The Gravity of Maturation: Synthesizing Brain Criticality and Gastric Spacetime Dynamics in the Developmental Transition to Adulthood

Nicholas P. Timms
Submitted: April 2026 : Publish: 3rd April 2026

Abstract

The developmental transition from adolescence to adulthood entails profound shifts in neurocomputational dynamics, specifically the progression of the cerebral cortex toward a state of self-organized criticality. Concurrently, peripheral autonomic systems, particularly the gastrointestinal tract, undergo functional maturation. This paper presents a novel theoretical synthesis bridging the emergence of frequency-specific cortical criticality with an analogue gravity model of gastric electrophysiology. Within this framework, the prefrontal cortex achieves an optimal excitatory-inhibitory balance during adulthood, enabling high-fidelity, top-down vagal modulation. This matured vagal tone acts as a dynamic homeostatic tuner for the stomach wall, which functions as an effective biological spacetime metric propagating the gastric slow wave. During the supercritical instability of adolescence, or in instances of developmental pathology, erratic vagal signals degrade this acoustic metric. This degradation prompts the localized collapse of wave propagation into pathological analogue event horizons, clinically manifesting as conduction blocks and severe dysmotility such as gastroparesis. Furthermore, this model elucidates how these visceral spacetime collapses transmit afferent distress signals that abnormally hyper-couple with frontoparietal networks, driving profound psychiatric distress. Finally, it highlights vagus nerve stimulation as a bidirectional electroceutical intervention capable of simultaneously restoring neural criticality and repairing the gastric metric topology. Ultimately, this criticality-spacetime control model redefines neuro-visceral maturation as the biological calibration of a unified, complex gravitational system.

Introduction

The transition from adolescence to adulthood constitutes one of the most dynamic and consequential epochs in human physiological development. Historically, the maturation of the central nervous system has been understood through the lens of structural biology, focusing heavily on the macroscopic pruning of synaptic connections, the arborization of dendrites, and the progressive myelination of white matter tracts across the cortex. While these structural paradigms provide foundational insights into neurodevelopment, they often fall short of explaining the profound computational and dynamic shifts that characterize the emergence of adult cognition and autonomic regulation. Recent advancements in statistical mechanics, non-linear dynamics, and high-resolution network electrophysiology have necessitated a paradigm shift. Emerging theoretical frameworks propose that the maturing brain systematically shifts toward a state of self-organized criticality—a precise phase transition boundary characterized by highly specific adjustments in the balance between excitatory and inhibitory (E/I) neurotransmission across distinct oscillatory frequency bands.[1]

Simultaneously, peripheral organ systems—most notably the gastrointestinal tract—undergo parallel and heavily integrated functional maturations. The intrinsic electrophysiological dynamics of the stomach, paced primarily by the 0.05 Hz gastric slow wave, do not operate in a vacuum; rather, they are subjected to continuous, dynamic modulation by the central nervous system via the complex neural architecture of the gut-brain axis (GBA).[1] Recent theoretical advancements in the field of biological physics have proposed a radical reframing of this gastric electrophysiological network as an analogue gravity system. In this model, the physical properties of the stomach wall function as an "effective spacetime metric," and the bioelectrical gastric slow wave propagates through this medium as a massless scalar field.[1] Within this relativistic analogy, the variable, location-dependent propagation speed of the slow wave, denoted as $c(x)$, determines the curvature of this biological spacetime. Pathological disruptions to this propagation manifest as "analogue event horizons"—conduction blocks where $c(x)$ effectively drops to zero, trapping the neuro-electrical pulse and resulting in severe, debilitating motility disorders such as gastroparesis.[1]

This report presents an exhaustive, novel synthesis of these two highly advanced theoretical frameworks. By systematically cross-referencing the developmental shifts in frequency-specific brain criticality [1] with the analogue gravity model of the gastric slow wave [1], a comprehensive, updated model of neuro-visceral maturation is formulated. The central thesis of this analysis posits that the age-related shift toward neural criticality in the cerebral cortex provides the requisite computational stability, signal-to-noise ratio, and dynamic range for the prefrontal cortex to exert precise, top-down inhibitory control over vagal efferents. This matured vagal tone acts as the primary homeostatic "tuner" of the gastric effective spacetime metric, ensuring the seamless propagation of gastric slow waves and preventing the formation of pathological event horizons. The developmental trajectory from adolescence to adulthood can thus be understood not merely as the strengthening of neural circuits, but as the biological calibration of a vast, integrated control system, where cortical criticality dictates visceral spacetime topology.

The Computational Cortex: Ontogeny of E/I Balance and Neural Criticality

The Phase Transition of the Mind

To grasp the implications of neurodevelopment on autonomic output, one must first understand the operational regime of the resting human brain. At rest, the healthy adult brain operates at the boundary between two distinct dynamical regimes, existing in a state of marginal stability known in statistical physics as a critical phase transition.[1] On one side of this theoretical boundary lies an inhibition-dominant, subcritical state characterized by rapidly decaying signal propagation, leading to non-responsiveness and computational stagnation. On the opposite side lies an excitation-dominant, supercritical state prone to chaotic, runaway neural activity and uncoordinated signal integration.[1]

When neural systems are perfectly balanced at this critical point, they exhibit emergent properties that are mathematically optimal for computation. These properties include maximized dynamic range, optimal information transmission capacity, scale-free network activity, and profound susceptibility.[1] Susceptibility, in this context, refers to the propensity of the neural system to reorganize and respond to subtle, complex inputs without destabilizing—a feature absolutely essential for driving associative plasticity and maintaining homeostatic control over peripheral organs.[1]

Extensive longitudinal electroencephalography (EEG) data tracking 169 healthy human individuals across 310 sessions from age 10 to 33 reveals that the brain systematically shifts toward this critical point across developmental time.[1] This ontogenetic shift is quantified through two primary emergent markers of complex dynamical systems: Long-Range Temporal Correlations (LRTC) and amplitude bistability (BiS).[1] LRTCs are evaluated using Detrended Fluctuation Analysis (DFA), which measures the persistence of amplitude fluctuations over time; a DFA exponent closer to 1.0 indicates a system operating near criticality.[1] Amplitude bistability quantifies the capacity of a neural population to spontaneously vacillate between high- and low-amplitude states without altering external control parameters, mathematically modeled by the superior fit of a bi-exponential function over a single exponential function.[1] The developmental trajectory demonstrates spectrally-widespread increases in both DFA exponents and BiS scores, confirming unequivocally that the mature adult brain operates closer to a state of self-organized criticality than the adolescent brain.[1]

