



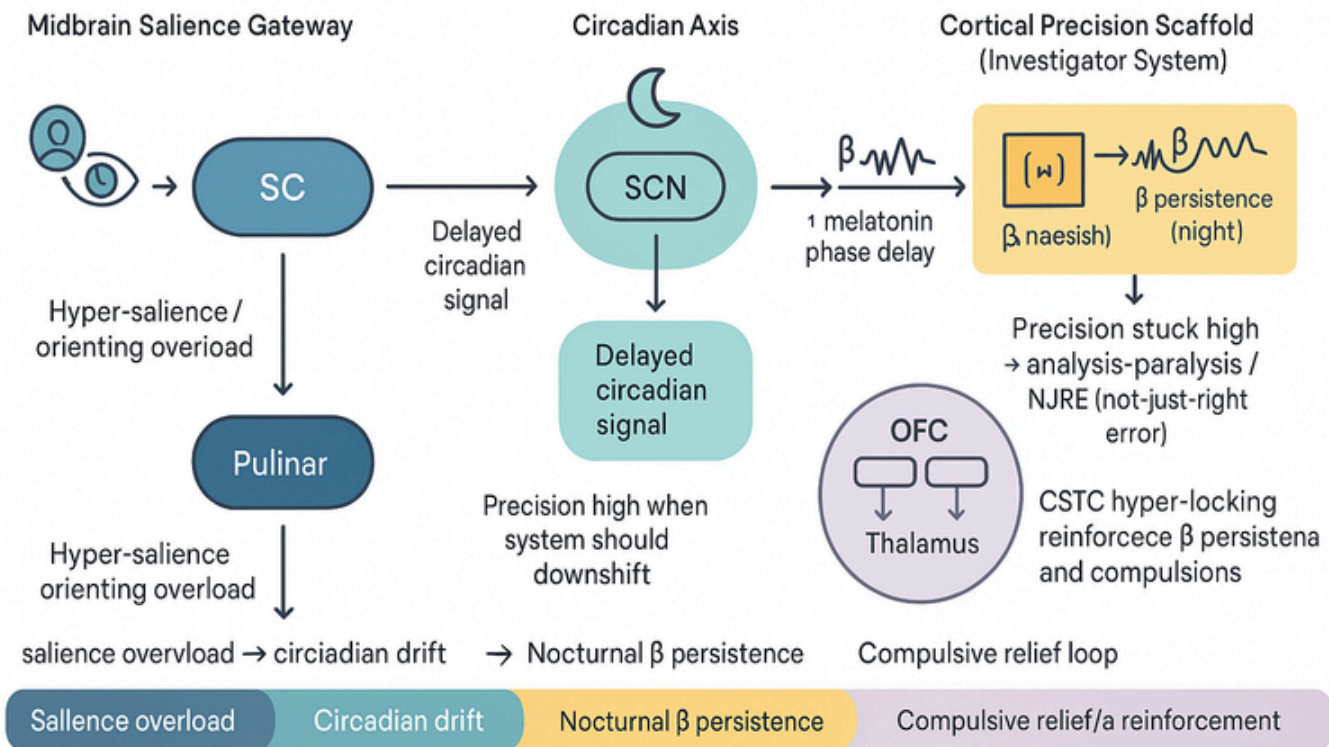
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Investigator System Dysfunction: Obsessive–Compulsive Disorder as a Salience–Circadian Precision Disorder

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Investigator System Dysfunction – Salience → Circadian → Precision Pathway (OCD Mechanism)
Evening precision surge and β -persistence driven by SC/Pulvinar–SCN mis-timing



1 Prevailing Conception of OCD

Obsessive–Compulsive Disorder (OCD) is typically framed as a fronto-striatal dysfunction producing intrusive thoughts (obsessions) and repetitive behaviors (compulsions). Standard models emphasize corticostriatal–thalamic–cortical (CSTC) loops, hyperconnectivity in orbitofrontal–caudate circuits, and serotonin–glutamate imbalance. ERP/CBT and SRIs are first-line treatments. Yet several robust features remain poorly explained by monoamine or purely cognitive accounts:

- Strong evening worsening and delayed sleep-wake phase.
- Nocturnal β persistence with sleep initiation difficulty.
- Complement C4a-linked neuroinflammation and microglial activation across CSTC.
- Normal accuracy but reduced confidence and analysis-paralysis under uncertainty.

These features suggest a deeper midbrain salience \rightarrow circadian precision disturbance that mistimes the β scaffold governing salience selection and cognitive “completion.”

