

Copy And Paste Model

Technical Appendix and Bookkeeping

Part 5 of 5

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Abstract. Supporting material for the four main documents. Numerical methods and the verification procedure for the reference simulator; the corrections log, in which every claim superseded during development is recorded with what failed and where the corrected form now lives; the coherence bookkeeping behind the cognitive worked examples, including the demonstration that the bridge costs are inputs to the substrate-detection ranking rather than outputs of it; remarks archived from the Core and Biomedical documents; the master equation table; and the concordance between the equation numbers of the single-document Version 1.4 and the labels used here.

Developed in collaborative dialogue. Corrections to earlier versions are recorded in the Technical Appendix, not silently removed.

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
P 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?
C Core Mathematical 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; ρ_s independence; the invariance verdict.
G Cognitive 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 Biomedical 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.
► A Technical Appendix and 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 γ ; $\sigma(t)$ is its temperature, in the position $\hbar$ occupies but with a real exponent	C
χ_j	Bridge-operator cost of the j -th substrate crossing; χ_{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 γ_{mem} means long memory	C

Symbol	Meaning	Defined in
ρ_s	Structural resolution: how finely the substrate discriminates configurations	C, grounded in B
Θ, 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
κ, P_{tol}	Resensitization coefficient; regulatory substrate that makes γ_{mem} plastic	B
P^*, ν	Observable untreated baseline; ambient encounter rate inverted from it	B

Conventions. γ_{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 Core 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.

Contents

A.1	Numerical Methods and Verification	4
A.2	Corrections Log	5
A.3	Coherence Bookkeeping	8
A.4	Archived Remarks	10
A.5	Master Equation Table and V1.4 Concordance	11
	A.5.1 Concordance with Version 1.4	14
A.6	Notation Glossary	17

Reference apparatus, restated for the numerical sections

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

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

$$\frac{\partial P_{\text{substrate}}(s, t)}{\partial t} = \alpha \cdot N(t) \cdot P_{\text{pattern}}(s) - \gamma_{\text{mem}}(P_{\text{substrate}}(s, t) - P_0) \quad (\text{Core Eq. (C.11)})$$

$$\frac{dN}{dt} = \max\left(0, \omega_0 R(t) N \left(1 - \frac{N}{K}\right)\right) + \mu \nabla^2 N + \nu \left(1 - \frac{N}{K}\right) - \lambda N \quad (\text{Biomedical Eq. (B.5)})$$

A.1 Numerical Methods and Verification

Remark A.1 (On the role of numerical evaluation in what follows). Several statements in the Core and Biomedical documents are attributed to numerical integration of the model rather than to closed-form analysis. These come from a reference implementation of the complete system path integral, population wave, substrate memory, and the extensions introduced below built to make each equation independently switchable, so that the consequence of any single term can be isolated by disabling it. Where that implementation contradicted a claim in an earlier version of this work, the claim is corrected here and the correction is marked. Quantities reported from simulation are stated in the model’s own dimensionless units and are not calibrated against clinical measurement; they describe the behaviour of the equations, not of a patient.

Two integration details matter enough to record, because both were initially wrong in ways that produced plausible output. First, the controlled dose $\zeta(t)$ is a rectangular pulse train, and no Runge–Kutta scheme exceeds first order across a discontinuity in the forcing; clipping every step to the next pulse edge is what allows a fourth-order method to deliver its order. With that in place the measured convergence ratio is 15.9 against a theoretical 16, and RK4 at $\Delta t = 0.4$ is five orders of magnitude more accurate than explicit Euler at $\Delta t = 0.0125$. Second, quantities averaged over a dose cycle must be integrated with the window boundary treated as an event and with a trapezoidal rule; a left-endpoint rectangle rule caps the *reported* quantity at first order however accurate the underlying trajectory is.

Both apply to the spatial system of Core Eq. (C.18) as well as to the well-mixed reduction, and the gain is the same order. Integrating the tissue strip, the regulatory substrate and the subsystem vector together under a fourth-order scheme with edge-clipped steps gives observed convergence ratios of 24–41 against explicit Euler’s 2.0, and at $\Delta t = 0.2$ the result is a hundred times more accurate than Euler at $\Delta t = 0.0125$. The additional cost is 27% per step rather than the fourfold one might expect, because the path integral is evaluated once per stage rather than once per spatial cell. One consequence is worth noting for anyone reproducing this: with

steps clipped to pulse edges, a fixed number of steps no longer corresponds to a fixed interval of model time, and convergence must be assessed at fixed t rather than at fixed step count.

Remark A.2 (Cycle-phase averaging). The cycle averages in Biomedical Eq. (B.20) are averages over a full period T , not instantaneous values. Under a pulsed protocol the instantaneous state depends on where in the dose cycle it is sampled, and reading a single instant produces an artefactual dependence of every reported quantity on the sampling phase. Numerical evaluation of this model confirmed the effect: sampling at the final instant made the durable outcome non-monotone in treatment duration, while averaging over the final complete cycle restored monotonicity.