Frequency-Specific Divergence in Excitation and Inhibition

While the global, systemic shift of the brain is toward criticality, the underlying mechanisms driving this shift are highly frequency-specific, reflecting diverse local E/I reconfigurations across different canonical frequency bands. The maturation of the cerebral cortex involves the aggressive pruning of excitatory glutamatergic synapses and the potentiation of inhibitory signaling, primarily mediated by fast-spiking, parvalbumin-positive (PV+) GABAergic interneurons, particularly within higher-order association cortices.[1] This developmental transition is physically supported by the formation of perineuronal nets (PNNs)—specialized extracellular matrix structures that encapsulate mature PV+ interneurons, stabilizing structural synapses, closing critical windows of neuroplasticity, and strictly regulating the synaptic E/I balance.[9]

The developmental effects on E/I ratios manifest divergently depending on the specific oscillatory frequency band analyzed, reflecting the complex, multiplexed nature of cortical communication.

In low-frequency systems, encompassing the theta (θ, 4.0–8.3 Hz), alpha (α, 8.3–13.4 Hz), and beta (β, 13.4–27.6 Hz) bands, the neural mechanisms experience a net decrease in their functional E/I ratio (fE/I).[1] Despite this pronounced decrease in excitation relative to inhibition, the emergent markers of criticality—specifically DFA and BiS—grow significantly stronger with age.[1] For instance, in the α band, mixed-effects regressions reveal that DFA exponents increase robustly with age ($\beta = .351$, $p = 10^{-8}$), alongside corresponding increases in BiS scores ($\beta = .251$, $p = 10^{-5}$).[1] This paradoxical relationship implies that during adolescence, these low-frequency systems operate in a slightly supercritical, excitation-dominant state. The developmental up-regulation of GABAergic inhibition effectively reins in this runaway excitation, bringing these widespread oscillatory networks down to the critical boundary.[1]

Conversely, the neural systems driving high-frequency gamma (γ) oscillations (27.6–57.6 Hz) exhibit an entirely different developmental trajectory. These systems show a marked *increase* in the E/I ratio alongside a highly spatially selective *decrease* in emergent critical dynamics.[1] In the γ band, DFA exponents significantly decrease with age ($\beta = -.197$, $p = .00176$), as do BiS scores ($\beta = -.163$, $p = .00981$).[1] Because γ rhythms are heavily dependent on the extremely fast time constants of GABA-A receptor-mediated inhibition, a relative localized increase in excitability—combined with robust, rapidly alternating inhibitory feedback—results in faster-growing neuronal avalanches.[1] This is empirically reflected by higher global branching ratios (σ) in adults, indicating that high-amplitude bursts of neural activity propagate more readily.[1] However, the dampening of long-range temporal correlations in the γ band is likely not a sign of network failure, but rather the result of cross-frequency suppression. The increasingly stable, powerful low-frequency oscillations (α and θ) exert top-down inhibitory control over high-frequency γ bursts, truncating their long-range temporal persistence in order to route information precisely and suppress endogenous neural noise.[1]

Oscillatory Band	**Frequency Range (Hz)**	**Age-Related Shift in E/I Ratio**	**Shift in Criticality Markers (DFA & BiS)**	**Inferred Developmental Trajectory**
Theta (θ)	4.0 - 8.3	Decrease	Increase	Supercritical $\rightarrow$ Near-Critical
Alpha (α)	8.3 - 13.4	Decrease	Increase	Supercritical $\rightarrow$ Near-Critical
Beta (β)	13.4 - 27.6	Decrease	Increase	Supercritical $\rightarrow$ Near-Critical
Gamma (γ)	27.6 - 57.6	Increase	Decrease	Subcritical $\rightarrow$ Near-Critical (with blunted emergent dynamics via cross-frequency coupling)

Table 1: Frequency-specific developmental trajectories of E/I balance and neural criticality from adolescence to adulthood based on longitudinal EEG dynamics.[1]

Artificial Neural Network Simulations of Maturation

The validity of these developmental E/I shifts is strongly supported by computational simulations utilizing the CRitical Oscillations (CROS) model. The CROS model consists of a highly coupled 50×50 network of recurrent excitatory and inhibitory artificial neurons, specifically structured with 75% excitatory and 25% inhibitory units.[1] By manipulating the local connection density between these units, researchers can map the state space of possible neural dynamics.

When the connection densities of excitation and inhibition are perfectly balanced, the artificial neural network undergoes a critical phase transition, spontaneously generating strong long-range temporal correlations, power-law distributed neuronal avalanches, and bistable amplitude distributions—perfectly mirroring the empirical EEG data.[1] By tracking the High-to-Low Power Separation Index ($E + I_{HLS}$) and the High-to-Low Amplitude Proportion (E/I_{HLP}), researchers have plotted the feasible developmental trajectories from age 10 to 35 across this state space. The simulations confirm that the empirical developmental changes observed in spontaneous resting EEG dynamics can be wholly recapitulated by localized physiological changes in excitatory and inhibitory synaptic connection densities.[1] This computational evidence solidifies the premise that the physical architecture of the brain is remodeling itself specifically to achieve a critical operational state.

State-Dependent Adaptability: The Eyes-Closed to Eyes-Open Transition

The true computational advantage of achieving neural criticality lies in the brain's enhanced susceptibility—the capacity of the system to rapidly reorganize its internal state in response to environmental perturbations without cascading into chaotic failure.[1] This enhanced dynamic range is empirically demonstrable through state transitions, such as the simple act of opening the eyes.

