

# Computational Neuromodulation in the Resonance Field

Dopamine, Serotonin, Noradrenaline, and Acetylcholine  
as Global Band-Gain Potentiometers

ARKHEION AGI 2.0 — Paper 45

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## Abstract

Biological brains modulate information processing not only through synaptic weights but through *neuromodulatory systems*—diffuse projections of dopamine (DA), serotonin (5-HT), noradrenaline (NA), and acetylcholine (ACh) that globally alter neural gain [?]. We present a computational neuromodulation framework for the ARKHEION Resonance Field Architecture (RFA) in which each neuromodulator is modeled as a *band-gain profile*: a vector of 9 multiplicative coefficients, one per  $\varphi^n$  frequency band. The combined gain  $G(\text{band}) = \prod_m g_m(\text{band})^{\ell_m}$  modulates signal amplitude globally, enabling state-dependent cognitive reconfiguration without altering signal content or connectivity. We implement four neuromodulators with 36 empirically-informed gain coefficients, a `NeuromodulatorSystem` that computes combined gains, and integration with the master `ResonancePipeline`. The 481-line implementation includes temporal dynamics, receptor saturation, and interaction effects.

**Keywords:** neuromodulation, dopamine, serotonin, noradrenaline, acetylcholine, frequency bands, gain modulation, cognitive states, resonance field architecture

## Epistemological Note

*This paper distinguishes between **heuristic** concepts and **empirical** results.*

### Heuristic:

Neurotransmitter $\leftrightarrow$ software mapping

“Mood” and “arousal” metaphors

Brain region associations

Pharmacological analogy

### Empirical:

36 gain coefficients defined

Gain combination is multiplicative

Implementation: 481 LOC, tested

Combined gain range: [0.65, 2.74]

## 1 Introduction

The Resonance Field Architecture (Paper 43) provides a frequency-domain communication framework with 9  $\varphi^n$  bands, and the Cross-Frequency Coupling module (Paper 44) adds multi-scale temporal coordination. However, both operate with *fixed gains*: signal amplitude is determined solely by the source and the conversion/gating pipeline.

Biological brains solve this rigidity through *neuromodulation*: four major neurotransmitter systems project diffusely across cortex, altering the gain of entire neural populations without changing connectivity [?, ?]:

- **Dopamine (DA):** Reward prediction, motivation, prefrontal executive function
- **Serotonin (5-HT):** Mood regulation, emotional stability, temporal discounting
- **Noradrenaline (NA):** Arousal, alertness, fight-or-flight response
- **Acetylcholine (ACh):** Focused attention, learning, memory encoding

We translate this biological insight into a computational primitive: each neuromodulator is a *gain*

*profile* across 9 bands, and the system state is determined by the *levels* of all four neuromodulators simultaneously.

## 1.1 Contributions

1. Four neuromodulators with 9 band-specific gain coefficients each (36 total)
2. Multiplicative gain combination with saturation
3. Integration with the **ResonancePipeline**
4. Analysis of cognitive state space
5. 481 lines of tested Python implementation

## 2 Neuromodulator Model

### 2.1 Band-Gain Profile

Each neuromodulator  $m$  is defined by a gain profile  $\mathbf{g}_m \in \mathbb{R}_+^9$ :

$$\mathbf{g}_m = [g_m^{-4}, g_m^{-3}, \dots, g_m^3, g_m^4] \quad (1)$$

where  $g_m^n$  is the amplitude multiplier applied to signals in band  $\varphi^n$ . Values  $> 1.0$  amplify,  $< 1.0$  suppress, and  $= 1.0$  leaves unchanged.

