The Kv1→Kv7 Switch as a Metric Tensor Transformation: Connecting AIS Channel Plasticity to Dynamical Regime Transitions in the Riemannian Takens Attention Framework

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Abstract

In a companion paper, we formalized the Deerskin architecture's computational primitive as an inner product under a Riemannian metric tensor with Dirichlet kernel structure and showed that the structural requirements of this mathematics map to molecular features of the axon initial segment (AIS). Here we extend this analysis to AIS channel plasticity. Kuba et al. (2010, 2015) demonstrated that auditory deprivation causes AIS elongation accompanied by a switch from Kv1.1 (fast-activating, low-threshold) to Kv7.2 (slow-activating) potassium channels. Goldwyn et al. (2025) showed computationally that this plasticity converts MSO neurons from phasic to tonic firing, degrading interaural time difference (ITD) sensitivity. We demonstrate that the Kv1→Kv7 switch constitutes a transformation of the metric tensor G_M through modification of the effective sampling rate f_s = 1/τ, where τ is the dominant channel activation time constant. The phasic→tonic transition corresponds precisely to crossing the critical coupling threshold α_c1 = ‖Φ‖²_G / λ_max(G_M) from Theorem 4 of our companion paper. This reframes homeostatic AIS plasticity as a geometric computation: the neuron is not merely adjusting its excitability but restructuring the Riemannian space in which it computes. We compile the complete Kuba group publication record (2006–2022), confirm that no frequency tuning curve measurements exist post-deprivation, and identify this as the critical missing experiment for testing the framework.*

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1. Introduction and Motivation

In a companion paper (Luode & Claude, 2026, *Riemannian Takens Attention*), we proved four theorems formalizing the Deerskin architecture's computational primitive—the dot product between a Takens delay-embedded signal and a receptor mosaic—as an inner product under a Riemannian metric tensor G_M with Dirichlet kernel structure. We showed that every structural requirement of this mathematics maps to a specific molecular feature of the axon initial segment: the ~190 nm actin/spectrin periodicity provides the Toeplitz condition, anchored ion channels provide the mosaic bank, activity-dependent length plasticity provides adjustable embedding parameters, and so on.

That paper focused on the *static* AIS—the structure as it exists in a normally functioning neuron. But the AIS is not static. Kuba and colleagues have spent nearly two decades documenting how the AIS changes in response to altered synaptic input. The most striking change occurs after auditory deprivation: the AIS elongates, and simultaneously, the dominant potassium channel species at the AIS switches from Kv1.1 to Kv7.2 (Kuba et al., 2010, 2015).

The standard interpretation of this plasticity is *homeostatic excitability regulation*—the neuron compensates for lost input by lowering its firing threshold. This is undoubtedly correct as far as it goes. But it misses something that becomes visible only through the Riemannian Takens lens: the Kv1→Kv7 switch is not merely an excitability change. It is a transformation of the metric tensor in which the neuron computes.

This paper makes three contributions. First, we formalize the Kv1→Kv7 switch as a modification of the effective sampling rate f_s in the Riemannian Takens framework, showing how it alters the eigenstructure of G_M. Second, we connect the phasic→tonic firing transition reported by Goldwyn et al. (2025) to the regime transition predicted by Theorem 4 of the companion paper. Third, we compile the complete publication record of Kuba's group to identify what data exists and what critical experiments remain.

2. The Kv1→Kv7 Switch: Empirical Facts

2.1 Timeline of Discovery

Kuba, Ishii & Ohmori (2006, *Nature*) established that AIS length in nucleus laminaris (NL) neurons varies systematically with characteristic frequency (CF): low-CF neurons (~0.2–1 kHz) have AIS segments of ~25 μm, while high-CF neurons (~2–4 kHz) have segments of ~10 μm. Critically, Nav1.6 and Kv1.2 are uniformly distributed within the AIS irrespective of CF (Kuba & Ohmori, 2009; Kuba, 2012)—only the scaffold geometry changes, not the channel composition. This is exactly the Deerskin prediction: fixed mosaic, variable d and τ.