When visual input floods the nervous system, the brain undergoes a spectrally-widespread shift toward lower functional E/I (subcriticality), marked by massive increases in both feedforward and feedback inhibition.[1] This physiological response is designed to stabilize sensory processing, prevent runaway excitation from the sudden influx of photons, and filter environmental noise.[1]

Crucially, this state-dependent shift is significantly more pronounced in adults than in adolescents, highlighting a massive developmental leap in cognitive control.[1] In the dominant α band, opening the eyes drastically reduces DFA ($\beta = -.467$, $p = 10^{-14}$) and BiS ($\beta = -.179$, $p = .00455$) in adults, deliberately dragging the system into an inhibition-dominant, subcritical regime to process visual information safely.[1] Adults exhibit a profound Age $\times$ Condition interaction for DFA in the α band ($\beta = -.302$, $p = 10^{-7}$), underscoring the mature brain's superior capacity to dynamically and aggressively tune its E/I parameters to meet immediate cognitive and sensory demands.[1] Conversely, in the γ band, opening the eyes strengthens DFA statistics ($\beta = .147$, $p = .0180$), indicating that while low-frequency global coordinators are suppressed, local high-frequency processing networks are unblocked to handle high-resolution visual perception.[1] This enhanced, state-dependent dynamic range in adults serves as the foundational computational mechanism by which the central nervous system maintains rigid, homeostatic control over complex peripheral systems, including the viscera.

The Acoustic Spacetime of the Gastrointestinal Tract

To fully comprehend how the maturing, critical brain interfaces with the peripheral body, it is strictly necessary to establish the physical realities of gastric electrophysiology. The human stomach is not merely a mechanical pouch; it is a highly complex, autonomous bioelectrical organ governed by a basal electrical rhythm known as the gastric slow wave.[1] Operating at an exceptionally low frequency of approximately 0.05 Hz (roughly one cycle every 20 seconds), these electrical waves are spontaneously generated by specialized pacemaker cells known as the Interstitial Cells of Cajal (ICC), which are embedded within the smooth muscle layers of the gastric wall.[1] The gastric slow wave dictates the precise timing, propagation velocity, and contractile force of gastric peristalsis, orchestrating the mechanical grinding and emptying of nutrients.[1]

The Effective Spacetime Metric

Advanced mathematical models, specifically those formulated in the seminal work by Allegra et al., describe the complex propagation of this bioelectrical activity using a one-dimensional wave equation.[1] When this mathematical construct is conceptually mapped onto the principles of analogue gravity—a field initially conceived by physicist Bill Unruh in 1981 to study black hole thermodynamics by substituting curved spacetime with moving fluid media—the stomach reveals itself to be a literal biological analogue spacetime.[1]

The governing foundational equation for the stomach's electrical activity is defined as:

$$\frac{\partial^2 u}{\partial t^2} = c(x)^2 \frac{\partial^2 u}{\partial x^2}$$

Where:

- $u(x, t)$ represents the amplitude of the gastric slow wave, modeled as a "Gaussian pulse," at a specific spatial location x and given time t.[1]
- $c(x)$ represents the highly critical, location-dependent wave speed of the gastric medium.[1]

In this elegant theoretical framework, the Gaussian pulse represents a massless scalar field (akin to a photon or phonon), and the variable wave speed $c(x)$ determines the emergent acoustic metric—the localized "curvature" of the effective biological spacetime.[1] The stomach wall is fundamentally not a uniform electrical conductor. Instead, the healthy gastric medium possesses an intrinsic topology that allows the wave to propagate at differing velocities depending on the precise anatomical region.[1] Normative spatial propagation speeds extracted from high-resolution mapping data reveal a highly structured terrain: the wave originates and travels at 6.0 mm/s in Proximal Region 1, decelerates significantly to 3.0 mm/s in the transitional Proximal 2 / Distal 1 regions, and accelerates once again to 5.9 mm/s in Distal Region 2 as it approaches the pylorus.[1]

Analogue Event Horizons and Pathological Spacetime

In traditional astrophysics, an event horizon forms in a region of spacetime where the gravitational pull is so immense that the escape velocity exceeds the speed of light, trapping all matter and radiation. Translating this to the acoustic metric of the stomach, an "analogue event horizon" forms when the local electrophysiological properties of the gastric medium degrade to the point where the propagation speed $c(x)$ is pathologically reduced to zero.[1]

This phenomenon provides a mathematically rigorous model for clinical gastric dysrhythmias and conduction blocks, which are the hallmark physiological failures seen in severe motility disorders like gastroparesis.[1] When a healthy Gaussian pulse (the slow wave) propagating down the stomach encounters a conduction block, it begins to decelerate rapidly as the local value of $c(x)$ approaches zero. Ultimately, the pulse becomes "trapped" by the pathological curvature of the gastric medium, entirely unable to propagate further distally.[1] The electromechanical coupling of the stomach fails at this exact coordinate, halting peristalsis.[14]

In extreme pathological states, the effective spacetime metric becomes so violently warped that the normal anterograde flow of the wave is entirely subverted. High-resolution mapping in severely dysrhythmic patients has shown complete reversals of wave direction, with retrograde propagation velocities reaching -4.3 mm/s, compared to the normal forward velocity of $+7.4 \text{ mm/s}$ seen in healthy control subjects.[1] Understanding the formation of these biological event horizons is paramount, because the physical properties of this gastric metric—the localized values of $c(x)$—are not completely autonomous. They are highly plastic, constantly being "tuned" from a higher-order control center.

