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Guardian System Dysfunction: ADHD–Inattentive Type as a Vestibular–Autonomic Timing Disorder

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1 Prevailing Conception of ADHD–Inattentive Type

The Inattentive subtype of ADHD (ADHD-I) has long been framed as a frontoparietal network underactivation problem—an executive dysfunction characterized by lapses of attention, mental drift, and poor sustained effort.

The dominant model emphasizes dopamine and norepinephrine insufficiency in prefrontal regions, leading to reduced working-memory precision and low motivation. Typical treatment focuses on stimulant enhancement of catecholamine tone.

- However, several recurrent clinical and physiological features are poorly explained by this cortical-centric framework:
- Dizziness, lightheadedness, or “floating” sensations during cognitive load or postural change.
- Fatigue and derealization that worsen on standing (orthostatic intolerance, POTS-like episodes).
- Salt craving, thirst, static shocks, and frequent urination, suggestive of ADH axis disruption.
- Preserved reasoning and creativity, yet chronic difficulty initiating or sustaining engagement—“I know what to do, I just can’t start.”
- Sleep fragmentation with hypovigilant states rather than hyperarousal.
- These signs point toward a failure not of executive planning but of body-to-brain timing and gating—the mechanism that decides when internal and external information is allowed into conscious processing.

This function aligns precisely with the Guardian System of the Native Brilliance architecture, whose core role is to stabilize access and veto ignition when self-location or physiological readiness is uncertain.

2 Native Brilliance Primer (Standard Across Series)

2.1 Predictive-Coding Scaffold (Brain ↔ Body)

The Native Brilliance (NB) architecture describes seven oscillatory systems coordinating predictive coding across brain and body.

Each governs a specific laminar lever, brain–body circuit, and Bayesian function:

NB System	Primary Role	Cortical Lever	Body Axis
Visionary	Comparator / Residual Maker	L1 + L2/3	Visual–circadian
Investigator	Precision Controller	L6–CT + L1	Saliency–circadian
Integrator	Evidence Curator / Boundary Maintainer	L4	Gut–vagal–immune
Guardian	Access / Veto Gate	Deep β holds (L5–L6 \leftrightarrow TRN)	Vestibular–autonomic
Pioneer	Match Amplifier / Broadcaster	L5	Motor–dopaminergic
Accountant	Capacity / Budget Gate	L6 \leftrightarrow TRN	Metabolic–hypothalamic
Persuader	Mode Switch / Reset	Neuromodulatory Hubs	Histamine–HPA axis

2.2 Lamina & Oscillations (What the Bands Do)

- α (8–12 Hz): inhibitory gating and sensory segmentation.
- β (15–25 Hz): timing precision, hold/veto control.
- θ (4–7 Hz): body–space integration and memory sampling.
- γ (30–80 Hz): content ignition.
- δ : metabolic reset / recovery.

Guardian control rhythms: θ – α coherence anchors body-in-space awareness; deep β bursts signal “stop/hold” commands through the subthalamic nucleus and thalamic reticular nucleus (TRN).

When vestibular or autonomic reliability drops, these rhythms desynchronize, producing lapses in engagement and spatial self-coherence.

2.3 System-of-Systems Snapshot

NB System	Dominant Oscillatory Failure	Core Clinical Correlate
Visionary	$\beta \rightarrow \gamma$ decoupling; α collapse	GAD / visual-circadian anxiety
Investigator	β persistence; phase delay	OCD / salience hypervigilance
Integrator	α collapse; cross-modal γ intrusion	Dyslexia / multisystem sensitivity
Guardian	θ - α desynchrony; weak β holds	ADHD-Inattentive / POTS spectrum
Pioneer	$\beta \rightarrow \gamma$ mistiming; MMP-9 \uparrow	ADHD-Hyperactive / tissue stress
Accountant	α gate loss; δ dominance	Autism / capacity collapse
Persuader	β ERD / histamine resets	Bipolar spectrum

2.4 Shared vs Type-Specific Biology

Most NB disturbances occur on a shared CIRS-like terrain (\uparrow C4a, \uparrow MMP-9, \uparrow TGF- β 1, \downarrow VIP/MSH/ADH).

The Guardian's specific signature centers on \downarrow ADH/AVP, \uparrow osmolality, and vestibular hypoperfusion, driving oscillatory instability in the TRN and deep β - θ coordination hubs.

Compromised osmoregulation blurs interoceptive precision, while degraded vestibular feedback undermines spatial self-consistency—together producing the drifting, unfocused, dissociative quality of ADHD-I.