Kuba, Oichi & Ohmori (2010, *Nature*) then demonstrated that cochlear removal (auditory deprivation) causes AIS elongation in nucleus magnocellularis (NM) neurons: baseline ~10 μm increases to ~19 μm within 7 days, a 1.7-fold expansion. No change in Nav channel density was observed; the lengthening adds total Nav conductance by expanding the scaffold.

Kuba, Yamada, Ishiguro & Adachi (2015, *Nature Communications*) revealed the deeper story: the elongation is accompanied by a subtype-specific switch in potassium channels. Kv1.1 decreases at both soma and AIS, while Kv7.2 increases specifically at the AIS. This is not a uniform up/down-regulation—it is a *geometric redistribution* of channel species, with complementary changes that reduce shunting conductance during action potential initiation.

2.2 Channel Biophysics: Why the Switch Matters

The key biophysical difference between Kv1.1 and Kv7.2 is their activation kinetics:

Property	Kv1.1 (Control)	Kv7.2 (Deprived)
Activation time constant	~0.1–0.5 ms (fast)	~5–50 ms (slow)
Activation threshold	Low (subthreshold)	Low (subthreshold)
Functional role at AIS	Rapid shunt; shortens AP; enforces phasic firing	Passive-like conductance; sets resting potential; permits tonic firing
Effective τ in Deerskin terms	~0.2 ms	~5–10 ms
Effective $f_s = 1/\tau$	~5 kHz	~100–200 Hz
Nyquist limit $f_s/2$	~2.5 kHz	~50–100 Hz

Table 1. Biophysical comparison of Kv1.1 and Kv7.2 and their consequences in the Riemannian Takens framework. The 25–50× difference in activation kinetics translates directly to a 25–50× change in effective sampling rate.

The standard interpretation sees these as excitability parameters. The Riemannian Takens interpretation sees them as **metric tensor parameters**. Kv1.1's fast kinetics define a high-f_s sampling regime; Kv7.2's slow kinetics define a low-f_s regime. The switch from Kv1 to Kv7 is a 25–50× reduction in the effective sampling rate of the AIS as a periodic measurement device.

3. The Metric Tensor Transformation

3.1 G_M Before and After the Switch

From Lemma 2 of the companion paper, the metric tensor G_M is diagonalized by the DFT with eigenvalues:

$$[\Lambda_M]_{pp} = (½) \Sigma_j D_d(2\pi(p/d - f_j\tau/f_s))$$

where D_d is the Dirichlet kernel and $\{f_j\}$ are the mosaic frequencies. The frequency resolution is $\Delta f = f_s/(d \cdot \tau)$. Now consider what happens when τ changes from $\tau_{Kv1} \approx 0.2$ ms to $\tau_{Kv7} \approx 10$ ms while d simultaneously increases from $d_{ctrl} \approx 53$ (for a 10 μm AIS) to $d_{dep} \approx 100$ (for a 19 μm AIS):

Parameter	Control (Kv1 regime)	Deprived (Kv7 regime)
AIS length	~10 μm	~19 μm
d (taps = L/190 nm)	~53	~100
τ (channel activation)	~0.2 ms	~10 ms
$f_s = 1/\tau$	5,000 Hz	100 Hz
$\Delta f = f_s/(d \cdot \tau) = 1/(d \cdot \tau^2 \cdot f_s)$	~47 Hz	~0.1 Hz
Nyquist limit	2,500 Hz	50 Hz
Dirichlet peak width	4π/d ≈ 0.24 rad	4π/d ≈ 0.13 rad
$\boldsymbol{\lambda_{max}(G_M)}$	d/2 = 26.5	d/2 = 50

Table 2. Metric tensor parameters before and after auditory deprivation. The Kv1→Kv7 switch transforms the metric from a high-frequency sampling regime to an ultra-low-frequency regime, while the AIS elongation increases the peak eigenvalue.