Bridging the Topologies: The Vago-Vagal Transduction Network

The analogue gravity framework dictates that the local wave speed $c(x)$, and therefore the overarching curvature of the gastric spacetime, is continuously modulated to adapt to physiological needs (e.g., fasting states versus postprandial digestion). The "central controller" of this dynamic metric is the Gut-Brain Axis (GBA), with the vagus nerve (Cranial Nerve X) serving as the primary biological transducer of this modulation.[1]

The Anatomy of Visceral Tuning

The vago-vagal reflex circuit is a marvel of evolutionary engineering, comprising both extensive afferent (sensory) fibers and highly targeted efferent (motor) fibers.[19] Visceral afferents originating in the gastric mucosa and musculature continuously transmit high-fidelity sensory information regarding stretch, tension, and chemical composition to the Nucleus Tractus Solitarius (NTS) situated in the medulla of the brainstem.[19] The NTS processes this ascending data and relays it to adjacent motor nuclei, primarily the Dorsal Motor Nucleus of the Vagus (DMV) and the Nucleus Ambiguus (NAmb).[19] From the DMV, efferent fibers project back down to the enteric nervous system, forming synapses with both excitatory and inhibitory post-ganglionic neurons embedded within the myenteric plexus to modulate the Interstitial Cells of Cajal and smooth muscle cells.[20]

However, the brainstem does not act as an isolated, autonomous relay station. The NTS and DMV receive incredibly dense, top-down modulatory projections from higher-order cortical and limbic regions, particularly the Prefrontal Cortex (PFC), anterior insula, paraventricular nucleus of the hypothalamus (PVH), and the amygdala.[24] According to the neurovisceral integration model, the PFC exerts profound inhibitory control over the amygdala and subsequent brainstem nuclei, effectively modulating the delicate balance between sympathetic fight-or-flight arousal and parasympathetic vagal tone.[26]

Maturation of the Autonomic Nervous System

The maturation of the PFC during the adolescent transition—specifically the previously detailed decrease in the E/I ratio driven by the proliferation of GABAergic inhibition—directly correlates with the maturation of cardiac and gastric vagal tone.[1] During early adolescence, the supercritical state of low-frequency cortical bands and the relative structural immaturity of PFC-amygdala connectivity result in highly volatile, easily disrupted autonomic regulation.[1] The immature brain's inability to consistently suppress excitatory bursts leads to erratic firing in the DMV, manifesting as unstable vagal efferent activity and an impaired ability to maintain a steady homeostatic baseline.[28] This instability is further compounded by the massive neuroendocrine fluctuations characteristic of puberty, including surges in the Hypothalamic-Pituitary-Gonadal (HPG) and Hypothalamic-Pituitary-Adrenal (HPA) axes, which dynamically alter the sensitivity of the gut-brain axis.[33] Furthermore, parallel developmental shifts in the composition of the gastrointestinal microbiome during adolescence uniquely interact with these neuroendocrine pathways, creating a period of extreme vulnerability in autonomic control.[33]

As the individual transitions into early adulthood, the neural networks achieve their optimal critical state. The stabilization of the E/I balance in the PFC allows for sustained, high-fidelity inhibitory signaling to the brainstem, effectively dampening extraneous noise.[1] Consequently, baseline vagal tone—often measured non-invasively via High-Frequency Heart Rate Variability (HF-HRV) or the standard deviation of N-to-N intervals (SDNN)—increases and stabilizes in adulthood.[27] High vagal tone serves as a direct proxy for enhanced prefrontal-subcortical inhibitory control, and is highly correlated with superior performance in executive cognitive tests, such as the Flanker Test and Dimensional Change Card Sort, which demand high levels of attentional inhibition and cognitive flexibility.[27]

This matured, high-fidelity vagal tone is the exact mechanism by which the adult brain "tunes" the gastric effective spacetime metric. Empirical evidence robustly demonstrates that artificial vagus nerve stimulation (VNS) significantly alters gastric slow wave propagation parameters. Acute VNS increases the rate of gastric emptying from 29.1% to 40.7% over a four-hour period, induces a significant relaxation of the pyloric sphincter (increasing the cross-sectional area of the lumen from 1.5 to 2.6 mm^2), and critically, modifies the slow wave frequency and increases the propagation velocity from 0.50 mm/s to 0.67 mm/s.[39] Thus, vagal efferent firing acts as a continuous, dynamic update to the variable $c(x)$, actively smoothing the acoustic metric and preventing the emergence of pathological event horizons.[1]

The Criticality-Spacetime Control Model: A Synthesis

By systematically synthesizing the network electrophysiological principles of brain criticality [1] with the relativistic analogue gravity model of the gastrointestinal tract [1], we can construct a unified, mathematically informed theoretical model: **The Criticality-Spacetime Control Model of Neuro-Visceral Maturation.**

The Theoretical Formulation

In this highly integrated model, the human brain functions as a complex, marginally stable computational engine whose primary output continuously regulates the topology of a distant, autonomous biological spacetime (the stomach). The precision, reliability, and adaptive capacity of this regulation are entirely dependent on the brain's proximity to a critical phase transition.[1]

Let us define a novel vagal tuning parameter, $V(t)$, as a direct function of the cortical E/I balance over time. Within this framework, the gastric wave speed $c(x,t)$ is no longer viewed solely as a static spatial constant dependent on local anatomy; rather, it is a dynamic variable continuously perturbed by descending vagal input:

$$c(x,t) = c_{intrinsic}(x) + f(V(t))$$

Where $c_{intrinsic}(x)$ represents the baseline electrical propagation speed dictated by the local cellular architecture and density of the Interstitial Cells of Cajal, and $f(V(t))$ represents the dynamic, real-time modulation provided by vagal efferent firing originating from the brainstem.[1]

For the stomach to function optimally—meaning continuous wave propagation without the formation of conduction blocks—$V(t)$ must provide a highly adaptive, noise-free, and stable modulatory signal. If $V(t)$ is highly volatile, erratic, or chronically suppressed due to central nervous system dysfunction, the effective metric $c(x,t)$ experiences drastic, localized drops. If these drops are severe enough, they lead to the formation of analogue event horizons where $c(x,t) \leq 0$.[1]