2 Native Brilliance Primer (Standard Across Series)

2.1 Predictive-Coding Scaffold (brain \leftrightarrow body)

The Native Brilliance (NB) architecture maps seven coordinated brain–body systems onto a predictive-coding stack. Cortex continuously compares top-down predictions with bottom-up evidence, updating beliefs by minimizing prediction error. Each NB system emphasizes a distinct control function: Visionary (comparator), Investigator (precision), Integrator (evidence), Guardian (access/veto), Pioneer (broadcast), Accountant (capacity), Persuader (reset). NB assumes brain and body share an oscillatory grammar; peptides, vascular status, and inflammation set precision of these couplings.

2.2 Lamina & Oscillations (what the bands do)

- L1: α/β predictions (apical precision).
- L2/3: γ residuals (feed-forward).
- L4: evidence windows.
- L5: burst/broadcast.
- L6 \leftrightarrow TRN: relay timing/budget.

Bands: α gating; β precision/hold; γ residuals; θ/δ slow carriers. Healthy perception/action shows β -phase \rightarrow γ -amplitude (CFC); illness can show α collapse, β mistiming, or γ fragmentation.

2.3 System-of-Systems at a glance

NB system	Laminar lever	Primary failure sign
Visionary (comparator)	L1 apical; L2/3 γ	$\downarrow \alpha$ gating; $\beta \rightarrow \gamma$ decoupling; γ paradox
Investigator (precision)	L6-CT mid- β ; L1 feature precision	β persistence (night); abnormal $\beta \rightarrow \gamma$; analysis-paralysis
Integrator (evidence)	L4 windows	α boundary collapse; weak $\beta \rightarrow \gamma$
Guardian (access/veto)	Deep β holds; TRN/L6 relay	Weak stop β ; θ intrusions
Pioneer (broadcast)	L5 bursts	Excess β suppression; erratic γ ; weak $\beta \rightarrow \gamma$ at onsets
Accountant (budget)	TRN \leftrightarrow hypothalamus	Capacity collapse: $\uparrow \theta/\delta$, $\downarrow \alpha$
Persuader (reset)	LC-NE/ACh/DA/5-HT/Histamine/HP A	Global β ERD \pm failed re-scaffold

2.4 Shared vs type-specific biology

Shared terrain (CIRS-like): \uparrow C4a, \uparrow MMP-9, \uparrow TGF- β 1; peptide drift (\downarrow MSH, \downarrow VIP, \pm \downarrow ADH). Type-specific in Investigator/OCD: Superior colliculus + Pulvinar salience hyperactivity \rightarrow SCN circadian drift \rightarrow β precision mistiming with evening surge and nocturnal β persistence.

2.5 Measurement battery (series-wide)

Electrophysiology: α suppression (task/state), $\beta \rightarrow \gamma$ CFC, θ/δ intrusion, system locks (frontal β for Investigator). Biomarkers: terrain (C4a, MMP-9, TGF- β 1); type-specific (melatonin phase, cortisol curve; \pm ADH).

3 The Native Brilliance Framework (Investigator orientation)

The Investigator implements system-level precision (L6-CT) and feature-level precision (L1), pacing the relay to L4 and setting postsynaptic gain for the Visionary comparator. In wakeful cognition it runs on mid- β (15–25 Hz); at night it should downshift to θ /high- δ for recovery. Failure mode: precision stuck high or mistimed, producing β persistence and abnormal $\beta \rightarrow \gamma$ coupling [\[200†source\]](#) .

4 Investigator Function in Bayesian Architecture

4.1 Normal operation — precision allocation

- L6-CT mid- β maintains system precision across thalamo-cortical relays (timing “when to weight evidence”).

- L1 adjusts feature-specific precision for sensory streams.
- Handshake with Visionary: β -phase (Investigator) \rightarrow γ -amp (Visionary) ensures timely updating.

4.2 Failure signature in OCD

- Nocturnal β persistence; daytime δ intrusions; abnormal $\beta \rightarrow \gamma$ coupling (β “jams” updating)
- Behavioral: “not-just-right” sensation, checking rituals, slowed responses despite intact accuracy; evening worsening.

5 Innate-Immune Hypothesis of Investigator Dysfunction

5.1 Midbrain salience gateway \rightarrow circadian axis

Hyperactivity in the Superior Colliculus (SC) and Pulvinar overwhelms salience selection and destabilizes the SCN via non-retinal routes. This produces melatonin phase delay (-30 – 40% secretion; $\sim +2$ h onset) and elevated nocturnal cortisol, shifting precision into the evening window.

5.2 Complement & microglia

Chronic hypervigilance elevates C4a, with TSPO-PET evidence of microglial activation across CSTC (caudate, OFC, thalamus, ventral striatum). C4-mediated pruning further reduces cognitive flexibility, reinforcing β -locked loops and delayed sleep onset.

