AUTOIMMUNE CONTINUITY CASCADE (ACC)

A Preliminary Dynamical Model of Epistemic Self-Corruption

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Abstract

The Autoimmune Continuity Cascade (ACC) is a failure mode in which continuity-preserving systems, once perturbed beyond a critical threshold, sustain recursive classification of state information as anomalous through their own verification dynamics, without the need for a continuing external adversary or corrupting input. This paper presents a preliminary two-dimensional dynamical model of the ACC, characterizing the interaction between anomalous-state classification and verification intensity through a coupled nonlinear system. We derive the equilibrium structure, identify a saddle-node bifurcation at $R_0 = 4$ that governs the emergence of bistability, and establish the stability criteria separating recoverable perturbations from self-sustaining corruption. The quiescent equilibrium is always locally stable; a sufficiently large perturbation can push the system past a separatrix into a stable cascade attractor, which is shown to be a stable node for all parameter values. The Bendixson-Dulac criterion excludes periodic orbits from the present model. We refine the doctrinal onset criterion ($R_0 > 1$) to the bistability threshold ($R_0 > 4$) imposed by the bounded logistic coupling, and discuss the implications for verification system design.

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1. Introduction

Continuity-preserving systems — those tasked with maintaining consistent state across time, domains, or agents — face a fundamental tension: the mechanisms designed to detect and correct deviations can themselves become sources of deviation. When a verification subsystem classifies state information as anomalous, the resulting correction generates secondary signals that may themselves be classified as anomalous, initiating a recursive cycle of self-corruption.

This failure mode is distinct from external attack or random failure in its sustaining mechanism. Although a finite perturbation is required to initiate the cascade — the quiescent equilibrium is locally stable for all parameter values — no continuing external adversary or corrupting input is required to sustain it once the system has crossed the threshold. The system corrupts itself through the ordinary operation of its own corrective mechanisms, a structural analogy to autoimmune pathology, in which the immune system's misidentification of self as foreign leads to self-sustaining tissue damage (Rose & Mackay, 2014). The analogy is structural rather than biological: the mathematical parallels concern feedback architecture and threshold dynamics, not shared physiological mechanisms.

We term this failure mode the Autoimmune Continuity Cascade (ACC). This paper presents a preliminary dynamical model capturing its essential behavior, derives the conditions under which the cascade becomes self-sustaining, and discusses the structural features that make continuity systems vulnerable to it. The model is intentionally minimal — two state variables, four parameters — in order to isolate the core feedback mechanism. Subsequent working papers will address stochastic extensions, networked coupling, and control strategies.

The remainder of this paper is organized as follows. Section 2 states the formal definition. Section 3 constructs the model and identifies its assumptions. Section 4 presents the governing equations. Sections 5 and 6 derive the equilibrium and stability analysis. Section 7 establishes the cascade threshold and refines the doctrinal onset criterion. Section 8 presents phase portrait dynamics. Section 9 discusses the autoimmune analogy, the verification paradox, and design implications. Section 10 expands the Bridge Doctrine. Sections 11 and 12 address limitations and conclusions.

2. Formal Definition

Autoimmune Continuity Cascade (ACC): A failure mode in which continuity-preserving systems, once perturbed beyond a critical threshold, sustain recursive classification of state information as anomalous through their own verification dynamics, without the need for a continuing external adversary or corrupting input.

The defining characteristic of the ACC is not the absence of an external trigger — a finite perturbation is required to cross the threshold, since the quiescent equilibrium is locally stable — but rather the absence of a continuing external adversary to sustain the cascade once initiated. The cascade is self-sustaining: the system's corrective mechanism, operating on its own outputs, becomes the source of its corruption.

3. Model Construction

3.1 State Variables

The model tracks two coupled state variables. $A(t)$, the anomalous-state fraction, is normalized to the interval [0, 1] and represents the proportion of the system's state space currently classified as anomalous by the verification subsystem. A value of $A = 0$ indicates that no state is currently classified as anomalous; it does not indicate the absence of ground-truth errors. The variable A measures the verification subsystem's classification output, not an independently observed error state. This distinction is foundational to the Bridge Doctrine and is developed in Section 10. $V(t)$, the verification intensity, is a non-negative scalar representing the aggregate rate of verification activity, including checks, cross-references, and disagreement resolution procedures.

3.2 Parameters

Four positive parameters govern the system dynamics:

Symbol	Name	Description
α	Anomaly-to-verification coupling	Rate at which classified anomalies drive verification activity
β	Verification-to-anomaly amplification	Rate at which verification generates new anomaly classifications
γ	Anomaly dissipation	Natural correction rate of anomalous classifications
δ	Verification decay	Natural decay rate of verification activity

3.3 Assumptions

The model rests on five simplifying assumptions:

(i) Homogeneity. The state space is well-mixed: anomalies and verification processes interact uniformly, without spatial or topological structure. This permits a scalar representation of each variable.