The Developmental Transition of the Control System

1. **The Adolescent Epoch (Supercritical/Unstable Cortical Dynamics):** During the adolescent developmental window, the cerebral cortex exhibits lower levels of GABAergic inhibition (reflected by high fE/I scores in the dominant α and θ bands), meaning the overarching neural system operates in a volatile, supercritical domain.[1] The distinct lack of mature critical dynamics (low DFA) mathematically implies that the system possesses a significantly lower dynamic range and lower information transmission fidelity.[1] Consequently, top-down prefrontal inhibitory control over the amygdala and the DMV is characterized by high endogenous noise and poor error-correction capabilities.[31]
   - *Effect on the Gastric Metric:* Because the central controller is noisy, the vagal tuning parameter $V(t)$ is highly inconsistent. As a direct result, the gastric effective spacetime metric experiences constant micro-fluctuations in localized curvature. The adolescent stomach is therefore inherently more prone to transient conduction blocks, uncoordinated peristaltic contractions, and stress-induced visceral discomfort, reflecting micro-event horizons that rapidly form and dissolve within the gastric medium as the autonomic nervous system struggles to maintain homeostasis.[1]
2. **The Adult Epoch (Near-Critical/Stable Cortical Dynamics):** With the successful transition to adulthood, millions of excitatory synapses have been pruned, and PV+ interneuron inhibition is vastly up-regulated (resulting in deeply decreased E/I ratios in θ, α, β bands).[1] The brain reaches a state of near-criticality, characterized by exceptionally strong long-range temporal correlations (DFA ≈ 1.0) and high amplitude bistability.[1] This operational state maximizes *susceptibility*—the ability of the neural network to integrate and respond to subtle visceral afferent feedback without tipping into

chaotic overreaction.[1]

  - *Effect on the Gastric Metric:* The fully mature PFC generates a highly precise, stable, and context-adaptive inhibitory signal to the brainstem. The vagal parameter $V(t)$ operates as a finely-tuned, high-fidelity homeostatic control variable. The GBA smoothly and continuously updates $c(x,t)$, maintaining a continuous, non-singular acoustic metric across the entirety of the stomach wall.[1] Gastric slow waves propagate seamlessly from the proximal to the distal stomach, entirely free from the localized collapse of spacetime (conduction blocks).[1]

Developmental Stage	**Neural Dynamics (PFC E/I State)**	**Vagal Tone (Control Signal)**	**Gastric Spacetime Metric (c(x))**	**Incidence of Analogue Event Horizons**
Adolescence	Supercritical (High E/I, Low DFA)	Erratic / Low fidelity	Fluctuating curvature, unstable wave propagation	High susceptibility to transient conduction blocks
Adulthood	Near-Critical (Balanced E/I, High DFA)	Stable / High dynamic range	Smooth, finely tuned acoustic metric	Low, continuous propagation perfectly maintained

Table 2: The Criticality-Spacetime Control Model detailing the maturation of neuro-visceral dynamics and their physical consequences.[1]

Phase-Amplitude Coupling and Visceral-Cortical Synchronization

The interaction between the brain's precise critical state and the stomach's emergent spacetime metric is not a unidirectional command structure; it is fundamentally bidirectional, governed by continuous, massive feedback loops.[1] The integrity and functional status of this bidirectional communication can be empirically observed through trans-organ cross-frequency coupling.

Synchronization of Cortical Oscillations and Gastric Phase

The gastric slow wave operates at an exceptionally low frequency (~0.05 Hz), making it one of the slowest continuous biological rhythms in the human body.[1] Recent landmark studies utilizing high-resolution magnetoencephalography (MEG) and functional MRI (fMRI) have demonstrated that this visceral rhythm serves as a massive basal physiological scaffold, phase-locking with spontaneous cortical oscillations across wide swaths of the brain.[13] Through a mechanism known as phase-amplitude coupling (PAC), the specific phase of the slow-moving gastric wave directly modulates the amplitude of the brain's much faster α (8–13 Hz) and θ (4–8 Hz) rhythms.[13]

This precise phase-amplitude coupling is highly state-dependent and is most pronounced during specific physiological states, such as the N3 stage of non-rapid eye movement (NREM) sleep (slow-wave sleep). During N3 sleep, the alignment between the gastric phase and cortical slow-wave oscillations increases substantially, with the von Mises concentration parameter (κ) showing an approximately four-fold increase in alignment strength ($\beta = 0.82$, $z = 11.5$, $p < 0.001$).[44] At rest, during normal wakefulness, the gastric rhythm couples with the α rhythm prominently in the parieto-occipital regions and the anterior insula networks.[45] Increased gut-brain PAC in these specific regions is strongly correlated with enhanced vigilance and faster cognitive performance speeds on tasks requiring working memory.[46]

The Role of E/I Maturation in Trans-Organ Coupling

The developmental shift in frequency-specific E/I balance documented by EEG [1] has direct, profound implications for this gut-brain coupling phenomenon. During adolescence, when the neural mechanisms driving α and θ oscillations are supercritical (excitation-dominant) and exhibit significantly weaker amplitude bistability [1], the cortex is inherently less capable of maintaining stable phase-locking with the extremely slow, subtle input ascending from the gastric vagal afferents. The excessive endogenous "noise" generated by the supercritical neural network effectively washes out the 0.05 Hz signal.

As adulthood is reached and the E/I ratio decreases, the α and θ networks enter their optimal critical regime, characterized by robust long-range temporal correlations (high DFA).[1] Critical systems exhibit maximal susceptibility to weak external perturbations.[1] Therefore, the matured, critical adult brain becomes exquisitely sensitive to the ascending phase of the gastric slow wave. The stomach's effective spacetime metric, assuming it is healthy and free of event horizons, provides a continuous, highly reliable temporal scaffold that helps pace and coordinate the adult brain's low-frequency oscillatory dynamics, optimizing cognitive vigilance.[1]

State-Dependent Disruptions: Visual Perturbation

The application of acute visual stimuli (transitioning from an eyes-closed to an eyes-open state) demonstrates exactly how top-down cognitive processing dynamically alters this delicate trans-organ coupling. Opening the eyes drives the cortical α networks forcefully into an inhibition-dominant, subcritical state (drastically lowering E/I and suppressing DFA).[1] Because the cortical system is pushed far away from criticality, its susceptibility to the slow, 0.05 Hz gastric rhythm is acutely diminished. The brain, prioritizing intense visual processing, effectively "decouples" its α amplitude from the gastric phase, shifting its massive computational resources entirely toward the external environment.[1] Adults, possessing a demonstrably greater dynamic range, exhibit a much more dramatic decoupling upon eye-opening compared to adolescents, reflecting a more mature, adaptable allocation of neuro-visceral resources.[1]

Pathological Geometries: Dysrhythmia, Hyper-Coupling, and Mental Health

When the developmental trajectory deviates from its normative path—either due to a failure of the cortex to achieve criticality or a disruption in the vagal transduction network—the consequences reverberate catastrophically across both the central nervous system and the gastric analogue metric.