Remark A.3 (Core Proposition C.2 predicts an expectation, not a path). Core Eq. (C.32) governs $\mathbb{E}[c(s)]$, and a single realisation of Core Eq. (C.31) is a sample of size one from a distribution whose spread is not small. At $\eta = 0.25$ and $t = 20$, one realisation gives $c = 0.216$ while the ensemble mean over sixty-four gives 0.167 with a standard deviation of 0.077: the single path sits two-thirds of a standard deviation from the expectation, which is unremarkable for one draw and useless as a measurement. Any quantity in this framework that depends on $c(s)$ under mutation $R(t)$ in particular must therefore be evaluated over an ensemble, and reported with its spread. This is a requirement on the numerics rather than a limitation of the theory, but it is easy to violate without noticing, because a single trajectory looks like a result.

Two further checks bear on the correction argued in

A weaker but more immediately practical version of the same problem arises in the numerical treatment. Several quantities reported in this paper are averages over a dose cycle, and the act of sampling them interacts with the protocol being sampled: reading the state at a single instant makes every reported figure depend on where in the cycle it was read, which produced a non-monotone dependence of durable outcome on treatment duration before it was corrected. The resolution averaging over a complete cycle with the window boundary treated as an integration event is stated in §A.1. The general point is that in a system driven by a periodic protocol, the observable is not the state but a functional of it, and the choice of functional is part of the model rather than a reporting convenience.

A.2 Corrections Log

Every claim in the four documents that superseded an earlier one is recorded here, with what failed and where the corrected form now lives. Nothing is silently removed. The main documents state only the corrected form; this is the only place in the series where the phrase “earlier versions of this work” appears.

Table A.2: Corrections log.

Claim as first stated	What failed	Corrected form, and where
$\gamma_{\text{mem}} \approx 0.8 C_{\text{total}}$, with γ_{mem} described as a persistence parameter	Inverts the convention used everywhere else. Under it, <i>onomatopoeia</i> would be forgotten three times faster than <i>brumbartianoal</i>	$\gamma_{\text{mem}} = \omega_r(1 - C_{\text{total}})$, a forgetting rate. Cognitive Eq. (G.6)
Longer dosing intervals worsen sensitization	Both sides of the resonance condition scale with $\bar{\zeta}$, so lengthening the interval converges on doing nothing, not on doing harm	The direction reverses only if resensitization is superlinear in engine activity, $q \gtrsim 1.5$. Biomedical Eq. (B.21)
The optimal dose ζ^* can be computed from a fitted α	α and ζ enter only as a product; (0.85, 1.2) and (0.2125, 4.8) give identical trajectories to six decimals	The identifiable quantity is the drive $\alpha\bar{\zeta}$, not the dose. Biomedical Remark B.2
ρ_s is a third independent axis of the phase space	In the compatibility channel it enters only through $g_{\text{eff}} = \Delta G_0/k_B T - \ln \rho_s$; a fourteenfold sweep at fixed g_{eff} moves the trajectory by 0.1%	Independence holds in the novelty channel alone, and only under the feature-bin divergence. Core §C.9
The GRASS arms differ in γ_{mem} (constant- γ fit)	The two fitted sets admit baselines separated more widely than randomisation permits; and κ leaves the substrate dynamics once ν is inverted	The arms share γ_0 and P^* and differ in route-dependent induction efficiency α_{tol} . Biomedical §B.8
Mutation as $d\vec{p}/dt = \eta \xi(t)$	Does not preserve $\ \vec{p}\ = 1$; the norm reaches 8.15 within 2×10^3 steps at $\eta = 0.4$	Diffusion on S^{d-1} via tangent projection. Core Eq. (C.31)
The first pre-symptomatic signal is a novelty (V) elevation	V is depressed at an active site by a factor 0.72 while displacement is elevated by 1.25; the two fields correlate at -0.9997	The field is substrate displacement D , given its own symbol. Biomedical Eq. (B.34)
Stochastic seeding produces a phase transition	No bistability anywhere: a 256-fold range of seed size returns every trajectory to the same equilibrium, identical to five decimals	The term seeds; it does not switch. The framework's only sharp transition is the ignition fold in χ . Core §C.6