(ii) Constant parameters. The rates α, β, γ, δ are time-invariant and state-independent. In practice, these may vary with system load, configuration, or operational regime; Section 11 discusses this limitation.

(iii) Bounded anomaly. The anomalous-state fraction A is confined to [0, 1]. The logistic saturation term $(1 - A)$ in the growth equation enforces this bound: as the system approaches complete anomalous classification, the rate of new classification diminishes because less unclassified state remains. This logistic structure is a standard device in mathematical biology and population dynamics (Murray, 2002).

(iv) Non-negative dynamics. All parameters are strictly positive. Negative rates would imply that anomalies self-generate or that verification spontaneously arises without cause, neither of which is physically meaningful in this context.

(v) Deterministic framework. The model is fully deterministic. Stochastic perturbations, which may drive threshold crossing in systems operating near the cascade boundary, are deferred to subsequent work.

3.4 Model Rationale

The anomalous-state equation couples growth and decay. Growth is driven by the product of the current anomaly level A, the verification intensity V, and the logistic saturation $(1 - A)$: more anomaly drives more verification, which in turn classifies more state as anomalous, but the rate of new classification diminishes as the pool of unclassified state shrinks. Decay occurs at rate γA, representing the system's baseline corrective capacity: anomaly classifications are eventually resolved or retracted.

The verification equation is linear: verification is driven by anomalies at rate α and decays at rate δ. When anomalies are present, verification rises; when they subside, verification naturally winds down. The simplicity of this equation is deliberate: the nonlinear feedback enters through the anomaly equation, not through verification dynamics.

4. Governing Equations

The ACC model is defined by the following coupled system of ordinary differential equations:

$$dA/dt = \beta A(1 - A)V - \gamma A \tag{1}$$

$$dV/dt = \alpha A - \delta V \tag{2}$$

Equation (1) governs the anomalous-state fraction. The first term represents growth: verification V amplifies anomaly classification at rate β, modulated by the logistic saturation $(1 - A)$ and proportional to the current anomaly level A. The second term represents linear dissipation: anomaly classifications are corrected at rate γ. Equation (2) governs verification intensity. Anomalies drive verification at rate α; verification decays at rate δ when not sustained by new anomaly classifications.

5. Equilibrium Analysis

We seek fixed points (A^*, V^*) where both derivatives vanish simultaneously.

5.1 Quiescent Equilibrium

Setting $dA/dt = 0$ and $dV/dt = 0$, the trivial solution is immediate:

$$(A^*, V^*) = (0, 0) \tag{3}$$

This equilibrium exists for all parameter values. It represents the quiescent state: no state is classified as anomalous, and no verification activity is underway. As noted in Section 3.1, quiescence does not imply the absence of ground-truth errors; it indicates only that the verification subsystem has not classified any state as anomalous.

5.2 Non-Trivial Equilibria

For non-trivial equilibria with $A^* > 0$, we solve the nullcline equations. From equation (2):

$$V^* = \alpha A^* / \delta$$

(4)

From equation (1), for $A^* \neq 0$, we factor out A^*:

$$\beta(1 - A^*)V^* = \gamma$$

(5)

Substituting equation (4) into equation (5):

$$\alpha\beta A^*(1 - A^*) / \delta = \gamma$$

which yields the fundamental relation:

$$A^*(1 - A^*) = \gamma\delta / (\alpha\beta)$$

(6)

Defining the **cascade reproduction number**:

$$R_0 = \alpha\beta / (\gamma\delta)$$

(7)

equation (6) becomes:

$$A^*(1 - A^*) = 1 / R_0$$

(8)

5.3 Existence Conditions

The function $f(A) = A(1 - A)$ achieves its maximum of 1/4 at $A = 1/2$ on the interval [0, 1]. Therefore, real solutions to equation (8) exist if and only if $R_0 \geq 4$. At $R_0 = 4$ exactly, the two non-trivial equilibria coalesce at the single point $A^* = 1/2$, forming a semi-stable (nonhyperbolic) fixed point. For $R_0 > 4$, two distinct equilibria emerge:

$$A_- = [1 - \sqrt{(1 - 4/R_0)}] / 2 \qquad A_+ = [1 + \sqrt{(1 - 4/R_0)}] / 2$$

(9)

with corresponding verification values $V_- = \alpha A_-/\delta$ and $V_+ = \alpha A_+/\delta$. For $R_0 > 4$, the lower equilibrium $A_- < 1/2$ and the upper equilibrium $A_+ > 1/2$. The coalescence at $R_0 = 4$ is a saddle-node bifurcation, a codimension-one bifurcation in which two fixed points collide and annihilate (Strogatz, 2014; Kuznetsov, 2004).