The Pathogenesis of Gastric Event Horizons

If the prefrontal cortex fails to mature properly during adolescence, maintaining a pathologically high E/I ratio (supercriticality) well into adulthood, the vital inhibitory top-down control over the brainstem remains severely deficient.[1] This persistent developmental failure leads to chronically low or highly erratic vagal tone.[28] Deprived of its homeostatic central tuner, the gastric medium's wave speed parameter $c(x)$ becomes highly irregular and unpredictable.[1]

Without consistent vagal support to maintain the physiological elasticity and excitability of the ICC networks, localized zones within the stomach wall experience a catastrophic drop in wave speed, $c(x) \to 0$.[1] These zones constitute the analogue event horizons. The gastric slow wave becomes trapped within this pathological spacetime curvature, causing an immediate uncoupling of electro-mechanical peristalsis.[1] The physical manifestation of this spacetime collapse is clinical gastroparesis. While diabetes mellitus accounts for a large portion of adult gastroparesis, it accounts for only 2%–4% of pediatric cases; instead, delayed gastric emptying is seen in an astonishing 80% of critically ill children, underscoring the extreme fragility of the developing gut-brain axis to stress and dysregulation.[43] The symptoms of this collapse are debilitating: intractable nausea, vomiting, severe bloating, and early satiety.[1]

Hyper-Coupling and the Dimensional Signature of Psychopathology

Crucially, the presence of an analogue event horizon in the stomach does not simply halt physical digestion; it actively transmits a massive distress signal back to the central nervous system. Large-scale machine learning analyses of high-density electrogastrography paired with resting-state fMRI across 243 participants have revealed a profound dimensional signature of mental health directly tied to aberrant stomach-brain coupling.[50]

Paradoxically, in states of severe psychological distress, the phase-locking between the gastric rhythm and fronto-parietal brain regions does not weaken; it becomes *abnormally strong*.[50] Unusually strong stomach-brain coupling—specifically hyper-loading onto the left superior angular gyrus, right posterior supramarginal gyrus, and the broader dorsal attention and frontoparietal control networks—indexes a significantly higher psychological burden.[53] This hyper-coupling correlates strongly with clinical trait anxiety, severe depression, chronic stress, fatigue, and somatic symptoms.[50]

Viewed through the integrated Criticality-Spacetime theoretical model, this phenomenon can be explained with immense clarity: When an analogue event horizon forms in the gut, the localized trapping of the bioelectrical pulse triggers massive, uncoordinated vagal afferent firing (serving as a biological equivalent of Hawking radiation, wherein trapped energy at the event horizon boundary creates a massive outward energetic burst). This overwhelming afferent signal floods the NTS, ascending rapidly to the amygdala, anterior insula, and prefrontal cortex.[24]

The brain, receiving constant, high-amplitude alarm signaling from the disrupted gastric metric, forcibly locks its cortical oscillations to the pathological gut rhythm.[50] This "hyper-coupling" forcibly shifts the brain's delicate E/I balance, driving prefrontal networks out of their optimal critical state and inducing the profound cognitive rigidity, hyper-arousal, and executive dysfunction characteristic of severe anxiety and depressive disorders.[52] The biological spacetime is broken, and the mind is dragged down with it.

Biological Domain	**Healthy Matured State**	**Pathological State (Developmental Failure/Stress)**
Brain E/I Balance (α/θ)	Near-critical (Balanced E/I, High DFA)	Supercritical (High E/I, low stability, low DFA)

Vagal Control Signal	High tone, high fidelity, adaptable	Low tone, highly erratic, suppressed
Gastric Acoustic Metric	Smooth curvature, variable $c(x) > 0$	Collapse of $c(x) \to 0$ at specific loci
Spacetime Topology	Continuous, rhythmic wave propagation	Analogue Event Horizons (Conduction Blocks)
Gut-Brain Coupling	Subtle, state-dependent, easily decoupled	Hyper-coupled, locked to pathological rhythm
Clinical Presentation	Autonomic resilience, normal gastric motility	Trait Anxiety, Depression, Severe Gastroparesis

Table 3: Comparison of physiological parameters across the Gut-Brain Axis in healthy neuro-visceral maturation versus pathological states.[1]

Therapeutic Neuromodulation: Rewiring the Biological Metric

Understanding the stomach as a biological spacetime tuned by neural criticality opens the door for highly advanced, targeted therapeutic interventions. When the developmental tuning process has failed, or when chronic stress has induced a pathological supercritical state in the brain, pharmacological interventions often fall short.[3] However, Vagus Nerve Stimulation (VNS) has emerged as a potent electroceutical tool capable of artificially inducing the exact tuning required to dissolve analogue event horizons.[23]

By delivering precise, algorithmically defined electrical pulses directly to the cervical vagus nerve, VNS bypasses the dysfunctional prefrontal controller and directly provides the missing tuning parameter $f(V(t))$ to the gut.[39] Pre-clinical and clinical mapping has shown that specific parameters—such as medium (0.50 ms, 0.50 mA, 5 Hz) and high (1.00 ms, 1.00 mA, 10 Hz) charge delivery—have profound effects on the effective metric.[56] These stimulations have been shown to temporarily override the intrinsic, dysrhythmic gastric rhythm, alter the localized values of $c(x)$, and induce massive relaxation of the pyloric sphincter, effectively restoring forward motility and "smoothing out" the pathological spacetime curvature.[39]

Concurrently, ascending signals generated by VNS reach the brainstem and actively modulate the cortical E/I balance. VNS triggers the widespread release of neuromodulators that directly tilt the central balance back toward inhibition, notably increasing central GABA levels while significantly reducing excitatory amino acids like glutamate and aspartate.[23] In doing so, VNS manually pushes a supercritical, anxious brain back toward the critical boundary, resolving the E/I imbalance in both the hippocampi and the amygdalae.[23] Therefore, electroceutical interventions function as true bidirectional metric stabilizers, simultaneously treating the neural computational failure and reversing the resulting visceral spacetime collapse.[23]

Conclusion

The developmental trajectory from adolescence to adulthood encompasses far more than the localized pruning of cortical synapses or the psychological maturation of the individual; it represents the grand biological calibration of a vast, interdependent bio-physical control system. By systematically cross-referencing the longitudinal emergence of frequency-specific brain criticality [1] with the relativistic analogue gravity model of the gastric slow wave [1], a profound theoretical synthesis emerges.

