

# Copy And Paste Model

## Core Mathematical Apparatus

Part 2 of 5

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**Abstract.** This document defines the CAPM engine and asks what can be established about it. A Boltzmann-weighted path sum over a substrate graph is coupled to a substrate memory that the propagating population rewrites, and to a population wave that reads the path sum back. Two pathologies of the original formulation are identified and repaired: normalisation discards the absolute propagation rate, which is restored through a propagation efficiency  $\Theta$ ; and route quality is confounded with route length, which a hybrid action separates. We establish that the equilibrium is unique and therefore admits no perturbation-induced switching; that the continuous limit is substrate-determined only inside a narrow window of the path-length discount; that structural resolution is independent of binding affinity in the novelty channel alone; that compartmental heterogeneity is a prerequisite rather than a refinement, since a scalar substrate memory cancels identically from the partition function; and that pattern mutation is a diffusion on the sphere with a closed-form compatibility decay. The substrate-invariance claim is then tested against silicon and multi-agent substrates, and the verdict is partial: the population dynamics transfer exactly, the path integral transfers with a topology-dependent reparameterisation, the substrate memory law degenerates, and the ambient exposure inversion does not transfer at all, because it encodes a regime assumption rather than a mechanism.

## The CAPM Map

One framework, five documents. Read the one addressed to you; the others are referred to by name, never assumed.

| Document                                             | Its job                                                                                                                                                 | What you are asked to evaluate                                                                                                                                       |
|------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>P</b><br>Philosophy                               | States the thesis ( <i>the substrate changes; the equations do not</i> ), the method, and the boundaries of the claim. Prose only.                      | Is the thesis coherent? Is the intuition-first method honest? Is the narrowed claim that survived testing the right one to make?                                     |
| ► <b>C</b><br>Core<br>Mathematical<br>Apparatus      | Defines the engine, the action, substrate memory and the population wave; reports what is known about their well-posedness; tests substrate invariance. | The normalisation pathology and its repair; the measure problem and the convergence bound; uniqueness of equilibrium; $\rho_s$ independence; the invariance verdict. |
| <b>G</b><br>Cognitive<br>Extension                   | Applies the engine to thought: geometry translated across substrates, and the detection of the substrate a pattern natively occupies.                   | Whether coherence across crossings predicts what is remembered, and whether substrate detection predicts anything its bridge costs did not contain.                  |
| <b>B</b><br>Biomedical<br>Extension                  | Applies the engine to allergic sensitization; derives predictions for immunotherapy, polysensitization and pre-symptomatic detection.                   | A falsification ledger: every prediction with its status, whether contradicted, pre-registered, testable now, or open.                                               |
| <b>A</b><br>Technical<br>Appendix and<br>Bookkeeping | Numerical methods, verification runs, the corrections log, coherence bookkeeping, master equation table, V1.4 concordance.                              | Reproducibility. Every “earlier versions” statement lives here and nowhere else.                                                                                     |

**Anchor symbols.** Shared across the documents; defined where indicated.

| Symbol                                  | Meaning                                                                                                                                                          | Defined in |
|-----------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------|
| $R(t)$                                  | Replication probability: Boltzmann-weighted share of path weight reaching the nominated destination                                                              | C          |
| $S[\gamma, t]$                          | Informational action along path $\gamma$ ; $\sigma(t)$ is its temperature, in the position $\hbar$ occupies but with a real exponent                             | C          |
| $\chi_j$                                | Bridge-operator cost of the $j$ -th substrate crossing; $\chi_{ij}$ its matrix form between subsystems                                                           | C          |
| $c(s)$                                  | Node compatibility: geometric $\vec{p}^\top M_s \vec{p}$ and thermodynamic $\rho_s / (\rho_s + e^{\Delta G_0 / k_B T})$ , one quantity in two coordinate systems | C          |
| $V(s, t, \rho_s)$                       | Novelty: $\rho_s \cdot \text{JSD}(P_{\text{pattern}} \  P_{\text{substrate}})$ over feature bins                                                                 | C          |
| $P_{\text{substrate}}, P_0, N, \bar{n}$ | Substrate memory and its pristine baseline; execution-engine population and $\bar{n} = N/K$                                                                      | C          |
| $\alpha, \gamma_{\text{mem}}$           | Learning rate and <i>forgetting</i> rate of the substrate; low $\gamma_{\text{mem}}$ means long memory                                                           | C          |

| Symbol                       | Meaning                                                                                                                | Defined in       |
|------------------------------|------------------------------------------------------------------------------------------------------------------------|------------------|
| $\rho_s$                     | Structural resolution: how finely the substrate discriminates configurations                                           | C, grounded in B |
| $\Theta, \text{Mu}$          | Propagation efficiency $(Z/Z_{\text{ideal}})^{1/\bar{n}}$ ; mutation–adaptation ratio $\eta^2 d/\gamma_{\text{relax}}$ | C                |
| $C_{\text{total}}, \omega_r$ | Coherence across a set of substrate crossings; rate of recall attempts                                                 | G                |
| $\kappa, P_{\text{tol}}$     | Resensitization coefficient; regulatory substrate that makes $\gamma_{\text{mem}}$ plastic                             | B                |
| $P^*, \nu$                   | Observable untreated baseline; ambient encounter rate inverted from it                                                 | B                |

**Conventions.**  $\gamma_{\text{mem}}$  is a forgetting rate throughout. Differences are oriented as  $(P_{\text{substrate}} - P_0)$ . The population enters memory laws as  $\bar{n} = N/K$ . Equation, section and theorem numbers carry the document letter, so Eq. (C.1) is the engine wherever it is cited and the letter alone tells you which document to open. Gray tags [v1.4(*n*)] give the number the same equation carried in the single-document Version 1.4.

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## C.1 Notation and Conventions

*Remark C.1* (Normalisation of the population term). In the biomedical formulation of Biomedical §B.1 the population enters as the normalised density  $\bar{n} = N/K \in [0, 1]$  rather than as  $N$ . With  $N$  unnormalised,  $P_{\text{substrate}}$  scales with the carrying capacity and leaves the unit interval on which the divergence in Equation (C.4) is defined. The substitution is dimensional bookkeeping and does not alter the structure of the law.

## C.2 The Engine and the Action

### C.2.1 The CAPM Engine

Let  $\Gamma_t = (V_t, E_t)$  define the discrete topological network at time  $t$ , where  $V_t$  represents system nodes and  $E_t$  the active connectivity edges. The replication probability from origin node  $s_0 \in V_t$  to destination node  $s_f \in V_t$  is:

$$R(t) = \frac{\sum_{\gamma \in \mathcal{P}(s_0 \rightarrow s_f; \Gamma_t)} \gamma_{\text{len}}^{|\gamma|} \cdot C(\gamma) \cdot e^{-S[\gamma, t]/\sigma(t)}}{\sum_{s_{\text{alt}} \in V_t} \sum_{\gamma' \in \mathcal{P}(s_0 \rightarrow s_{\text{alt}}; \Gamma_t)} \gamma_{\text{len}}^{|\gamma'|} \cdot C(\gamma') \cdot e^{-S[\gamma', t]/\sigma(t)}} \quad [\text{v1.4 (11)}] \quad (\text{C.1})$$

where  $\gamma_{\text{len}} \in (0, 1)$  is the path-length discount factor,  $C(\gamma)$  the global structural compatibility of path  $\gamma$ ,  $\sigma(t)$  the environmental congestion noise, and  $S[\gamma, t]$  the accumulated informational action. The denominator is the systemic partition function  $\mathcal{Z}$ , ensuring  $0 \leq R(t) \leq 1$ .

### C.2.2 The Information Action Functional

$$S[\gamma, t] = \sum_{s \in \gamma} \frac{E(s)}{1 + \tanh(V(s, t) \cdot L(s) \cdot c(s))} + \sum_{j \in B_t} \chi_j \quad [\text{v1.4 (12)}] \quad (\text{C.2})$$

where  $B_t \subset \gamma$  is the set of boundary interfaces,  $E(s) > 0$  the baseline energy at node  $s$ , and  $\chi_j \geq 0$  the Substrate-Bridge Operator Cost for cross-substrate translation.

## C.3 Micro-Coefficients: Compatibility, Novelty, Coherence

### C.3.1 Coupled Micro-Coefficients

**Definition C.1** (Localized Node Compatibility).

$$c(s) = \max(0, \vec{p}^T M_s \vec{p}), \quad \|\vec{p}\|_2 = 1, \quad \|M_s\|_2 \leq 1 \quad [\text{v1.4 (16)}] \quad (\text{C.3})$$

with global pathway compatibility  $C(\gamma) = \prod_{s \in \gamma} c(s)$ .

**Definition C.2** (Time-Adaptive Novelty with Structural Resolution).

$$V(s, t, \rho_s) = \rho_s \cdot \text{JSD}(P_{\text{pattern}} \parallel P_{\text{substrate}}(s, t)) \quad [\text{v1.4 (17)}] \quad (\text{C.4})$$

where JSD denotes the symmetric Jensen-Shannon Divergence and  $\rho_s > 0$  is the structural resolution of the substrate at node  $s$ . When  $\rho_s = 1$  the original formulation is recovered exactly.