3 The Native Brilliance Framework (Guardian Orientation)

The Guardian System governs Bayesian access control—it determines whether the brain has sufficient confidence in self-location and bodily readiness to ignite conscious processing or action.

It operates as a real-time gate linking vestibular certainty with autonomic capacity.

3.1 Normal Operation

- Vestibular feedback from semicircular canals and otoliths stabilizes a reference frame for self-world alignment (“I am here; this is me”).
- Autonomic signals (baroreflex, osmolality, vagal tone) supply a physiological budget (“I can afford to engage”).
- The TRN- β network integrates these streams: if both are reliable, it releases the gate (allow); if either is uncertain, it issues a β hold (veto).
- θ - α coupling links body-space awareness to cortical readiness; deep β synchrony times the transition from resting to task mode.

3.2 Failure Signature in ADHD-Inattentive / POTS

When vestibular or autonomic precision collapses:

- θ - α desynchrony: floating or “unanchored” sensations; task drift.
- β hold weakness: difficulty maintaining engagement; premature disengagements.
- Orthostatic sensitivity: standing triggers dizziness or derealization.
- ADH ↓ / osmolality ↑: thirst, static shocks, urinary frequency.
- Low vagal tone (HRV ↓): poor state transitions between rest and focus.

Cognitively this appears as “attention without anchor.” The person can perceive and plan but cannot sustain ignition—attention slides off targets as if gravity were missing.

Physiologically it manifests as autonomic lightness—a brain and body out of sync with their spatial and energetic boundaries.

4 Guardian Function in Bayesian Architecture

4.1 Normal Operation — The Access/Veto Gate

Within the Bayesian brain, access control is not about attention allocation (Visionary) or precision tuning (Investigator); it’s about deciding whether the system is confident enough in its self-reference and physiological budget to allow ignition.

The Guardian thus serves as the bridge between interoception and perception—where bodily state sets the gain for cognitive access.

Mechanistically:

- Vestibular nuclei → Parieto-Insular Vestibular Cortex (PIVC) provide self-motion certainty (“where I am and whether I’m moving”).
- Baroreceptor and osmoreceptor afferents → Nucleus Tractus Solitarius (NTS) supply physiological readiness signals (blood volume, pressure, hydration).
- Integration hub: TRN + deep β synchrony in basal ganglia and thalamocortical loops determine gating threshold.
- Decision rule:
 - If vestibular reliability (R_v) and autonomic readiness (R_a) are both high, gate opens (access).
 - If either is low, gate closes (veto).
 - Mathematically: $P(\text{access}) \propto R_v \times R_a$.
 - This gate determines whether cortical γ bursts (content ignition) are allowed to propagate or remain suppressed.

4.2 Oscillatory Correlates

Band	Guardian Role	Failure Mode	Symptom Manifestation
θ (4–7 Hz)	Body–space integration	Desynchronized	Spaciness, depersonalization
α (8–12 Hz)	Vestibular–visual gating	Collapsed or unstable	Motion sensitivity, dizziness
β (15–25 Hz)	Hold / Veto precision	Weak or mistimed	Poor sustained focus
γ (30–80 Hz)	Conscious ignition	Fragmented	Shallow or inconsistent awareness

Healthy Guardian rhythm shows **θ–α** nesting (body-space coherence) and **β–γ** phasic locking (gated ignition).

In ADHD-I, both are degraded: **θ–α** drift yields disembodiment; **β** instability yields unreliable ignition.

5 Innate-Immune Hypothesis of Guardian Dysfunction

5.1 Autonomic–Vestibular Terrain

The Guardian system’s failure in ADHD-I maps directly onto Shoemaker’s biotoxin cascade, where chronic inflammatory signaling damages neurovascular and neuropeptide balance.

Key elements:

Biomarker / Peptide	Direction	Functional Impact
ADH / AVP	↓	Osmolality ↑ → thirst, urination, static shocks, orthostatic intolerance
VIP	↓	Autonomic repair ↓; vagal tone ↓; vestibular hypoperfusion
C4a	↑	Capillary hypoperfusion; microglial activation
TGF-β1	↑	Fibrotic remodeling of neurovascular junctions
MMP-9	↑	BBB permeability; endothelial instability

The hypothalamus–pituitary–posterior axis controls both ADH and AVP, which govern fluid balance and stress readiness.

Inflammatory cytokines (IL-6, TNF- α) and complement activation (C4a) downregulate these peptides, degrading interoceptive precision.

Meanwhile, vestibular nuclei in the brainstem depend on consistent oxygenation; capillary hypoperfusion (C4a-driven) disrupts their firing stability.