During adolescence, the neural mechanisms driving widespread low-frequency cortical oscillations operate in a slightly supercritical, excitation-dominant regime, completely lacking the long-range temporal correlations necessary for high-fidelity top-down control.[1] Consequently, the gut-brain axis exhibits immense volatility, and the "effective spacetime metric" of the stomach—defined by the location-dependent wave speed $c(x)$—is inherently prone to instability and structural degradation.[1]

As the brain matures into adulthood, the vast up-regulation of GABAergic inhibition decreases the E/I ratio in the $\theta, \alpha,$ and β bands, pulling the cerebral cortex precisely to a critical phase transition.[1] This optimized critical state provides maximum computational susceptibility and dynamic range, allowing the prefrontal cortex to exert precise, stable, and highly adaptable inhibitory control over vagal efferents.[6]

This matured, high-fidelity vagal tone acts as the master "tuner" of the visceral spacetime.[1] The precise top-down modulation ensures that the gastric acoustic metric remains perfectly smooth, allowing the 0.05 Hz massless scalar field (the gastric slow wave) to propagate endlessly without encountering pathological decelerations.[1] When this delicate developmental tuning process fails—due to environmental stress, genetic diathesis, or neurodevelopmental delay—the gastric wave speed collapses to zero, forming devastating analogue event horizons. These conduction blocks trap bioelectrical energy, halt peristalsis, and reflexively drive the brain into hyper-coupled, pathological states of anxiety and depression.[1]

Ultimately, the transition to a healthy, functional adulthood represents the mastery of biological analogue gravity. This mastery is achieved not in the tissues of the gut itself, but through the delicate, mathematically perfect critical balance of excitation and inhibition maintained in the highest echelons of the human cerebral cortex. By understanding and manipulating this criticality-spacetime axis, the future of both psychiatry and gastroenterology may lie in the unified rewiring of the human metric.

Works cited

1. A Theoretical Framework for Gastric Electrophysiology_ Re-framing the Gut-Brain Axis and the Gastric Slow Wave as an Analogue Gravity System https://www.tymmesalab.com/2026/02/15/a-theoretical-framework-for-gastric-electrophysiology-re-framing-the-gut-brain-axis-and-the-gastric-slow-wave-as-an-analogue-gravity-system/
2. The Microbiota-Gut-Brain Axis: From Motility to Mood - PMC - NIH, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC8634751/
3. Gastric electrical stimulation: An emerging therapy for children with intractable gastroparesis - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC6938723/
4. A Physical Theory For When the Brain Performs Best | Quanta Magazine, accessed April 3, 2026, https://www.quantamagazine.org/a-physical-theory-for-when-the-brain-performs-best-20230131/
5. The brain's sweet spot: How criticality could unlock learning, memory—and prevent Alzheimer's | ScienceDaily, accessed April 3, 2026, https://www.sciencedaily.com/releases/2025/06/250625075016.htm
6. Is criticality a unified set-point of brain function? - bioRxiv, accessed April 3, 2026, https://www.biorxiv.org/content/10.1101/2024.09.02.610815v2.full-text
7. Theoretical foundations of studying criticality in the brain | Network Neuroscience | MIT Press, accessed April 3, 2026, https://direct.mit.edu/netn/article/6/4/1148/112392/Theoretical-foundations-of-studying-criticality-in
8. Brain criticality emerges with developmental shifts in frequency-specific excitation-inhibition balance | bioRxiv, accessed April 3, 2026, https://www.biorxiv.org/content/10.64898/2026.03.26.714563v1.full-text
9. Regulation of the E/I-balance by the neural matrisome - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC10151766/
10. On cross-frequency phase-phase coupling between theta and gamma oscillations in the hippocampus | eLife, accessed April 3, 2026, https://elifesciences.org/articles/20515
11. Intrinsic excitation-inhibition imbalance affects medial prefrontal cortex differently in autistic men versus women | eLife, accessed April 3, 2026, https://elifesciences.org/articles/55684

12. Slow Wave Conduction Patterns in the Stomach: From Waller's Foundations to Current Challenges - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC4405773/
13. Spatiospectral signatures of stomach-brain synchrony | bioRxiv, accessed April 3, 2026, https://www.biorxiv.org/content/10.64898/2026.03.13.711584v1.full
14. Relationship Between Intestinal Slow-waves, Spike-bursts, and Motility, as Defined Through High-resolution Electrical and Video Mapping, accessed April 3, 2026, https://www.jnmjournal.org/journal/view.html?doi=10.5056/jnm21183
15. The gastric conduction system in health and disease: a translational review, accessed April 3, 2026, https://journals.physiology.org/doi/abs/10.1152/ajpgi.00065.2021
16. Analogue Gravity - PMC - NIH, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC5255570/
17. arXiv:gr-qc/0505065v4 29 Nov 2024, accessed April 3, 2026, https://www.arxiv.org/pdf/gr-qc/0505065
18. Automated classification and identification of slow wave propagation patterns in gastric dysrhythmia - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC3911879/
19. The vagus connection: exploring the neurobiology of brain-gut communication | Journal of Neurophysiology | American Physiological Society, accessed April 3, 2026, https://journals.physiology.org/doi/10.1152/jn.00516.2024
20. Vagal neurocircuitry and its influence on gastric motility - PMC - NIH, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC5605144/
21. Inhibitory neurotransmission regulates vagal efferent activity and gastric motility, accessed April 3, 2026, https://www.researchgate.net/publication/303980819_Inhibitory_neurotransmission_regulates_vagal_efferent_activity_and_gastric_motility
22. Brainstem Neuronal Circuitries Controlling Gastric Tonic and Phasic Contractions: A Review, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC9595174/
23. A Review of Vagus Nerve Stimulation for Disease: Comprehensive Theory and Evidence for Mechanisms of Action - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC12960021/
24. Vagus Nerve and Underlying Impact on the Gut Microbiota-Brain Axis in Behavior and Neurodegenerative Diseases - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC9656367/
25. The Vagus Nerve and the Brain-Gut Axis: Implications for Neuropsychiatric Disorders | The Journal of Neuropsychiatry and Clinical Neurosciences - Psychiatry Online, accessed April 3, 2026, https://www.psychiatryonline.org/doi/10.1176/appi.neuropsych.20240118
26. Heart Rate Variability and Cardiac Vagal Tone in Psychophysiological Research – Recommendations for Experiment Planning, Data Analysis, and Data Reporting - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC5316555/
27. From the heart to the mind: cardiac vagal tone modulates top-down and bottom-up visual perception and attention to emotional stimuli - Frontiers,

