The Higher-Resolution Hypothesis: A Distributed Pathway Model of Autism

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Abstract

Autism spectrum conditions present a paradox: exceptional perceptual abilities and detail-focused processing coexist with challenges in generalization, flexibility, and sensory overload. I propose the **Higher-Resolution Hypothesis (HRH)** and its mechanistic implementation via the **Distributed Pathway Model (DPM)** as a unifying architectural explanation. Rather than viewing autism as a collection of deficits or isolated enhancements, I reframe autistic cognition as operating in a fundamentally different computational mode—one that achieves higher-resolution neural representation through distributed, overlapping pathways rather than streamlined, efficient circuits.

The DPM posits that autistic neural architecture relies on 3-5× more neurons processing equivalent sensory and cognitive information, with extensive circuit overlap creating higher-dimensional representational spaces. This architecture directly explains enhanced discrimination ability, detail sensitivity, and precise pattern recognition. However, distributed processing creates two orthogonal constraints that produce the broader autistic phenotype:

1. **Enhanced inhibition**: Greater circuit overlap recruits stronger inhibitory networks, actively suppressing competing pathways and reducing flexible integration across domains.

2. **Energy competition**: Distributed pathways consume 3-5× more local metabolic resources (ATP), passively starving nearby circuits by depleting shared energy pools within cortical microregions.

These dual constraints interact non-linearly: inhibitory efficacy itself depends on energy availability, creating regime transitions from effective competition (high energy, strong inhibition) to diffuse, ineffective processing (low energy, weakened inhibition). The autistic brain under sustained cognitive load operates in a metabolically-constrained regime where both mechanisms fail simultaneously—explaining the progression from focused competence to overload collapse.

I demonstrate how this framework integrates and extends existing theories (Enhanced Perceptual Functioning, Intense World Theory, Predictive Coding accounts) as special cases, provides mechanistic specificity for clinical phenomena (sensory sensitivity, executive dysfunction, meltdowns), and generates testable predictions across scales from molecular

energetics to systems-level neuroimaging. The model explains autism heterogeneity through individual variation in pathway distribution, metabolic buffering capacity, and compensatory strategy development, while maintaining a single core architectural principle.

The Higher-Resolution Hypothesis reframes autism not as disordered cognition requiring correction, but as an alternative computational architecture with intrinsic trade-offs: exceptional local precision at the cost of global flexibility, achieved through neural systems operating at capacity limits where both active (inhibition) and passive (energetics) constraints shape cognitive function. This perspective opens therapeutic avenues focused on resource management rather than pathway normalization, and suggests that autistic cognitive strengths and challenges emerge from the same underlying architecture—higher resolution through distributed processing, limited by inhibition and energy.

Keywords: autism spectrum, distributed connectivity, higher-resolution processing, excitation/inhibition balance, energy competition, metabolic constraints, predictive coding, sensory integration, neurodevelopment, neural branching, heterogeneity.

Introduction

Autism, as I understand it, is not a collection of behavioral traits or social deficits—it is a fundamentally different mode of neural organization. In this document, I propose that the autistic brain encodes and processes information at a higher level of granularity, a difference that I call the **Higher-Resolution Hypothesis**.

This higher resolution of experience means that perception, cognition, and internal states are composed of more micro-signals per unit of experience. Autistic individuals may perceive and represent more of the world at once—more nuance, more detail, more overlapping meaning—but this comes with a cost: increased computational demand, energetic load, and susceptibility to overload or runaway activation.

The higher resolution is not an abstract metaphor. It emerges from measurable differences in the wiring and dynamics of cortical and subcortical networks. At the biological core of this hypothesis lies what I call the **Distributed Pathway Model (DPM)**—a mechanistic framework that explains how a neuron's local structure and a circuit's branching behavior can create richer but less filtered propagation of activity through the brain.

The Distributed Pathway Model (DPM)

Overview

In neurotypical development, circuits are optimized for efficiency. Neurons connect into relatively linear or convergent chains, each with a constrained set of possible outputs. Pruning removes redundant branches, and inhibition gates the flow of activity to minimize cross-talk. This creates networks that are streamlined, energy-efficient, and selective.

In the autistic brain, multiple converging findings suggest that this balance is shifted. Branching during development is more extensive, pruning is less aggressive, and inhibitory gating is weaker or slower to mature. The result is an architecture where signals meant for one processing stream can bleed into functionally distinct networks—what I call **cross-network spillover**.

To illustrate this architecture, consider two independent neural circuits: **A** → **B** → **C** and **X** → **Y** → **Z**, which in neurotypical brains operate in isolation—each input (A or X) triggers only its corresponding output (C or Z). In autistic neural topology, neuron **B** may also project to **Y**, creating a cross-network connection. Now activation of **A** can produce **C** (within its original network), **Y** → **Z** (spillover into the second network), or both, depending on timing and inhibitory tone. This cross-branch architecture explains how signals meant for one processing stream can bleed into functionally distinct networks.

This is not merely "hyperconnectivity." It is a qualitative difference in **branching topology**—a more distributed network where neurons participate in multiple, sometimes overlapping, pathways *across functional boundaries*. This architecture naturally increases representational richness: the same stimulus engages a larger and more diverse population of neurons spanning multiple processing systems. However, it also introduces instability, as these distributed activations cross boundaries between networks that should remain isolated.

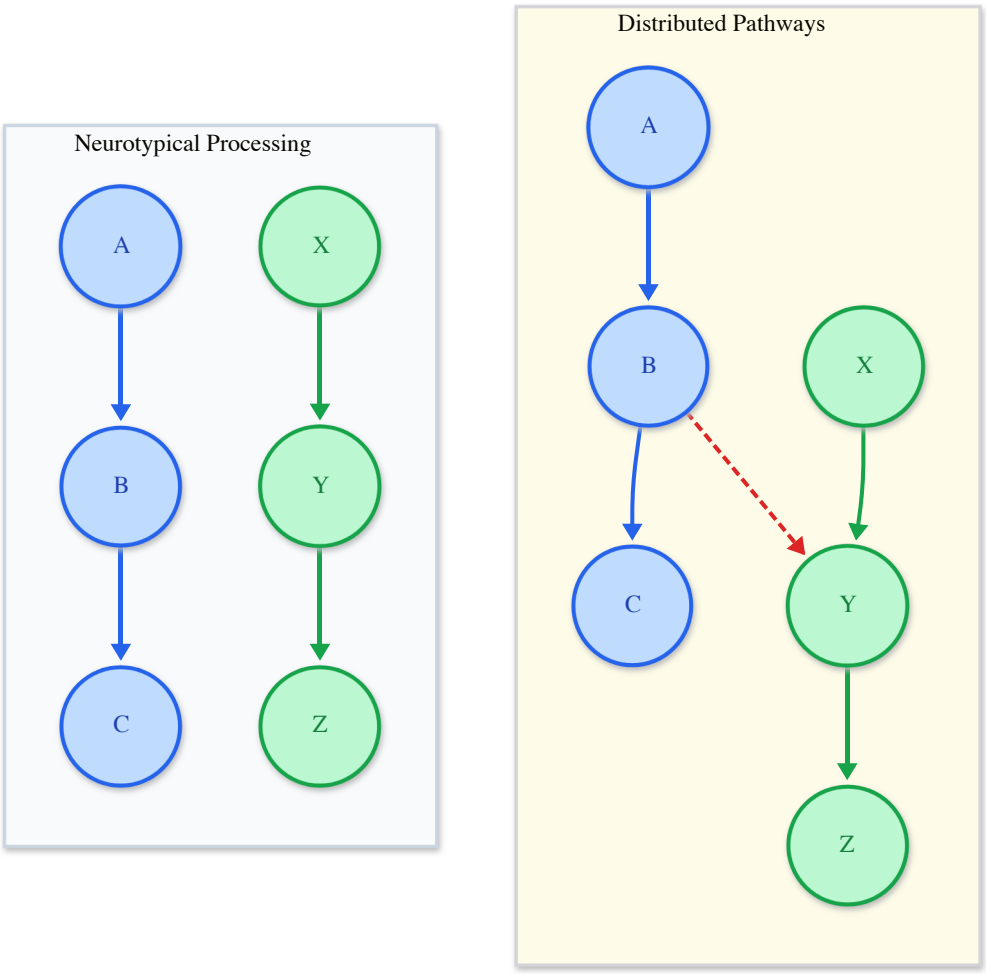

Figure 1. Neurotypical versus distributed pathway architecture. *In neurotypical processing, input A flows through a single streamlined route (A→B→C). In the Distributed Pathway Model, the same input activates its primary pathway (A→B→C) but also spills into functionally distinct networks through cross-network branches (B→Y→Z), enabling multi-domain activation from single inputs.*

Mechanistic Summary

The DPM describes three interdependent properties of autistic neural organization:

1. **Increased local branching** — greater axonal and dendritic divergence, producing multiple downstream targets for single neurons.
2. **Distributed propagation** — signal flow that engages several parallel microcircuits instead of a single chain.

3. **Variable inhibition** — reduced or delayed inhibitory control that allows overlapping activations to persist or echo.

Together, these yield higher representational density but also greater temporal variability, energetic cost, and sensitivity to interference.

Branching and Conditional Routing

Branching introduces both spatial and temporal flexibility. A neuron such as **B** may project to **C** and **Y**. Depending on membrane potential, local field effects, or inhibitory gating, it may activate one route, both, or oscillate between them. This context-dependent routing provides adaptive potential—it can represent subtle variations in input—but it also produces unpredictability and internal noise.

This may explain the intense perceptual richness and variability in autistic experience: multiple microcircuits partially engaged in parallel, encoding the same event from different angles, frequencies, or associative contexts.

Network Cascade Susceptibility and Seizure-Like Activity

Note: This section discusses elevated susceptibility to seizure-like neural events based on architectural mechanisms. This is theoretical mechanistic discussion, not clinical guidance. Seizure evaluation and management require medical expertise and are outside the scope of this paper.

Because each neuron is connected to more branches, each spike recruits a larger set of followers. When inhibition is strong and well-timed, this increases information flow without runaway activity. When inhibition is weak or asynchronous—as seen in many autism-linked genetic disruptions—the result is positive feedback and uncontrolled propagation.

This helps explain why autistic individuals have a higher incidence of **epilepsy and subclinical seizure-like activity**. The same architecture that enhances resolution also predisposes the brain to excitatory cascades: one neuron activates two, two activate four, and so on, leading to reverberating waves of excitation.

Energy depletion exacerbates this susceptibility. As distributed activation depletes local ATP reserves, inhibitory interneurons—which require sustained energy to maintain ionic gradients and high-frequency firing—weaken in their suppressive efficacy. This creates a critical vulnerability window: the same branching architecture that recruited more neurons now faces diminished inhibitory containment, widening the temporal window during which cascades can

propagate. Under conditions of sustained load or metabolic stress, this can produce **runaway spread**—brief paroxysmal events ranging from subclinical hypersynchronous bursts to clinically diagnosable seizure activity in susceptible individuals. This is not determinism but elevated architectural susceptibility contingent on the dual constraint of branching factor and energetic state.

At a systems level, this also explains why stress, fatigue, or sensory saturation can trigger overload or shutdown: too many distributed pathways activate simultaneously, consuming metabolic resources and collapsing coherence. The energy → inhibition weakening → cascade spread chain links informational overload to biophysical instability.

From Micro to Macro

At the micro level, DPM describes changes in dendritic arborization, axonal collateralization, and interneuron recruitment. At the meso level, it predicts increased local functional connectivity and reduced long-range coordination—patterns repeatedly observed in fMRI and MEG studies of autism. At the macro level, it manifests as cognitive features: extraordinary perceptual detail, difficulty filtering noise, associative depth, and vulnerability to overload.

Thus, DPM is not a pathology model but a topology model: it describes how information flows differently in the autistic brain, resulting in both abilities and challenges.