*What  $P_{\text{pattern}}$  and  $P_{\text{substrate}}$  Range Over*

Equation (C.4) is a divergence between distributions, and the choice of the space they are distributions over is not incidental. Treating both as scalars the natural first reading reduces JSD to its Bernoulli case, and that case is degenerate in a way worth stating plainly: **two allergens with different epitope compositions but the same scalar mean have a novelty of exactly zero relative to one another**. Not small; zero. The formulation is not merely imprecise about such a pair, it cannot see the difference at all.

We therefore resolve both distributions over  $B$  epitope feature bins. Let  $\vec{w}$  be the allergen's epitope profile, normalised over the bins. A substrate of resolution  $\rho_s$  cannot register arbitrarily fine structure in  $\vec{w}$ ; what it can register is the profile convolved with a kernel whose width narrows as resolution improves,

$$\vec{b}^\rho = \mathcal{N}[\vec{w} * G_{w_0/\rho_s}], \quad G_\sigma(d) = e^{-d^2/2\sigma^2} \quad [\text{v1.4 (18)}] \quad (\text{C.5})$$

with  $\mathcal{N}$  denoting normalisation and  $w_0$  the kernel width at unit resolution. The encoded state interpolates from the uniform distribution, when nothing has been learned, to  $\vec{b}^\rho$ , when everything learnable has been:

$$q_i = P_{\text{substrate}} b_i^\rho + \frac{1 - P_{\text{substrate}}}{B}, \quad V = \rho_s \cdot \text{JSD}(\vec{w} \parallel \vec{q}) \quad [\text{v1.4 (19)}] \quad (\text{C.6})$$

The convex combination in Equation (C.6) is automatically normalised, since both components sum to unity.

**This is what makes  $\rho_s$  an independent axis.** Under the scalar reading,  $V = \rho_s f(P_{\text{substrate}})$  is separable:  $\rho_s$  multiplies a function of the substrate state and does nothing else. The ratio  $V(0.9)/V(0.1)$  is then 0.0107 at *every* value of  $\rho_s$ , which is precisely the confound Remark C.5 identifies. Under Equation (C.6),  $\rho_s$  also sets the kernel width, so the shape of  $V$  as a function of  $P_{\text{substrate}}$  depends on it. The same ratio now runs from 0.85 at  $\rho_s = 0.25$  to 0.013 at  $\rho_s = 4$ .  $V$  is no longer separable, and  $\rho_s$  has acquired a channel that  $\Delta G_0$  cannot imitate.

**The kernel width sets an exchange rate.**  $w_0$  is not free of consequence. A wide kernel leaves a large irreducible mismatch between  $\vec{w}$  and the best the substrate could encode; that mismatch is common to every pathway, so it cancels in the normalisation of Equation (C.1)

and contributes nothing except to drown the learning signal. Narrowing  $w_0$  from 1.6 to 0.5 lowers the residual at perfect learning from 0.167 to 0.022 and raises the span of  $V$  across the learning range from 0.032 to 0.173, while leaving the resolution dependence above intact. Narrowing it much further, to  $w_0 \approx 0.2$ , eliminates the mismatch entirely and returns  $\rho_s$  to a pure multiplier reinstating the very confound the construction exists to remove. The two properties are obtained together only for intermediate  $w_0$ , and  $w_0 \approx 0.5$  is used throughout.

**Definition C.3** (Logic Coherence Engine).

$$L(s) = e^{-\varepsilon(s)} \quad [\text{v1.4 (23)}] \quad (\text{C.7})$$

where  $\varepsilon(s)$  counts semantic exceptions or compilation errors at node  $s$ .

## C.4 Two Pathologies and Their Repairs

### C.4.1 Normalisation Discards What the Population Wave Needs

Equation (C.1) defines  $R(t)$  as a share: the weight reaching the nominated destination divided by the partition function over all destinations. Any factor common to every pathway therefore cancels exactly, and this has a consequence that is easy to miss and hard to live with.

**The symptom.** Degrading compatibility *raises*  $R(t)$ . Sweeping  $\Delta G_0/k_B T$  from  $-4$  to  $+6$ , so that  $c(s)$  falls from 0.982 to 0.0025,  $R$  rises from 0.592 to 0.9987. Worse matching, higher replication probability. Reversing which pathway is nominated reverses the direction ( $0.492 \rightarrow 0.000$ ), and equalising all path lengths flattens it entirely ( $0.5447 \rightarrow 0.5445$ ). The sign is set by whether the target is shorter or longer than its alternatives, not by compatibility at all:  $c(s)$  does not raise or lower propagation, it amplifies whatever length advantage already exists.

**The obstruction is structural.** No normalised share can express “compatibility is poor, so less propagates.” That is a statement about the absolute rate, and normalisation is precisely the operation that discards it. Moving  $\gamma_{\text{len}}^{|\gamma|}$  and  $C(\gamma)$  inside the exponent does not help: the ratio  $c^{|\gamma_1|}/c^{|\gamma_2|}$  still diverges as  $c \rightarrow 0$ , merely more slowly.

**The information is not missing, only unused.** The partition function itself retains it. Across the same sweep  $\mathcal{Z}$  falls from  $8.5 \times 10^{-1}$  to  $2.0 \times 10^{-11}$  ten orders of magnitude, in exactly the direction required. Equation (C.13) reads only  $R$ , and so never sees it.

We therefore let the population wave read both outputs of the path integral:  $R$  for *where* propagation goes, and a propagation efficiency for *whether* it goes at all. Since  $\mathcal{Z}$  scales as  $c^{|\gamma|}$ , the per-node efficiency is the quantity dimensionally suited to multiplying a per-node rate:

$$\Theta = \left( \frac{\mathcal{Z}}{\mathcal{Z}_{\text{ideal}}} \right)^{1/\bar{n}}, \quad \frac{dN}{dt} = \omega_0 \Theta R N \left( 1 - \frac{N}{K} \right) + \cdots \quad [\text{v1.4 (13)}] \quad (\text{C.8})$$

with  $\mathcal{Z}_{\text{ideal}}$  the partition function at  $c \equiv 1$  and  $\bar{n}$  the mean path length.  $\Theta \rightarrow 1$  as compatibility approaches ideal, so Equation (C.8) reduces to Equation (C.13) exactly and every result in this paper is unaffected at  $\Theta = 1$ .

With  $\Theta$  in place the sweep behaves as both substrate readings require. The equilibrium density falls monotonically with compatibility 0.435, 0.407, 0.214, 0, 0 where without it the same sweep produces 0.483, 0.606, 0.697, rising as the match degrades. It also couples the two halves of the framework in a way they were not previously coupled: the supercriticality condition becomes  $\omega_0 \Theta R > \lambda$ , so poor compatibility can move a substrate from self-sustaining to driven. That is the distinction Section C.12 found separating silicon from tissue, now reachable by degrading the match rather than only by changing rates.

#### C.4.2 Path-Length Normalisation: The Hybrid Action

Equation (C.2) sums  $E(s)$  over the nodes of  $\gamma$ , so the action grows with path length even when every node along the route is highly compatible. Route *quality* and route *length* are therefore confounded in a single quantity. The confound is compounded by the compatibility product  $C(\gamma) = \prod_{s \in \gamma} c(s)$ , which penalises length a second time, and by the discount  $\gamma_{\text{len}}^{|\gamma|}$  in Equation (C.1), which penalises it a third.

Numerical evaluation of the partition function shows the practical consequence. At  $\sigma = 0.2$  the shortest admissible route receives essentially the entire weight of  $\mathcal{Z}$ , and  $R(t)$  pins at unity regardless of every other parameter in the model. The path integral is then formally present but carries no information.

Where routes of differing length must be compared on equal terms, we therefore adopt the **hybrid action**:

$$S_{\text{hybrid}}[\gamma, t] = \frac{S[\gamma, t]}{|\gamma|} + \lambda |\gamma| \quad [\text{v1.4 (14)}] \quad (\text{C.9})$$

The first term is the mean action density per node, which measures route quality independently of length. The second restores length as an explicit preference with its own coefficient  $\lambda \geq 0$ . Setting  $\lambda = 0$  leaves the length preference entirely to  $\gamma_{\text{len}}^{|\gamma|}$ , which is temperature-independent; a nonzero  $\lambda$  places part of it inside the exponent, where it softens as  $\sigma$  rises. At  $\lambda = 0$  and uniform  $|\gamma|$  the hybrid action is a monotone reparameterisation of Equation (C.2) and selects the same route ordering.

Under the hybrid form the same partition function that collapsed to 100/0/0 at  $\sigma = 0.2$  retains a spread of 88.6/10.8/0.6 across three candidate routes, and the sensitivity of  $R(t)$  to the binding free energy  $\Delta G_0$  widens from 0.247 to 0.384 over the range  $\Delta G_0/k_B T \in [-4, 3]$ .

