15. **Han et al. (2020)**. High-Frequency Synchronization Improves Firing Rate Contrast and Information Transmission Efficiency in E/I Neuronal Networks. *Neural Plasticity*. doi:10.1155/2020/8823111
    High-frequency synchronization in E/I network models enhances firing-rate contrast and information-encoding efficiency, so the E-I gamma rhythm sharpens gain/contrast to improve information transmission. [c]
16. **Susin & Destexhe (2021)**. Integration, coincidence detection and resonance in networks of spiking neurons expressing Gamma oscillations and asynchronous states. *PLOS Computational Biology*. doi:10.1371/journal.pcbi.1009416
    PING/ING gamma states modulate network responsiveness and resonance to afferent stimuli relative to the asynchronous mode, making the E-I gamma cycle the vehicle for gating stimulus integration and gain. [m]

Phase coding & spike timing (26)

1. **Traub, Whittington & Jefferys (1997)**. Gamma Oscillation Model Predicts Intensity Coding by Phase Rather than Frequency. *Neural Computation*. doi:10.1162/neco.1997.9.6.1251
    A distributed pyramidal-interneuron network where interneuron spike doublets set a zero-phase temporal framework predicts stimulus intensity is coded by gamma phase rather than frequency, making the E-I gamma cycle the reference for a phase code that also supports feature binding. [s]
2. **Tiesinga et al. (2002)**. Information transfer in entrained cortical neurons. *Network: Computation in Neural Systems*. doi:10.1088/0954-898x/13/1/302

When pyramidal cells are entrained by synchronized inhibitory gamma input, mutual information between the number of inputs and the output-spike phase lag rises, so the E-I gamma cycle acts as a phase code carrying afferent information. [c]

3. **Hasenstaub et al. (2005)**. Inhibitory Postsynaptic Potentials Carry Synchronized Frequency Information in Active Cortical Networks. *Neuron*. doi:10.1016/j.neuron.2005.06.016
   Shows rhythmic IPSPs from the interneuron network carry synchronized gamma-frequency timing information to pyramidal cells, providing the inhibitory clock that times cortical spiking. [m]
4. **Fries, Nikolić & Singer (2007)**. The gamma cycle. *Trends in Neurosciences*. doi:10.1016/j.tins.2007.05.005
   Proposes the gamma cycle produced by the E-I loop as a temporal frame that orders pyramidal spikes by excitation strength, supplying a phase-of-firing code and reference for downstream reading. [m]
5. **Tiesinga, Fellous & Sejnowski (2008)**. Regulation of spike timing in visual cortical circuits. *Nature Reviews Neuroscience*. doi:10.1038/nrn2315
   Review argues interneuron-generated gamma regulates the precise spike timing of pyramidal cells in visual cortical circuits, framing rhythmic inhibition as the mechanism that sets a spike-timing code. [m]
6. **Morita et al. (2008)**. Recurrent Synaptic Input and the Timing of Gamma-Frequency-Modulated Firing of Pyramidal Cells during Neocortical "UP" States. *The Journal of Neuroscience*. doi:10.1523/jneurosci.3948-07.2008
   Dynamic-clamp shows pyramidal gamma-phase firing during UP states requires low-latency FS->RS inhibition rather than recurrent excitation, making the PING inhibitory drive the vehicle for gamma-timed pyramidal spiking that maximizes representational capacity. [s]
7. **Mazzoni et al. (2008)**. Encoding of Naturalistic Stimuli by Local Field Potential Spectra in Networks of Excitatory and Inhibitory Neurons. *PLoS Computational Biology*. doi:10.1371/journal.pcbi.1000239
   A sparse E-I network encodes static input rates into gamma-range oscillations generated by excitatory-inhibitory interaction, so gamma from the E-I loop serves as the code carrying sensory information in the LFP. [c]
8. **Edward (2010)**. Neuronal biophysics modulate the ability of gamma oscillations to control response timing. *Frontiers in Neuroscience*. doi:10.3389/conf.fnins.2010.03.00004
   Neuronal biophysics determine how synchronized inhibitory gamma enforces temporal precision and controls the timing of postsynaptic responses, making the interneuron-gamma inhibition the vehicle for spike-timing control. [s]
9. **Miconi & VanRullen (2010)**. The Gamma Slideshow: Object-Based Perceptual Cycles in a Model of the Visual Cortex. *Frontiers in Human Neuroscience*. doi:10.3389/fnhum.2010.00205
   In a V1 model, feedback interneuron inhibition generating gamma combines with lateral Gestalt connections so different objects fire on successive gamma cycles, using the PING cycle as a phase reference that temporally segments the scene into perceptual cycles. [c]