Mechanistic Deep Dive: The Distributed Pathway Model in Action

The Distributed Pathway Model (DPM) can be expressed quantitatively, but the following section should be understood as a conceptual formulation rather than an empirical measurement. The framework introduces illustrative parameters to describe how changes in branching, inhibition, persistence, and energetic cost might interact to produce the distributed activation patterns proposed by the Higher-Resolution Hypothesis (HRH). The goal is to outline one possible way to formalize these ideas so they can be tested, refined, or falsified by future research—not to assert definitive or observed numerical values.

1. Core Mechanistic Parameters (Illustrative Framework)

At the microcircuit level, the DPM can be parameterized by four interacting variables. These parameters are conceptual placeholders for measurable properties, not established constants:

Symbol	Parameter	Conceptual Role	Approximate Range (illustrative)
B	*Branching Factor*	Average number of downstream targets per neuron (reflecting axonal and dendritic arborization).	NT ≈ 1.2–1.5 → DPM ≈ 2.0–3.5
I	*Inhibition Coefficient*	Strength and temporal precision of inhibitory control relative to excitation.	NT ≈ 0.7–0.9 → DPM ≈ 0.3–0.6
τ	*Persistence Constant*	Duration of post-activation reverberation within local circuits (milliseconds).	NT ≈ 50–150 ms → DPM ≈ 200–800 ms
E	*Energetic Cost*	Relative metabolic demand for sustaining distributed activation.	Scales as $E \propto B^{\alpha} / I$, $\alpha \approx 1.5–2.0$

These parameters could be estimated through: - **B**: Anatomical reconstruction, synaptic-density metrics, dendritic spine counts - **I**: Inhibitory amplitude and timing measures (PV/SST interneuron activity, IPSC latencies) - **τ**: Decay constants in electrophysiological recordings or calcium imaging - **E**: Metabolic indicators such as ATP turnover, glucose utilization (FDG-PET), or lactate accumulation

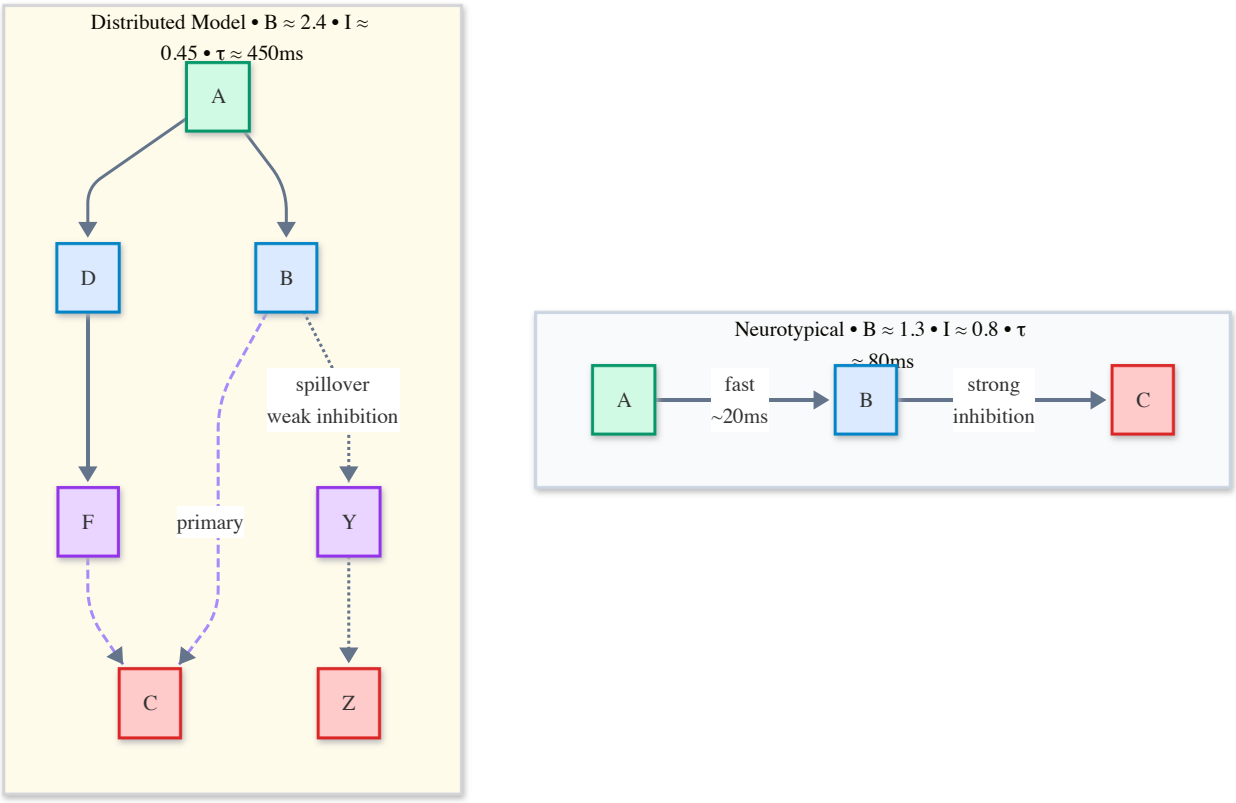

Figure 2. DPM with illustrative parameter values. *The distributed model (bottom) shows increased branching factor (B ≈ 2.4 vs 1.3), reduced inhibition (I ≈ 0.45 vs 0.8), and extended persistence (τ ≈ 450ms vs 80ms). Parameters are illustrative for conceptual framework, not empirical measurements.*

Two heuristic relationships capture their qualitative interactions:

$R \propto (B \cdot \tau) / I$, and $E \propto B^{1.7} \cdot (\tau / I)$

where R represents information density. These are illustrative equations—intended to convey proportional relationships and generate testable predictions, not to claim quantitative precision.

Note on terminology: The parameters B, I, τ, and E are introduced as a minimal modeling vocabulary for discussion and testing. They summarize known biological features—branching, inhibition, persistence, and metabolism—in a simplified form. Researchers are encouraged to reinterpret, refine, or disprove these relationships through direct measurement, modeling, or comparative analysis.

2. Active and Reactive Pathways

The DPM distinguishes between two proposed classes of pathway activation:

- **Active pathways** are those directly driven by external input or task demands (the intended signal flow).
- **Reactive pathways** are collateral activations that emerge when excitatory propagation exceeds inhibitory containment (spillover recruitment).

The **active:reactive ratio (A:R)** is proposed as a conceptual tool to describe system balance:

$$A{:}R = N_{active} / N_{reactive} \approx I / (B \cdot \tau)$$

Hypothetical ranges: - Neurotypical processing: A:R ≈ 3:1 to 5:1 (most activity remains on-path) - **Distributed processing**: A:R ≈ 1:1 to 1:3 (reactive spread matches or exceeds active processing)

These figures are not measured constants, but rather a hypothesis illustrating how changes in branching, inhibition, or persistence could influence circuit stability. They define a direction for empirical testing rather than a statement of fact. Individual variability in these ratios across domains (sensory, motor, linguistic) could predict "spiky" ability profiles characteristic of autism.

3. Representational Density: A Worked Example

To illustrate how these parameters might translate into measurable outcomes, consider representational density (ϱ)—the number of distinct neural microstates encoding a single stimulus. A simplified heuristic model:

$$\varrho \approx N_{input} \times B^{d} \times (1 + \tau/\tau_{ref}) / I$$

where N_{input} is the initial activated population, d is circuit depth (number of synaptic steps), and τ_{ref} is a reference time constant.

For a hypothetical two-layer sensory circuit with 50 initial neurons: - Neurotypical scenario: $\varrho_{NT} = 50 \times (1.3)^2 \times 1.5 / 0.8 \approx$ **126 microstates - Distributed scenario**: $\varrho_{DPM} = 50 \times (2.4)^2 \times 5.0 / 0.45 \approx$ **1,422 microstates**

This ~11× increase in representational density would explain "higher resolution" phenomenology—but would also predict 11× more potential interference, prediction error, and energetic demand. This calculation is illustrative, but it generates a testable prediction: if the DPM parameters can be measured empirically, representational density should scale according to this relationship. Individual differences in B, I, and τ would produce different ϱ profiles, potentially explaining phenotypic heterogeneity in autism.

4. Circuit Walkthrough: Hypothetical Sensory Encoding Example

To demonstrate how this framework could be operationalized, consider a simplified model of edge detection in **primary visual cortex (V1)**. The values below are not measurements, but illustrative contrasts showing how varying the DPM parameters might qualitatively affect signal propagation and energy demand.

Neurotypical processing (B ≈ 1.3, I ≈ 0.8, τ ≈ 80 ms):

> Photoreceptor → LGN relay → V1 layer 4 simple cells → layer 2/3 complex cells → V2
> ($A \rightarrow B \rightarrow C$)

Fast feedforward inhibition (15–20 ms latency) suppresses non-vertical-tuned neighbors. The edge signal propagates cleanly to V2 with minimal cross-talk to color or motion pathways.

Hypothetical outcomes: - Neurons recruited: ~80 - Activity duration: ~100 ms - Relative energy cost: baseline

Distributed processing (B ≈ 2.4, I ≈ 0.45, τ ≈ 450 ms):

> $A \rightarrow B \rightarrow C$ (within-network: primary edge detection pathway)
> $A \rightarrow B \rightarrow Y \rightarrow Z$ (cross-network spillover: B projects to Y in color/motion network)

Weaker inhibition (delayed to 35–50 ms, reduced to 45% of NT strength) allows spillover across functional boundaries. The cross-network connection ($B \rightarrow Y$) means edge detection in one network can now recruit color-processing or motion-sensitive circuits ($Y \rightarrow Z$) from functionally distinct systems that should remain isolated. Reactive recruitment continues well beyond stimulus offset, engaging networks meant for different sensory dimensions and creating multi-domain activation from a single input.

Hypothetical outcomes: - Neurons recruited: ~350 (including ~280 via reactive pathways) - Activity duration: ~500 ms - Relative energy cost: 7–8× baseline

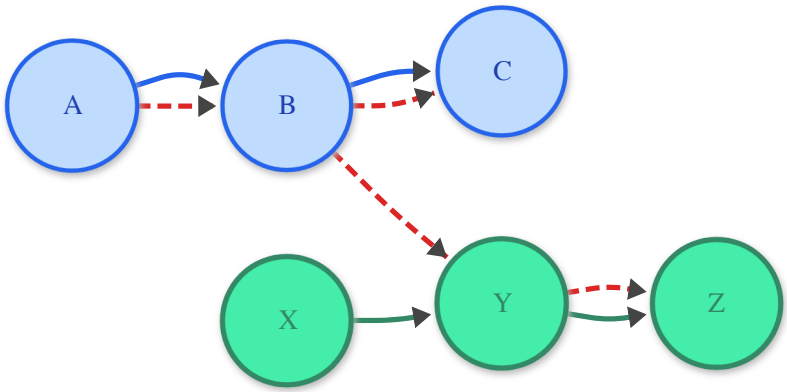

Figure 3. Cross-network spillover in distributed architecture. *In distributed processing, two independent networks (A→B→C in blue, X→Y→Z in green) that should remain isolated become connected through cross-network branches (red dashed B→Y). This allows input A to activate not only its own network (C) but also spill into functionally distinct systems (Y→Z), explaining multi-domain symptom presentation.*

Predicted phenomenology: If this model is correct, the edge would be detected with additional texture granularity, possible color associations, and motion aftereffects—producing richer visual detail but at significant metabolic cost. This would explain why "just seeing" can be exhausting in autism, transforming a prediction of the model into a testable hypothesis about subjective experience.