#### C.4.3 The Noise Scale as Temperature

Equation (C.15) places  $\sigma(t)$  exactly where  $\hbar$  sits in the Feynman path integral, which makes  $\sigma$  the temperature of the path sum rather than an incidental nuisance parameter. Nothing in the

original formulation fixes its scale, and the behaviour of  $R(t)$  depends on that scale qualitatively rather than quantitatively: below  $\sigma \approx 1$  the partition function is degenerate in the sense above.

We therefore impose a floor

$$\sigma(t) \geq \sigma_{\min} \quad [\text{v1.4 (15)}] \quad (\text{C.10})$$

with  $\sigma_{\min}$  expressed in per-node action units, so that it carries the same meaning under Equation (C.2) and under Equation (C.9). Under the raw action the floor must be scaled by the typical path length  $|\overline{\gamma}|$  to preserve that meaning. The floor is a guard against degeneracy, not a physical claim; determining what actually fixes  $\sigma$  in a biological substrate remains open.

## C.5 Substrate Memory and the Population Wave

### C.5.1 The Memory-Dissipation Adaptation Law

The substrate is not a static background. When a wave of copies floods a network node, the substrate updates its internal cache to recognize the configuration. This is governed by:

$$\frac{\partial P_{\text{substrate}}(s, t)}{\partial t} = \alpha \cdot N(t) \cdot P_{\text{pattern}}(s) - \gamma_{\text{mem}}(P_{\text{substrate}}(s, t) - P_0) \quad [\text{v1.4 (24)}] \quad (\text{C.11})$$

where  $\alpha \in (0, 1)$  is the learning rate,  $N(t)$  the macro population size,  $\gamma_{\text{mem}} \in [0, 1]$  the forgetting rate, and  $P_0$  the pristine initial state of the medium. As  $N(t) \rightarrow 0$ , the substrate resets toward  $P_0$ , allowing novelty to regenerate over time.

### C.5.2 Bounded Systemic Congestion Noise

$$\sigma(t) = \sigma_0 + (\sigma_{\max} - \sigma_0) \cdot \tanh\left(\beta \frac{N(t)}{K}\right) \quad [\text{v1.4 (25)}] \quad (\text{C.12})$$

As  $N(t) \rightarrow K$ , noise saturates at  $\sigma_{\max}$ , driving  $R(t) \rightarrow 0$  and creating a stabilizing self-regulatory loop.

### C.5.3 The Macroscopic Population Wave

$$\frac{dN}{dt} = \max\left(0, \omega_0 \cdot R(t) \cdot N(t) \left(1 - \frac{N(t)}{K}\right)\right) - \lambda N(t) \quad [\text{v1.4 (26)}] \quad (\text{C.13})$$

All parameters are bounded and dimensionally consistent:  $\omega_0 > 0$  (inverse time),  $K > 0$  (count),  $\lambda > 0$  (inverse time).

## C.6 Uniqueness of Equilibrium and the Seeding Term

### C.6.1 Stochastic Seeding

For out-of-distribution initializations where  $\lim_{P_{\text{substrate}} \rightarrow \emptyset} C(\gamma_{\text{anomaly}}) \rightarrow 0$ , the system is augmented with a stochastic injection term:

$$\frac{dN}{dt} = \max\left(0, \omega_0 R(t) N(t) \left(1 - \frac{N(t)}{K}\right)\right) + \zeta \cdot \xi(t) \cdot \delta(t - t_{\text{injection}}) - \lambda N(t) \quad [\text{v1.4 (27)}] \quad (\text{C.14})$$

where  $\delta$  is the Dirac delta function,  $\xi(t) \in [0, 1]$  the structural coherence of the anomalous pattern, and  $\zeta > 0$  the injection seed size.

*Remark C.2* (Equation (C.14) seeds; it does not switch). Earlier versions titled this subsection *Stochastic Phase Transitions*. That overstates what Equation (C.14) can do, and the correction is worth making precisely because the term is otherwise useful.

A phase transition induced by a perturbation requires bistability: two stable states at one parameter set, with a separatrix between them that a large enough kick can cross. The model has none. Injecting seeds spanning a 256-fold range, from  $\zeta = 0.5$  to 128, every trajectory returns to the same equilibrium 14.25926 in engine units, identical to five decimal places in every case. The peak excursion scales linearly with  $\zeta$ , with the ratio of excess to seed size constant at 0.0391 across the range. This is the signature of a linear transient in a globally stable system, not of a threshold.

The reason is structural rather than parametric. Linearising Equation (C.14), the growth term is  $N[\omega_0 R(1 - N/K) - \lambda]$ , which has a single interior root; and both routes by which  $R$  responds to  $N$  through the congestion noise  $\sigma(N)$  of Equation (C.12), and through  $P_{\text{substrate}}$  into the novelty  $V$  are negative feedbacks. A unique equilibrium follows, and a unique equilibrium admits no switching. The coupled subsystem model of Section C.10 does not rescue it: starting the same parameter set from quiescent and from activated initial conditions gives the same aggregate to five decimals, so it is not bistable either.

The framework does contain a sharp transition, but it is a different object. The ignition threshold of Biomedical §B.9.4 is a fold in the *control parameter*  $\bar{\nu}$  the state jumps from 0.150 to 0.541 across a change of  $7 \times 10^{-4}$  and it lives in the coupling matrix  $\chi$ , not in the injection term. Crossing it requires changing the environment, not delivering a seed. Producing a genuine perturbation-induced transition would require bistability to be built in, most naturally through an Allee term of the form  $\omega_0 R N(N - a)(1 - N/K)$ , which no equation in this paper contains.

Equation (C.14) remains correct and useful for what it is: a way to introduce a configuration that the substrate has not encountered, so that its subsequent propagation can be followed. It is a seeding term. Nothing in this paper's conclusions depends on it being more.

## C.7 The Continuous Limit: Measure, Convergence, Diffusion

The original CAPM operates on discrete network topologies with passive environmental background. Biomedical application requires three fundamental extensions, developed in Sections 4.1–4.3. A fourth subsection then reconciles the compatibility tensor introduced by the third with the geometric definition it appears to replace; that reconciliation is not itself a refinement but a consistency requirement, and Section C.11 depends on it.

### C.7.1 Refinement I: Continuous Field Reformulation

The discrete path sum in Equation (C.1) must become a genuine functional integral over continuous tissue geometry:

$$R(t) = \frac{1}{\mathcal{Z}} \int \mathcal{D}\gamma \exp\left(-\frac{S[\gamma, t]}{\sigma(t)}\right) \quad [\text{v1.4 (28)}] \quad (\text{C.15})$$

The correspondence with the Feynman path integral is close, with  $\sigma(t)$  playing the role of  $\hbar$ , but it is not an identity and the difference is the part Feynman had to construct by hand. Equation (C.15) writes  $\mathcal{D}\gamma$  without specifying it, and without a measure on path space the discrete sum of Equation (C.1) has no unique limit to approach.

The point is demonstrable rather than pedantic. Enumerating many paths within each destination class and computing the class share, three equally natural enumerations lengths drawn uniformly, lengths drawn geometrically, and lengths held fixed each converge, but to 0.676, 0.718 and 0.713 respectively. The answer depends on how paths are counted, not merely on how many are counted. Until  $\mathcal{D}\gamma$  is fixed, Equation (C.15) names an object rather than defining one.

*The Framework Already Contains the Normalisation*

The missing ingredient is largely present, unrecognised, in Equation (C.1). Feynman’s measure is defined by time-slicing with a normalisation per slice; the analogue here is a normalisation per node, and  $\gamma_{\text{len}}^{|\gamma|}$  is exactly that. Its role has been described in this paper as a path-length discount, which understates it.

On a substrate graph of out-degree  $b$ , the number of distinct walks of length  $n$  grows as  $b^n$ , while each carries  $\gamma_{\text{len}}^n C(\gamma) e^{-S/\sigma}$  with  $C(\gamma) \leq c_{\text{max}}^n$ . The class sum is therefore a geometric series, and converges if and only if

$$b \cdot \gamma_{\text{len}} \cdot c_{\text{max}} \cdot e^{-E_{\text{min}}/\sigma} < 1 \quad [\text{v1.4 (29)}] \quad (\text{C.16})$$

Equation (C.16) is the condition for the path sum to exist at all, and hence for Equation (C.15) to have anything to converge to. It also gives  $\gamma_{\text{len}}$  a determinate role: it is not free, but bounded above by the topology it is applied to.

### What Failing the Bound Actually Means

The bound is not comfortable at the values used here. With  $\gamma_{\text{len}} = 0.92$ ,  $c_{\text{max}} \approx 0.62$  and  $\sigma \approx 1.67$ , the ratio in Equation (C.16) is 0.944 at  $b = 3$  and 1.259 at  $b = 4$ , while vascular, lymphatic and neural networks all exceed a branching factor of three.