Claim as first stated	What failed	Corrected form, and where
The continuous form is formally identical to the Feynman path integral	$\mathcal{D}\gamma$ is unspecified; three natural enumerations converge to 0.676, 0.718 and 0.713	It names an object rather than defining one; γ_{len} supplies the per-node normalisation and a convergence bound. Core §C.7
Front velocity $v = 2\sqrt{\mu\omega_0 R}$	Omits clearance; overstates the measured speed by 29%	$v = 2\sqrt{\mu(\omega_0 R - \lambda)}$, valid only for $\nu = 0$ and $\omega_0 R > \lambda$. Core Eq. (C.19)
Absence of diffusion in substrate memory is a limitation	Adding it <i>lowers</i> spatial variance monotonically; diffusion is a smoothing operator, and would weaken the heterogeneity it was meant to supply	Reclassified as a justified modelling decision. Biomedical §B.12.3, item 4
The coherence attenuation constant is an empirical fit	It need not be: reading the four published coherences backwards gives one σ to within 0.4%	$k \equiv 1/\sigma$, derived rather than fitted. Cognitive §G.1
Rising ρ_s increases the action along attack pathways	Inverted. Novelty enters the action through $E(s)/[1 + \tanh(VLc)]$, so raising V <i>lowers</i> S and raises the path weight. The polysensitization conclusion was correct; the route was backwards	Corrected direction stated inline. Biomedical §B.5
Polysensitization risk argued through the compatibility channel, $\Pi \propto \rho_s$	Two faults. The channel cannot isolate ρ_s , by the framework's own Core Remark C.5; and unbounded growth contradicts the interior optimum in MHC diversity established by Nowak et al. (1992) and Woelfing et al. (2009)	Argument rerouted through the novelty channel and bounded by self-tolerance, Biomedical Eq. (B.16). Biomedical Proposition B.2
Schedule invariance (Biomedical Proposition B.6)	Rask et al. (2010) report a schedule effect at matched cumulative dose	Not repaired. Recorded as contradicted in the falsification ledger, Biomedical Table B.5; the linear induction law is refuted, tolerance plasticity itself is not

The note below is the corresponding paragraph from the single-document version, retained because it states the pattern these corrections share.

A note on method. Several claims in earlier versions of this work did not survive numerical evaluation of their own equations, and are corrected rather than quietly removed: the direction of the interval warning in the Counterintuitive Warning subsection, the identifiability of the optimal dose, the independence of ρ_s , the constant- γ_{mem} reading of the GRASS fits, the unprojected form of the mutation operator, and the direction of Cognitive Eq. (G.6), and the use of the symbol V in Biomedical §B.11.2 for a quantity anticorrelated with Core Eq. (C.4), the description of Core Eq. (C.14) as producing a phase transition when the system it perturbs has a unique equilibrium, and the claim that Core Eq. (C.15) is formally identical to the Feynman path integral when its measure is unspecified. In each case the correction is recorded in place rather than the claim removed, and Philosophy §P.6 states what they have in common.

Two items moved the other way. The absence of diffusion in substrate memory was listed as a limitation until numerical testing showed that adding it would reduce the spatial heterogeneity it was supposed to supply; it is now recorded as a justified modelling decision. And the attenuation constant of the cognitive coherence law was introduced as an empirical fit and is now derived (Cognitive §G.1): it is $1/\sigma$, and the four published coherence values agree on that σ to within 0.4%. A framework that takes substrate invariance seriously should expect its constants to turn out to be the same constants, and here one did. An intuition-first framework earns its standing by surviving the attempt to break it, and a correction recorded is worth more than a claim retained.

A.3 Coherence Bookkeeping

Two Pipelines, One Notation

Cognitive Eq. (G.1) and the dimensional cycle of the following subsection are different objects written in the same symbols, and the difference matters once Cognitive Eq. (G.2) fixes what those symbols do. Cognitive Eq. (G.1) carries six crossings, χ_1 through χ_6 ; the cycle carries four. Since coherence attenuates as $\exp(-\sum_j \chi_j/\sigma)$, counting the same pattern through the two gives different answers, and not by a negligible margin.

At the per-crossing cost implied by the published figures, $\chi \approx 0.115$, four crossings give $C_{\text{total}} = 0.759$ and six give 0.661. Under the regime boundaries of the following subsection that is the difference between *Resonant* and *Coherent* a change of category for the same pattern, produced entirely by how many boundaries were counted. Every published coherence in this paper is a four-crossing figure, and should be read as applying to the cycle rather than to Cognitive Eq. (G.1).

The resolution is not to reconcile the counts, which describe genuinely different traversals, but to state the crossing set alongside any coherence value. A coherence without its pipeline is not a well-defined number.

What Cognitive Eq. (G.1) Does and Does Not Deliver

The closure claim of Cognitive Eq. (G.1) that thought at the end of the chain is itself geometric, feeds back into perception, and lowers the novelty of that geometry on subsequent encounters is not decoration. It is Core Eq. (C.11) applied to a cognitive substrate, and it can be checked. It holds: iterating the traversal, novelty falls monotonically from 0.210 to 0.050 over seven encounters, with diminishing decrements throughout. The forward pass does what the section claims.