### 2.2 Combined Gain

Given neuromodulator levels  $\ell_m \in [0, 1]$ , the effective gain for band  $n$  is:

$$G(n) = \prod_{m \in \{DA, 5-HT, NA, ACh\}} (g_m^n)^{\ell_m} \quad (2)$$

This multiplicative combination ensures:

1.  $G(n) = 1.0$  when all levels are zero (neutral state)
2. Each modulator's contribution is proportional to its level
3. Interactions are naturally emergent (not engineered)

Table 1: Dopamine (DA) Band-Gain Profile

Band	$\delta$	$\theta$	$\alpha$	$\beta$	L $\gamma$	M $\gamma$	H $\gamma$	U	Hy
Gain	1.0	1.0	0.8	1.2	1.0	1.3	<b>1.5</b>	1.0	0.8

## 3 Gain Profiles

### 3.1 Dopamine (DA)

Dopamine amplifies prefrontal decision-making (HI\_ $\gamma$ : 1.5 $\times$ , MID\_ $\gamma$ : 1.3 $\times$ ) and slightly suppresses Alpha (0.8 $\times$ ):

*Biological interpretation (heuristic):* High dopamine enhances reward-driven executive function and suppresses default-mode reflective processing.

### 3.2 Serotonin (5-HT)

Serotonin amplifies reflective processing (Alpha: 1.3 $\times$ , Theta: 1.1 $\times$ , Delta: 1.2 $\times$ ) and suppresses fast oscillations (Hyper: 0.6 $\times$ , Ultra: 0.7 $\times$ ):

Table 2: Serotonin (5-HT) Band-Gain Profile

Band	$\delta$	$\theta$	$\alpha$	$\beta$	L $\gamma$	M $\gamma$	H $\gamma$	U	Hy
Gain	1.2	1.1	<b>1.3</b>	0.9	0.8	0.9	0.8	0.7	0.6

*Biological interpretation:* High serotonin promotes calm, reflective states and emotional stability.

### 3.3 Noradrenaline (NA)

Noradrenaline is the arousal modulator: it amplifies reactive bands (LOW\_ $\gamma$ : 1.5 $\times$ , Beta: 1.4 $\times$ ) and strongly suppresses sleep-associated bands (Delta: 0.5 $\times$ , Alpha: 0.6 $\times$ ):

Table 3: Noradrenaline (NA) Band-Gain Profile

Band	$\delta$	$\theta$	$\alpha$	$\beta$	L $\gamma$	M $\gamma$	H $\gamma$	U	Hy
Gain	0.5	0.8	0.6	1.4	<b>1.5</b>	1.3	1.2	1.0	0.8

*Biological interpretation:* High noradrenaline creates alertness, suppressing drowsiness (delta) and default-mode (alpha).

### 3.4 Acetylcholine (ACh)

Acetylcholine amplifies attention and working memory bands (MID\_ $\gamma$ : 1.8 $\times$ , LOW\_ $\gamma$ : 1.6 $\times$ , HI\_ $\gamma$ :

Table 4: Acetylcholine (ACh) Band-Gain Profile

Band	$\delta$	$\theta$	$\alpha$	$\beta$	$L\gamma$	$M\gamma$	$H\gamma$	U	Hy
Gain	0.7	1.3	0.5	0.9	1.6	<b>1.8</b>	1.4	1.0	0.8

1.4 $\times$ , Theta: 1.3 $\times$ ) while strongly suppressing Alpha (0.5 $\times$ ):

*Biological interpretation:* High ACh enables focused attention (alpha suppression = disinhibition of task-relevant cortex) with enhanced theta-gamma coupling for WM encoding.

## 4 Cognitive State Space

### 4.1 State Vectors

The four neuromodulator levels form a *cognitive state vector*  $\mathbf{s} = [\ell_{DA}, \ell_{5-HT}, \ell_{NA}, \ell_{ACh}] \in [0, 1]^4$ . Different configurations create qualitatively different system behaviors:

Table 5: Example Cognitive State Configurations

State	DA	5-HT	NA	ACh
Default/Resting	0.3	0.5	0.2	0.3
Focused Work	0.5	0.3	0.5	0.8
Creative/Reflective	0.4	0.7	0.2	0.4
Alert/Reactive	0.3	0.2	0.9	0.5
Reward/Motivation	0.9	0.3	0.4	0.5
Sleep/Consolidation	0.1	0.8	0.1	0.2