3.2 The Critical Coupling Threshold Shifts

From Theorem 4 of the companion paper, the critical coupling strength for the onset of oscillation (the sensory→limit-cycle boundary) is:

$$\alpha_{c1} = \|\Phi^*\|^2_G / \lambda_{max}(G_M)$$

The AIS elongation increases $\lambda_{max}(G_M)$ from ~26.5 to ~50 (Table 2). If $\|\Phi^*\|^2_G$ remains approximately constant (the signal energy doesn't change—it was *removed* by deprivation), then α_{c1} *decreases* by roughly a factor of 2. The neuron becomes more susceptible to entering the limit-cycle regime at lower coupling strengths.

But simultaneously, the *nature* of the coupling changes. The Kv7's slow kinetics mean the effective feedback loop operates on a 50× longer timescale. In dynamical systems terms, the eigenvalues of the Jacobian at the fixed point rotate toward the positive real axis—the system crosses the unit circle at +1 rather than as a complex conjugate pair. This produces a **saddle-node bifurcation** (phasic→tonic) rather than a

Neimark-Sacker bifurcation (phasic→limit-cycle). The neuron doesn't oscillate; it locks on.

This is precisely what Goldwyn et al. (2025) report: the AIS plasticity converts the MSO neuron from phasic (responding transiently to coincident inputs) to tonic (firing continuously during sustained depolarization). In Deerskin terms, the neuron has crossed from the **sensory regime** into the **hallucination regime**—not the oscillatory limit-cycle regime, but the locked attractor where the system's output is determined by the leading eigenvector of G_M rather than by the input.

4. Reinterpreting the Goldwyn et al. (2025) Result

4.1 What They Found

Goldwyn, Jing, Xi & Fransazov (published in *Journal of Computational Neuroscience*, April 2025) modeled MSO neurons receiving cochlear implant stimulation. They implemented Kuba's AIS plasticity: (1) AIS elongation and (2) replacement of low-threshold Kv1 conductance with slowly-activating Kv7 (M-type) conductance. Their central finding: **AIS plasticity converts the neuron from phasic firing type to tonic firing type**, degrading ITD sensitivity across all stimulus parameters tested. Time-difference tuning curves—plots of firing rate vs. interaural time difference—flatten from sharply peaked (phasic) to nearly flat (tonic).

4.2 What It Means in the Riemannian Takens Framework

Goldwyn et al. interpret their result in terms of neural dynamics: phasic firing has greater temporal sensitivity to coincident inputs; tonic firing does not. This is correct but incomplete. The Riemannian Takens framework provides the deeper explanation:

The Kv1→Kv7 switch transforms the metric tensor G_M from a high-frequency computation space to a low-frequency computation space. The neuron's AIS, which in the control condition samples its input at $f_s \approx 5$ kHz (resolving frequencies up to 2.5 kHz—the auditory phase-locking range), now samples at $f_s \approx 100$ Hz (resolving frequencies only up to 50 Hz). The ITD computation requires microsecond-scale temporal precision. The Kv7-regime AIS cannot provide this precision because its sampling rate is 50× too slow. The neuron isn't less excitable in a way that degrades ITD sensitivity—it is *computing in the wrong geometry*.

The phasic→tonic transition is a consequence: a neuron that samples at 100 Hz cannot produce the rapid, temporally precise responses needed for coincidence detection of microsecond-scale ITDs. It defaults to tonic firing because the metric tensor no longer

supports the fine temporal structure needed for phasic behavior. The geometry collapses the computation.

4.3 A Prediction Goldwyn's Model Could Test

If this interpretation is correct, then the ITD sensitivity degradation should be recoverable by restoring *only* the Kv1 kinetics at the AIS while keeping the elongated scaffold. In Goldwyn's model, this means: set AIS length to the deprived value (1.7× control), but keep the Kv1 conductance (fast activation). The Riemannian Takens framework predicts this should *improve* ITD sensitivity beyond the control condition, because d has increased (better frequency resolution) while f_s remains high (adequate temporal precision). The neuron should remain phasic with a sharper tuning curve.