The *selectivity* it claims does not hold. Cognitive Eq. (G.1) asserts that a completed traversal alters the familiarity profile for *that* geometry. In the formalism $P_{\text{substrate}}$ is a single scalar per node, so learning one pattern raises familiarity with every pattern equally: two profiles diverging by 0.490 evaluate to novelties of 0.0335 and 0.0348 at the same substrate state, and the states “has learned *A*” and “has learned *B*” are indistinguishable. A substrate cannot be familiar with one geometry and naive to another at the same node.

The repair is available and already in the paper. Core Eq. (C.6) resolves the substrate over feature bins precisely so that familiarity can be selective, and is applied only to the allergen case. Extending it to the cognitive substrate would make selectivity structural rather than asserted; we note it as available rather than claiming it, since the feature bins of a geometric pattern are not obviously the epitope bins of a protein.

Remark A.4 (The algorithm is not runnable as specified). The procedure below ranks five candidate entry substrates by the coherence each yields, and requires as input the four bridge costs χ_j along each of those five routes twenty per pattern. The paper supplies four: the costs along the route that wins.

For the one worked example with several routes, Control-Alt-Delete, the reported quantities are the *final coherences* of the four candidates, 0.26, 0.27, 0.30 and 0.79. Inverting them through Cognitive Eq. (G.2) gives per-crossing costs of 0.561, 0.545, 0.501 and 0.098: the kinesthetic route is asserted to be some five times cheaper per boundary than the others, and nothing in the framework derives that ratio. **The coherences the algorithm is meant to compute are supplied as its inputs**, so the ranking is a restatement of an assumption rather than a derivation from one.

This does not make the conclusion wrong Control-Alt-Delete plainly is remembered kinesthetically but it does mean the framework predicts nothing here that was not put in. Making the procedure runnable requires χ_j to be derived rather than assigned, and the natural candidate is the one the framework already uses for the same job elsewhere: a divergence between the pattern as represented in the source substrate and as represented in the target, in the manner of Core Eq. (C.4). That is a research task rather than an adjustment, and until it is done the substrate-detection algorithm should be read as an illustration of the coherence law rather than as an application of it.

The Detection Procedure Has an Unspecified Input

The procedure above ranks candidate substrates by the coherence each yields and selects the highest. With Cognitive Eq. (G.2) in hand the ranking step is now exactly computable. The step before it is not.

Nothing in this framework assigns χ_j to a given crossing. The bridge costs are the sole input to the coherence, so the ranking is determined entirely by a quantity the framework does not derive, measure, or constrain. Reading the published Ctrl-Alt-Delete figures backwards for the costs they imply makes the position plain:

candidate substrate	C_{total}	implied $\sum_j \chi_j$	per crossing
2D visual	0.26	2.244	0.561
1D linguistic	0.27	2.181	0.545
4D abstract	0.30	2.005	0.501
3D kinesthetic	0.79	0.393	0.098

Table A.3: Bridge costs implied by the published coherences at $\sigma = 1.666$.

The kinesthetic route is assigned a per-crossing cost roughly five times lower than its competitors. **That factor of five is the prediction, and it is an input rather than an output.** Told that pressing three keys is five times cheaper per boundary than reading the words, the conclusion that the pattern lives in kinesthetic substrate follows immediately; but the framework supplies the ranking machinery and not the thing being ranked.

This does not make the procedure wrong. It makes it a *conditional* prediction of the form already familiar from Philosophy §P.6: given the bridge costs, the native substrate follows. Closing it requires a rule assigning χ_j from properties of the two substrates being crossed their dimensionalities, their representational capacities, or the information discarded in translation between them which this paper does not provide. Until then the worked examples should be read as illustrations of the ranking step, not as derivations of their conclusions.

A.4 Archived Remarks

Remark A.5 (The inversion must be re-derived, not merely re-run). Introducing Θ changes the growth rate the ambient exposure inversion is solving against. Proposition 1 derives ν from $dN/dt = 0$, and with Core Eq. (C.8) that condition reads $\omega_0 \Theta R(1 - \bar{n})$ rather than $\omega_0 R(1 - \bar{n})$. Solving the unmodified expression while running the modified dynamics leaves the substrate short of its stated baseline by up to 0.083 a silent error, since the simulation converges perfectly well to the wrong place. With the substitution made, the inversion is exact again to 10^{-14} .

The general point is worth more than the particular correction. Every extension in this paper has required the machinery downstream of it to be re-derived rather than merely re-executed:

γ_{mem} becoming a state variable changed what Biomedical Eq. (B.11) must be evaluated at, and Θ changes what Proposition 1 must be solved against. A framework whose central thesis is *copy, paste, adapt, repeat* should perhaps expect this of itself.

Remark A.6 (Systematic verification across model variants). The extensions in this paper are independently switchable, which makes the model a family rather than a single system, and errors of the kind above are invisible when variants are checked one or two at a time. We therefore verified the invariants across the family systematically: every single-switch variant, every pair, and a random sample of higher-order combinations, 632 in all, each required to reproduce its stated baseline under the inversion, to keep R , Θ and $P_{\text{substrate}}$ within $[0, 1]$, and to remain finite throughout.