10. **Womelsdorf et al. (2012)**. Orientation selectivity and noise correlation in awake monkey area V1 are modulated by the gamma cycle. *Proceedings of the National Academy of Sciences*. doi:10.1073/pnas.1114223109
Orientation selectivity and noise correlations vary across the gamma cycle, so the E-I-driven gamma phase acts as a temporal reference that maximizes stimulus-selective, low-noise spiking for downstream readout. [c]
11. **Nikolić, Fries & Singer (2013)**. Gamma oscillations: precise temporal coordination without a metronome. *Trends in Cognitive Sciences*. doi:10.1016/j.tics.2012.12.003
Argues gamma provides precise, self-organized temporal coordination of spikes without an external clock, casting the E-I gamma cycle as a flexible timing reference for neural coordination and communication. [m]
12. **Sancristóbal et al. (2013)**. Emergent bimodal firing patterns implement different encoding strategies during gamma-band oscillations. *Frontiers in Computational Neuroscience*. doi:10.3389/fncom.2013.00018
In a balanced E-I network expressing gamma, emergent bimodal firing lets neurons co-encode input rate and LFP gamma phase, making the E-I gamma cycle the vehicle for a phase-based encoding strategy. [m+s]
13. **Jadi & Sejnowski (2014)**. Cortical oscillations arise from contextual interactions that regulate sparse coding. *Proceedings of the National Academy of Sciences*. doi:10.1073/pnas.1405300111
Model cortical circuit shows the balance of mono- vs disynaptic drive to inhibitory neurons regulates gamma power/frequency and hence pyramidal spike timing, linking interneuron-driven oscillation to a stimulus-modulated spike-timing code. [m]
14. **Lowet et al. (2015)**. Input-Dependent Frequency Modulation of Cortical Gamma Oscillations Shapes Spatial Synchronization and Enables Phase Coding. *PLOS Computational Biology*. doi:10.1371/journal.pcbi.1004072
Input-dependent gamma-frequency modulation in an E-I network sets phase relations that carry stimulus information (phase coding) and organize spatial synchronization, making the PING gamma cycle the vehicle for temporal coding. [m]
15. **Li & Cleland (2017)**. A coupled-oscillator model of olfactory bulb gamma oscillations. *PLOS Computational Biology*. doi:10.1371/journal.pcbi.1005760
In the olfactory bulb, a pyramidal-resonance interneuron-network gamma (PRING) provides a zero-phase common clock that phase-restricts informative principal-cell spikes, making the E-I gamma cycle the vehicle for a phase-of-firing code. [s]
16. **Shin & Moore (2018)**. Persistent Gamma Spiking in Non-Sensory Fast-Spiking Cells Predicts Perceptual Success. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/374314
A non-sensory fast-spiking interneuron subtype whose regular gamma-periodic firing (the FS-driven gamma template) persists across stimulus onset predicts perceptual detection, arguing the E-I gamma clock provides the temporal reference that enables perception. [s]
17. **Onorato et al. (2019)**. A distinct class of bursting neurons with strong gamma synchronization and stimulus selectivity in monkey V1. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/583955
A bursting excitatory 'pacemaker' cell class in monkey V1 whose gamma phase-locking

(via rhythmic inhibition from interneurons, i.e. the E-I loop) is highly predictive of its orientation tuning, arguing gamma-phase relative to the E-I rhythm encodes and transmits stimulus information. [s]