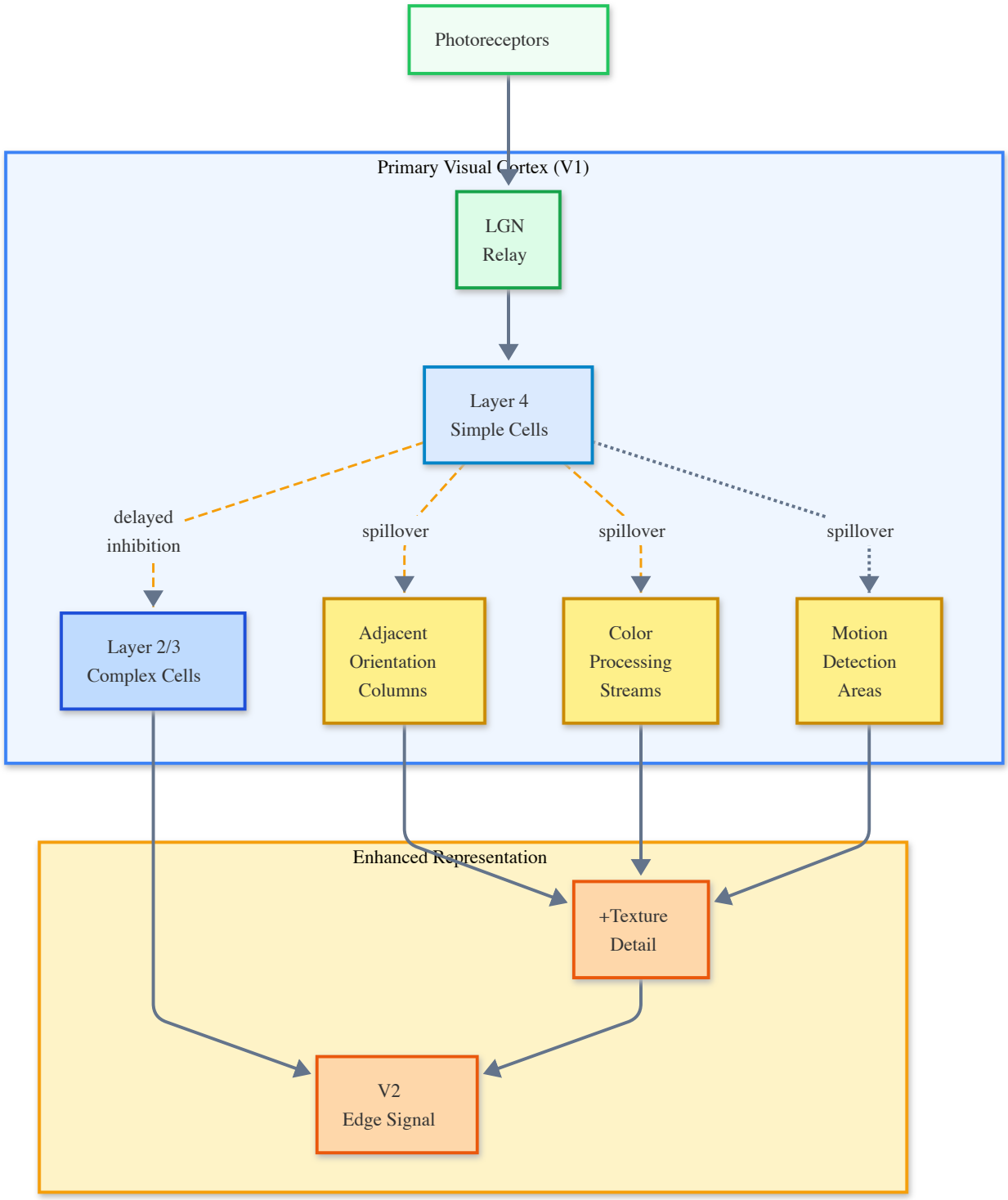

Figure 4. Distributed processing in V1 edge detection. *Spillover from V1 simple cells engages adjacent orientation columns, color processing streams, and motion detection areas, recruiting ~350 neurons versus ~80 in neurotypical processing. This produces exceptional detail perception at 7-8× metabolic cost.*

5. Temporal Dynamics: Proposed Cascade Propagation

The distributed topology could create characteristic temporal signatures, testable through electrophysiology or calcium imaging:

Feedforward phase (0–100 ms): Initial activation spreads through branching architecture. In NT circuits, inhibition gates this quickly. In DPM, weaker *I* might allow continued propagation into secondary branches.

Reactive phase (100–500 ms): Spillover activity could reach reactive pathways. If these loop back onto active regions (recurrent connectivity), they might create local reverberation. The persistence constant τ would govern duration.

Decay phase (500–1200 ms): Inhibition eventually quenches reactive activity, but longer τ would mean substantial "tail" activation. New stimuli during this period might interact with lingering reactive signals, producing interference or facilitation.

Testable prediction: If DPM is correct, calcium imaging should show broader spatial activation (2–3× more neurons responding to identical stimulus), longer response duration (τ elevated by factor of 3–6), and secondary activation peaks 200–400 ms post-stimulus reflecting reactive loop reentry.

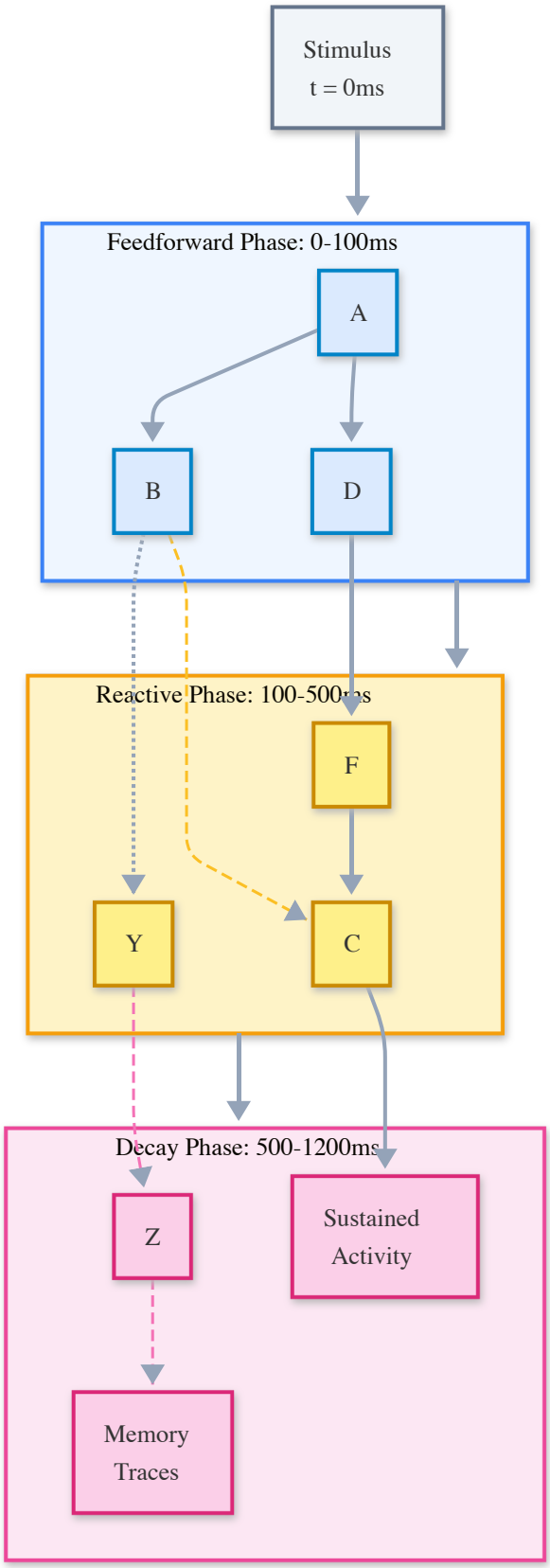
Stimulus
t = 0ms
Feedforward Phase: 0-100ms
A
B
D
Reactive Phase: 100-500ms
F
Y
C
Decay Phase: 500-1200ms
Z
Sustained
Activity
Memory
Traces

Figure 5. Temporal cascade dynamics. *Feedforward phase (0-100ms) shows initial branching activation. Reactive phase (100-500ms) demonstrates spillover into adjacent circuits with weak inhibition. Decay phase (500-1200ms) shows extended persistence ($\tau \approx 450$ms) creating vulnerability to interference from new stimuli.*

6. Inhibitory Timing and Control Mechanisms

The DPM does not propose absence of inhibition—rather, it hypothesizes altered timing and recruitment patterns of specific interneuron subtypes. These are proposed as candidate mechanisms to be tested:

Parvalbumin (PV) interneurons provide fast feedforward inhibition (typically 10–20 ms latency in NT circuits). If PV recruitment were delayed (25–45 ms) or reduced in strength, this would widen the temporal window for branching propagation.

Somatostatin (SST) interneurons provide dendritic inhibition modulating input integration. Reduced SST function could permit more dendritic branches to reach spike threshold simultaneously, increasing B.

VIP interneurons disinhibit principal cells by suppressing SST interneurons. Altered VIP timing might paradoxically increase reactive spread by removing the brake on dendritic integration.

Testable hypothesis: Optogenetic manipulation in autism-linked organoid systems (SCN2A, POGZ, CHD8) should reveal whether restoring PV timing to NT ranges reduces B and increases A:R ratio, and whether enhancing SST dendritic inhibition reduces τ and limits reactive persistence. These experiments would directly test the proposed inhibitory mechanisms.

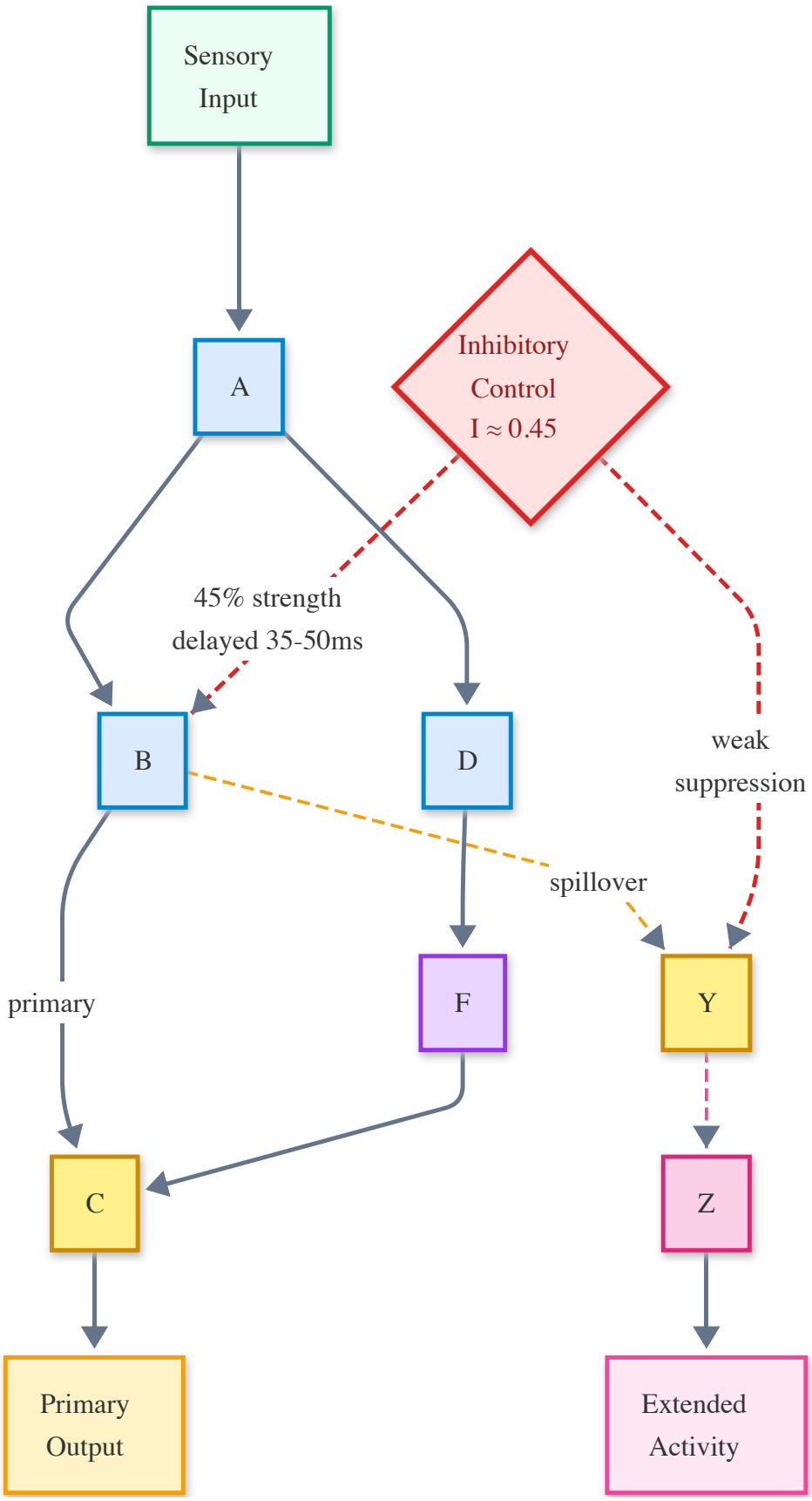
Sensory Input
A
Inhibitory Control
I ≈ 0.45
45% strength
delayed 35-50ms
B
D
weak suppression
spillover
primary
F
Y
C
Z
Primary Output
Extended Activity