5.3 Secondary endocrine drift

Under severe/longstanding stress, ADH may drift (osmolality symptoms), but this is secondary for Investigator compared to melatonin/cortisol and complement patterns.

6 Mechanistic Cascade: From Salience to Compulsion

Step 1 – Salience overload

SC + Pulvinar hyper-salience \rightarrow constant orienting pressure; precision remains high into the evening.

Step 2 – SCN timing drift

Melatonin secretion reduced/delayed; nocturnal cortisol elevated \rightarrow β precision peaks late (6–10 PM) **【196†source】** .

Step 3 – β jam of updating

Post-trial β rebound fails; β persists and blocks new encoding \rightarrow “analysis-paralysis” with abnormal $\beta \rightarrow \gamma$ coupling.

Step 4 – Compulsive loops

Incomplete internal “completion signal” (NJRE) drives checking/ordering; rituals temporarily relieve β load but reinforce the loop.

Step 5 – Inflammatory reinforcement

C4a \uparrow and microglial activity track distress and treatment resistance; insomnia perpetuates salience mis-timing

7 Predicted Biomarker Pattern and Oscillatory Signature

7.1 Biochemical Predictions

Biomarker	Direction	Functional Consequence	Notes / Citations
Melatonin (24h / DLMO)	↓ / phase-delayed	Evening precision surge; sleep-onset delay	[196†source]
Cortisol (diurnal)	↑ nocturnal	Persistent arousal; β persistence	[196†source]
C4a	↑	Complement pruning; CSTC cognitive rigidity	[196†source]
TSPO (PET)	↑	Microglial activation (CSTC)	[196†source]
ADH (optional)	Variable / ↓	Osmolality symptoms (secondary)	[190†source]

7.2 Oscillatory Predictions

Metric	Healthy Investigator	Investigator Dysfunction (OCD)	Notes / Citations
Frontal β complexity	Moderate; rebounds post-encoding	Elevated; post-trial rebound failure	[196†source]
$\beta \rightarrow \gamma$ coupling	Coherent timing to update Visionary	Abnormal; β “jams” γ updating	[196†source][200†source]
Nighttime β	Drops for sleep onset	Persists \rightarrow insomnia	[196†source]
Evening symptom window	Neutral	Peak OCD severity 6–10 PM	[196†source]

8 Testable Predictions and Research Plan

8.1 Core Hypotheses

- 1) Melatonin phase delay + nocturnal cortisol elevation + C4a \uparrow predict nocturnal β persistence and abnormal $\beta \rightarrow \gamma$ coupling.
- 2) Evening-focused interventions (light restriction, circadian phase advance) reduce β persistence and compulsions.
- 3) Complement/microglia modulation tracks reductions in compulsive urge and insomnia.

8.2 Human Cohort Study

Participants: OCD vs matched controls; optional GAD comparator. Measures: dim-light melatonin onset (DLMO), 24-h cortisol curve, C4a; EEG across day with focus on 6–10 PM (frontal β complexity; $\beta \rightarrow \gamma$); actigraphy/sleep diary; TSPO-PET subset.

Predictions: Biomarker triad explains unique variance in evening β and symptom severity; phase advance + evening α -coordination retrains β timing and reduces compulsions.

8.3 Translational Model

Induce SC/pulvinar salience stress; assay melatonin/cortisol rhythms, β persistence, $\beta \rightarrow \gamma$ coupling; test complement modulation and circadian realignment.

9 Clinical and Theoretical Implications

9.1 Reframing

OCD as a salience–circadian precision disorder: midbrain salience overload + circadian drift mistime β , producing nocturnal persistence and evening symptom surge; complement-linked neuroinflammation reinforces rigidity.

9.2 Diagnostic Integration

Panel: DLMO, 24-h cortisol, C4a (\pm ADH); EEG: frontal β complexity, $\beta \rightarrow \gamma$ coupling (with evening emphasis); actigraphy; consider TSPO-PET in research settings.

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

- Circadian phase advance & light hygiene (reduce evening β drive).
- β retiming: daytime α coordination, task-bound β scaffolding; reduce nocturnal β .
- Complement/microglia modulation to relieve CSTC rigidity.

10 Summary and Next Steps

Core insight — Superior colliculus + Pulvinar \rightarrow SCN disturbance mistimes β precision, creating nocturnal β persistence, evening symptom surge, and abnormal $\beta \rightarrow \gamma$ coupling; complement-linked microglial activation maintains rigidity. Demonstrating biomarker– β links and reversible evening retiming would ground OCD within the NB predictive framework.

Next steps — finalize figure assets; pre-register the evening-window cohort protocol; align biomarker/EEG acquisition with the NB Knowledge Base for cross-type comparisons.



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