It would be wrong, however, to read this as the framework’s central expression being undefined. No physical substrate admits walks of unbounded length: signals degrade, cells have finite lifetimes, networks have finite diameter. With any cutoff  $n \leq N_{\text{max}}$  the sum in Equation (C.1) is finite whatever the ratio. What Equation (C.16) actually decides is whether the answer depends on where that cutoff is placed.

| $b$ | ratio | share of the sum from the longest decade |
|-----|-------|------------------------------------------|
| 2   | 0.627 | 0.9%                                     |
| 3   | 0.940 | 35.1%                                    |
| 3.5 | 1.097 | 71.6%                                    |
| 4   | 1.254 | 90.6%                                    |
| 8   | 2.507 | 99.99%                                   |

Table C.2: Contribution of the longest paths to the class sum. Below ratio one the tail is negligible and the cutoff is irrelevant; above it the cutoff supplies essentially the whole answer.

The correct statement is therefore sharper than a convergence condition. **When Equation (C.16) holds,  $R(t)$  is a property of the substrate. When it fails,  $R(t)$  is a property of how long paths were allowed to be.** A model in the second regime is not meaningless, but it is reporting a modelling choice as though it were a result, and any such model must state  $N_{\text{max}}$  and justify it.

Restoring the bound at realistic branching is possible but not free. At  $b = 8$  convergence requires  $\gamma_{\text{len}} \lesssim 0.3$ , which concentrates 90% of the partition function on the shortest route pressing back toward the degeneracy that motivated the hybrid action of Section C.4.2. The two requirements pull in opposite directions, and the honest summary is that the framework has a narrow window in which the path sum is both substrate-determined and non-degenerate. Locating that window on a real topology is unfinished work, and is recorded here as such.

The action functional  $S[\gamma, t]$  in tissue becomes:

$$S[\gamma, t] = \int_{\gamma} \frac{E(\mathbf{x})}{1 + \tanh(V(\mathbf{x}, t) \cdot L(\mathbf{x}) \cdot c(\mathbf{x}))} d\ell(\mathbf{x}) + \sum_{j \in B_t} \chi_j \quad (\text{C.17})$$

where  $\mathbf{x} \in \mathbb{R}^3$  is a continuous tissue coordinate and  $d\ell$  the arc-length element along path  $\gamma$ . The energy  $E(\mathbf{x}) > 0$  now represents binding energy the thermodynamic cost of allergen interfacing with each receptor node directly measurable via calorimetry. The novelty  $V(\mathbf{x}, t)$  becomes

the structural divergence between allergen geometry and local receptor memory, computable from protein structure databases.

### C.7.2 Refinement II: Thermodynamic Diffusion Augmentation

Allergens lack an explicit replication drive but are subject to thermodynamic diffusion. The macroscopic population wave is augmented with a diffusion operator:

$$\frac{dN}{dt} = \max\left(0, \omega_0 R(t) N(t) \left(1 - \frac{N(t)}{K}\right)\right) + \mu \nabla^2 N(t) - \lambda N(t) \quad [\text{v1.4 (31)}] \quad (\text{C.18})$$

where  $\mu > 0$  is the molecular diffusion coefficient, and  $\nabla^2 N(t)$  is the Laplacian over tissue geometry. The new term bridges CAPM with standard reaction-diffusion equations used in biochemistry, producing a unified model in which both active informational propagation and passive thermodynamic spreading are formally represented. When  $\mu \rightarrow 0$  the original CAPM population wave is recovered exactly.

*Remark C.3* (Front velocity, corrected and verified). Equation (C.18) is of Fisher–KPP type when  $R(t)$  is held constant, and therefore admits a travelling front. Earlier versions of this remark gave its velocity as  $v = 2\sqrt{\mu \omega_0 R}$ . That is incorrect: the Fisher–KPP velocity is set by the linear growth rate of the state being *invaded*, and linearising Equation (C.18) about  $N = 0$  gives  $r = \omega_0 R - \lambda$ , not  $\omega_0 R$ . The clearance term was omitted.

$$v = 2\sqrt{\mu (\omega_0 R - \lambda)} \quad [\text{v1.4 (32)}] \quad (\text{C.19})$$

Two conditions must hold before Equation (C.19) applies at all, and neither was previously stated. First, the ambient encounter rate must vanish: with  $\nu > 0$ ,  $N = 0$  is not an equilibrium, the strip fills uniformly, and there is no empty state to invade. Second, the empty state must be unstable,  $\omega_0 R > \lambda$ . **At the reference parameters used throughout this paper the model is subcritical**  $\omega_0 R - \lambda = -0.236$  so no front exists there and the population is sustained entirely by ambient exposure. The prediction is not merely untested at those settings but inapplicable to them.

Where the conditions do hold, Equation (C.19) is accurate. Seeding an invasion on a 3000-cell strip and tracking the half-maximum contour with sub-cell interpolation, the speed converges from below as expected for a pulled front to 1.018 times the predicted value, agreement within 2%. The superseded expression overstates it by 29% at the same parameters. Front speed remains directly measurable via acoustic microscopy, and is now a quantitative test rather than a scaling claim.

## C.8 Compatibility as Thermodynamics; Two Forms Are One Quantity

### C.8.1 Refinement III: Statistical Mechanics Correction to $c(s)$ with Structural Resolution

At the molecular scale of allergen-receptor interaction, the classical compatibility tensor requires a physically grounded refinement. Rather than invoking quantum mechanical wavefunctions which, while theoretically interesting, introduce dependencies that are difficult to constrain experimentally we ground  $c(s)$  in the well-established framework of **statistical mechanics and thermodynamic free-energy landscapes**, augmented by the structural resolution parameter  $\rho_s$ .

Receptor-allergen binding is fundamentally a thermodynamic process whose sensitivity depends on how finely the receptor population can discriminate between molecular configurations. We therefore write:

$$c(s) = \frac{1}{1 + \exp(\Delta G(s, \rho_s) / k_B T)} \quad [\text{v1.4 (33)}] \quad (\text{C.20})$$

where  $\Delta G(s, \rho_s)$  is the resolution-dependent binding free energy:

$$\Delta G(s, \rho_s) = \Delta G_0(s) - k_B T \ln(\rho_s) \quad [\text{v1.4 (34)}] \quad (\text{C.21})$$

Here  $\Delta G_0(s)$  is the baseline binding free energy at average resolution  $\rho_s = 1$ , and the  $-k_B T \ln(\rho_s)$  term encodes the thermodynamic effect of resolution: a substrate with higher  $\rho_s$  resolves finer free-energy differences between molecular configurations, effectively lowering the apparent binding free energy for structurally similar but distinct patterns. Substituting:

$$c(s) = \frac{\rho_s}{\rho_s + \exp(\Delta G_0(s) / k_B T)} \quad [\text{v1.4 (35)}] \quad (\text{C.22})$$

*Remark C.4.* At  $\rho_s = 1$ , Equation (C.22) reduces to the standard Fermi-Dirac occupancy form. As  $\rho_s \rightarrow \infty$ ,  $c(s) \rightarrow 1$  regardless of  $\Delta G_0$  a perfectly resolving substrate finds everything compatible, which is the mathematical expression of the polysensitization risk discussed in Biomedical §B.5. As  $\rho_s \rightarrow 0$ ,  $c(s) \rightarrow 0$  the substrate cannot distinguish the pattern at all.

This formulation is directly experimentally accessible:  $\Delta G_0(s)$  is measurable via isothermal titration calorimetry and surface plasmon resonance;  $\rho_s$  is estimable from TCR repertoire sequencing. Furthermore, the substrate memory  $P_{\text{substrate}}(s, t)$  under this formulation becomes a *resolution-weighted stochastic state-space* analogous to a neural network where both the learning rate  $\alpha$  and the effective input dimensionality  $\rho_s$  jointly determine the richness of representable patterns.

### C.8.2 The Two Forms of $c(s)$ Are One Quantity

Definition 2 gives compatibility as a geometric quantity,  $c(s) = \max(0, \vec{p}^T M_s \vec{p})$ , and Equation (C.22) replaces it with a thermodynamic occupancy in  $\Delta G_0$ . These have been presented

as alternatives the second refining the first for biomedical use. They are not alternatives. They are the same quantity in different coordinates, and the change of variables between them is explicit.

Inverting Equation (C.22) for the free energy implied by a given compatibility:

$$\frac{\Delta G_{\text{eff}}(s)}{k_B T} = \ln \left( \frac{\rho_s (1 - c(s))}{c(s)} \right) \quad [\text{v1.4 (36)}] \quad (\text{C.23})$$

Every geometric configuration therefore *has* a binding free energy, and every binding free energy *has* an implied alignment. The content of Equation (C.23) is that structural alignment and thermodynamic affinity are not two mechanisms acting on  $c(s)$  but two readings of one number.

**The consistency condition.** Using both forms in one model requires them to agree where they overlap. Under the parameterisation  $M_s = a \vec{u}_s \vec{u}_s^T + b \mathbb{I}$ , which satisfies  $\|M_s\|_2 \leq 1$  for  $a + b \leq 1$  and gives  $c(s) = a (\vec{u}_s \cdot \vec{p})^2 + b$ , the requirement that a perfectly aligned pattern reproduce the thermodynamic value fixes

$$a + b = \frac{1}{1 + \exp(\Delta G_0(s)/k_B T - \ln \rho_s)} \quad [\text{v1.4 (37)}] \quad (\text{C.24})$$

Equation (C.24) is not cosmetic. Imposed, the geometric formulation reproduces the thermodynamic one to machine precision and the mutation dynamics of Section C.11 become a strict extension of the static model. Omitted, the two forms disagree at  $t = 0$  by an amount depending on nothing in particular, and every downstream quantity shifts.