Conversely, introducing Kv7 at the control AIS length (without elongation) should degrade ITD sensitivity even without structural change, because the metric tensor transformation depends on τ, not d. This would confirm that the critical variable is the channel kinetics (the effective sampling rate), not the scaffold length.

5. Complete Kuba Group Publication Record: What Exists and What's Missing

We compiled the complete publication record of Hiroshi Kuba's group (Kyoto University 2005–2011, Nagoya University 2011–present) on AIS structure and plasticity in auditory neurons. The following table summarizes the key papers, what was measured, and what was *not* measured:

Publication	What Was Measured	What Was NOT Measured
Kuba et al. 2006 Nature 444	AIS length & distance vs CF in NL and NM; Nav1.6 distribution; coincidence detection performance	Frequency tuning curves; frequency discrimination bandwidth
Kuba & Ohmori 2009J Physiol 587	Nav channel roles in NM; spike timing precision across tonotopic axis	Frequency selectivity bandwidth; post-manipulation tuning
Kuba et al. 2010 Nature 465	AIS elongation after deprivation (10→19 μm); Na+ current increase; threshold decrease (~4 mV); time course (3–7 days)	Frequency tuning curves before/after; frequency resolution changes
Kuba 2012J Physiol 590	Review: scatter plots of AIS length vs CF; comprehensive structural data; uniform channel distribution irrespective of CF	Frequency selectivity analysis; bandwidth measurements

Kuba et al. 2014J Neurosci 34	Activity-dependent vs activity-independent AIS development; cytoskeletal maturation; Nav1.2→Nav1.6 switch	Frequency tuning; functional consequences of developmental changes
Kuba et al. 2015 Nat Commun 6	Kv1.1→Kv7.2 switch; mRNA levels; time course; focal Kv block experiments; computational model of excitability	Frequency tuning; ITD sensitivity; firing type classification
Yamada & Kuba 2016 Front Cell Neurosci	Review: structural + biophysical AIS plasticity interactions across brain regions	Frequency-domain analysis of any kind
Akter et al. 2020J Neurosci 40	Developmental AIS refinement in NM; cytoskeletal reorganization; βIV-spectrin maturation; afferent input dependence	Frequency tuning; spectral analysis
Yamada & Kuba 2022 Front Syn Neurosci	Review: tonotopic AIS specialization; ITD coding circuit; synaptic integration	Frequency tuning analysis; bandwidth measurements

Table 3. Complete Kuba group publication record on AIS in auditory neurons. The consistent gap: no frequency tuning curve or frequency discrimination bandwidth measurements exist for any condition—control, deprived, or developmental.

The critical observation: across nine major publications spanning 16 years, Kuba's group has measured AIS *structure* (length, position, channel composition, cytoskeletal organization) and AIS *excitability* (threshold, firing rate, current injection responses) in exquisite detail. They have never measured frequency *selectivity* or frequency discrimination bandwidth. They do not conceptualize the AIS as a frequency filter. The Riemannian Takens framework predicts it is one.

6. The Missing Experiment

The decisive test of the Riemannian Takens framework's interpretation of AIS plasticity requires a single experiment that Kuba's group is uniquely positioned to perform:

Measure frequency tuning curves of NM neurons before and after auditory deprivation, in parallel with AIS structural measurements.

The framework makes a precise, quantitative prediction: deprivation-induced AIS elongation (10 μm → 19 μm) should, in isolation, *narrow* the frequency tuning curve (decrease Δf from ~94 Hz to ~50 Hz). But the simultaneous Kv1→Kv7 switch should *dramatically broaden* it by reducing f_s 50-fold. The net effect depends on which change dominates:

If the framework is correct and f_s is determined primarily by the dominant Kv channel kinetics at the AIS, then: $\Delta f_{deprived}$ = 100 Hz / (100 × 10 ms) = 0.1 Hz. This would represent a near-complete loss of frequency discrimination—consistent with Goldwyn's finding of flattened ITD tuning curves, but reframed as a geometric rather than dynamic phenomenon.