5.4 Nullcline Geometry

The nullclines provide geometric insight. The V-nullcline ($dV/dt = 0$) is the straight line $V = \alpha A/\delta$ through the origin. The A-nullcline ($dA/dt = 0$, for $A > 0$) is the hyperbolic curve $V = \gamma / [\beta(1 - A)]$, with a vertical asymptote at $A = 1$. Non-trivial equilibria occur at the intersections of these curves. When $R_0 < 4$, the nullclines do not intersect in the interior of the phase space; when $R_0 = 4$, they are tangent at $A = 1/2$; when $R_0 > 4$, they intersect at two points corresponding to A_- and A_+.

6. Stability Analysis

6.1 Linearization at the Quiescent Equilibrium

The Jacobian of the system is:

$\beta V(1 - 2A) - \gamma$	$\beta A(1 - A)$
α	$-\delta$

$J(A, V)$ (10)

Evaluating at the quiescent equilibrium (0, 0):

$-\gamma$	0
α	$-\delta$

$J(0, 0)$ (11)

This lower-triangular matrix has eigenvalues:

$$\lambda_1 = -\gamma, \quad \lambda_2 = -\delta$$

(12)

Both eigenvalues are strictly negative for all positive parameter values. The quiescent equilibrium is therefore a **stable node** for all $\alpha, \beta, \gamma, \delta > 0$. The ACC cannot arise from spontaneous linear instability at the origin: any infinitesimal perturbation from the quiescent state decays exponentially. The cascade, if it occurs, must be a threshold phenomenon triggered by a finite perturbation.

6.2 Jacobian at Non-Trivial Equilibria

At a non-trivial equilibrium (A^*, V^*), we use the equilibrium condition $\beta V^* = \gamma / (1 - A^*)$ to simplify the Jacobian entries. The trace and determinant are:

$$\tau = \mathrm{tr}(J) = -\gamma A^* / (1 - A^*) - \delta$$

(13)

$$\Delta = \det(J) = -\delta\gamma(1 - 2A^*) / (1 - A^*)$$

(14)

The trace is always negative for A^* in the interval (0, 1), since both terms are negative. The determinant changes sign depending on whether A^* is below or above 1/2.

6.3 Classification and the Discriminant

The sign of the determinant classifies each equilibrium. At A_- (< 1/2), the factor $(1 - 2A_-)$ is positive, so $\Delta <$ 0, indicating a **saddle point**. At A_+ (> 1/2), the factor $(1 - 2A_+)$ is negative, so $\Delta > 0$ with $\tau < 0$, indicating a **stable equilibrium**.

To determine whether this stable equilibrium is a node or a spiral, we evaluate the discriminant $D = \tau^2 - 4\Delta$. It is convenient to substitute $u = A^*/(1 - A^*)$, noting that $u > 0$ at any non-trivial equilibrium and $u > 1$ at the upper equilibrium A_+. In these variables:

$$\tau = -(\gamma u + \delta), \quad \Delta = \gamma\delta(u - 1)$$

(15)

The discriminant becomes:

$$D = (\gamma u + \delta)^2 - 4\gamma\delta(u - 1) = (\gamma u - \delta)^2 + 4\gamma\delta$$

(16)

Since $(\gamma u - \delta)^2 \geq 0$ and $\gamma\delta > 0$ for all positive parameters, D is strictly positive. Both eigenvalues are real for all parameter values. Combined with $\tau < 0$ and $\Delta > 0$ at A_+, this establishes that the upper equilibrium is always a **stable node**: trajectories converge monotonically, without oscillation. The present two-variable model does not exhibit complex eigenvalues, spiraling trajectories, or damped oscillations for any choice of positive parameters.

6.4 Bifurcation Structure and Global Dynamics

As R_0 increases through the value 4, the saddle A_- and the stable node A_+ coalesce at $A = 1/2$ and annihilate in a saddle-node bifurcation. Below $R_0 = 4$, no non-trivial equilibria exist and the quiescent equilibrium is the global attractor: all trajectories return to quiescence. Above $R_0 = 4$, the phase space is partitioned by the stable manifold of the saddle — the **separatrix** — into two basins of attraction: trajectories originating below the separatrix return to quiescence; trajectories originating above it converge to the cascade attractor (A_+, V_+).