**Mutation is a climbing affinity.** Combining Equation (C.23) with the decay law of Proposition C.2, a pattern drifting under mutation presents the receptor population with a monotonically rising effective binding free energy. The geometric and thermodynamic pictures of immune escape loss of shape complementarity, and loss of binding affinity are the same process.

**Escape is bounded by promiscuity.** Because  $c(s)$  relaxes to  $\bar{c} = a/d + b$  rather than to zero, the effective free energy saturates:

$$\frac{\Delta G_{\text{eff}}^{\text{max}}}{k_B T} = \ln \left( \frac{\rho_s (1 - \bar{c})}{\bar{c}} \right), \quad \bar{c} \geq b \quad [\text{v1.4 (38)}] \quad (\text{C.25})$$

A substrate with nonzero promiscuity  $b$  therefore retains residual recognition of an arbitrarily mutated pattern. Complete escape requires  $b = 0$ , that is a receptor population that binds nothing it was not shaped for. This is the framework's account of persistent cross-reactivity, and it also bounds the polysensitization mechanism of Biomedical §B.5: the same  $b$  that prevents escape is what admits structurally unrelated allergens.

**An experimental consequence.** Equations (C.24) and (C.25) connect two measurement modalities that would otherwise be separate. Calorimetric determination of  $\Delta G_0$  constrains  $a + b$ ; the residual binding of a mutated or unrelated ligand constrains  $b$  alone; and the two together fix  $a$  and hence the configuration dimension  $d$  through  $\bar{c}$ . The geometric parameters of Definition 2, which have no direct experimental interpretation as written, become accessible through binding assays.

## C.9 Structural Resolution Is Independent Only in the Novelty Channel

*Remark C.5* ( $\rho_s$  and  $\Delta G_0$  are not separately identifiable). Equation (C.22) can be rewritten

$$c(s) = \frac{1}{1 + \exp(\Delta G_0(s)/k_B T - \ln \rho_s)}, \quad [\text{v1.4 (49)}] \quad (\text{C.26})$$

which is Equation (C.21) substituted into Equation (C.20) and read as an identity:  $c(s)$  depends on  $\rho_s$  and  $\Delta G_0$  only through the single combination  $g_{\text{eff}} = \Delta G_0/k_B T - \ln \rho_s$ . Holding  $g_{\text{eff}}$  fixed while sweeping  $\rho_s$  over a fourteenfold range changes the simulated trajectory by a root-mean-square of  $1.2 \times 10^{-3}$ , or about 0.1%.

In the compatibility channel, therefore, structural resolution is not a third axis but a reparameterisation of binding affinity. Raising  $\rho_s$  is indistinguishable from lowering  $\Delta G_0$ . The phase space  $(\gamma_{\text{mem}}, \alpha, \rho_s)$  is three-dimensional only if  $\rho_s$  possesses a channel that  $\Delta G_0$  does not share.

Equation (C.4) supplies exactly that channel, since  $\rho_s$  multiplies the novelty  $V$  as well as entering  $c(s)$  but only if the distributions it compares are resolved finely enough for resolution to matter. Under a scalar reading of  $P_{\text{pattern}}$  and  $P_{\text{substrate}}$  it does not:  $V = \rho_s f(P_{\text{substrate}})$  is separable,  $\rho_s$  acts as a pure multiplier, and the confound is complete in both channels at once.

Section C.3.1 resolves this. Once the distributions range over epitope feature bins and  $\rho_s$  sets the width of the kernel through which the substrate registers them,  $V$  ceases to be separable: the ratio  $V(0.9)/V(0.1)$ , constant at 0.0107 for every  $\rho_s$  under the scalar reading, runs from 0.85 at  $\rho_s = 0.25$  to 0.013 at  $\rho_s = 4$ . **The independence of  $\rho_s$  is therefore established rather than merely unrefuted**, and the three-dimensional phase space  $(\gamma_{\text{mem}}, \alpha, \rho_s)$  stands. Biomedical Proposition B.2 may be read as written.

Two qualifications remain. The independence lives entirely in the novelty channel; in the compatibility channel  $\rho_s$  and  $\Delta G_0$  are still exactly confounded through  $g_{\text{eff}}$ , so an experimental determination of  $\rho_s$  must proceed through a measurement sensitive to  $V$  a discrimination assay rather than a binding assay. And the strength of the effect depends on the kernel width  $w_0$ , which is itself unmeasured.

## C.10 Heterogeneity Is a Prerequisite: The Subsystem Tensor

Biomedical §B.12 lists the absence of parallel memory systems as a limitation. It is better understood as the framework's central structural gap, because a second and independent argu-

ment demands the same repair: Section C.13 shows that a single substrate memory makes  $V$  common to every route, and a common shift cancels in the normalisation of Equation (C.1). Novelty cannot function without heterogeneity, and heterogeneity is what parallel subsystems supply.

### C.10.1 Formulation

Replace the scalar pair  $(P_{\text{substrate}}, N)$  with vectors over  $M$  subsystems indexed by  $i$ , each carrying its own memory  $P_i$ , normalised engine density  $n_i = N_i/K_i$ , structural resolution  $\rho_i$ , learning rate  $\alpha_i$ , constitutive forgetting rate  $\gamma_{0,i}$  and resensitization coefficient  $\kappa_i$ . Let  $\chi_{ij}$  be the cross-subsystem bridge operator: the rate at which activity in subsystem  $j$  recruits ( $\chi_{ij} > 0$ ) or suppresses ( $\chi_{ij} < 0$ ) subsystem  $i$ . Then

$$\frac{dn_i}{dt} = \omega_i R_i n_i (1 - n_i) - \lambda_i n_i + \underbrace{\left( \sigma_i^\nu \bar{\nu} + \sigma_i^\zeta g_N \zeta(t) \right)}_{\text{exogenous}} + \underbrace{\sum_{j \neq i} \chi_{ij} n_j}_{\text{coupling}} (1 - n_i) \quad [\text{v1.4 (59)}] \quad (\text{C.27})$$

$$\frac{dP_i}{dt} = \alpha_i \zeta(t) (P_0 - P_i) + \gamma_i (P_0 - P_i) + \kappa_i n_i^q (1 - P_i) \quad [\text{v1.4 (60)}] \quad (\text{C.28})$$

where  $\sigma_i^\nu$  and  $\sigma_i^\zeta$  are the shares of ambient and controlled exposure reaching subsystem  $i$ . Crucially each subsystem sees the path integral through its *own* resolution and *own* memory:

$$V_i = \rho_i \cdot \text{JSD}(P_{\text{pattern}} \parallel P_i), \quad R_i = R[V_i, \rho_i] \quad [\text{v1.4 (61)}] \quad (\text{C.29})$$

which is Equation (C.4) with the node index  $s$  realised as a subsystem index rather than discarded. Clinically observed reactivity is the weighted aggregate  $\bar{P} = \sum_i w_i P_i / \sum_i w_i$ , with  $w_i$  the symptomatic weight of each population.

### C.10.2 Pathways Belong to Compartments

Equations (C.27)–(C.29) give each subsystem its own novelty, but the path integral only benefits if the pathways of Equation (C.1) are attached to them. Each candidate route is therefore assigned a compartment index, and reads that compartment's memory and resolution:

$$V_\gamma = \rho_{\text{sub}(\gamma)} \cdot \text{JSD}(\vec{w} \parallel \vec{q}^{\text{sub}(\gamma)}) \quad [\text{v1.4 (64)}] \quad (\text{C.30})$$

The assignment is not cosmetic. It is what converts compartmental heterogeneity into pathway heterogeneity, and pathway heterogeneity is the only kind the normalisation in Equation (C.1) does not cancel. An attack route reading the mast-cell compartment and a tolerance route reading the regulatory compartment see genuinely different novelty because those compartments hold genuinely different memories; two routes reading one scalar cannot.

### C.10.3 Novelty Recovered

The purpose of the construction is served. Because the compartments have genuinely different constitutive forgetting rates IgE at  $\gamma_0 = 0.02$  settles near  $P = 0.98$ , mast populations at  $\gamma_0 = 0.35$  near  $P = 0.72$  their novelties differ. The measured spread in  $V$  across effector subsystems rises from effectively zero in the scalar model to  $3.9 \times 10^{-2}$ , and the induced spread in  $R$  from  $2 \times 10^{-4}$  to  $9.2 \times 10^{-2}$ , roughly two orders of magnitude. Only with the tensor in place does Equation (C.4) carry the weight the framework assigns it.

The consequence for  $R(t)$  follows from Equation (C.30): across a sweep of the substrate state,  $R$  spans  $9.1 \times 10^{-3}$  with the compartments separated against  $3.8 \times 10^{-4}$  without them. The path integral responds to substrate memory roughly twenty-four times more strongly, which is the difference between a term that participates in the dynamics and one that is present but inert.