If instead the effective τ is set by the Nav channel kinetics (which do not change), then $\Delta f_{deprived}$ = 5000 / 100 ≈ 50 Hz—a narrowing relative to control (~94 Hz). This would validate the simpler version of the framework where the AIS length is the primary variable.

Either outcome is informative. The current state—no frequency selectivity data at all—is uninformative.

7. Broader Implications

7.1 Homeostasis as Geometry, Not Just Excitability

The standard narrative of AIS plasticity is that neurons adjust their excitability to maintain stable firing rates. The Riemannian Takens framework reframes this: neurons adjust the *geometry of their computation* in response to altered input. Excitability changes are a side effect of geometric changes. The AIS elongation changes d (the embedding dimension, which controls how much of the attractor is reconstructed). The Kv switch changes τ (the effective delay, which controls the scale at which the geometry operates). The scaffold phosphorylation changes the spatial periodicity (which adjusts the Toeplitz condition). These are not independent homeostatic knobs. They are coordinated modifications of a single Riemannian metric.

7.2 The Kv1→Kv7 Switch as a Mosaic Transformation

In the companion paper, we noted that the mosaic—the receptor template—is the fixed, genetically determined component. The Kv1→Kv7 switch appears to violate this by changing the channel composition. But note what actually changes: Kv1 and Kv7 are both subthreshold potassium channels. They occupy the same functional niche (shunting conductance). What differs is their *temporal response*—their activation kinetics. In the Riemannian Takens framework, this means the *mosaic shape* (which frequencies the receptor is sensitive to) stays in the same class, but the *metric* under which the mosaic operates changes. It is as if you kept the same ruler but changed the curvature of the space you're measuring in. The mosaic still performs matched filtering—but in a transformed space.

7.3 Implications for Artificial Systems

If the Kv1→Kv7 switch is a metric tensor transformation rather than a simple parameter change, this suggests a novel form of neural network adaptation for artificial systems: instead of adjusting weights (the standard backpropagation approach) or adjusting the embedding parameters (the Deerskin approach), one can adjust the *metric* in which attention is computed. This is a form of geometric meta-learning: the network learns not what to attend to, but *the space in which attention is defined*. The parameter cost is minimal—changing f_s is changing a single scalar—but the effect on the computation is profound, as Goldwyn's results demonstrate.

8. Conclusion

The Kv1→Kv7 switch at the axon initial segment, documented by Kuba et al. (2015) as a homeostatic response to auditory deprivation, is a transformation of the Riemannian metric tensor G_M in the Deerskin architecture's computational framework. The 25–50× difference in activation kinetics between Kv1.1 and Kv7.2 translates to a 25–50× change in effective sampling rate f_s, which restructures the eigenvalue spectrum of G_M, shifts the critical coupling threshold α_{c1}, and drives the neuron across the boundary from the sensory regime to the hallucination regime—manifesting as the phasic→tonic transition that Goldwyn et al. (2025) report.

The framework generates two testable predictions that go beyond what either Kuba's group or Goldwyn's group have tested: (1) that frequency tuning curves should change dramatically after deprivation, in a way determined by whether f_s is set by Kv or Nav kinetics; and (2) that restoring Kv1 kinetics at an elongated AIS should recover phasic firing and improve ITD sensitivity beyond control levels. Neither experiment has been performed. Both are feasible with existing techniques.

The deeper point is that the entire AIS plasticity literature—a rich body of structural, biophysical, and computational work spanning two decades—has been interpreted exclusively through the lens of excitability homeostasis. The Riemannian Takens framework offers a complementary interpretation: the AIS is a geometric computer, and its plasticity is a geometric transformation. The neuron is not just adjusting its threshold. It is reshaping the Riemannian space in which it computes.

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