18. **Feng et al. (2019)**. Gamma Oscillations in the Basolateral Amygdala: Biophysical Mechanisms and Computational Consequences. *eneuro*. doi:10.1523/eneuro.0388-18.2018
    A biophysical BLA model shows gamma arising from reciprocal principal-cell/fast-spiking-interneuron interaction entrains individual neurons' spike timing, making the PING cycle a temporal reference that structures amygdalar spiking. [c]
19. **Meneghetti et al. (2020)**. Thalamic inputs determine functionally distinct gamma bands in mouse primary visual cortex. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/2020.07.09.194811
    A recurrent E-I network reproduces a broadband gamma channel encoding high-contrast information distinct from the thalamic narrowband, so the E-I gamma band functions as a code for complementary visual features (preprint of the eNeuro study). [c]
20. **Meneghetti et al. (2021)**. Narrow and Broad γ Bands Process Complementary Visual Information in Mouse Primary Visual Cortex. *eneuro*. doi:10.1523/eneuro.0106-21.2021
    Broadband gamma arising from cortical excitatory-inhibitory interplay carries high-contrast information (complementary to thalamic narrowband), so the E-I gamma band is the vehicle for encoding distinct visual features. [c]
21. **Quast et al. (2023)**. Rapid synaptic and gamma rhythm signature of mouse critical period plasticity. *Proceedings of the National Academy of Sciences*. doi:10.1073/pnas.2123182120
    Computational TC model shows an interneuronal (PV) gamma rhythm dissociates thalamic input from cortical spiking, driving spike-timing-dependent plasticity that opens critical-period plasticity, using ING timing as the plasticity gate. [s]
22. **Cattani et al. (2023)**. Basolateral amygdala oscillations enable fear learning in a biophysical model. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/2023.04.28.538604
    Preprint of the BLA fear-learning model showing interneuron-generated theta-gamma rhythms shape spike-timing-dependent plasticity, so the E-I rhythms enable associative learning by organizing precise spike timing. [c]
23. **Cattani et al. (2024)**. Basolateral amygdala oscillations enable fear learning in a biophysical model. *eLife*. doi:10.7554/elife.89519
    A biophysical BLA model shows PV/SOM/VIP interneurons generate theta-gamma rhythms that shape spike-timing-dependent plasticity to build a fear circuit, so interneuron-based rhythms enable learning by structuring precise spike timing. [c]
24. **Chalkiadakis et al. (2025)**. The role of feedforward and feedback inhibition in modulating theta-gamma cross-frequency interactions in neural circuits. *PLOS Computational Biology*. doi:10.1371/journal.pcbi.1013363
    Feedback-inhibition PING versus feedforward ING gamma set the directionality of theta-gamma cross-frequency coupling that organizes memory firing sequences, making the E-I gamma mechanism the vehicle for phase-organized timing. [s]
25. **Chalkiadakis et al. (2025)**. The Role of Feedforward and Feedback Inhibition in Modulating Theta-Gamma Cross-Frequency Interactions. *Qeios*. doi:10.32388/rih5uu

This preprint shows PING feedback versus ING feedforward inhibition set the directionality of theta-gamma cross-frequency coupling that organizes neuronal firing sequences, making the E-I gamma motif the vehicle for phase-organized timing. [s]

26. **Tucker & Luu (2026)**. Excitatory-inhibitory resonance in cognition stabilizes synaptic traces in memory. *Cerebral Cortex*. doi:10.1093/cercor/bhag044
Representation is carried by phase alignment of pyramidal-interneuron network gamma (PING), and excitatory-inhibitory resonance across laminae stabilizes synaptic memory traces. [s]

Binding by synchrony (12)