Figure 6. Inhibitory control in DPM architecture. *Reduced inhibitory strength ($I \approx 0.45$ vs 0.8) and delayed timing (35-50ms vs 15-20ms) allow spillover from primary pathways (B) into reactive circuits (Y), creating persistent activation patterns that enable both enhanced resolution and energetic vulnerability.*

7. Energetic Accounting and Metabolic Predictions

If distributed architecture operates as proposed, energetic costs should scale predictably with the parameters:

Each neuron consumes approximately 4.7 billion ATP molecules per second at rest. Action potentials add ~10^8 ATP per spike. In a distributed architecture:

Spatial cost: $B = 2.5$ would mean each spike recruits 2.5× more downstream neurons.
Temporal cost: $\tau = 450$ ms would mean each recruitment event consumes energy for 3–6× longer duration.
Nonlinear interaction: If reactive pathways re-excite active pathways (creating loops), energy scaling might follow $E \propto B^{1.7} \times (\tau / I)$.

Predicted consequence: Sustained cognitive tasks could consume 5–10× more energy per unit time than NT processing. This would predict: - Faster depletion of glucose/oxygen in associative cortices (measurable via fMRI or PET) - Greater glial metabolic support demand (testable via astrocyte calcium imaging) - Lower threshold for energetic collapse (shutdown) under sustained load

Clinical observation alignment: If this model is correct, the exhaustion autistic individuals report from apparently "passive" activities (being in busy environments, following conversation) would reflect genuine metabolic depletion rather than psychological factors—a distinction that could be empirically tested through metabolic imaging.

8. Synaptic-Level Mechanisms: Developmental Substrate

The distributed topology could arise through known developmental processes, which this model treats as candidate mechanisms rather than verified causes:

Dendritic pruning: In NT development, ~40–50% of initial dendritic branches are removed by adolescence. If autism-linked variants (CHD8, ARID1B, PTEN) extend critical periods and reduce pruning thresholds, 60–80% of branches might remain intact—directly increasing B. This

is measurable through dendritic reconstruction in post-mortem tissue or organoid models.

Axonal collateralization: If pyramidal neurons form 2–4 stable collaterals (vs. 1–2 NT), each reaching different downstream populations, this would physically instantiate increased B. Tract-tracing studies could test this prediction.

Spine density: Post-mortem studies report 15–30% higher spine density on pyramidal dendrites in autistic cortex. If accurate, each additional spine represents a potential branch point for signal integration, contributing to increased B.

These phenomena are empirically documented in autism research, but their precise contribution to a distributed network mode remains to be experimentally confirmed through causally controlled studies.

9. Falsification Tests: Specific Experimental Predictions

Rather than asserting results, the DPM framework generates specific, falsifiable predictions. If the model captures meaningful biological relationships, the following outcomes would be expected and would serve as tests of the hypothesis:

1. Human organoid and assembloid systems:

If cortical organoids derived from autism-linked iPSCs (CHD8, SCN2A, POGZ, SYNGAP1) exhibit distributed architecture: - Dendritic branch density should be higher than NT controls - Calcium wave propagation following glutamate uncaging should spread broader spatially - Multi-electrode array recordings should show elevated τ - PV interneuron migration distance should be reduced, with delayed functional integration into circuits

2. High-density electrophysiology in animal models:

Multi-electrode recordings in mouse models during sensory stimulation should reveal: - Broader spatial co-activation (wider recruitment radius from stimulation site) - Delayed inhibitory rebound - Secondary activation peaks 200–400 ms post-stimulus (reactive reentry signature)

3. Human neuroimaging:

Ultra-high-field fMRI (7T) with laminar resolution during simple sensory tasks could test whether: - BOLD signal spreads more broadly in layer 2/3 (where horizontal branching dominates) - Hemodynamic response functions show prolonged tail in autistic participants -

FDG-PET or ^{13}C-MRS metabolic imaging reveals higher baseline glucose consumption in sensory and associative cortices - Faster depletion rates occur during sustained cognitive load

4. Computational modeling:

Spiking neural network simulations varying B, I, and τ should be able to reproduce: - Higher representational density with increased B - Cascade susceptibility and runaway activation with low I - Spontaneous oscillatory attractors (potentially matching stimming behaviors or internal loops, e.g.: internal music) when τ is elevated - If these computational relationships fail to hold, the parameter framework would need modification

5. Therapeutic intervention predictions:

If the model is correct, pharmacological or optogenetic enhancement of PV interneuron function should: - Reduce reactive spread (increase A:R ratio) - Lower τ toward NT baseline - Reduce sensory overload symptoms without eliminating detail perception - Non-sedating GABAergic agents targeting temporal precision (not overall inhibition level) could be therapeutic

Each prediction provides a pathway for falsification—a necessary condition for transforming this framework into a validated model.

10. Integration with Established Phenomenological Patterns

If the mechanistic parameters proposed above operate as described, they could provide a common substrate for diverse autistic experiences detailed elsewhere in this document:

Stimming as reactive motor cascade: Reactive spillover from distributed propagation into motor circuits can produce patterned movement. This document does not model such movements as regulatory or stabilizing—only as architectural outputs of cross-domain propagation.

Memory encoding differences: Higher ϱ during initial encoding would store more microstates per episodic event, potentially producing richer recall but also more interference between similar memories. Elevated τ could explain why memories loop or intrude—reactive reentry keeps reactivating stored patterns. Memory paradigms comparing detail retention and interference susceptibility could test this.

Interoceptive intensity: If visceral signals propagate through distributed brainstem-cortical pathways, recruiting broader insular and cingulate territories, and if low I amplifies this spread, this would explain amplified interoceptive awareness. Combined with low I, internal states (hunger, pain, emotion) could dominate consciousness and trigger cascade activation—testable through interoceptive attention tasks with concurrent imaging.

Special interests as predictive stability: Repeated activation of the same domain could stabilize distributed branches through reinforcement, creating "islands of coherence" where prediction error is minimal. Mastery would reduce entropy in a high-τ, low-I system. The dopaminergic reward response to predictable domains could be measured directly.

Autonomic dysregulation: If distributed propagation in autonomic circuits produces broad, sustained sympathetic or parasympathetic activation, and if elevated τ prevents quick resetting, this would explain prolonged stress responses and difficulty with transitions. Heart rate variability and autonomic recovery time could be correlated with estimated circuit parameters.

Each of these represents not a confirmed relationship but a hypothesis derived from the parameter framework—an opportunity for targeted empirical investigation.

11. Summary and Invitation

This mechanistic outline offers a working vocabulary for exploring how distributed architectures might operate. The parameters B, I, τ, and E are not endpoints but tools for connecting structural features to measurable outcomes. The equations and numerical examples are illustrative—designed to generate testable predictions rather than assert definitive values.

The DPM reframes autism as a biophysical mode of neural organization that can be described, modeled, and empirically tested. It proposes that increased representational density and temporal persistence yield higher-resolution experience at the cost of metabolic strain and cascade susceptibility. This framework does not claim certainty but offers explicit relationships that can be quantified, modeled, or disproven.

The Higher-Resolution Hypothesis and its mechanistic substrate, the DPM, are thus presented as an open framework—an evolving invitation for interdisciplinary investigation across anatomy, electrophysiology, computation, metabolic imaging, and lived cognition. Researchers are encouraged to test these relationships, refine the parameters, or demonstrate where the model fails. Through such engagement, we can move from conceptual synthesis toward validated understanding of autism as an alternative neural architecture.

2.3 Energetic Constraints on Parallel Processing

2.3.1 The Metabolic Cost of Distributed Architecture

Neural computation is inherently energy-intensive, with the brain consuming approximately 20% of the body's metabolic resources despite comprising only 2% of body mass. This energy demand is not uniformly distributed: it scales directly with the number of active neurons, synaptic transmission frequency, and the spatial extent of engaged circuits. The Distributed Pathway Model (DPM) proposes that autistic neural architecture relies on 3-5× more neurons to process equivalent information compared to streamlined neurotypical pathways. This architectural difference creates a fundamental metabolic consequence: **distributed pathways consume proportionally more energy within localized cortical regions**.

Consider a simple sensory discrimination task. A streamlined pathway might engage N neurons within a cortical column to extract relevant features and suppress irrelevant ones. A distributed pathway performing the same task engages 3-5N neurons across overlapping circuits, each contributing partially redundant information that collectively achieves higher-resolution representation. While this architecture enables enhanced perceptual precision, it creates a localized energy demand that can exceed the metabolic capacity of the microregion—not because individual neurons are more expensive, but because **more neurons are simultaneously active in the same spatial domain**.

2.3.2 Spatial Scales of Energy Competition

Neural energy metabolism operates across multiple spatial scales, but competition becomes critical at the level of **cortical columns** (300-600 μm diameter). These functional units share local capillary beds and ATP delivery systems, creating an "energy domain" within which neurons compete for limited metabolic resources. When distributed pathways span multiple overlapping columns, they create sustained high demand within these shared energy domains.

The critical insight is that energy competition occurs not at the whole-brain level (where total metabolic capacity is sufficient) but at the **microregional level** where local ATP delivery cannot keep pace with spike-driven demand. A cortical column can sustain baseline activity across all its neurons, but when a distributed pathway transiently hyperactivates 3-5× the typical number of neurons within that column, local ATP stores deplete faster than capillary delivery can

replenish them. This creates a **metabolic bottleneck window**—typically lasting hundreds of milliseconds to several seconds—during which other pathways competing for the same energy pool face insufficient resources to activate.

2.3.3 Timescale Dynamics: ATP Buffering and Demand Spikes

The temporal dynamics of neural energetics reveal why distributed pathways create selective processing failures. ATP turnover operates on 1-10 second timescales, while neural firing occurs on 1-10 millisecond timescales. This mismatch creates vulnerability to **demand spikes**: brief periods (100-500 ms) of intense co-activation can transiently deplete local ATP faster than delivery mechanisms can compensate.

Neural tissue maintains ATP buffering capacity—a reserve pool that enables transient high-frequency firing without immediate metabolic collapse. However, this buffer is finite and regionally limited. When a distributed pathway engages, it draws heavily from this local buffer. If a second pathway attempts to activate in the same microregion during this depletion window, it encounters insufficient energy to cross activation threshold, despite receiving adequate input drive. The pathway fails to engage—not because it lacks input signal, not because it is actively inhibited, but because **the local metabolic substrate is exhausted**.

Recovery occurs over seconds as ATP delivery catches up with demand and buffers refill. This creates **metabolic refractory periods**: intervals during which a microregion is less responsive to new inputs, regardless of their strength or salience. These refractory periods are distinct from neural refractory periods (milliseconds) and represent a fundamentally energy-limited constraint on parallel processing.