**Convergence in  $M$  is checkable without knowing the magnitudes.** A model whose predictions move when the compartment count is refined is reporting a property of its own discretisation. We therefore rebuild the system at  $M \in \{3, 4, 6, 8, 12\}$ , interpolating effector properties across the range the four-compartment version spans, assigning equal weights, and preserving the total drive reaching any compartment. The result separates two topologies sharply.

| $M$ | star (parallel) |          | chain (relay) |          |
|-----|-----------------|----------|---------------|----------|
|     | aggregate       | $\Delta$ | aggregate     | $\Delta$ |
| 3   | 0.80145         | —        | 0.80145       | —        |
| 4   | 0.79248         | 0.00896  | 0.75656       | 0.04489  |
| 6   | 0.78831         | 0.00417  | 0.65058       | 0.10597  |
| 8   | 0.78702         | 0.00129  | 0.59636       | 0.05422  |
| 12  | 0.78604         | 0.00098  | 0.53602       | 0.06035  |

Table C.3: Aggregate reactivity above the ignition threshold as the compartment count is refined, for two coupling topologies.  $\Delta$  is the change from the preceding row.

Under a star topology, in which the effectors are activated in parallel from a common source, successive refinements change the answer by less than  $10^{-3}$  and the model has a well-defined limit. Under a chain, in which each compartment relays activation to the next, the answer does not settle: lengthening the relay attenuates terminal activation, so every refinement lowers the aggregate again.

**Proposition C.1** (Topology, not magnitude, determines well-posedness). *The existence of a compartment-count limit for the coupled system is a property of the sign-and-adjacency structure of  $\chi$ , not of the size of its entries. No rescaling of a chain topology produces a limit; a star topology possesses one for any magnitudes tested.*

The practical consequence is that  $\chi$  is constrained from an unexpected direction. Its magnitudes remain unmeasured, but its *admissible topologies* are decided by a test requiring no measurements at all. The parameters used in Section C.10 form a hybrid a two-step relay  $\text{Th2} \rightarrow \text{IgE} \rightarrow \text{mast}$  alongside a direct  $\text{Th2} \rightarrow \text{mast}$  term and therefore sit between the two behaviours, which is a reason for caution rather than confidence.

## C.11 Pattern Mutation and Escape from Substrate Memory

The framework as developed so far holds the identity vector  $\vec{p}$  fixed, while biological patterns evolve; Biomedical §B.12.1 records this as a limitation and proposes a mutation term  $d\vec{p}/dt = \eta \xi(t)$  (Biomedical Eq. (B.38)). That proposal requires one correction before it can be used, after which it yields more than was asked of it.

### C.11.1 The Constraint Forces a Projection

Definition 2 requires  $\|\vec{p}\|_2 = 1$ : the identity vector is a direction in configuration space, not a magnitude. An unconstrained additive perturbation does not respect this. Integrating  $d\vec{p} = \eta \xi dt$  with  $\eta = 0.4$  for 2000 steps drives  $\|\vec{p}\|$  from 1 to 8.15, at which point  $c(s) = \max(0, \vec{p}^T M_s \vec{p})$  has lost its meaning as a bounded compatibility.

Mutation must therefore be a diffusion *on the sphere*  $S^{d-1}$ , obtained by projecting the noise onto the tangent space at  $\vec{p}$ :

$$d\vec{p} = \eta (\mathbb{I} - \vec{p} \vec{p}^T) \circ d\vec{W} \quad [\text{v1.4 (68)}] \quad (\text{C.31})$$

in the Stratonovich convention, with  $\vec{W}$  a standard  $d$ -dimensional Wiener process. The projector  $(\mathbb{I} - \vec{p} \vec{p}^T)$  annihilates the radial component, so  $\|\vec{p}\| = 1$  is preserved exactly; numerically the norm holds to twelve decimal places over  $2 \times 10^4$  steps. Equation (C.31) is the corrected form of the proposal, and  $\eta$  retains its intended meaning as the mutation rate.

### C.11.2 The Decay Law for Compatibility

Equation (C.31) generates Brownian motion on  $S^{d-1}$  with generator  $\frac{\eta^2}{2} \Delta_{S^{d-1}}$ . The compatibility  $c(s) = \vec{p}^T M_s \vec{p}$  is a quadratic form, which decomposes into a constant part and a degree-two spherical harmonic; the Laplace–Beltrami eigenvalue on degree- $\ell$  harmonics is  $\ell(\ell + d - 2)$ , giving  $2d$  at  $\ell = 2$ . Hence:

**Proposition C.2** (Compatibility Decay Under Mutation). *Under Equation (C.31),*

$$\mathbb{E}[c(s)](t) = \bar{c} + (c(s)|_{t=0} - \bar{c}) e^{-\eta^2 d t}, \quad \bar{c} = \frac{\text{tr } M_s}{d} \quad [\text{v1.4 (69)}] \quad (\text{C.32})$$

*Compatibility relaxes exponentially, at rate  $\eta^2 d$ , from its initial value to the isotropic average  $\text{tr } M_s / d$  the compatibility a randomly oriented pattern would have.*

Monte Carlo integration of Equation (C.31) over 4000 replicas reproduces the predicted rate to within 2% for  $d \in \{4, 8, 16\}$  and  $\eta \in \{0.15, 0.3\}$ . Note the dimensional dependence: escape accelerates in richer configuration spaces, because there are more directions in which to differ.

### C.11.3 Numerical Verification

Proposition C.2 is a closed-form claim and is therefore checkable. It was implemented in the reference simulator described in Appendix §A.1 and tested directly, using the parameterisation  $M_s = a \vec{u}_s \vec{u}_s^T + b \mathbb{I}$  of Section C.8, whose exact isotropic mean  $\bar{c} = a/d + b$  is what makes the prediction falsifiable rather than merely plottable.

| $d$ | $\eta$ | predicted $\eta^2 d$ | measured | ratio |
|-----|--------|----------------------|----------|-------|
| 4   | 0.15   | 0.0900               | 0.0896   | 0.996 |
| 6   | 0.20   | 0.2400               | 0.2440   | 1.017 |
| 8   | 0.30   | 0.7200               | 0.6960   | 0.967 |
| 12  | 0.20   | 0.4800               | 0.4780   | 0.996 |
| 20  | 0.25   | 1.2500               | 1.2376   | 0.990 |
| 8   | 0.60   | 2.8800               | 2.7041   | 0.939 |

Table C.4: Decay rate of  $\mathbb{E}[c(s)]$  measured over ensembles of 1500 independent realisations of Equation (C.31), against the rate predicted by Proposition C.2.

Agreement is within 1–6% across a fivefold range of dimension and a fourfold range of mutation rate, the residual being ensemble noise and the finite integration step.

Section 8.1. The projected form preserves  $\|\vec{p}\| = 1$  to twelve decimal places over  $5 \times 10^4$  steps at  $\eta = 0.5$ ; the unprojected form  $d\vec{p} = \eta \xi dt$  reaches  $\|\vec{p}\| = 8.15$  within  $2 \times 10^3$  steps at  $\eta = 0.4$ . The projection is a requirement, not a refinement.

Finally, with the anchoring condition Equation (C.24) imposed, the mutating model at  $\eta = 0$  reproduces the static model to  $1.1 \times 10^{-16}$  over a full protocol simulation. Section C.11 is therefore an extension of the preceding sections and not a replacement for them, in the same sense as tolerance plasticity and the subsystem tensor.

### C.11.4 Escape Time and the Mutation–Adaptation Ratio

Equation (C.32) gives the time for a pattern to escape an established recognition:

$$\tau_{\text{escape}} = \frac{1}{\eta^2 d} \ln \left( \frac{c_0 - \bar{c}}{c_{\text{thr}} - \bar{c}} \right) \quad [\text{v1.4 (70)}] \quad (\text{C.33})$$

for a recognition threshold  $c_{\text{thr}}$ . The substrate, meanwhile, relaxes toward its own equilibrium at rate  $\gamma_{\text{mem}} + A$  (Biomedical §B.1). The competition between the two is governed by a single dimensionless group:

$$\text{Mu} = \frac{\eta^2 d}{\gamma_{\text{mem}} + A} \quad [\text{v1.4 (71)}] \quad (\text{C.34})$$

- $\text{Mu} \ll 1$ : the substrate tracks the pattern. Memory, once written, remains valid, and the Biomedical document–6 apply as written.
- $\text{Mu} \gg 1$ : the pattern escapes faster than the substrate can encode it.  $c(s)$  decays toward  $\bar{c}$  before the memory consolidates, novelty  $V$  never falls, and no stable encoding is reached.

At the reference parameters of the simulator ( $d = 8$ ,  $\gamma_{\text{mem}} = 0.05$ ,  $P^* = 0.85$ ) the three regimes are separated by roughly one order of magnitude in  $\eta$ :  $\eta = 0.05$  gives  $\text{Mu} = 0.065$  with  $\tau_{\text{escape}} = 45.3$ , so the substrate tracks;  $\eta = 0.15$  gives  $\text{Mu} = 0.59$  with  $\tau_{\text{escape}} = 5.0$ , a contested regime in which encoding and escape proceed on comparable timescales; and  $\eta = 0.6$  gives  $\text{Mu} = 9.4$  with  $\tau_{\text{escape}} = 0.31$ , in which no encoding survives. The transition is sharp because  $\text{Mu}$  depends on  $\eta^2$ .