1. **Singer (1993)**. Synchronization of Cortical Activity and Its Putative Role in Information Processing and Learning. *Annual Review of Physiology*. doi:10.1146/annurev.physiol.55.1.349
Singer's foundational review arguing that synchronization of cortical activity (temporal correlation among assemblies) serves feature binding and information processing/learning, the origin of the binding-by-synchrony hypothesis. [m]
2. **Gray (1994)**. Synchronous oscillations in neuronal systems: Mechanisms and functions. *Journal of Computational Neuroscience*. doi:10.1007/bf00962716
Reviews synchronous oscillations as the mechanism for perceptual feature binding, with the gamma rhythm arising from local excitatory-inhibitory network interactions grouping co-active neurons. [m]
3. **Traub et al. (1996)**. A mechanism for generation of long-range synchronous fast oscillations in the cortex. *Nature*. doi:10.1038/383621a0
Describes an E-I network mechanism producing long-range zero-lag synchronous fast oscillations, supplying the synchronization substrate for binding features across distant cortical sites. [m]
4. **Eckhorn et al. (2004)**. Dynamic cortical cooperation related to visual perception. *Proceedings of the International Joint Conference on Neural Networks, 2003.*. doi:10.1109/ijcnn.2003.1223931
Perceptually modulated gamma synchrony (generated by inhibitory feedback loops) among neural groups binds figure features while decoupling figure from background, supporting the binding-by-synchronization account of visual perception. [s]
5. **Tort et al. (2007)**. On the formation of gamma-coherent cell assemblies by oriens lacunosum-moleculare interneurons in the hippocampus. *Proceedings of the National Academy of Sciences*. doi:10.1073/pnas.0705708104
Hippocampal network model shows O-LM interneurons produce gamma coherence that couples anatomically distinct pyramidal modules into gamma-coherent cell assemblies, an interneuron-driven synchrony mechanism for binding distant cells. [m]
6. **Arthur & Boahen (2007)**. Synchrony in Silicon: The Gamma Rhythm. *IEEE Transactions on Neural Networks*. doi:10.1109/tnn.2007.900238
A silicon interneuron network synchronizing in the gamma band via shunting inhibition entrains model excitatory principal neurons to implement object binding, instantiating the interneuron-network gamma mechanism as the vehicle for feature binding. [s]
7. **Thomas & Rufin (2010)**. Gamma oscillations decompose the visual scene into object-based perceptual cycles: a computational model. *Journal of Vision*. doi:10.1167/10.7.1270

A V1 model with feedback inhibition, refractory periods and gestalt lateral connections generates gamma cycles that both bind an object's features by co-firing and act as a winner-take-all gate, decomposing the scene into successive object-based perceptual cycles. [s]

8. **Folias et al. (2013)**. Synchronisation hubs in the visual cortex may arise from strong rhythmic inhibition during gamma oscillations. *European Journal of Neuroscience*. doi:10.1111/ejn.12287
   Strong rhythmic inhibition during gamma makes certain visual-cortex neurons synchronization hubs that entrain promiscuously with others, so the E-I gamma mechanism sets which neurons synchronize while preserving orientation selectivity. [c]
9. **Cornford et al. (2018)**. Dendritic NMDA receptors in parvalbumin neurons enable strong and stable neuronal assemblies. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/279505
   Dendritic NMDARs give PV cells supralinear (coincidence-sensitive) integration of feedback excitation so that reciprocal E-I recruitment strengthens and stabilizes principal-cell assemblies, the gamma-generating loop thereby binding neurons into robust assemblies and explaining sensory-gating deficits when it fails. [s]
10. **Barbosa et al. (2021)**. Across-area synchronization supports feature integration in working memory. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/2021.06.09.447667
    PING gamma (fast excitation, slow feedback inhibition) holds memory items at distinct phases, and weak cross-area coupling synchronizes the two PING networks to bind color and location features in working memory. [s]
11. **Barbosa et al. (2021)**. Across-Area Synchronization Supports Feature Integration in a Biophysical Network Model of Working Memory. *Frontiers in Neural Circuits*. doi:10.3389/fncir.2021.716965
    Reciprocally coupled color and location attractor networks, each generating gamma via recurrent excitation and feedback inhibition, bind features through cross-area gamma synchrony, making PING gamma the vehicle for feature binding in working memory. [s]
12. **Ursino & Pirazzini (2024)**. Construction of a Hierarchical Organization in Semantic Memory: A Model Based on Neural Masses and Gamma-Band Synchronization. *Cognitive Computation*. doi:10.1007/s12559-023-10202-y
    In a neural-mass attractor model of semantic memory, GABAergic-interneuron/E-I balance produces gamma synchronization that binds shared features into hierarchical concept representations, making the PING rhythm the vehicle for feature binding. [c]