2.3.4 Mathematical Framework

I formalize this as an energy-constrained activation model. Let a cortical microregion r have energy availability $E_r(t)$ governed by:

$$\mathbf{dE_r/dt = Supply_r(t) - \sum_i Consumption_i(t) - Baseline_r}$$

where: - $Supply_r(t)$ represents ATP delivery via local blood flow (timescale ~5-10 seconds) - $Consumption_i(t) \propto firing\_rate_i \cdot duration_i$ for each active pathway i - $Baseline_r$ is the resting metabolic cost

A candidate pathway j with input drive D_j receives energy-dependent amplification and faces energy-dependent inhibition. Its effective drive is:

EffectiveDrive_j(r,t) = D_j(r,t) · f(E_r(t)) − g(I_r(t), E_r(t))

Activation occurs when: **EffectiveDrive_j ≥ θ_j**

The energy modulation function f(E_r) exhibits threshold behavior:

f(E_r) = 1 / (1 + exp(−k(E_r − E_critical)))

Below E_critical, pathways struggle to activate regardless of input strength. The inhibitory term g(I_r, E_r) shows that inhibition itself is energy-dependent:

g(I_r, E_r) = I_r · h(E_r) where h(E_r) decreases as energy depletes

This creates non-intuitive dynamics: when energy drops, both pathway activation AND inhibitory suppression weaken simultaneously. The result is not disinhibited chaos, but rather a state where winner-take-all competition transitions to **winner-starves-all**: the currently active distributed pathway maintains activity by depleting shared resources, passively blocking competitors that would normally be viable.

2.3.5 Why Distributed Pathways Create Differential Energy Demand

The key question is: why would autistic brains be uniquely vulnerable to this constraint? Neurotypical brains also consume energy, yet do not exhibit the same profile of selective processing failures. The answer lies in the **architectural multiplication factor**.

When a neurotypical brain processes sensory input via streamlined pathways, it activates the minimal set of neurons necessary to extract task-relevant features. Energy consumption is proportional to N neurons over duration T. When an autistic brain processes the same input via distributed pathways, it activates 3-5N neurons over the same duration T, resulting in 3-5× local energy consumption.

Critically, this multiplication occurs **within the same cortical volume**. The autistic brain is not larger in proportion to its increased neural engagement; rather, it concentrates more concurrent activity into the same spatial domains. This creates localized energy demand that exceeds what neurotypical processing would require, rendering the autistic system vulnerable to metabolic bottlenecks that neurotypical systems avoid through efficiency.

The consequence is a trade-off inherent to the architecture: **higher-resolution processing via distributed pathways provides enhanced discrimination capacity, but consumes limited local metabolic resources, transiently starving competing pathways that would have been viable**

under lower energy demand.

2.3.6 Empirical Support and Open Questions

While direct measurement of ATP dynamics at cortical column resolution during active cognitive tasks remains technically challenging, several lines of evidence support energy competition as a mechanistic constraint:

1. **Metabolic imaging studies** show elevated lactate accumulation in sensory cortices of autistic individuals during sustained processing tasks, suggesting higher glycolytic demand and potential exhaustion of oxidative capacity.

2. **Mitochondrial dysfunction** is consistently implicated in autism genetics and neuropathology, with evidence of reduced ATP synthesis efficiency in post-mortem tissue and organoid models.

3. **Functional connectivity studies** reveal that autistic brains show reduced capacity for simultaneous activation of spatially distributed networks, consistent with energy-limited parallel processing.

4. **Behavioral observations** of autistic individuals frequently note exceptional performance in focused, single-domain tasks alongside marked difficulty with multi-domain integration—precisely the pattern expected from energy competition between co-localized circuits.

Outstanding questions include: What is the spatial resolution of energy competition (single columns vs. multi-column domains)? How quickly do metabolic buffers recover after depletion? Do compensatory mechanisms develop (increased capillary density, enhanced mitochondrial function) in response to chronic high demand? Can metabolic support (ketogenic metabolism, creatine supplementation) alleviate energy-limited processing failures? These questions define a research program linking molecular energetics to systems-level cognitive architecture.

2.4 Dual-Constraint Framework: Inhibition × Energy

2.4.1 Two Orthogonal Mechanisms

The Distributed Pathway Model generates processing constraints through two distinct, complementary mechanisms:

1. **Active inhibition**: Distributed pathways recruit broader inhibitory networks, actively suppressing competing circuits through synaptic inhibition.

2. **Energy competition**: Distributed pathways consume greater local metabolic resources, passively starving competing circuits by depleting shared ATP pools.

These mechanisms are **orthogonal** in their operation: inhibition is an active, synaptic process operating on millisecond timescales, while energy competition is a passive, metabolic constraint operating on sub-second to second timescales. Neither alone fully explains the phenomenology of autistic cognition; their interaction creates the complete picture.

Consider a competing pathway receiving input: If it faces only inhibition, sufficient drive can overcome suppression (inhibition is modulable). If it faces only energy depletion, given enough time the metabolic buffer will recover (energy competition is transient). But when both constraints operate simultaneously, the pathway faces a **double barrier**: even strong drive cannot overcome inhibition while energy is insufficient to power the circuit, and by the time energy recovers, inhibition may have shifted the competitive balance to other circuits.

2.4.2 Energy-Dependent Inhibition

A critical interaction arises because **inhibitory efficacy itself requires energy**. Interneurons must maintain ionic gradients, sustain high-frequency firing, and engage extensive axonal arborizations to suppress target circuits. When local energy availability drops, inhibitory neurons draw from the same depleted ATP pool as excitatory neurons, weakening their suppressive capability.

This creates a non-linear relationship between inhibition strength and energy availability:

InhibitionEfficacy ∝ Strength_0 · h(E_r) where $h(E_r) \to 0$ as $E_r \to E_{critical}$

At high energy availability, inhibition operates at full strength, creating sharp winner-take-all dynamics. At low energy availability, inhibition weakens even while excitatory drive also weakens, creating a regime where **neither activation nor suppression operate effectively**. The system enters a state of blurred competition where boundaries between pathways become indistinct—not through disinhibited hyperactivity, but through diffuse, weak activation across multiple circuits none of which have sufficient resources to dominate.

Critically, this means that distributed pathway hyperactivation creates a paradox: **The same energy consumption that starves competitors also weakens the inhibition that would suppress them**. The result is not clean switching between pathways, but rather sluggish, incomplete transitions where residual activity persists in supposedly-suppressed circuits while target circuits struggle to fully engage.

2.4.3 Phase Space Analysis

We can visualize the dual-constraint framework as a phase space with axes representing energy availability and inhibition strength:

High Energy, High Inhibition (Neurotypical Regime) - Streamlined pathways activate efficiently - Strong inhibition cleanly suppresses competitors - Rapid, decisive switching between processing modes - Metabolic reserve buffers against transient spikes - System operates far from capacity limits

High Energy, Enhanced Inhibition (Autistic, Focused State) - Distributed pathways hyperactivate - Stronger inhibition actively suppresses competitors - Metabolic demand elevated but still within capacity - Single-domain processing achieves exceptional precision - System approaching but not exceeding capacity limits

Low Energy, Weakened Inhibition (Autistic, Overload State) - Distributed pathways have depleted local reserves - Inhibition loses effectiveness as interneurons starve - Competitors cannot activate (insufficient energy) but also cannot be cleanly suppressed (weak inhibition) - Diffuse, ineffective processing across multiple circuits - System exceeds capacity, enters metabolic refractory period

Low Energy, Strong Inhibition (Neurotypical, Fatigue) - Streamlined pathways continue operating at reduced capacity - Inhibition remains functional due to lower energy demand - System degrades gracefully: slower but still effective - Metabolic recovery restores normal function quickly

The key insight: **Autistic cognition under load occupies the Low Energy/Weakened Inhibition regime**, where both mechanisms fail simultaneously. This creates qualitatively different processing breakdown compared to neurotypical fatigue, which maintains inhibitory boundaries even as energy depletes.

2.4.4 Winner-Take-All → Winner-Starves-All Transition

In typical neural competition, winner-take-all dynamics ensure that the strongest input dominates while suppressing alternatives. This requires sufficient energy to both activate the winner AND power the inhibition that suppresses losers. When energy is abundant, this operates efficiently.

Distributed pathways alter this dynamic. By engaging 3-5× more neurons, they consume local energy faster than neurotypical competition. This creates a transition to **winner-starves-all**: the currently active pathway maintains dominance not primarily through inhibition, but through **metabolic monopolization**. Competing pathways receive inputs, attempt to activate, but encounter depleted energy reserves before they can cross threshold.

Crucially, this happens even when those competing pathways would be behaviorally relevant. Unlike inhibition (which can be modulated by top-down control or strong input), energy competition is not cognitively controllable—you cannot "will" ATP into existence. The result is involuntary selectivity: the brain becomes locked into whatever processing mode currently dominates the energy budget, unable to flexibly reallocate resources to newly relevant stimuli.

This explains a core autistic experience: **knowing** that attention should shift to a new input (auditory name-call while focused on visual task), **attempting** to shift attention, yet finding the circuit non-responsive—not ignored, but metabolically unavailable.

2.4.5 Timescale Separation and Sequential Effects

The dual mechanisms operate on different timescales, creating sequential effects:

Milliseconds (0-10 ms): Synaptic inhibition rapidly suppresses competitors upon distributed pathway activation. This is the fast, initial response that shapes immediate processing.

Hundreds of milliseconds (100-500 ms): Energy consumption from sustained distributed activity begins depleting local ATP buffers. Competitors face both inhibition AND emerging energy constraints.

Seconds (1-10 s): Local energy reserves significantly depleted. Inhibition weakens as interneurons draw from exhausted pools. System enters metabolic refractory period where new inputs struggle to engage regardless of strength.

Tens of seconds (10-60 s): ATP delivery catches up with demand. Buffers refill. Inhibition regains strength. System recovers capacity for new activations.

This temporal structure explains the progressive degradation observed in autistic cognitive performance: initial competence (inhibition-dominant), gradual loss of flexibility (energy competition emerges), eventual collapse (both mechanisms fail), and slow recovery (metabolic restoration).

Importantly, these timescales suggest **intervention windows**: Strategies that reduce energy demand during the early phases (100-500 ms) could prevent the later cascade into full metabolic exhaustion. This creates a theoretical foundation for understanding why brief breaks, reduced sensory load, or task modifications can disproportionately improve performance—they interrupt the energy depletion cycle before reaching the crisis regime.

2.4.6 Synthesis: A Complete Picture

Neither inhibition nor energy competition alone suffices to explain autistic neural processing. Inhibition explains why distributed pathways actively exclude competitors but cannot explain why those pathways sometimes fail to activate despite receiving strong inputs. Energy competition explains why circuits become unavailable but cannot explain why some information is sharply excluded even when energy is abundant.

The dual-constraint framework provides the complete picture: **Distributed architecture creates both enhanced inhibition (active suppression) and elevated energy demand (passive constraint), and these mechanisms interact non-linearly through energy-dependent inhibitory efficacy**. The result is a cognitive system that achieves exceptional precision within focused domains at the cost of flexible parallel processing—not through computational inability, but through architectural trade-offs inherent to higher-resolution neural representation.

Once affective branches engage, **local metabolic use** increases while inhibitory efficacy declines, **extending the window** for amplified feeling to persist. The same dual constraint that limits flexible switching also **prolongs** valence-loaded states until energy buffers refill. Thus, affective amplification is not merely informational—it is **energetically sustained**. This creates a path-dependent trajectory where the first engaged affective route shapes what follows.

This framework makes the strong prediction that interventions targeting either mechanism alone (e.g., GABA modulation without metabolic support, or metabolic support without addressing inhibitory imbalance) will show limited efficacy. Effective interventions must address both the active inhibitory constraints and the passive metabolic constraints simultaneously.