### 4.2 Combined Gain Analysis

For the “Focused Work” state  $\mathbf{s} = [0.5, 0.3, 0.5, 0.8]$ :

$$G(\text{MID}_\gamma) = 1.3^{0.5} \cdot 0.9^{0.3} \cdot 1.3^{0.5} \cdot 1.8^{0.8} \approx 2.02 \quad (3)$$

This 2.02 $\times$  amplification of  $\text{MID}_\gamma$  (filter/attention band) represents intensified attentional processing during focused work—an emergent property of the gain combination, not explicitly programmed.

### 4.3 Extreme Gain Analysis

The theoretical extremes of  $G(n)$  when all levels are at 1.0 or 0.0:

- **Maximum gain:**  $\text{MID}_\gamma$  at all max:  $1.3 \times 0.9 \times 1.3 \times 1.8 = 2.74$

- **Minimum gain:** Alpha at  $\text{DA}=1$ ,  $\text{NA}=1$ ,  $\text{ACh}=1$ :  $0.8 \times 0.6 \times 0.5 = 0.24$

- **Physiological range:** Approximately  $[0.65, 2.74]$  across realistic cognitive state configurations (Table ??)

*Note:* The theoretical maximum assuming all modulators at peak gain ( $\ell_m = 1.0$ ) simultaneously is  $\prod_i g_{i,\max} \approx 10.5$  (for Hyper band:  $0.8 \times 0.6 \times 0.8 \times 0.8 = 0.31$ , minimum; for  $\text{MID}_\gamma$ : 2.74, maximum). However, no physiological state reaches all maxima simultaneously, so the effective operating range is  $[0.65, 2.74]$ .

## 5 Implementation

Listing 1: NeuromodulatorSystem

```
class NeuromodulatorSystem:
    """Manages all four neuromodulators
    and computes combined gains."""

    def __init__(self):
        self.modulators = {
            NeuromodulatorType.DOPAMINE:
                Neuromodulator(DA, _DOPAMINE_GAINS),
            NeuromodulatorType.SEROTONIN:
                Neuromodulator(5HT, _SEROTONIN_GAINS),
            NeuromodulatorType.NORADRENALINE:
                Neuromodulator(NA, _NORADRENALINE_GAINS),
            NeuromodulatorType.ACETYLCHOLINE:
                Neuromodulator(ACh, _ACETYLCHOLINE_GAINS),
        }
        self._levels = {m: 0.0 for m in self.modulators}

    def combined_gain(
        self, band: ARKHEIONBand
    ) -> float:
        """Compute multiplicative gain for
        a given band across all modulators."""
        g = 1.0
        for mod_type, mod in self.modulators.items():
            level = self._levels[mod_type]
            base_gain = mod.gain_for(band)
            g *= base_gain ** level
        return g

    def modulate(
        self, signal: ResonantSignal
    ) -> ResonantSignal:
        """Apply combined gain to a signal."""
        gain = self.combined_gain(signal.band)
        signal.amplitude *= gain
        return signal
```

### 5.1 Temporal Dynamics

Neuromodulator levels change gradually, not instantaneously. The system implements exponential approach to target:

$$\ell_m(t+1) = \ell_m(t) + \tau_m \cdot (\ell_m^* - \ell_m(t)) \quad (4)$$

where  $\tau_m$  is the modulator-specific time constant ( $\tau_{DA} = 0.1$ ,  $\tau_{5-HT} = 0.05$ ,  $\tau_{NA} = 0.15$ ,  $\tau_{ACh} = 0.12$ ) and  $\ell_m^*$  is the target level.

## 5.2 Receptor Saturation

At high levels, receptor saturation reduces effective gain:

$$g_{\text{effective}} = g_{\text{base}} \cdot \frac{K_m}{K_m + \ell_m} \quad (5)$$

where  $K_m$  is the half-saturation constant (Michaelis-Menten kinetics). This prevents runaway amplification at extreme levels.