Causal / optogenetic PV–gamma & circuit performance (17)

1. **Fuchs et al. (2007)**. Recruitment of Parvalbumin-Positive Interneurons Determines Hippocampal Function and Associated Behavior. *Neuron*. doi:10.1016/j.neuron.2007.01.031
   Genetically altering fast AMPA drive to parvalbumin interneurons disrupts hippocampal gamma and associated behavior, causally linking PV-interneuron recruitment (the PING driver) to network function. [m]
2. **Sohal et al. (2009)**. Parvalbumin neurons and gamma rhythms enhance cortical circuit performance. *Nature*. doi:10.1038/nature07991

Optogenetically drives PV interneurons to generate gamma and shows this enhances cortical signal transmission by reducing noise, causally tying PV-gamma to improved circuit performance. [m]

3. **Cardin et al. (2009)**. Driving fast-spiking cells induces gamma rhythm and controls sensory responses. *Nature*. doi:10.1038/nature08002
   Optogenetic driving of fast-spiking PV interneurons selectively amplifies gamma and the timing of sensory input relative to the gamma cycle controls the amplitude/precision of evoked responses, the founding causal FS-gamma demonstration. [m]
4. **Korotkova et al. (2010)**. NMDA Receptor Ablation on Parvalbumin-Positive Interneurons Impairs Hippocampal Synchrony, Spatial Representations, and Working Memory. *Neuron*. doi:10.1016/j.neuron.2010.09.017
   Cell-type-specific NMDA-receptor ablation on parvalbumin interneurons degrades hippocampal gamma synchrony, spatial coding, and working memory, giving causal evidence that PV-interneuron gamma from the E-I loop supports cognitive performance (title-inferred, empty abstract). [c]
5. **Yizhar et al. (2011)**. Neocortical excitation/inhibition balance in information processing and social dysfunction. *Nature*. doi:10.1038/nature10360
   Optogenetically elevating cellular E/I balance in prefrontal cortex raises 30-80 Hz gamma power and causally impairs information processing and social behavior, tying inhibition-generated gamma to circuit and behavioral performance. [c]
6. **Carlén et al. (2012)**. A critical role for NMDA receptors in parvalbumin interneurons for gamma rhythm induction and behavior. *Molecular Psychiatry*. doi:10.1038/mp.2011.31
   Deletes NMDA receptors selectively in PV interneurons, causally disrupting PV-driven gamma induction and producing specific cognitive deficits, directly linking the PV-gamma E-I mechanism to behavioural performance. [m]
7. **Siegle, Pritchett & Moore (2014)**. Gamma-range synchronization of fast-spiking interneurons can enhance detection of tactile stimuli. *Nature Neuroscience*. doi:10.1038/nn.3797
   Optogenetic FS-gamma enhances detection of less-salient tactile stimuli specifically when excitation arrives in the 20-25 ms window after FS synchronization, causally tying the interneuron-set inhibitory window to perceptual performance. [m]
8. **Cho et al. (2015)**. Gamma Rhythms Link Prefrontal Interneuron Dysfunction with Cognitive Inflexibility in Dlx5/6+/− Mice. *Neuron*. doi:10.1016/j.neuron.2015.02.019
   Optogenetically driving prefrontal fast-spiking interneurons at gamma restores cognitive flexibility in Dlx5/6 mutant mice, causally linking FSIN-generated gamma to cognition. [m]
9. **Kim et al. (2016)**. Prefrontal Parvalbumin Neurons in Control of Attention. *Cell*. doi:10.1016/j.cell.2015.11.038
   Optogenetically silencing vs gamma-synchronizing prefrontal FS-PV interneurons causally impairs vs improves attentional behavior, tying PV-driven gamma and pyramidal phase-locking to attention. [m+s]
10. **Shin & Moore (2019)**. Persistent Gamma Spiking in SI Nonsensory Fast Spiking Cells Predicts Perceptual Success. *Neuron*. doi:10.1016/j.neuron.2019.06.014