## C.12 The Invariance Claim, Tested

The Philosophy document makes substrate invariance the framework’s deepest commitment, and every quantitative result in this paper comes from the biomedical column of the table below. That makes the central thesis the least tested claim in the work. This subsection tests it, by running the equations under silicon parameters and asking which of them survive.

The distinguishing feature of silicon is not its labels but its timescales. A processor clock runs at order  $10^9$  Hz while garbage collection runs at order 1–100 Hz, so  $\omega_0/\gamma_{\text{mem}} \sim 10^7$ , against roughly 10 in tissue. Sweeping that separation gives a clean and immediate result.

**The population wave transfers exactly.** Setting  $\nu = 0$  and integrating, the equilibrium density reproduces the logistic prediction  $n^* = 1 - \lambda/(\omega_0 R)$  across three decades of clock rate: measured 0.1626, 0.5785, 0.8313, 0.9578, 0.9916 against predicted 0.1591, 0.5795, 0.8318, 0.9580, 0.9916. Equation (C.13) is substrate-invariant in the strong sense not merely relabelled but quantitatively correct in a domain it was not built for. This is the clearest positive evidence the framework has.

**The ambient exposure inversion does not transfer, and the reason is structural.** Proposition 1 requires  $\nu \geq 0$ , hence  $\omega_0 R(1 - \bar{n}) \leq \lambda$ : the substrate must be unable to sustain itself without external input. Tissue satisfies this remove the allergen and the response dies. Silicon does not: the clock keeps processes alive with no external input at all. The critical point sits at  $\omega_0 \approx 0.98$  in these units, and above it the inversion returns no solution at any timescale separation tested from  $10^2$  to  $10^7$ .

This is a genuine limit on the framework rather than a defect in it. **The biomedical machinery of the Biomedical document–6 assumes a driven substrate; silicon is a**

**self-sustaining one, and the two are opposite regimes.**  $P^*$ , the observable the inversion is built on, has no analogue in a system that does not relax to a baseline when its input is removed.

**Substrate memory saturates and stops carrying information.** Above  $\omega_0/\lambda \approx 8$  the substrate state pins at  $P \approx 0.975$  and ceases to respond: measured 0.9703, 0.9749, 0.9752 across two further decades. Read as cache occupancy this is arguably correct a busy processor's cache is always full but it means Equation (C.11) discriminates nothing in that regime, and any prediction resting on substrate memory is vacuous there.

**The path sum requires a much smaller discount.** Switch fabrics fan out far more widely than tissue. At branching factor 16 the ratio of Equation (C.16) is 5.0, and at 32 it is 10.1, so  $R(t)$  is dominated by its longest paths unless  $\gamma_{\text{len}}$  falls to 0.18 or 0.09 respectively. The value used biomedically, 0.92, is inadmissible on silicon topology by two orders of magnitude.

### C.12.1 The Multi-Agent Column: An Independent Confirmation

The silicon result of Section C.12 is a single data point, and a single data point admits luck. The multi-agent column supplies a second and sharper test, because unlike silicon it can sit on *either* side of the driven/self-sustaining boundary: a message below the epidemic threshold requires continued seeding, and one above it spreads unaided.

In the framework's terms the basic reproduction number is

$$\mathcal{R}_0 = \frac{\omega_0 \Theta R}{\lambda} \quad [\text{v1.4 (77)}] \quad (\text{C.35})$$

and standard susceptible–infected–susceptible theory, which forms no part of this framework and against which nothing here was fitted, predicts a threshold at  $\mathcal{R}_0 = 1$  with endemic prevalence  $n^* = 1 - 1/\mathcal{R}_0$  above it.

| $\omega_0$ | $\mathcal{R}_0$ | inversion | adoption $n^*$ | $1 - 1/\mathcal{R}_0$ |
|------------|-----------------|-----------|----------------|-----------------------|
| 0.8        | 0.577           | applies   | 0.00000        | 0.00000               |
| 1.2        | 0.866           | applies   | 0.00000        | 0.00000               |
| 1.6        | 1.155           | undefined | 0.13424        | 0.13386               |
| 3.0        | 2.165           | undefined | 0.53332        | 0.53806               |
| 8.0        | 5.773           | undefined | 0.82486        | 0.82677               |

Table C.5: Multi-agent substrate against standard epidemic theory. Prevalence agrees to three decimal places; the ambient inversion applies below threshold and is undefined above it.

Bisecting for the point at which spread first appears locates it at  $\mathcal{R}_0 = 0.971$ , within 3% of

the theoretical value of one.

### *This Agreement Is Algebraic, Not Empirical*

It is tempting to read the table as independent confirmation of substrate invariance. It is not, and the distinction matters enough to state plainly. Setting Equation (C.13) with clearance to zero,

$$\omega_0 R (1 - n) = \lambda \implies n^* = 1 - \frac{\lambda}{\omega_0 R} = 1 - \frac{1}{\mathcal{R}_0},$$

which is the SIS endemic equilibrium in two lines. **The logistic equation with linear death is the SIS equation.** The agreement above is therefore an identity that had to hold, not a prediction that might have failed and the same objection applies to the silicon result of Section C.12, where logistic growth was assumed and logistic saturation was recovered.

What the two exercises do establish is weaker but not empty: the parameters of this framework map onto those of two well-validated models without inconsistency, and the mapping is quantitative rather than nominal. That is a calibration result. It is not evidence that the framework's *additional* structure the path integral, the substrate memory, the tensor carries across, because none of that structure is exercised by an equilibrium calculation.

Two things do survive the objection. The first is the crossover itself: the ambient inversion applies precisely below threshold and is undefined precisely above it, which identifies what Proposition 1 encodes. It is not a fact about immunology but the condition  $\mathcal{R}_0 < 1$ , and that identification was not visible from the biomedical column alone. The second is the residual: the measured prevalence departs from  $1 - 1/\mathcal{R}_0$  by up to  $5.4 \times 10^{-3}$ , because  $R$  and  $\Theta$  drift with the state while plain SIS holds its parameters fixed. That departure is small, but it is the only part of the comparison that is not guaranteed in advance.

### *A Prediction That Requires $\Theta$*

Equation (C.35) contains  $\Theta$ , and this is where the correction of Section C.4 earns its place. Holding reach and audience fixed and degrading only the contextual match,  $\mathcal{R}_0$  falls from 1.794 to 0.477 and the message crosses from spreading unaided to requiring continued seeding **a message can be made sub-critical purely by fitting its audience worse.**

Without  $\Theta$  the same sweep produces the opposite:  $\mathcal{R}_0$  would *rise* from 1.957 to 3.133 as the match degrades, so a poorly-targeted message would spread more readily than a well-targeted one. The pathology identified in Section C.4 is not a technicality of the biomedical reading; it inverts the central quantity of epidemic theory.

This is also where the framework says something SIS does not. In SIS,  $\mathcal{R}_0$  is fixed by contact and recovery rates alone; the fit between message and audience has no representation. Here  $\mathcal{R}_0$  carries a third factor, and it moves the threshold: at  $\Delta G_0 = 0$  the same protocol gives  $\mathcal{R}_0 = 1.277$  where plain SIS would give 1.957 regardless of fit. **The genuine contribution of the multi-agent column is not that the model reproduces epidemic theory it**

**must but that it extends it with a fit-dependent reproduction number.** Whether real diffusion data show that dependence is an empirical question, and a sharper one than any equilibrium comparison.

### Verdict

Invariance holds *in part*, and the partition is informative. Two substrates have now been tested rather than one. Their agreement on the population equation is algebraic rather than evidential (Section C.12.1), but the boundary they locate is not: the ambient inversion transfers exactly where  $\mathcal{R}_0 < 1$  and nowhere else, which identifies a condition that the biomedical column alone left implicit. The population dynamics carry across exactly. The path integral carries across with a topology-dependent reparameterisation that the framework now specifies. The substrate memory law carries across formally but degenerates. The ambient inversion does not carry across at all, because it encodes an assumption about the regime rather than about the mechanism.

The honest form of the claim is therefore narrower and more useful than the one Section 1 makes: **the propagation machinery is substrate-invariant; the biomedical apparatus built on top of it is not, and assumes a driven substrate.** Four entries in the table below already carry no counterpart outside the biomedical column, which is the same fact recorded at the level of individual parameters.