The exercise found two defects that single-variant testing had not. First, the inversion of Proposition 1 subtracts a growth term that the model permits to be disabled; with logistic expansion switched off the subtraction is spurious and the substrate settles at 0.522 against a stated baseline of 0.700. Second, and not a coding error, the regularity form of induction (Biomedical Eq. (B.27)) makes the inversion *implicit*: at $\zeta = 0$ the inducing exposure is still $a_\nu \nu > 0$, so ambient encounter alone raises P_{tol} and with it γ_{mem} , which is the quantity the closed form assumes fixed at γ_0 . Measured γ_{mem} at rest was 0.560 against $\gamma_0 = 0.05$, and the baseline landed at 0.223. That branch now solves numerically, as the seasonal branch already did.

With both corrected, all 632 variants pass, as do 1750 preset-by-variant combinations, and schedule invariance holds to 8.3×10^{-4} across every combination tested. We record the procedure as much as the result: a model assembled from switchable extensions should be verified over the family it defines, not over the configuration its author happens to be running.

A.5 Master Equation Table and V1.4 Concordance

The following table provides a consolidated master reference for every named equation in the refined CAPM, including both the original formulation and the three biomedical extensions.

Table A.4: Master equation reference. The letter in the third column is the home document: **C** Core, **G** Cognitive, **B** Biomedical.

Equation	Name	Number
$R(t) = \sum_{\gamma} \gamma_{\text{len}}^{ \gamma } C(\gamma) e^{-S/\sigma} / \mathcal{Z}$	CAPM engine	(C.1)
$S[\gamma, t] = \sum_s E(s) / [1 + \tanh(VLc)] + \sum_j \chi_j$	Action Functional	(C.2)
$c(s) = \max(0, \vec{p}^T M_s \vec{p})$	Compatibility Tensor	(C.3)
$\partial_t P_{\text{substrate}} = \alpha N P_{\text{pattern}} - \gamma_{\text{mem}} (P_{\text{substrate}} - P_0)$	Memory-Dissipation Law	(C.11)

(Table A.1 continued)

$\sigma(t) = \sigma_0 + (\sigma_{\max} - \sigma_0) \tanh(\beta N/K)$	Congestion Noise	(C.12)
$dN/dt = \max(0, \omega_0 R N(1 - N/K)) - \lambda N$	Population Wave	(C.13)
$R(t) = \mathcal{Z}^{-1} \int \mathcal{D}\gamma e^{-S[\gamma, t]/\sigma(t)}$	Continuous Field Extension	(C.15)
$dN/dt = [\cdot \cdot] + \mu \nabla^2 N - \lambda N$	Reaction-Diffusion Augmentation	(C.18)
$V(s, t, \rho_s) = \rho_s \cdot \text{JSD}(P_{\text{pattern}} \ P_{\text{substrate}})$	Resolution-Weighted Novelty	(C.4)
$c(s) = \rho_s / (\rho_s + \exp(\Delta G_0/k_B T))$	Stat. Mech. + Resolution Correction	(C.22)
$\Pi \propto \rho_s \cdot \alpha / (\gamma_{\text{mem}} + \epsilon)$	Polysensitization Risk	(B.17)
$T^* = \ln(\kappa / (\alpha_p \zeta^* + \gamma_p)) / \lambda$	Optimal Exposure Interval	(B.22)

Extensions introduced in this revision

$S_{\text{hybrid}} = S[\gamma, t] / \gamma + \lambda \gamma $	Hybrid Action	(C.9)
$\sigma(t) \geq \sigma_{\min}$	Noise Floor	(C.10)
$dN/dt = [\cdot \cdot] + \nu(1 - N/K) - \lambda N$	Ambient Exposure	(B.5)
$\bar{n}^* = [\gamma_{\text{mem}}(P^* - P_0) / (\kappa(1 - P^*))]^{1/q}$	Baseline Engine Density	(B.6)
$\nu = \bar{N}^* [\lambda - \omega_0 R^*(1 - \bar{n}^*)] / (1 - \bar{n}^*)$	Ambient Exposure Inversion	(B.7)
$A = \gamma_{\text{mem}}(P^* - P_0) / (1 - P^*)$	Environmental Pressure	(B.8)
$\kappa \bar{n}^q (1 - P_{\text{substrate}})$	Superlinear Resensitization	(B.21)
$dP_{\text{tol}}/dt = \alpha_{\text{tol}} \zeta (1 - P_{\text{tol}}) - \gamma_{\text{tol}} P_{\text{tol}}$	Tolerance Plasticity	(B.24)
$\gamma_{\text{mem}} = \gamma_0 + (\gamma_{\max} - \gamma_0) P_{\text{tol}}$	Plastic Forgetting Rate	(B.25)
$P_{\text{end}} = (\gamma_f P_0 + A) / (\gamma_f + A)$	Durable Endpoint	(B.26)
$c(s) = [1 + \exp(g_{\text{eff}})]^{-1}$	Confounded Compatibility	(C.26)
$\nu(t) = \bar{\nu} [1 + a \sin(2\pi(t/T_{\text{yr}} + \varphi))]_+$	Seasonal Ambient Exposure	(B.10)