5.2 The TRN and Autonomic Gating

The thalamic reticular nucleus (TRN)—the Guardian’s cortical gearbox—requires tight cholinergic and GABAergic control to maintain rhythmic gating.

When VIP and ADH drop, acetylcholine tone falls and GABAergic precision wanes, leading to wider, noisier relay windows.

The result: weak veto control (β bursts mistimed) and unstable ignition (γ desynchronization).

Clinically this expresses as attentional drift, autonomic hypersensitivity, and fatigue despite normal cortical power.

5.3 Genetic and Environmental Modifiers

- HLA-DR susceptibility alleles: predispose to chronic inflammatory response with peptide suppression.
- AVPR1B polymorphisms: alter vasopressin receptor sensitivity; correlate with attention lapses and orthostatic reactivity.
- Environmental cofactors: chronic mold, infection, dehydration, or microgravity-like vestibular deprivation all mimic Guardian dysfunction through similar neuroimmune channels.

6 Mechanistic Cascade: From Vestibular–Autonomic Drift to Inattentive Behavior

Step 1 – Biotoxin or stress exposure:

Pattern-recognition receptors trigger cytokine release \rightarrow complement C4a \uparrow \rightarrow vascular leak \rightarrow hypothalamic peptide suppression (\downarrow ADH, \downarrow VIP).

Step 2 – Osmolality and vestibular instability:

ADH \downarrow \rightarrow plasma osmolality \uparrow \rightarrow variable blood volume and perfusion \rightarrow hypoxia in vestibular nuclei and TRN \rightarrow α desynchrony.

Step 3 – TRN gating failure:

With VIP and GABA tone low, TRN relay windows widen; β bursts become mistimed; γ ignition fails to lock to β phase.

Step 4 – Autonomic feedback:

Vagal tone \downarrow ; orthostatic stress \uparrow ; baroreceptor reliability \downarrow \rightarrow brain interprets physiological uncertainty as

environmental uncertainty.

Step 5 – Cognitive manifestation:

- Focus requires gating confidence; when absent, attention slips.
- Working memory collapses under orthostatic load.
- Sensation of “floating away” or “seeing but not engaging” appears.

Step 6 – Behavioral adaptation:

The individual unconsciously limits exposure to upright, stimulating, or fast-changing environments.

Movement or music may temporarily restore coherence (vestibular stimulation re-entrains α), but fatigue returns as autonomic load builds.

Step 7 – Chronic compensation:

Recurrent micro-hypoperfusion fosters cortical hypoarousal, leading to habitual inattention, slow response initiation, and dreamy dissociation—the characteristic ADHD-I profile.

7 Predicted Biomarker Pattern and Oscillatory Signature

7.1 Biochemical Predictions

Marker	Direction	Functional Consequence	Interpretive Note
ADH / AVP	↓	Osmolality ↑ → orthostatic intolerance, thirst, urinary frequency, static shocks	Core Guardian deficit
VIP	↓	Autonomic repair ↓ ; vagal tone ↓ ; vestibular hypoperfusion	Failsafe peptide for TRN rhythm
C4a	↑	Capillary hypoperfusion; neurovascular leak	Correlates with dizziness / cognitive fog
TGF-β1	↑	Fibrotic remodeling → rigid vascular response	Chronic compensation pattern
MMP-9	↑	BBB permeability → TRN instability	Links inflammation ↔ oscillatory noise
Osmolality	↑	Fluid imbalance → vestibular error signals	Tracks symptom intensity
HRV (vagal tone)	↓	Weak parasympathetic containment	Predicts lapses in sustained attention

Composite “Guardian signature”:

↓ ADH × ↓ VIP × ↑ C4a → θ - α desynchrony + β -hold weakness → inconsistent access control.

7.2 Oscillatory Predictions

Metric	Healthy Guardian	Dysfunctional Guardian (ADHD-I)	Functional Outcome
θ - α coupling	Strong body-space coherence	Desynchronized	“Floating” / dissociation
α power (vestibular hubs)	Stable, lateralized	Variable; suppressed upright	Dizziness, blurred focus
β burst timing (TRN / BG)	Precise; event-locked	Weak / mistimed	Poor sustained engagement
β - γ coupling	Coherent ignition	Unstable; short bursts	“Micro-attention” pattern
HRV-EEG coherence	High	Low	Uncoupled body-brain rhythms

Predicted covariance:

$\alpha_{PIVC} \propto VIP_{\alpha} \{ PIVC \} \propto VIP_{\alpha} PIVC \propto VIP$ and $\beta_{TRN} \propto ADH \times vagal\ tone_{\beta} \{ TRN \} \propto ADH \times vagal\ tone_{\beta} TRN \propto ADH \times vagal\ tone$.