Claims perceptual detection success is predicted by sustained gamma-regular spiking of a nonsensory FS interneuron subtype, casting FS-mediated E-I gamma as the temporal reference whose regularity gates perceptual performance. [m]

11. **Tan et al. (2019)**. Gamma oscillations in somatosensory cortex recruit prefrontal and descending serotonergic pathways in aversion and nociception. *Nature Communications*. doi:10.1038/s41467-019-08873-z
    Optogenetic activation of S1 PV interneurons induces gamma that causally enhances nociceptive sensitivity and aversive behavior, tying PV-driven gamma to a behavioural pain-processing outcome. [s]
12. **Cho et al. (2019)**. Interhemispheric gamma synchrony between parvalbumin interneurons supports behavioral adaptation. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/784330
    Out-of-phase optogenetic disruption of parvalbumin-interneuron ~40 Hz interhemispheric synchrony causally impairs behavioral adaptation, showing PV-driven gamma synchrony is required for prefrontal communication supporting learning. [c]
13. **Cho et al. (2020)**. Cross-hemispheric gamma synchrony between prefrontal parvalbumin interneurons supports behavioral adaptation during rule shift learning. *Nature Neuroscience*. doi:10.1038/s41593-020-0647-1
    Cross-hemispheric gamma synchrony between prefrontal PV interneurons is causally required (out-of-phase optogenetic disruption causes perseveration) for behavioral adaptation, casting PV-gamma coherence as the vehicle for flexible rule-shift learning. [m]
14. **Patrono et al. (2022)**. The role of optogenetic stimulations of parvalbumin-positive interneurons in the prefrontal cortex and the ventral hippocampus on an acute MK801 model of schizophrenia-like cognitive inflexibility. *bioRxiv (Cold Spring Harbor Laboratory)*. doi:10.1101/2022.03.25.485752
    Optogenetically driving PFC and ventral-hippocampal PV interneurons is used to rescue MK801-induced cognitive inflexibility, arguing that PV-generated prefrontal gamma (and its theta-coupled cross-regional synchronization) causally supports attentional set-shifting behavior. [s]
15. **Dalal & Haddad (2022)**. Upstream γ-synchronization enhances odor processing in downstream neurons. *Cell Reports*. doi:10.1016/j.celrep.2022.110693
    Optogenetically increasing mitral-cell gamma-synchrony (via GABAergic granule cells) causally enhances downstream piriform odor coding despite lower firing rates, giving direct causal evidence that inhibition-based gamma-synchrony improves inter-areal sensory transmission. [c]
16. **Ferguson, Glick & Huguenard (2023)**. Prefrontal PV interneurons facilitate attention and are linked to attentional dysfunction in a mouse model of absence epilepsy. *eLife*. doi:10.7554/elife.78349
    Gamma-frequency optogenetic stimulation of prefrontal PV interneurons rescues attention deficits in Scn8a+/- mice, giving causal evidence that PV-driven gamma from the E-I loop underlies attentional performance. [s]
17. **Knowles (2024)**. Attentional Deficits and Absence Epilepsy: A Tale of 2 Interneuronopathies. *Epilepsy Currents*. doi:10.1177/15357597241251709

Attention deficits track PV-interneuron hypoactivity and reduced cue-evoked gamma, and gamma-frequency optogenetic PVIN stimulation rescues attentional performance, making the PING-type PV->pyramidal gamma loop the causal vehicle for attention. [s]

Reviews & theory (mechanism → function) (18)