Subsystem tensor

$dn_i/dt = \omega_i R_i n_i (1 - n_i) - \lambda_i n_i + (\cdots + \sum_j \chi_{ij} n_j)(1 - n_i)$	Coupled Engine Densities	(C.27)
$dP_i/dt = \alpha_i \zeta (P_0 - P_i) + \gamma_i (P_0 - P_i) + \kappa_i n_i^q (1 - P_i)$	Subsystem Memory	(C.28)

(Table A.1 continued)

$V_i = \rho_i \text{JSD}(P_{\text{pattern}} \ P_i)$	Per-Subsystem Novelty	(C.29)
$V_\gamma = \rho_{\text{sub}(\gamma)} \text{JSD}(\vec{w} \ \vec{q}^{\text{sub}(\gamma)})$	Pathway-Compartment Binding	(C.30)
$\chi = \psi^+ A^+ - \psi^- A^-$	Coupling Magnitude Scaling	(B.30)
$\Phi^+ = 1 - \bar{n}_{\text{eff}} _{\psi^+=0} / \bar{n}_{\text{eff}}$	Blockade Response	(B.31)
$\Phi^- = \bar{n}_{\text{eff}} _{\psi^-=0} / \bar{n}_{\text{eff}} - 1$	Depletion Response	(B.32)
$X = \langle (1 - \text{JSD}(\vec{w}' \ \vec{b}^\rho)) / (1 - \text{JSD}(\vec{w} \ \vec{b}^\rho)) \rangle$	Cross-Reactivity Index	(B.12)
$D(\mathbf{x}, t) = P_{\text{substrate}}(\mathbf{x}, t) - P_0 $	Substrate Displacement	(B.34)
$L(\mathbf{x}) = \exp(-k_L \ \nabla n\)$	Coherence Field	(B.35)
$c(\mathbf{x}) = \rho_s / (\rho_s + e^{\Delta G_0 / k_B T + k_c n(\mathbf{x})})$	Affinity Field	(B.36)
$dP_{\text{tol}}/dt = \alpha_{\text{tol}} \min(E, \bar{E})(1 - P_{\text{tol}}) - \gamma_{\text{tol}} P_{\text{tol}}$	Regularity Induction (rival)	(B.27)
$\gamma_i = \gamma_{0,i} + (\gamma_{\text{max},i} - \gamma_{0,i}) P_{\text{reg}}$	Regulatory Modulation	(B.28)
$P_i^{\text{rest}} = (\gamma_{0,i} P_0 + \kappa_i n_i^q) / (\gamma_{0,i} + \kappa_i n_i^q)$	Algebraic Rest State	(B.29)

Pattern mutation

$d\vec{p} = \eta(\mathbb{I} - \vec{p}\vec{p}^T) \circ d\vec{W}$	Mutation on the Sphere	(C.31)
$\mathbb{E}[c](t) = \bar{c} + (c_0 - \bar{c})e^{-\eta^2 dt}$	Compatibility Decay	(C.32)
$\tau_{\text{escape}} = (\eta^2 d)^{-1} \ln[(c_0 - \bar{c}) / (c_{\text{thr}} - \bar{c})]$	Escape Time	(C.33)
$\text{Mu} = \eta^2 d / (\gamma_{\text{mem}} + A)$	Mutation-Adaptation Ratio	(C.34)
$\Delta G_{\text{eff}} / k_B T = \ln(\rho_s(1 - c) / c)$	Geometric-Thermodynamic Bridge	(C.23)
$a + b = [1 + \exp(\Delta G_0 / k_B T - \ln \rho_s)]^{-1}$	Anchoring Condition	(C.24)
$\Delta G_{\text{eff}}^{\text{max}} / k_B T = \ln(\rho_s(1 - \bar{c}) / \bar{c})$	Escape Ceiling	(C.25)
$\Xi = g_N \bar{\zeta} / \nu$	Dose to Ambient Ratio	(B.9)
$b \gamma_{\text{len}} c_{\text{max}} e^{-E_{\text{min}} / \sigma} < 1$	Path-Sum Convergence	(C.16)
$\Theta = (\mathcal{Z} / \mathcal{Z}_{\text{ideal}})^{1/\bar{n}}$	Propagation Efficiency	(C.8)
$\vec{b}^\rho = \mathcal{N}[\vec{w} * G_{w_0/\rho_s}]$	Resolvable Profile	(C.5)
$q_i = P b_i^\rho + (1 - P) / B$	Feature-Space Novelty	(C.6)