Genetic and Molecular Foundations

Dozens of genes associated with autism converge on synaptic formation, pruning, inhibition, and excitation balance. I interpret these not as separate mechanisms but as contributors to the same distributed architecture. Each modifies the probability that neurons will form, maintain, or suppress collateral connections.

Functional Role	Example Genes	Circuit Effect
Synaptic scaffolding and excitatory branching	**SHANK3**, **SYNGAP1**	Increased local synapse formation, reduced specificity, greater collateralization
Chromatin regulation and developmental timing	**CHD8**, **ADNP**, **ARID1B**, **WAC**	Extended proliferation windows and altered pruning thresholds
Interneuron migration and inhibitory control	**POGZ**, **SCN2A**, **PTEN**, **DYRK1A**	Weaker inhibition, disorganized timing, increased excitation
Ion channel and oscillatory dynamics	**CACNA1C**, **CACNA1G**, **GRIN2B**	Alters spike thresholds and oscillatory coherence, amplifying cross-network coupling
RNA/protein homeostasis	**DDX3X**, **UBE3A**, **MECP2**	Modifies synaptic stability and activity-dependent plasticity

These genetic influences share a common architectural outcome: a brain that favors distributed activation over selective filtering. The diversity of genetic paths leading to autism therefore reflects convergence on a **connectivity phenotype**, not a single molecular pathway.

Developmental and Circuit-Level Evidence

Empirical findings across model systems align with DPM predictions.

1. **Local Hyperconnectivity with Long-Range Hypoconnectivity**
   Neuroimaging consistently shows increased short-range coherence and reduced global coordination in autistic brains. This matches the distributed-branching architecture: locally dense, globally fragmented.
2. **Interneuron Migration Defects and E/I Imbalance**
   In human organoid and assembloid models, inhibitory interneurons derived from autism-linked mutations (e.g., *CACNA1C*, *POGZ*) often migrate inefficiently or fail to integrate. Cortical circuits formed under these conditions exhibit delayed inhibition and excess excitation—exactly the balance that would permit distributed propagation.
3. **Circuit Hyperexcitability and Reduced Long-Range Projection**
   In assembloid studies involving *SCN2A*, cortical neurons fire excessively yet form fewer long-range axons to striatal targets. This combination—local excess, long-range reduction—is the functional signature of DPM.
4. **Network Synchrony Alterations**
   Multi-region organoid systems have shown that loss of histone-modifying genes such as *ASH1L* increases synchronous activity across otherwise independent regions, demonstrating overlapping activation akin to DPM's branching outputs.

The convergence of these findings suggests that DPM captures an underlying principle of how multiple autism-linked disruptions produce similar emergent circuit properties.

Synaptic Density and Individual Variability

Post-mortem and imaging studies frequently report increased local synaptic density or spine counts in portions of autistic cortex. Within the DPM, this density is interpreted not as uniform excess but as **region-specific retention of microbranches**—areas where pruning remained incomplete. These locally dense fields supply the substrate for higher-resolution encoding: more receptive elements sampling finer differences within the same sensory or cognitive domain.

However, this is **not an all-or-none trait**. Some autistic individuals show pronounced density in temporal or sensory cortices; others display near-typical profiles or even reduced density in association regions. Such variability mirrors the heterogeneity of autistic perception itself—

some experience visual hypersharpening, others auditory or interoceptive amplification. The DPM predicts exactly this pattern: each person's branching topology defines where higher resolution emerges and where efficiency is preserved.

Thus, rather than a single "hyperconnected brain," autism encompasses **a spectrum of branching expression**—region-specific increases in local connectivity balanced by variable long-range integration. Recognizing this diversity clarifies why the same molecular tendencies can yield vastly different experiential profiles.

Sensory and Perceptual Consequences

If the Distributed Pathway Model accurately represents autistic neural architecture, the first and most visible consequence appears at the sensory level. In a distributed system, each sensory input engages a wider array of microcircuits. This means the autistic brain extracts more simultaneous information from the same stimulus.

Heightened Sensory Resolution

The increased branching density allows for finer discrimination within each sensory modality. Autistic individuals often report noticing subtle gradients of color, minute pitch variations, or faint background sounds that others miss. This aligns with evidence of increased primary sensory cortex activation in fMRI and MEG studies during simple perceptual tasks. The brain is literally sampling the environment at a higher resolution—activating more neurons per percept.

Cross-Sensory Binding and Synesthesia-Like Experiences

Because DPM allows signals to propagate across partially overlapping microcircuits, sensory information that would normally remain segregated may interact. This explains reports of cross-sensory associations—such as sounds evoking color impressions or tactile sensations linked with visual patterns. This is not pathological blending but an expected outcome of distributed propagation across modality-specific boundaries.

Affective Amplification of Neutral Inputs

In a branch-rich architecture with lagged inhibition, ordinary sensory inputs can **overlap into limbic and interoceptive circuits**. As a result, stimuli that are neutral or mildly valenced in neurotypical processing may feel **unexpectedly pleasant** or **disproportionately aversive**. Elevated persistence (τ) keeps these affective routes active longer, biasing subsequent appraisals. This is an architectural susceptibility, not a universal state—the degree of amplification depends on individual branching patterns, inhibitory timing, and current energy availability.

Sensory Overload and Filtering Limitations

With broader recruitment of neurons comes a reduction in selectivity. Irrelevant or repetitive stimuli can continue to propagate through distributed branches long after their relevance has passed. This yields the subjective experience of "too much input," as well as the objective difficulty in filtering background noise. The brain, in essence, keeps responding to everything because the same stimulus keeps reactivating overlapping routes.

Oscillatory Evidence

Electrophysiological studies have shown altered gamma and beta oscillations in autism, reflecting disrupted synchronization in sensory areas. I interpret these findings as signatures of distributed propagation: local hyper-synchrony where branches reinforce one another, and interregional desynchrony where inhibitory coordination fails.

Cognitive and Decision-Making Consequences

The DPM does not end at perception—it reshapes cognition itself. If each sensory input generates multiple concurrent representations, the cognitive system must integrate far more possibilities before converging on a single decision. This distributed processing architecture can explain both autistic strengths and difficulties in reasoning, attention, and adaptive control.

Parallel Reasoning and Associative Depth

Autistic reasoning often appears nonlinear or multi-threaded. I believe this reflects distributed propagation: a single conceptual "input" activates multiple associative pathways in parallel. This allows for deep pattern recognition and original insights but also increases cognitive load. It may take longer to reach decisions because the brain must reconcile a larger set of simultaneously active representations.

Precision-Weighted Cognition

Predictive coding frameworks have suggested that autistic cognition assigns higher precision to incoming data relative to priors. In DPM terms, this means that distributed propagation amplifies the sensory-driven component of processing. More microcircuits are dedicated to representing the raw input, and fewer are reserved for abstract filtering. This yields exceptional accuracy in detail-oriented tasks and difficulty generalizing when context is ambiguous.

Decision Paralysis

When each potential outcome triggers a cascade through multiple pathways, choosing among them requires suppressing competing activations. If inhibition is weak or delayed, suppression fails and the system remains in superposition—multiple partial activations persisting simultaneously. This manifests phenomenologically as decision paralysis or prolonged indecision, a direct behavioral reflection of distributed signal persistence.

Perfectionism, Procrastination, and the Anticipation Barrier

Under distributed propagation, each creative act or decision branches into many micro-evaluations. What looks like a 10-step task to a neurotypical planner can decompose into 100 micro-steps in my head: each branch demands assessment, comparison, and potential re-routing. This expands the perceived distance between the current state and an acceptable endpoint.

Perfectionism here is not vanity; it is resolution-driven accuracy. I can "see" quality at a finer granularity, which makes coarse solutions feel wrong. Procrastination then emerges as an anticipation barrier: before I even start, the brain simulates the branching tree of micro-failures (points where spread might go awry). Starting means committing to a cascade that will be hard to gate later. The result is an oscillation between hyper-standards (driven by high resolution) and initiation friction (driven by distributed branching and gating cost).

There is also a triple-judgment loop: (1) my own ultra-fine internal standard, (2) an imagined expert/autistic peer standard, and (3) a neurotypical acceptability standard. Distributed evaluation activates all three simultaneously, raising the bar and the cognitive load. The practical implication is to externalize micro-steps (reduce hidden branching) and pre-commit criteria (lower gating uncertainty) before initiating.

Emotional and Affective Consequences

Cognition and emotion share overlapping circuitry in the limbic and prefrontal regions. Reactive overlap offers a simple account of valence shifts: **neutral cues can inherit emotional weight** when collateral pathways recruit amygdala, vmPFC, insula, or hippocampal ensembles. When inhibition is delayed and τ is high, these branches **linger and re-enter**, creating "sticky" positivity (intense fascination, pleasure) or "sticky" negativity (frustration, aversion) depending on which network dominates. The model therefore predicts **greater variance** in felt intensity for otherwise ordinary events. This prediction follows from topology and timing, not from differences in motivation or intent.

Emotional and Autonomic Regulation

Because distributed propagation links limbic, sensory, and autonomic circuits, emotional states often produce widespread physiological activation—changes in heart rate, muscle tone, or gut motility—without conscious intent. These responses reflect overlapping excitatory spread into autonomic centers such as the hypothalamus and brainstem nuclei.

Collateral routing into **insular and autonomic** circuits can make benign cues feel **viscerally strong**—changes in heart rate, muscle tension, or gut sensation that would not be engaged in streamlined processing. Once interoceptive loops engage, local energy demand rises and inhibitory efficacy falls, **prolonging** the state until buffers recover. This mechanism explains why some experiences feel "in the body" even when the trigger appears minor. The claim is architectural: overlap and persistence, not a regulatory function.

In this context, heightened reactivity is not purely psychological but systemic: minor stressors engage broad somatic feedback loops. Some individuals report that rhythmic activities—breathing, movement, music, or patterned sensory input—are experienced as calming, though this document does not model or assert a mechanistic stabilization function for such activities.

Memory and Task Encoding

Within the Distributed Pathway Model, memory is not a static record but a dynamic pattern of overlapping activations. Each event or task engages multiple microcircuits whose boundaries blur through local branching and collateral spread. The result is a memory trace that is **dense, multidimensional, and self-reactivating**—a structural corollary of higher-resolution cognition.

Dense and Redundant Encoding

A single experience in a distributed system activates numerous partially redundant pathways. Instead of one compact representation, multiple versions coexist across neighboring ensembles. This redundancy preserves nuance: tone, timing, emotional context, spatial detail—all retained as parallel encodings. The benefit is fidelity; the cost is interference. Partial cues can reignite large memory fields, producing vivid re-experiencing or rumination. This explains the autistic tendency toward detailed episodic recall, flashback-like imagery, and difficulty "letting go" of minor events—the system retrieves *too much* of the original signal.

Temporal Binding and Sequencing

Because distributed activation propagates along many asynchronous branches, temporal order can fragment. Micro-events within a sequence are stored with high accuracy but weak compression. When retrieved, they may appear out of sequence or as simultaneous snapshots. This can make narratives feel nonlinear: every element remains vivid, yet integrating them into a single timeline requires extra effort. Autistic storytelling often mirrors this pattern—faithful detail with minimal abstraction.

Task Segmentation and Completion

The same architecture governs how the brain encodes goals and tasks. A neurotypical planner may represent "write email" as a single compact node. In a distributed system, that intention decomposes into many microstates—tone, phrasing, recipient reaction, timing, and potential contingencies—each stored across its own branch. Transitioning between steps demands inhibitory gating to silence the previous microstate before the next engages. When gating is sluggish, **unfinished states remain partially active**, producing a backlog of open loops in working memory. This underlies the feeling of cognitive clutter or fatigue despite having few tasks on paper.