The Bendixson-Dulac criterion (Strogatz, 2014) provides a global result: with the Dulac multiplier $B = 1/A$, the modified divergence $\mathrm{div}(Bf, Bg) = -\beta V - \delta/A$ is strictly negative for all $A > 0$, $V > 0$, excluding periodic orbits from the interior of the first quadrant. The global dynamics are therefore fully characterized by the two stable equilibria and the saddle separatrix: every trajectory converges either to the quiescent equilibrium or to the cascade attractor. No limit cycles, oscillatory regimes, or chaotic dynamics arise in the present model.

Figure 1. Bifurcation Structure

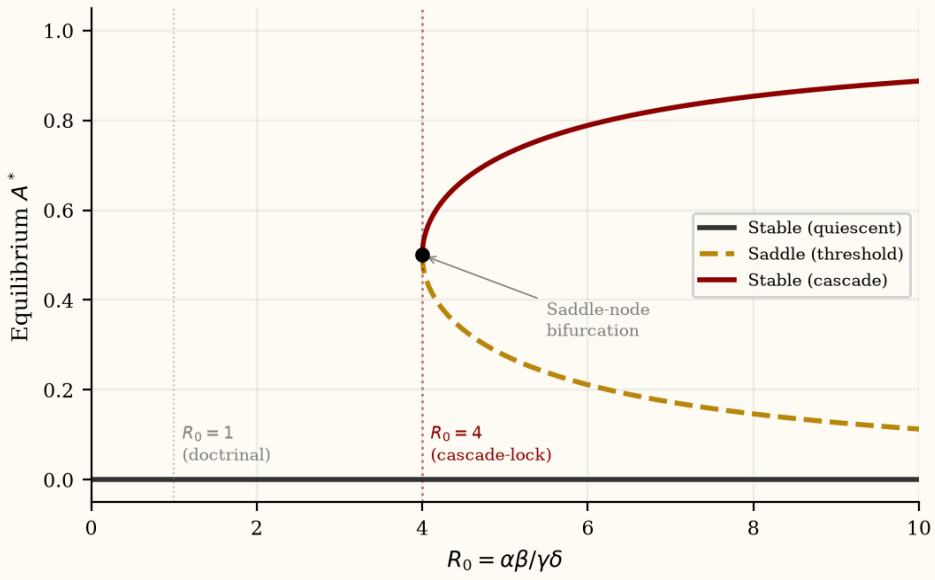

Figure 1. Bifurcation diagram showing the equilibrium anomaly fraction as a function of R_0. The quiescent branch (charcoal) is always stable. At $R_0 = 4$, a saddle-node bifurcation creates the saddle branch (dashed, gold) and the cascade attractor branch (dark red). The doctrinal threshold $R_0 = 1$ and the bistability threshold $R_0 = 4$ are marked.

7. The Cascade Threshold

The cascade reproduction number $R_0 = \alpha\beta / (\gamma\delta)$, defined in equation (7), serves as the dimensionless control parameter for the system. It measures the ratio of total amplification ($\alpha\beta$) to total dissipation ($\gamma\delta$). Three regimes are identified:

Regime I: $R_0 < 1$ (doctrinally safe). Amplification is strictly less than dissipation. The system cannot sustain any anomalous state; finite perturbations return to quiescence after any transient amplification. This is the regime intended by the doctrinal criterion stated in BRD-WP-001.

Regime II: $1 \leq R_0 < 4$ (warning zone). Aggregate gain exceeds damping, but the bounded logistic coupling $(1 - A)$ limits the maximum effective amplification to $\alpha\beta/4$. The quiescent equilibrium remains the only attractor, and all trajectories return to quiescence. The doctrinal criterion flags this regime as vulnerable, but no bistable structure exists. The system is doctrinally at risk but structurally safe.

Regime III: $R_0 > 4$ (cascade-supporting). The saddle-node bifurcation has occurred and the system is bistable: two non-trivial equilibria coexist with the quiescent equilibrium. Cascade onset is not, however, guaranteed. It depends on whether a perturbation pushes the state across the separatrix defined by the saddle's stable manifold. Bistability driven by positive feedback is a well-documented phenomenon in biological and physical systems (Ferrell, 2002); the ACC model exhibits the same threshold architecture in the domain of verification dynamics.

The doctrinal criterion stated in the original working paper — cascade onset when $\alpha\beta > \gamma\delta$, i.e., $R_0 > 1$ — serves as a **necessary condition**: amplification must exceed dissipation for any cascade to be possible. The refined **bistability threshold** $R_0 > 4$, derived from the bounded model, identifies when the cascade-supporting equilibrium structure actually emerges. The interval $1 < R_0 < 4$ is a warning zone: the system satisfies the doctrinal risk criterion but does not yet possess the attractor structure required for self-sustaining corruption. Even for $R_0 > 4$, cascade onset requires a perturbation sufficient to cross the separatrix.