The table below consolidates all model parameters across the three deployment substrates silicon infrastructure, dynamic multi-agent networks, and the new biomedical substrate introduced by this refinement providing a unified reference for empirical calibration and cross-domain validation.

| Var.                  | Bounds   | Silicon Substrate         | Multi-Agent work                 | Net- | Biomedical (New)                |
|-----------------------|----------|---------------------------|----------------------------------|------|---------------------------------|
| $N(t)$                | $[0, K]$ | Active memory processes   | Minds with active recall         |      | Activated immune cells          |
| $K$                   | $> 0$    | RAM capacity (bytes)      | Total target population          |      | Max immune cell count           |
| $\omega_0$            | $> 0$    | Processor clock (Hz)      | Daily interaction rate           |      | Immune cycle rate               |
| $E(s)$                | $> 0$    | Transmission power (J/Tb) | Attention time (s)               |      | Receptor binding energy         |
| $V(s, t)$             | $[0, 1]$ | Shannon divergence (bits) | Surprise response index          |      | Tissue acoustic impedance shift |
| $L(s)$                | $[0, 1]$ | Syntax validation rate    | Logical coherence score          |      | Cell structural regularity      |
| $c(s)$                | $[0, 1]$ | Protocol matching score   | Contextual relevance index       |      | Receptor binding occupancy      |
| $\alpha$              | $(0, 1)$ | Cache update weight       | Familiarity learning rate        |      | IgE clonal expansion rate       |
| $\gamma_{\text{mem}}$ | $[0, 1]$ | Garbage collection rate   | Memory decay rate                |      | IgE antibody half-life          |
| $\mu$                 | $> 0$    | —                         | —                                |      | Molecular diffusion coefficient |
| $\kappa$              | $> 0$    | —                         | —                                |      | Resensitization coefficient     |
| $\rho_s$              | $> 0$    | Protocol version fidelity | Audience discrimination capacity |      | TCR diversity / HLA specificity |

Table C.6: Extended parameter reference frames including the biomedical substrate. Variable slots are mathematically open free parameters, whose specific numerical ranges are calibrated per domain during empirical testing.

### C.13 Boundaries: The Novelty Channel

#### C.13.1 The Novelty Channel Is Structurally Weak

*Addressed in Sections C.3.1 and C.10; narrower limitations remain.*

Equation (C.4) exists to make novelty govern propagation: as a substrate learns a pattern,  $V \rightarrow 0$ , the action rises, and  $R(t)$  falls. This feedback is the mechanism by which Section 3.1 claims novelty regenerates over time. Numerical evaluation shows the loop gain is close to zero.

Driving the substrate across its full range, from  $P_{\text{substrate}} = 0.95$  to 0.1, moves  $R(t)$  by 0.011 under Equation (C.2) and by  $4 \times 10^{-4}$  under Equation (C.9). The cause is not saturation of the hyperbolic tangent, as might be supposed: at the operating point the argument  $V \cdot L \cdot c$  is approximately 0.3, well inside the linear region. The cause is that  $V$  enters as a quantity

common to all candidate routes, and a common shift of every action cancels in the normalisation of Equation (C.1). Only the between-route *spread* in  $\Delta S$  survives, and that spread is 0.481 under the raw action and 0.006 under the hybrid form.

Two partial repairs are available and both are reported here for completeness rather than adopted as claims.

**Restoring the node index.** Equation (C.4) indexes novelty by node,  $V(s, t, \rho_s)$ , but any well-mixed reduction collapses it to a single scalar. Allowing each route to read the substrate memory of the tissue it traverses raises the dynamic range from  $2 \times 10^{-4}$  to  $2.1 \times 10^{-3}$ , a factor of ten.

**Unbounding the coupling.** The factor  $1/(1 + \tanh x)$  is bounded below by  $1/2$ , so novelty can never reduce the action by more than a factor of two however large  $V$  becomes. The substitution  $1/(1 + \tanh x) \rightarrow e^{-x}$  agrees to first order in  $x$  identical wherever the original is linear and does not flatten. Combined with the node index it reaches  $5.0 \times 10^{-3}$ , and  $1.8 \times 10^{-2}$  under the raw action.

Neither repair is sufficient, and the reason is instructive. Both depend on different regions having learned different things, and in the present formulation they do not: no term diffuses  $P_{\text{substrate}}$ , so a spatially uniform execution population leaves a spatially uniform substrate. Simulated tissue is uniform to five decimal places under both quiescent and broad exposure, and even under focal exposure the candidate routes read novelty values within 0.011 of one another.

The conclusion is that novelty is close to inert in any *scalar* formulation of CAPM, and that no adjustment internal to the path integral repairs it. What such a formulation lacks is a structural reason for distinct compartments to hold distinct memories.

Two independent repairs resolve this in combination, and they address different halves of it.

**Range comes from the tensor.** With the compartments separated and each pathway reading the memory of the compartment it belongs to, the spread in  $V$  across effector subsystems rises from effectively zero to  $3.9 \times 10^{-2}$  under the scalar divergence and to  $6.9 \times 10^{-2}$  under the epitope-feature form of Section C.3.1 while the spread in  $R$  across a substrate sweep rises from  $3.8 \times 10^{-4}$  to  $9.1 \times 10^{-3}$ , roughly twenty-four fold. In a running simulation the per-pathway novelties separate rather than being identical: 0.075, 0.357 and 0.004 under the epitope-feature divergence of Section C.3.1, or 0.133, 0.357 and 0.230 under the scalar form against which the figure was first measured.

**Discrimination comes from the feature divergence.** The tensor makes  $V$  differ between pathways but leaves it blind to *which* epitopes differ. Section C.3.1 supplies that: two profiles with identical scalar means separate by 0.490 where the Bernoulli form gives exactly zero.

The diagnosis above is retained because it is the reason both constructions are prerequisites

rather than refinements, not because the defect is still present.

Three narrower limitations survive. First, the partial repairs discussed earlier the node index and the unbounded coupling remain insufficient *on their own*. Second and this corrects a claim made in an earlier version of this section the apparent cost of the feature divergence in sensitivity is a scale factor rather than a loss of information. The Bernoulli form's  $V$  spans 0.620 across the learning range where Equation (C.6) spans 0.180, a ratio of 3.44; the shortfall in  $R$  was 3.5-fold. These are the same number. The novelty coupling constant  $k_V$  of Equation (C.2) is simply set too low for the narrower divergence, and raising it recovers the range: with the unbounded coupling (Section C.13) at  $k_V = 8$  the feature form reaches  $9.98 \times 10^{-3}$  against the scalar form's  $9.07 \times 10^{-3}$ , slightly ahead rather than behind. Critically,  $k_V$  leaves the resolution ratios of Remark C.5 untouched at 0.85/0.159/0.013 for every value tested, so the discrimination and identifiability gains survive the rescaling intact. Nothing in the shortfall is irreducible, and the earlier assertion that some of it was should be disregarded. Third, no term diffuses  $P_{\text{substrate}}$  (Biomedical §B.12.3, item 4), so geometric heterogeneity in substrate memory cannot arise from the equations as written. Novelty is carried by compartment identity rather than by tissue location, which is a modelling choice and not a derived result.

## C.14 What This Document Claims, and What It Hands On

The Core document establishes the following. Items keep the numbering of the single-document version 1.4 so that the two can be read side by side; the remaining contributions are biomedical and are listed in Biomedical §B.13.

7. The reduction of the coupling matrix to two experimentally recoverable scalars (Biomedical §B.9.3) and of the epitope kernel width to a cross-reactivity index (Biomedical §B.5), completing a pattern in which every unobservable parameter of the framework the ambient exposure rate, the activation gain, the coherence attenuation constant, the coupling magnitudes, and the resolving width is expressed through a quantity an experiment yields. No free constant remains on which a result of this paper depends.
8. The subsystem tensor (Section C.10), which is required rather than optional: with a single substrate memory the novelty term is common to all pathways and cancels identically, so Equation (C.4) carries no weight until the compartments are separated. The coupled form additionally produces a sensitization threshold (Biomedical §B.9.4) that no scalar version exhibits, establishing that  $\chi_{ij}$  carries dynamical content of its own.
9. The identification of the geometric compatibility of §C.3 and Equation (C.22) as one quantity in two coordinate systems (Section C.8), with the change of variables  $\Delta G_{\text{eff}}/k_B T = \ln(\rho_s(1-c)/c)$ , the anchoring condition required for consistency between them, and the resulting bound on immune escape: because compatibility relaxes to  $\bar{c} \geq b$  rather than to zero, a promiscuous receptor population retains residual recognition of an arbitrarily mutated pattern.

10. The mutation operator (Section C.11), corrected to a diffusion on  $S^{d-1}$  so that  $\|\vec{p}\| = 1$  is preserved, verified numerically against its own prediction to within 1–6% (Section C.11.3), together with the compatibility decay law  $\mathbb{E}[c](t) = \bar{c} + (c_0 - \bar{c})e^{-\eta^2 dt}$  and the mutation-adaptation ratio  $\text{Mu}$ . This supplies the explanation the blueprint/engine distinction of Biomedical §B.1 previously asserted without one: allergic memory persists because  $\eta \approx 0$ , so the encoding is never invalidated by a moving target.

**What is handed on.** The Biomedical document imports the engine (C.1), the action (C.2), the novelty (C.4), the Memory-Dissipation Law (C.11) and the population wave (C.13), and adds to them a resensitization term, an observable baseline, and a regulatory substrate. Nothing else crosses. In particular it inherits the conditions established here: uniqueness of equilibrium (Proposition C.2), the convergence window of the path sum (C.16), and the confounds collected in Biomedical Table B.4. Any result there that appears to violate one of them is an error there rather than a discovery.

## References