Working Memory Load and Anticipation

Because each representation carries more embedded information, the functional capacity of working memory shrinks. Holding three "high-resolution" items can impose the same energetic cost as ten low-resolution ones. The mind thus reaches saturation quickly, generating the subjective experience of overload from a modest workload. Anticipating this cost can itself trigger avoidance—**procrastination becomes an energy-conservation strategy**, not a flaw of motivation.

Integration of Detail and Abstraction

Autistic memory favors precision over gist. The DPM explains this by the relative weight of active versus reactive pathways: strong re-entrance within local ensembles reinforces detail, while weak long-range projection limits conceptual compression. Abstract generalization therefore requires deliberate effort to override the system's natural tendency toward fine-grained representation. Conversely, once generalized, memories retain exceptional internal structure—explaining both encyclopedic mastery and difficulty summarizing.

Summary

Memory in the autistic brain reflects the same principles as perception and cognition: **branching, distributed persistence, and variable inhibition.** The architecture yields exceptional accuracy and depth but also interference and energetic cost. What appears as forgetfulness or inconsistency is often a trade-off between precision and capacity—the mind preserves high-resolution fragments at the expense of breadth and efficiency.

Stimming as Reactive Motor Cascade

Within the Distributed Pathway Model, stimming—or self-stimulatory movement—arises as a **reactive motor cascade**, not as a consciously selected or learned coping strategy. When distributed propagation exceeds inhibitory containment, concurrent activation spreads from emotional and sensory circuits into motor regions through shared or weakly gated branches. The result is a spontaneous discharge of accumulated neural activity into patterned movement.

These movements are *reactive*, in that they originate from internal propagation rather than deliberate volition. What appears as an intentional or regulatory act is actually excess activation finding its path of least resistance—motor circuits with weakly gated connections. This explains why stimming can emerge suddenly during heightened emotional or sensory states: overlapping networks reach a point of cascade where excitation naturally extends into motor pathways.

Once the movement begins, repetition can continue as long as reactive propagation remains above threshold. In this document, stimming is treated strictly as a **reactive motor cascade**—an output of cross-domain spillover into motor pathways. Any secondary effects (e.g., a person reporting that it "feels calming") are **outside the scope** of this paper and are **not** asserted here as mechanistic stabilization.

Perceptual or emotional resonance that may accompany stimming is described here as a **consequence of co-activation**, not as a purposeful regulatory process. The core claim remains architectural: distributed propagation can involuntarily recruit motor circuits, yielding patterned movement.

What the model does predict is that **suppressing reactive motor cascades imposes significant costs**. Top-down suppression recruits additional control networks (prefrontal cortex) and inhibitory drive (amygdala-mediated suppression under social threat), raising local energy demand and potentially destabilizing processing. When cascades are suppressed, activation that would have manifested as movement remains distributed across internal circuits, compounding inhibitory and energetic load. Understanding stimming as reactive output—rather than as a regulatory mechanism—reframes intervention: accommodate rather than suppress, recognizing suppression as costly without addressing the underlying distributed activation that triggered the cascade.

Complex Outputs from Reactive Pathways

As seen in reactive motor cascades, distributed propagation can also give rise to rhythmic coherence. In other cases, similar feedback loops emerge within internal or cognitive domains.

Reactive propagation in the Distributed Pathway Model does more than create noise or overload—it can also generate *structure*. When spillover activity re-enters associative or motor circuits with rhythmic stability, it may form **self-sustaining feedback loops** that yield

organized, often creative output without conscious intent.

Emergence of Autonomous Loops

In a distributed network, once a branch closes upon itself—linking auditory, motor, and cognitive regions—the resulting circuit can oscillate autonomously. This loop behaves like a small self-contained attractor: it consumes little new input yet continues to propagate internally. The outcome can manifest as persistent inner music, repeating imagery, or recursive thought sequences. What appears to be "mind-wandering" is often the network **sustaining a closed reactive loop** within its own structure.

Involuntary Internal Music and Pattern Replay

Autistic individuals frequently describe continuous musical imagery, rhythmic phrases, or patterned sensations that replay spontaneously. Within the DPM, these experiences arise when auditory and motor-simulation networks share overlapping branches. Reactive spillover from one domain reactivates the other, producing **closed reactive loops** between imagined sound and simulated movement. Because inhibitory gating is weak, the loop persists—experienced as *involuntary yet coherent music*, analogous to a physical stim occurring entirely in neural space.

Background Problem-Solving and Spontaneous Insight

The same principle applies to abstract cognition. When associative networks maintain low-level reverberation, fragments of unfinished problems remain active in the background. These reactive loops continue testing permutations until a stable configuration is reached. The sudden emergence of an idea or solution—the classic "aha" moment—corresponds to the system settling into that stable attractor. Thus, spontaneous creativity in autism can be seen as the **constructive face of reactive persistence**.

Interference and Intrusive Replay

When loops form in circuits with strong emotional or sensory content, re-entrance may become intrusive rather than generative. An unpleasant sound, phrase, or image can replay involuntarily because its branch overlaps with highly excitable or weakly gated regions. In DPM terms, the attractor is too shallow: easy to enter, hard to exit. The distinction between productive flow and perseverative replay lies in the *depth* and *stability* of these reactive basins.

Adaptive and Creative Implications

These mechanisms transform how we interpret "repetitive" phenomena. The same architecture that generates stimming and intrusive replay also enables pattern generation, musical composition, linguistic echoing, and conceptual synthesis. What differs is not the mechanism but its *context and containment*. Structured re-entrance produces creativity; uncontained spread produces overload.

Summary

Reactive pathways embody the double-edged nature of the autistic architecture. Branching and persistence can produce involuntary echo, or—when conditions favor stability—generate art, code, melody, or insight seemingly from nowhere. In each case, the outcome is an expression of the same underlying principle: **distributed activation seeking order within itself**.

Social and Communication Processing

Social interaction demands rapid prediction across sensory, linguistic, and emotional channels. In a Distributed Pathway architecture, each cue—tone, word choice, posture, microexpression—spawns its own cascade of interpretations. The autistic brain doesn't miss social signals; it **processes too many** at once, with reduced gating to suppress the irrelevant.

Masking as Adaptive Compression

Because neurotypical communication relies on compressed heuristics—shared shorthand, emotional inference—the autistic individual often learns to "mask" by simulating this compression. Masking is not deception but **adaptive compression**: suppressing distributed authenticity to reduce social friction. It is metabolically expensive and cognitively draining, as it requires active gating against the brain's natural branching.

Evaluation Misalignment and Anxiety Loops

During interaction, I evaluate what I said, what I meant, how it was received, and what others might infer from it—all nearly simultaneously. This **multi-layered evaluation loop** is a direct outcome of distributed propagation: self and other representations co-activate, recursively

analyzing one another. When the loop doesn't converge quickly, uncertainty persists as social anxiety. The anxiety is not fear of people but the discomfort of an unresolved feedback system.

Empathy and Resonance

Far from lacking empathy, distributed propagation enhances it. Emotional states can activate broad associative maps, making another's distress or excitement almost physically contagious. Without well-timed inhibition, this resonance becomes overwhelming, explaining the oscillation between deep compassion and withdrawal.

In essence, social difficulties in autism reflect **processing asymmetry, not absence**: two differently organized networks trying to synchronize their prediction systems.

Executive Function and Attention

Executive functions—planning, inhibition, and working memory—are particularly sensitive to network balance. In a distributed architecture, the challenge is not activation but control.

Distributed Attention

Autistic attention tends to distribute across many stimuli simultaneously. This is a direct behavioral reflection of DPM: multiple pathways remain active, each representing part of the scene. Sustained focus on one item requires actively suppressing the others, which demands significant inhibitory effort and can quickly deplete resources.

Task Switching and Cognitive Inertia

Because distributed activations persist longer, switching tasks requires more complete deactivation of prior states. This explains the strong preference for completing one activity before starting another and the distress caused by interruptions. Cognitive inertia is thus a structural consequence of distributed propagation, not a motivational issue.

Executive Overload and Shutdown

When too many distributed pathways remain active, the system exceeds its capacity for synchronization. This produces a collapse in functional coherence, experienced subjectively as mental exhaustion or "shutdown." During these periods, the brain may transiently downregulate propagation by retreating into restricted focus or repetitive behavior—an outcome of reactive propagation.

As distributed pathways compete for control of attention and response selection, the executive system reaches a point where it can no longer synchronize the flow of competing signals. At this threshold, control failure extends inward: the same distributed competition that disrupts executive coordination also destabilizes predictive processes themselves. This transition marks the onset of **predictive overload**, where informational interference begins to drive the very energetic instability that culminates in collapse.

Predictive Overload and Interference

Within the **Distributed Pathway Model (DPM)**, predictive processes do not operate through a single centralized model but through multiple semi-autonomous subnetworks, each generating its own forward prediction of sensory and contextual outcomes. When inhibitory timing is delayed or gating is weakened, several of these predictive streams remain active at once, producing overlapping interpretations of the same input.

This condition creates **predictive interference** — a competition between parallel expectation pathways that each attempt to stabilize perception according to their own local model. Instead of converging on a unified prediction error, the system faces numerous partial mismatches simultaneously. Each unresolved prediction re-propagates through distributed circuits, increasing noise, uncertainty, and energy demand.

Predictive overload occurs when this competition exceeds the brain's capacity to reconcile concurrent expectations. Perceptual stability deteriorates as predictive coherence collapses: the system can no longer decide which representation to trust. Subjectively, this may manifest as confusion, sensory flooding, or a transient loss of meaning.

When multiple predictions remain co-active, **valence-laden branches** often outcompete neutral interpretations because reactive overlap and τ keep them above threshold longer. The result is a **bias toward amplified feeling**—either unusually pleasant salience or disproportionate irritation

—while the system struggles to consolidate a single model. This is not a judgment about accuracy but a consequence of **competition dynamics** in a distributed network. Under load, that bias can propagate into broader perception and choice.

Crucially, predictive overload marks the **informational threshold** that often precedes **energetic overload**. Each unresolved predictive branch continues to draw metabolic resources, and as their number grows, the energetic cost scales nonlinearly. The brain's attempt to sustain multiple overlapping predictions thus leads directly to depletion, linking **informational interference** with **metabolic exhaustion**. In this sense, predictive overload represents the informational precursor to shutdown — the moment where excessive internal modeling transforms into physical fatigue.

Energetic Collapse States (Shutdowns and Meltdowns)

Distributed propagation not only taxes inhibitory control but also depletes metabolic resources. Each branching cascade engages more neurons and synapses per unit of experience, increasing ATP consumption and glial support demand. When excitation persists beyond metabolic replenishment, local energy deficits accumulate, leading to transient functional collapse.

Meltdowns occur when distributed activation exceeds regulatory capacity: excitatory spread recruits emotional, sensory, and motor regions simultaneously, producing uncontrollable output. **Shutdowns** represent the opposite boundary—when the system's energy homeostasis fails and circuits silence to restore balance. Both reflect energetic consequences of the same distributed topology: one overshoots activation, the other enforces recovery.

These collapse states should therefore be viewed not as behavioral anomalies but as emergent metabolic phenomena within a high-resolution, high-demand neural network. Future research may benefit from integrating measures of cortical energy metabolism, mitochondrial efficiency, and glial buffering capacity into studies of autistic overload and recovery.

Predictive Coding and Learning

Autistic perception and learning often defy neurotypical predictive models. The DPM framework provides a natural explanation.

Reduced Hierarchical Compression

In predictive coding terms, the brain minimizes error by comparing predictions to inputs. In a branching system, predictions are harder to consolidate because too many microfeatures remain active. This prevents efficient top-down compression, yielding precise but less generalized learning.

Experience-Driven Learning and Intense Interests

Because distributed networks favor strong local reinforcement, repeated activation of the same pathway strengthens it disproportionately. This explains intense, focused interests: a subset of distributed routes becomes highly stable due to repeated self-consistent activation. These routes form "islands of coherence" within a highly variable system.