1. **Buzsáki & Chrobak (1995)**. Temporal structure in spatially organized neuronal ensembles: a role for interneuronal networks. *Current Opinion in Neurobiology*. doi:10.1016/0959-4388(95)80012-3
   Reviews how interneuronal networks impose temporal structure on spatially organized ensembles, arguing inhibition-based oscillations provide the timing reference for ensemble coding. [m]
2. **Jefferys, Traub & Whittington (1996)**. Neuronal networks for induced '40 Hz' rhythms. *Trends in Neurosciences*. doi:10.1016/s0166-2236(96)10023-0
   Reviews simulations showing interneuron and E-I networks generate induced 40 Hz rhythms, linking that mechanism to higher functions such as feature binding and lower-level phase coding. [m+s]
3. **Whittington et al. (2000)**. Inhibition-based rhythms: experimental and mathematical observations on network dynamics. *International Journal of Psychophysiology*. doi:10.1016/s0167-8760(00)00173-2
   Reviews experimental and mathematical work on inhibition-based (interneuron-network) rhythm generation, establishing the E-I/inhibitory mechanism that underlies gamma's temporal-coordination functions. [m]
4. **Whittington & Traub (2003)**. Interneuron Diversity series: Inhibitory interneurons and network oscillations in vitro. *Trends in Neurosciences*. doi:10.1016/j.tins.2003.09.016
   Reviews how mutually connected inhibitory interneuron networks generate gamma/beta rhythms in vitro, framing the interneuron E-I mechanism as the substrate for temporal coordination of cortical activity. [m]
5. **Tiesinga & Sejnowski (2009)**. Cortical Enlightenment: Are Attentional Gamma Oscillations Driven by ING or PING?. *Neuron*. doi:10.1016/j.neuron.2009.09.009
   Perspective dissecting whether attentional gamma is generated by ING vs PING interneuron mechanisms, i.e. which E-I circuit motif underlies the attention-related gamma rhythm. [m+s]
6. **Wang (2010)**. Neurophysiological and Computational Principles of Cortical Rhythms in Cognition. *Physiological Reviews*. doi:10.1152/physrev.00035.2008
   Wang's review synthesizing how gamma and theta rhythms arise from interneuronal networks and reciprocal excitatory-inhibitory loops and how these synchronous rhythms sculpt temporal coordination across cognitive functions. [m+s]
7. **Whittington et al. (2011)**. Multiple origins of the cortical gamma rhythm. *Developmental Neurobiology*. doi:10.1002/dneu.20814
   Reviews how inhibition-based (interneuron/E-I) gamma provides shared temporal structure and argues different interneuron mechanisms of gamma generation carry distinct functional correlates including assembly formation and inter-regional communication. [m]
8. **Uhlhaas et al. (2011)**. A new look at gamma? High- (>60 Hz) γ-band activity in cortical networks: Function, mechanisms and impairment. *Progress in Biophysics and*

Molecular Biology. doi:10.1016/j.pbiomolbio.2010.10.004
Review arguing that high-frequency (>60 Hz) gamma generated by the E-I interneuron loop supports cortical computation and is disrupted in disease, surveying mechanism-to-function links across several cognitive domains. [c]

9. **Ainsworth et al. (2012)**. Rates and Rhythms: A Synergistic View of Frequency and Temporal Coding in Neuronal Networks. *Neuron*. doi:10.1016/j.neuron.2012.08.004
Reviews how inhibition-based rhythms including gamma synergistically combine rate and temporal (phase) coding, arguing the E-I oscillation structures information across frequency and timing. [m]
10. **Buzsáki & Wang (2012)**. Mechanisms of Gamma Oscillations. *Annual Review of Neuroscience*. doi:10.1146/annurev-neuro-062111-150444
Canonical review establishing that gamma rhythmogenesis is inextricably tied to perisomatic inhibition emerging from coordinated excitation-inhibition, framing the mechanism that later functional claims depend on. [m]
11. **Bosman, Lansink & Pennartz (2014)**. Functions of gamma-band synchronization in cognition: from single circuits to functional diversity across cortical and subcortical systems. *European Journal of Neuroscience*. doi:10.1111/ejn.12606
Review arguing that inhibition-excitation gamma generation arises from a limited set of circuit motifs that support many cognitive functions (perception, attention, memory, motivation, behavioral control) rather than a single universal one. [s]
12. **Kann, Papageorgiou & Draguhn (2014)**. Highly Energized Inhibitory Interneurons are a Central Element for Information Processing in Cortical Networks. *Journal of Cerebral Blood Flow & Metabolism*. doi:10.1038/jcbfm.2014.104
Reviews how energetically specialized fast-spiking PV interneurons provide the 'clockwork' rhythmic inhibition of the E-I loop that generates gamma as a central element for cortical information processing. [s]
13. **Womelsdorf et al. (2014)**. Dynamic circuit motifs underlying rhythmic gain control, gating and integration. *Nature Neuroscience*. doi:10.1038/nn.3764
Review casting recurrent E-I interneuron circuit motifs as the substrate for rhythmic gain control, gating and temporal integration, linking the PING mechanism to multiple routing/gating functions. [m]
14. **Ray & Maunsell (2015)**. Do gamma oscillations play a role in cerebral cortex?. *Trends in Cognitive Sciences*. doi:10.1016/j.tics.2014.12.002
Critically reviews whether E-I-generated gamma actually supports functions such as inter-areal communication and phase coding, weighing the mechanism-to-function link across attention, memory and routing accounts. [m]
15. **Pittman-Polletta et al. (2015)**. Brain Rhythms Connect Impaired Inhibition to Altered Cognition in Schizophrenia. *Biological Psychiatry*. doi:10.1016/j.biopsych.2015.02.005
Review theorizing that fast-spiking-interneuron dysfunction degrades PING-generated gamma, and that this altered temporal structure links impaired inhibition to the cognitive/perceptual deficits of schizophrenia across multiple functions. [c]
16. **Sohal (2016)**. How Close Are We to Understanding What (if Anything) γ Oscillations Do in Cortical Circuits?. *The Journal of Neuroscience*. doi:10.1523/