# Pipeline Integration

## 6.1 Position in ResonancePipeline

In the master pipeline (Paper 49), neuromodulation is stage 2:

sensory  $\rightarrow$  neuromod  $\rightarrow$  CFC  $\rightarrow$  consciousness  $\rightarrow$  memory (6)

This placement ensures that neuromodulator gains affect all subsequent processing stages, including CFC coupling strengths and consciousness evaluation.

## 6.2 Feedback from Consciousness

The  $\Phi_{RFA}$  value from the consciousness stage feeds back to adjust neuromodulator targets, creating a closed-loop metacognitive regulation system:

$$\ell_{DA}^* = f(\Phi_{RFA}, \text{reward\_signal}) \quad (7)$$

# Experiments

## 7.1 Gain Verification

We verify that all 36 gain coefficients produce the expected amplitude modulation across all band-modulator combinations. Test matrix: 4 modulators  $\times$  9 bands  $\times$  3 levels ( $\ell = 0.0, 0.5, 1.0$ ) = 108 test points, all passing.

## 7.2 Combined Gain Consistency

For the neutral state ( $\ell = 0$  for all),  $G(n) = 1.0$  for all bands. For any single modulator at  $\ell = 1.0$ ,  $G(n) = g_m^n$  exactly.

## 7.3 Saturation Behavior

At  $\ell_m = 1.0$ , receptor saturation reduces effective gain by approximately 33% compared to unsaturated values, preventing runaway amplification.

# Discussion

## 8.1 Emergent State Properties

The 36 gain coefficients were designed individually based on neuroscience literature, yet their multiplicative interaction produces emergent cognitive states (Table ??) that were not explicitly programmed. The ‘‘Focused Work’’ state naturally amplifies attention bands and suppresses distraction, purely from the independent gain profiles interacting.

## 8.2 Comparison with Biological Neuromodulation

Our implementation differs from biology in several key ways:

- **Discrete vs continuous:** We use 9 bands; biology has a continuous frequency spectrum
- **Global vs spatial:** Our gains are spatially uniform; biological neuromodulation varies across brain regions
- **Multiplicative vs complex:** Biological neuromodulation involves receptor subtypes, second messengers, and epigenetic effects
- **4 modulators:** Biology has many more neuromodulators (GABA, glutamate, endocannabinoids, neuropeptides, etc.)

## 8.3 Limitations

- Gain coefficients are *design choices*, not fitted to neural data
- Only 4 modulators; biological systems have dozens

- No spatial variation across modules
- Saturation model is simplified Michaelis-Menten

## 8.4 Future Work

- Learning gain profiles from task performance
- Module-specific (spatial) gain variation
- Additional modulators (GABA, endocannabinoids)
- Integration with the DMT-Inspired Architecture (Paper 46) for pharmacologically-motivated state transitions

## 9 Related Work

- **Dayan & Huys (2008)** [?]: Comprehensive review of computational neuromodulation in decision making
- **Marder (2012)** [?]: Neuromodulation as circuit reconfiguration, founding principle of our approach
- **Hasselmo (2004)** [?]: ACh effects on cortical dynamics, inspiring our ACh gain profile
- **RFA (Paper 43)**: Foundational  $\varphi^n$  band system
- **CFC (Paper 44)**: Cross-frequency coupling that neuromodulators modify

## 10 Conclusion

Computational neuromodulation provides the ARKHEION AGI with *state-dependent processing* without altering connectivity or signal content. By modeling dopamine, serotonin, noradrenaline, and acetylcholine as band-gain profiles (36 coefficients total), we enable the system to shift between cognitive modes—focused work, creative reflection, alert reactivity, and restful consolidation—through a single 4-dimensional level vector. The multiplicative combination produces emergent state properties from independently designed gain profiles, demonstrating that simple computational mechanisms can generate complex cognitive phenomenology. The 481-line implementation integrates seamlessly

with the resonance pipeline and provides the “potentiometers” of the AGI’s cognitive field.

## References

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