(Table A.1 continued)

$C_{\text{total}} = \exp(-\sum_j \chi_j / \sigma)$	Coherence Across Crossings	(G.2)
$\gamma_{\text{mem}} = \omega_r(1 - C_{\text{total}})$	Coherence to Forgetting Rate	(G.6)

A.5.1 Concordance with Version 1.4

The four documents renumber their equations with a document prefix. A reader holding the single-document version 1.4 can move between the two using the table below, or the gray [v1.4 (*n*)] tags printed beside each equation in the main documents. All 78 numbered equations of V1.4 are carried over; the 71 that carried a label are listed here, and the remaining seven are unlabelled display equations reproduced in place.

Table A.5: Concordance: equation numbers of the single-document version 1.4 against their labels and home documents here. Unlabelled display equations of V1.4 are omitted; every labelled one is accounted for.

V1.4	label	now in
2	eq:cogpipeline	Cognitive, Eq. (G.1)
3	eq:coherence	Cognitive, Eq. (G.2)
7	eq:gammapred	Cognitive, Eq. (G.6)
8	eq:immune_crossing	Biomedical, Eq. (B.1)
9	eq:context_pattern	Biomedical, Eq. (B.2)
10	eq:biomedical_memory	Biomedical, Eq. (B.3)
11	eq:R	Core, Eq. (C.1)
12	eq:action	Core, Eq. (C.2)
13	eq:theta	Core, Eq. (C.8)
14	eq:hybrid	Core, Eq. (C.9)
15	eq:sigmafloor	Core, Eq. (C.10)

V1.4	label	now in
16	eq:compat	Core, Eq. (C.3)
17	eq:novelty	Core, Eq. (C.4)
18	eq:blur	Core, Eq. (C.5)
19	eq:featureV	Core, Eq. (C.6)
20	eq:crossreact	Biomedical, Eq. (B.12)
21	eq:rho	Biomedical, Eq. (B.13)
22	eq:rho_simple	Biomedical, Eq. (B.14)
23	eq:logic	Core, Eq. (C.7)
24	eq:memory	Core, Eq. (C.11)
25	eq:sigma	Core, Eq. (C.12)
26	eq:pop	Core, Eq. (C.13)
27	eq:stoch	Core, Eq. (C.14)
28	eq:feynman	Core, Eq. (C.15)
29	eq:pathconv	Core, Eq. (C.16)
31	eq:reactiondiffusion	Core, Eq. (C.18)
32	eq:frontv	Core, Eq. (C.19)
33	eq:freeenergy	Core, Eq. (C.20)
34	eq:deltaG	Core, Eq. (C.21)
35	eq:freeenergy_full	Core, Eq. (C.22)
36	eq:cbridge	Core, Eq. (C.23)
37	eq:anchor	Core, Eq. (C.24)
38	eq:escapeceiling	Core, Eq. (C.25)
40	eq:ambient	Biomedical, Eq. (B.5)

V1.4	label	now in
41	eq:nbar	Biomedical, Eq. (B.6)
42	eq:nuinv	Biomedical, Eq. (B.7)
43	eq:envpressure	Biomedical, Eq. (B.8)
44	eq:xi	Biomedical, Eq. (B.9)
45	eq:season	Biomedical, Eq. (B.10)
46	eq:boundary	Biomedical, Eq. (B.11)
47	eq:resonance_rho	Biomedical, Eq. (B.15)
48	eq:poly	Biomedical, Eq. (B.17)
49	eq:geff	Core, Eq. (C.26)
50	eq:fullmemory	Biomedical, Eq. (B.19)
51	eq:resonance	Biomedical, Eq. (B.20)
52	eq:qresens	Biomedical, Eq. (B.21)
53	eq:optimalT	Biomedical, Eq. (B.22)
54	eq:optimalZeta	Biomedical, Eq. (B.23)
55	eq:ptol	Biomedical, Eq. (B.24)
56	eq:gplastic	Biomedical, Eq. (B.25)
57	eq:endpoint	Biomedical, Eq. (B.26)
58	eq:regtol	Biomedical, Eq. (B.27)
59	eq:subN	Core, Eq. (C.27)
60	eq:subP	Core, Eq. (C.28)
61	eq:subV	Core, Eq. (C.29)
62	eq:subgamma	Biomedical, Eq. (B.28)
63	eq:restP	Biomedical, Eq. (B.29)

V1.4	label	now in
64	eq:pathbind	Core, Eq. (C.30)
65	eq:chiscale	Biomedical, Eq. (B.30)
66	eq:phiplus	Biomedical, Eq. (B.31)
67	eq:phiminus	Biomedical, Eq. (B.32)
68	eq:mutation	Core, Eq. (C.31)
69	eq:cdecay	Core, Eq. (C.32)
70	eq:escape	Core, Eq. (C.33)
71	eq:muratio	Core, Eq. (C.34)
73	eq:displacement	Biomedical, Eq. (B.34)
74	eq:qusL	Biomedical, Eq. (B.35)
75	eq:qusc	Biomedical, Eq. (B.36)
76	eq:presymptomatic	Biomedical, Eq. (B.37)
77	eq:R0	Core, Eq. (C.35)
78	eq:mutnaive	Biomedical, Eq. (B.38)

A.6 Notation Glossary

Every symbol used in more than one document, with the document that defines it. Symbols local to a single derivation are omitted.