jneurosci.0990-16.2016

Sohal reviews the many candidate mechanisms by which E-I gamma oscillations could modulate input responses, activity patterns and output efficacy, arguing about how the gamma mechanism links to cortical function across several proposed roles. [m]

17. **Han, Shapley & Xing (2022)**. Gamma rhythms in the visual cortex: functions and mechanisms. *Cognitive Neurodynamics*. doi:10.1007/s11571-021-09767-x
    A review of gamma in visual cortex weighing candidate functions against E-I and thalamic generating mechanisms, spanning attention, encoding, and communication accounts of the rhythm. [c]
18. **Sohal (2022)**. Transforming Discoveries About Cortical Microcircuits and Gamma Oscillations Into New Treatments for Cognitive Deficits in Schizophrenia. *American Journal of Psychiatry*. doi:10.1176/appi.ajp.20220147
    Sohal's overview argues that PV-interneuron-generated cortical gamma, deficient in schizophrenia, causally supports cognition, citing mouse optogenetic studies where disrupting or enhancing synchronized gamma reproduces or ameliorates cognitive deficits. [m]

Appendix: adjacent critiques (not corpus members)

These are the major skeptical papers on gamma/synchrony function. They are *not* in the frozen corpus and are excluded from every count and recall figure above: each questions a functional claim without naming the E–I/PING loop as the generator, so it fails the mechanism-as-vehicle test. They are recorded here for the later review. The disposition tag marks whether the build surfaced and rejected the paper, or never reached it.

1. **Shadlen & Movshon (1999)**. Synchrony Unbound: A Critical Evaluation of the Temporal Binding Hypothesis. *Neuron*. doi:10.1016/s0896-6273(00)80822-3 (surfaced, judged out)
2. **Thiele & Stoner (2003)**. Neuronal synchrony does not correlate with motion coherence in cortical area MT. *Nature*. doi:10.1038/nature01285 (not surfaced)
3. **Palanca & DeAngelis (2005)**. Does Neuronal Synchrony Underlie Visual Feature Grouping?. *Neuron*. doi:10.1016/j.neuron.2005.03.002 (not surfaced)
4. **Ray & Maunsell (2010)**. Differences in Gamma Frequencies across Visual Cortex Restrict Their Possible Use in Computation. *Neuron*. doi:10.1016/j.neuron.2010.08.004 (surfaced, judged out)
5. **Merker (2013)**. Cortical gamma oscillations: the functional key is activation, not cognition. *Neuroscience & Biobehavioral Reviews*. doi:10.1016/j.neubiorev.2013.01.013 (not surfaced)
6. **Ray & Maunsell (2015)**. Do gamma oscillations play a role in cerebral cortex?. *Trends in Cognitive Sciences*. doi:10.1016/j.tics.2014.12.002 (surfaced, judged out)