Restoration of either peptide or vestibular reliability should normalize both.

8 Testable Predictions and Research Plan

8.1 Core Hypotheses

1. Peptide triad (\downarrow ADH / \downarrow VIP / \uparrow C4a) predicts orthostatic intolerance and θ - α desynchrony in ADHD-I.
2. Vestibular-autonomic rehabilitation (balance training + tVNS) restores θ - α coherence and reduces inattention scores.
3. Shoemaker peptide normalization (VIP / ADH restoration) strengthens β -hold precision and sustained attention.

8.2 Human Cohort Study

- Participants: ADHD-I (n = 50) vs controls (n = 50).
- Measures: Plasma ADH, VIP, C4a, TGF- β 1, MMP-9, osmolality; HRV; EEG (θ - α coherence, β bursts, β - γ CFC) during seated vs standing tasks.
- Predictions: Triad explains > 40 % variance in θ - α desynchrony and β -hold instability; upright posture exaggerates differences.

8.3 Translational Animal Model

- Design: Chronic ADH suppression + vestibular stress in rodents.
- Measures: Blood osmolality, TRN β power, α coherence (PIVC), behavioral “orientation drift.”
- Interventions: ADH analogue, VIP analogue, vagal stimulation.
- Expected: Peptide replacement \rightarrow α re-entrainment \rightarrow restored balance and focused behavior.

8.4 Therapeutic Translation Study

Intervention Arm	Mechanistic Target	Expected Biomarker Shift	EEG Outcome
VIP nasal + ADH support	Restore autonomic precision	VIP \uparrow ADH \uparrow C4a \downarrow osmolality \downarrow	θ - α coherence \uparrow β -hold stability \uparrow
tVNS + balance training	Vagal re-entrainment + vestibular reliability	HRV \uparrow α power \uparrow	Vestibular α stabilized
Hydration + salt protocols	Rebuild volume and baroreflex	Osmolality \rightarrow normal	Fewer orthostatic β lapses
Anti-inflammatory / CIRS protocol	Lower C4a / MMP-9	C4a \downarrow MMP-9 \downarrow	β -noise \downarrow coherence \uparrow

9 Clinical and Theoretical Implications

9.1 Reframing ADHD–Inattentive

ADHD-I is recast as a vestibular–autonomic timing disorder, not merely cortical underactivation.

The Guardian system’s failure to synchronize θ - α (body-space) and β (access control) explains both inattentive drift and somatic instability.

This reframing integrates neurocognitive and physiological findings—attention, balance, and osmoregulation as one loop.

9.2 Diagnostic Integration

- Peptide Panel: ADH, VIP, C4a, TGF- β 1, MMP-9, osmolality.
- EEG Metrics: θ - α coherence (PIVC-ACC), β burst timing (TRN).
- Autonomic Indices: HRV, tilt response, baroreflex gain.

Together they provide a measurable signature of Guardian dysfunction.

9.3 Therapeutic Implications (hypothesis-guided, not clinical advice)

- Vestibular rehabilitation (balance boards, ocular fixation training).
- Autonomic repair (tVNS, hydration protocols, gradual tilt conditioning).
- Peptide restoration (VIP/ADH support following CIRS guidelines).
- Oscillatory training (θ - α entrainment, β stability neurofeedback).
- Environmental control (minimize biotoxin exposure to reduce C4a load).

9.4 Conceptual Significance

The Guardian framework shows that attention is an embodied gating process, not a purely mental resource.

In this view, the inattentive phenotype reflects a Bayesian gate deprived of vestibular certainty and physiological confidence.

It bridges cognitive science, autonomic medicine, and neuroimmunology into a unified model of access control.

10 Summary and Next Steps

Core Insight — ADHD–Inattentive Type arises from a Guardian System failure: θ - α desynchrony and β -hold instability driven by vestibular–autonomic mistrust.

Biotoxin-related ADH / VIP suppression and C4a-linked hypoperfusion disrupt TRN timing, producing drift, orthostatic symptoms, and disengagement.

Next Steps —

1. Integrate vestibular and autonomic testing (EEG + HRV + peptide panels) into the Native Brilliance assessment pipeline.
2. Develop θ - α entrainment modules to restore body–space coherence and improve sustained access.
3. Pilot combined balance / vagal rehabilitation protocols with biomarker tracking.
4. Correlate VIP / ADH normalization with EEG β -stability and behavioral improvement.
5. Extend analysis to related conditions (POTS, chronic fatigue, derealization syndrome) to map the Guardian's broader protective role.



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