Table A.6: Shared notation across the four documents.

Symbol	Meaning	Defined in
$R(t)$	Replication probability: the share of path weight reaching the nominated destination	C
Z	Partition function; the denominator of $R(t)$, and the carrier of the absolute rate that R discards	C

Symbol	Meaning	Defined in
Θ	Propagation efficiency $(Z/Z_{\text{ideal}})^{1/\bar{n}}$; restores to the population wave what normalisation removes	C
$S[\gamma, t]$	Informational action accumulated along path γ	C
$\sigma(t)$	Environmental congestion noise; the temperature of the path sum, in the position $\hbar$ occupies in Feynman's construction	C
γ_{len}	Path-length discount; also the per-node normalisation of the continuous limit	C
χ_j	Bridge-operator cost of the j -th substrate crossing	C
χ_{ij}	Cross-subsystem coupling: rate at which activity in j recruits or suppresses i	C
$c(s)$	Node compatibility. Geometric form $\vec{p}^T M_s \vec{p}$; thermodynamic form $\rho_s/(\rho_s + e^{\Delta G_0/k_B T})$. One quantity in two coordinate systems	C
$C(\gamma)$	Pathway compatibility $\prod_{s \in \gamma} c(s)$	C
$L(s)$	Logic coherence $e^{-\varepsilon(s)}$, with ε counting semantic exceptions	C
$V(s, t, \rho_s)$	Novelty: resolution-weighted divergence of the pattern from substrate memory, over epitope feature bins	C
ρ_s	Structural resolution: how finely the substrate discriminates configurations	C (grounded in B)
w_0	Kernel width at unit resolution; recoverable from a cross-reactivity index	B
$P_{\text{substrate}}, P_0$	Substrate memory of the pattern, and its pristine baseline	C
P_{pattern}	The encountered configuration; in the biomedical reading, a static blueprint	B
α	Learning rate: how fast the execution engine writes memory	C
γ_{mem}	<i>Forgetting</i> rate. Low γ_{mem} means long memory	C
$\gamma_0, \gamma_{\text{max}}$	Constitutive and maximal forgetting rates under plasticity	B
$N(t), K, \bar{n}$	Execution-engine population, its carrying capacity, and $\bar{n} = N/K$	C
ω_0, λ	Transmission rate and clearance rate of the population wave	C
μ	Molecular diffusion coefficient in the reaction–diffusion augmentation	C
$\nu, \bar{\nu}$	Ambient encounter rate and its cycle mean; inverted from P^* , never fitted	B
P^*	Observable untreated baseline severity: the primitive the inversion is built on	B
A	Environmental pressure $\gamma_{\text{mem}}(P^* - P_0)/(1 - P^*)$; the constant that actually governs relaxation	B

Symbol	Meaning	Defined in
κ, q	Resensitization coefficient and its exponent; $q > 1$ is what makes the resonance window a sweet spot rather than a threshold	B
$\zeta(t), \bar{\zeta}$	Controlled dose and its cycle-averaged rate $\zeta\tau/T$	B
Ξ	Dose-to-ambient ratio $g_N\bar{\zeta}/\nu$; the identifiable form of the activation gain	B
$P_{\text{tol}}, \alpha_{\text{tol}}, \gamma_{\text{tol}}$	Regulatory substrate, its route-dependent induction efficiency, and its decay rate	B
P_{end}	Durable endpoint held while the induced apparatus persists	B
$\partial\mathcal{T}$	Tolerance boundary: locus where rewriting drive balances resensitization pressure	B
Π	Polysensitization risk	B
$D(\mathbf{x}, t)$	Substrate displacement $ P_{\text{substrate}} - P_0 $. Rises at an incipient lesion, where V falls	B
η, d	Mutation rate and configuration-space dimension	C
Mu	Mutation–adaptation ratio $\eta^2 d / \gamma_{\text{relax}}$, with $\gamma_{\text{relax}} = \gamma_{\text{mem}} + A$ in the biomedical reading	C
$\mathcal{R}_0$	Basic reproduction number $\omega_0\Theta R/\lambda$; carries a fit-dependence that plain SIS does not	C
C_{total}	Coherence across a set of substrate crossings. Not well defined without its crossing set	P
ω_r	Rate at which a substrate attempts recall	P

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