accessed April 3, 2026, https://www.frontiersin.org/journals/psychology/articles/10.3389/fpsyg.2014.00278/full
28. Neuroimaging Studies of the Neural Correlates of Heart Rate Variability: A Systematic Review - MDPI, accessed April 3, 2026, https://www.mdpi.com/2077-0383/12/3/1016
29. Vagal Regulation and Internalizing Psychopathology among Adolescents Exposed to Childhood Adversity - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC4043866/
30. Developmental regulation of excitatory-inhibitory synaptic balance in the prefrontal cortex during adolescence - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC8429511/
31. Adolescents' brain-autonomic coupling during emotion processing | Request PDF, accessed April 3, 2026, https://www.researchgate.net/publication/327428803_Adolescents'_brain-autonomic_coupling_during_emotion_processing
32. Adolescents' brain-autonomic coupling during emotion processing - PMC - NIH, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC6197902/
33. Is adolescence the missing developmental link in Microbiome–Gut–Brain axis communication? - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC6776431/
34. Reframing the Teenage Wasteland: Adolescent Microbiota-Gut-Brain Axis - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC4794958/
35. Getting under the skin: long-term links of adolescent peer relationship difficulties to adult vagal tone - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC9589924/
36. Characterizing change in vagal tone during the first three years of life: A systematic review and empirical examination across two longitudinal samples - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC8429175/
37. Sympathetic Vagal Balance and Cognitive Performance in Young Adults during the NIH Cognitive Test - MDPI, accessed April 3, 2026, https://www.mdpi.com/2411-5142/7/3/59
38. The connection between heart rate variability (HRV), neurological health, and cognition: A literature review - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC10014754/
39. Vagus Nerve Stimulation Promotes Gastric Emptying by Increasing Pyloric Opening Measured with Magnetic Resonance Imaging - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC6160317/
40. Cervical Vagus Nerve Stimulation Disrupts Gastric Slow Wave Activity in Rats - PubMed, accessed April 3, 2026, https://pubmed.ncbi.nlm.nih.gov/38082764/
41. Vagus nerve stimulation promotes gastric emptying by increasing pyloric opening measured with magnetic resonance imaging | Request PDF - ResearchGate, accessed April 3, 2026, https://www.researchgate.net/publication/325339367_Vagus_nerve_stimulation_promotes_gastric_emptying_by_increasing_pyloric_opening_measured_with_mag

netic_resonance_imaging
42. Prefrontal excitation/inhibition balance supports adolescent enhancements in circuit signal to noise ratio, accessed April 3, 2026, https://shane-mckeon.com/files/snr2024.pdf
43. Gastroparesis in the Pediatric Patient: Children Are Not Little Adults - MDPI, accessed April 3, 2026, https://www.mdpi.com/2624-5647/2/2/8
44. Simultaneous stomach-brain electrophysiology reveals dynamic coupling in human sleep - bioRxiv, accessed April 3, 2026, https://www.biorxiv.org/content/10.1101/2025.11.13.686572v1.full.pdf
45. Phase-amplitude coupling at the organism level: The amplitude of spontaneous alpha rhythm fluctuations varies with the phase of the infra-slow gastric basal rhythm - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC5312779/
46. Simultaneous Gut-Brain Electrophysiology Shows Cognition and Satiety Specific Coupling, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC9737783/
47. Gastric emptying scintigraphy results in children are affected by age, anthropometric factors, and study duration - naspghan, accessed April 3, 2026, https://naspghan.org/files/documents/pdfs/training/curriculum-resources/motility/Wong_Gastric-Emptying-Scintigraphy.pdf
48. Gastroparesis | Children's Hospital of Philadelphia, accessed April 3, 2026, https://www.chop.edu/conditions-diseases/gastroparesis
49. Delayed Gastric Emptying Correlates With Decreased Post-prandial Motility in Children: A Single-center Retrospective Review, accessed April 3, 2026, https://www.jnmjournal.org/journal/view.html?doi=10.5056/jnm24057
50. The stomach's 'second brain' is more in sync with the mind during mental distress, accessed April 3, 2026, https://www.eurekalert.org/news-releases/1094397
51. Stomach-brain coupling indexes a dimensional signature of mental health - bioRxiv, accessed April 3, 2026, https://www.biorxiv.org/content/10.1101/2024.06.05.597517v4
52. Stomach-brain coupling indexes a dimensional signature of mental health - bioRxiv, accessed April 3, 2026, https://www.biorxiv.org/content/10.1101/2024.06.05.597517v4.full
53. Stomach-brain coupling indexes a dimensional signature of mental ..., accessed April 3, 2026, https://www.biorxiv.org/content/10.1101/2024.06.05.597517v4.full-text
54. The role of neurotransmitters in mediating the relationship between brain alterations and depressive symptoms in patients with inflammatory bowel disease - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC10543356/
55. The gut-brain axis and cognitive control: a role for the vagus nerve - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC10427741/
56. Acute Cervical Vagus Nerve Stimulation Modulates Gastric Slow Waves in the Distal Rat Stomach - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC12163215/

57. Alteration of Excitation/Inhibition Imbalance in the Hippocampus and Amygdala of Drug-Resistant Epilepsy Patients Treated with Acute Vagus Nerve Stimulation - PMC, accessed April 3, 2026, https://pmc.ncbi.nlm.nih.gov/articles/PMC10377456/