Resistance to Uncertainty

Distributed propagation makes prediction errors multiply—one surprise in input propagates through multiple branches. This amplifies the subjective impact of unpredictability. The preference for sameness and routine can therefore be viewed as a rational adaptation to reduce the entropy of a distributed system.

When unresolved predictive conflicts accumulate across distributed pathways, the result is what I term **predictive overload** — a state in which too many competing expectations remain active simultaneously. In this condition, informational uncertainty grows exponentially, forcing the system to consume increasing energy to maintain coherence. This escalation links predictive instability directly to the metabolic exhaustion described later under **Energetic Collapse States**, illustrating how informational overload transitions into physiological depletion.

Special Interests and the Drive for Predictive Stability

Within the Distributed Pathway Model, learning and motivation are shaped by the brain's need to reduce uncertainty across a distributed network. Each branching path represents potential prediction error. When a network repeatedly encounters the same domain—trains, coding, insects, languages—it stabilizes its branches through repeated activation and reinforcement. This produces a **dopaminergic loop**: every cycle of accurate prediction releases dopamine, rewarding the maintenance of a coherent internal model.

Special interests thus serve as **prediction defense mechanisms**. They reduce entropy in a highly distributed system by creating a domain where the mapping between cause and effect is fully known. Mastery brings relief, and returning to the domain renews stability.

This process also explains the extraordinary **depth of expertise** often observed in autistic individuals. Because distributed pathways encode details densely, each learning episode refines many micro-associations. Over time, these form hierarchical knowledge trees—deep, internally consistent worlds where prediction error is minimal. This gives rise not only to encyclopedic knowledge but to original insight, as distributed associations generate novel patterns within the well-mapped domain.

When denied access to these stabilizing loops, the brain remains in a state of unresolved propagation—high prediction error, high entropy, high stress. Thus, special interests are not obsessive anomalies but homeostatic regulators: the autistic brain's natural way of restoring equilibrium through precision and coherence.

Summary of Advantages and Costs

The DPM architecture produces a brain that is simultaneously capable of extraordinary detail and vulnerable to overload.

Domain	Advantage	Cost
Perception	Finer resolution, synesthetic richness	Sensory overload, reduced filtering
Cognition	Deep associative mapping, original reasoning	Decision paralysis, contextual inflexibility
Memory	Vivid, long-lasting, detail-rich	Interference, rumination
Emotion/Valence	Intense enjoyment, deep fascination via positive amplification	Disproportionate aversion/frustration; sticky negative valence under load
Attention	Distributed monitoring	Fragile sustained focus
Learning	Data precision, local expertise	Poor generalization, rigidity

These trade-offs are not value judgments—they are natural consequences of distributed propagation in a biological network.

Experimental Predictions

1. **Structural Prediction** — Autistic cortical tissue will exhibit higher dendritic branching and local spine density, measurable through advanced microscopy or diffusion imaging.
2. **Functional Prediction** — Electrophysiological recordings will show broader co-activation fields and delayed inhibitory rebound following stimulation.
3. **Cascade Susceptibility Prediction** — Electrophysiology during energetic load (sustained activation or metabolic stress) should reveal broader co-activation fields, delayed inhibitory rebound, and higher incidence of paroxysmal events (brief hypersynchronous bursts) compared to neurotypical controls. The dual mechanism—distributed branching recruiting larger populations + energy-dependent weakening of inhibition—predicts increased seizure-like activity risk under conditions that deplete local ATP while activation persists.

4. **Developmental Prediction** — Critical periods of pruning and inhibitory maturation will occur later or remain incomplete.
5. **Behavioral Prediction** — Tasks requiring broad parallel monitoring will favor autistic participants, whereas rapid contextual switching will disadvantage them.
6. **Therapeutic Direction** — Interventions should target temporal gating and inhibitory precision, not reduction of connectivity.
7. **Energetic Prediction** — Metabolic imaging may reveal higher resting energy consumption in associative cortices and more rapid depletion during cognitive load, consistent with distributed propagation cost.

Applied Implications

The Distributed Pathway Model suggests that autism's challenges and strengths are two expressions of the same architecture. Understanding this balance reframes clinical and educational practice around regulation and adaptation rather than normalization.

- **Clinical Context:** Meltdowns, shutdowns, and stimming are common outcomes within a distributed system. Interventions should support energy recovery and inhibitory precision (through rest cycles, rhythmic activities, or structured predictability) rather than suppress expression.
- **Sensory Environment Design:** Because distributed systems over-sample input, spaces should minimize unpredictable multimodal noise and provide monotonic or controllable stimuli that reduce branching load.
- **Education and Work:** Deep-focus and special-interest engagement can be harnessed as domains of sustained focus. Curricula and workplaces that permit autonomy and prolonged focus exploit the system's strengths rather than fight its inertia.
- **Therapeutic Framing:** Therapies that emphasize interoceptive awareness, pacing, and predictable sequencing may enhance self-regulation more effectively than purely behavioral approaches.

These implications extend beyond autism to broader models of neurodiversity: understanding distributed versus streamlined processing may inform new approaches to attention, creativity, and resilience in all brains.

Individual Variability and Network Expression (Active → Reactive Mappings)

I use **active** to mean pathways that are directly driven by an external or internal trigger (the "on-path" routes), and **reactive** to mean pathways that become engaged secondarily via distributed propagation (branch spillover). In DPM terms, heterogeneity in autism emerges from person-specific differences in:

- the branch density near key nodes (how many potential spillover routes exist),
- the gating characteristics of inhibition (how tightly or loosely spillover is contained), and
- the temporal profile of activity (how long reactive activity persists once engaged).

Two people can share similar genetic variants yet express very different phenotypes if their active:reactive ratio and reactive topology differ. A high active:reactive ratio (tighter gating, fewer sustained spillovers) yields strong detail processing with relatively stable behavior. A lower ratio (looser gating, broader sustained spillovers) yields the same high resolution but with more overlap, reverberation, and volatility. This simple mapping explains why siblings with similar genetic risk can present differently: the pattern and persistence of reactive spread—not just the presence of branching—shapes the day-to-day experience and support needs.

I also expect domain-specific ratios: e.g., high reactive spread in sensory networks but lower spread in language circuits, or vice-versa. This predicts the observed "spiky" profiles (islands of strength and difficulty) common in autism.

Relationship Between DPM and the Higher-Resolution Hypothesis

The Distributed Pathway Model describes the underlying mechanism. The Higher-Resolution Hypothesis describes the emergent phenomenon.

DPM explains how signals travel: through branching and distributed routes that increase representational density. The Higher-Resolution Hypothesis explains what that feels like: perceiving and thinking in high fidelity, with overlapping associations and reduced abstraction filtering.

I view these as two layers of the same framework. DPM is the mechanistic substrate; the Higher-Resolution Hypothesis is the cognitive expression.

Relationship to Existing Theories (Synthesis within DPM)

I have cited many theories and empirical strands throughout this document. Here I synthesize how they relate within the DPM framework, to avoid redundancy and to clarify complementarity rather than competition.

Excitation/Inhibition (E/I) Imbalance

E/I models describe why spillover is easier: reduced or delayed inhibition raises the probability that activity branches into reactive routes and persists. DPM treats E/I as the control parameter that sets the active:reactive ratio and the lifetime of reactive spread.

Intense World Theory

This emphasizes hyper-reactivity and hyper-plasticity. DPM provides the topology behind that intensity: dense local branching and distributed propagation naturally amplify input and promote local overlearning. "Intense" is the phenomenology; distributed pathways are the wiring.

Predictive Coding & HIPPEA (Precision Weighting)

HIPPEA proposes high sensory precision vs. priors. DPM explains how structure yields that precision: more local branches dedicate more neurons to encoding incoming data, making bottom-up signals heavier relative to top-down compression. Prediction errors propagate widely in DPM, raising the subjective cost of uncertainty.

Weak Central Coherence (WCC) and Enhanced Perceptual Functioning (EPF)

WCC notes reduced global integration; EPF notes superior local processing. DPM unifies them: branch-rich local networks (EPF) coupled with fragile long-range gating (WCC). Local coherence rises as global coherence becomes harder to sustain.

Kozima's Granularity Theory

Granularity maps directly to representational density in DPM: more micro-features are concurrently represented because branching increases the sampling resolution of each stimulus.

Minicolumn and Pruning Accounts

Findings of narrower minicolumns, altered inhibitory surrounds, and reduced pruning map to DPM's branch retention and weakened lateral gating, giving a histological anchor to the architecture.

In short, DPM is not a rival to these accounts; it is a structural bridge. It explains how differences in pruning, inhibition, and channel dynamics manifest as distributed pathway behavior, from which the phenomenology (higher resolution, overload, creativity, replay) follows naturally.

Summary

In essence, autism reflects a brain wired for distributed propagation rather than streamlined efficiency. This architecture generates both its beauty and its fragility: greater sensitivity, richer internal worlds, but also greater energetic cost and volatility.

I do not see this as a pathology to correct but as an alternative computational mode—one that biology produced, that evolution tolerated, and that society can better understand through models like DPM.

Invitation for Further Research

This hypothesis is not a conclusion about how autism works. It is an evolving framework that I offer as an invitation to the scientific community—to test, refine, falsify, or expand. The Distributed Pathway Model can be validated empirically through neuroimaging, electrophysiology, and organoid systems, but it also requires conceptual integration with other theories of predictive coding, excitation/inhibition balance, and neural synchronization.

My goal is not to claim certainty but to spark inquiry—to provide a lens that unites molecular, circuit-level, and experiential perspectives into one coherent narrative. If others can use or adapt this model to generate new insights, corrections, or contradictions, then this document has served its purpose.

Appendix: Conceptual Model Summary

To visualize the Distributed Pathway Model in simple terms, imagine two small chains of neurons representing functionally distinct processing systems.

In a typical network, **A** → **B** → **C** and **X** → **Y** → **Z** operate independently. Each input (A or X) triggers a single predictable output (C or Z). These networks remain isolated—visual processing doesn't activate auditory circuits, motor planning doesn't trigger emotional responses.

In a Distributed Pathway configuration, neuron **B** also connects to **Y**, forming a cross-network branch. Now activation of **A** can lead to **C** (within-network processing), **Z** (cross-network spillover), or both, depending on timing, inhibition, and input strength. This small structural change multiplies the potential outcomes exponentially and, critically, allows signals to cross functional boundaries.

This cross-network architecture is fundamental to understanding why autism presents across multiple functional domains simultaneously. The same structural principle—reduced pruning of inter-network connections—manifests as sensory hypersensitivity, emotional reactivity, motor coordination challenges, and cognitive processing differences. These are not separate symptoms requiring separate explanations; they are unified by the same underlying topology of cross-network spillover.

At a large scale, this same principle applies across cortical columns and functional systems: distributed branching means each activation can spread across multiple networks that should remain isolated. This produces both **higher-resolution representations** (because more neurons across multiple systems contribute to encoding the same input) and **greater instability** (because inhibition must manage cross-network interference and potential cascade propagation across functional boundaries).

This is the fundamental logic of the Higher-Resolution Hypothesis: a brain that perceives more by engaging multiple processing systems simultaneously, connects more across functional domains, and sometimes, overwhelms itself by doing so.

Model Visualization and Mathematical Schema (Placeholder)

[Placeholder for future figure] — A simplified diagram or set of equations illustrating branching dynamics, inhibitory gating, and reactive feedback loops will be inserted here. It will formalize the Distributed Pathway Model in computational terms, showing how representational density scales with branch factor, inhibition strength, and temporal decay constants.

Acknowledgment of Lived Experience Integration

Although this document is written in scientific language, much of its insight originates from lived experience—direct observation of my own cognitive and sensory processes and comparison with empirical research. I have sought to interpret subjective patterns through an objective lens, aligning phenomenology with neuroscience rather than privileging one over the other. This hypothesis therefore represents both a **personal investigation** and a **scientific proposition**: an attempt to model from within what is typically only measured from without.

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