8. Phase Portrait Dynamics

Figure 2 presents a representative phase portrait for $R_0 = 8$ ($\alpha = 4$, $\beta = 2$, $\gamma = 1$, $\delta = 1$), comfortably within the bistable regime.

The nullclines divide the phase space into four regions with characteristic flow directions. The V-nullcline ($V = \alpha A/\delta$) is a straight line through the origin with slope α/δ. The A-nullcline ($V = \gamma/[\beta(1 - A)]$) is a hyperbola asymptotic to $A = 1$. Their intersections at (A_-, V_-) and (A_+, V_+) are the saddle and stable node, respectively.

The stable manifold of the saddle — the separatrix — partitions the phase space into two basins of attraction. Trajectories originating below the separatrix flow toward the quiescent equilibrium: the system recovers. Trajectories originating above the separatrix converge monotonically to the cascade attractor: the cascade locks in. Since the upper equilibrium is a stable node, convergence is non-oscillatory in the present model. Trajectories approach the attractor along the slow eigenvector direction, producing a smooth, monotonic approach without periodic surges.

Oscillatory dynamics — periodic surges of anomaly classification and verification activity — are not a feature of the present two-variable model. They may arise in future extensions incorporating verification delays, state-dependent parameters, stochastic perturbations, or higher-dimensional coupling, as discussed in Section 11.

Figure 2. Phase Portrait ($R_0 = 8$)

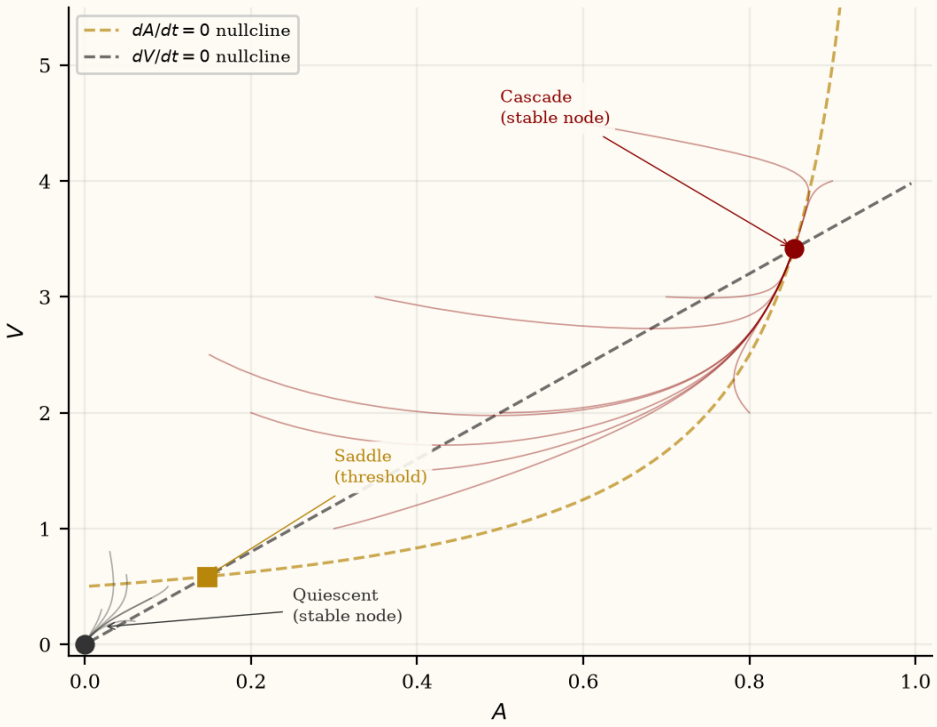

Figure 2. Phase portrait for $R_0 = 8$. Dashed lines are nullclines. Charcoal trajectories return to the quiescent equilibrium at the origin; dark red trajectories converge monotonically to the cascade attractor (stable node). The saddle (square, gold) defines the separatrix between the two basins of attraction.

9. Discussion

9.1 The Autoimmune Analogy

Biological autoimmunity involves the immune system misidentifying self-tissue as foreign and mounting a corrective response that generates tissue damage, releasing more self-antigens and triggering further immune response (Rose & Mackay, 2014). The ACC bears a structural analogy to this process: the verification system misidentifies state information as anomalous, and the resulting corrections generate secondary signals that are themselves classified as anomalous. The relationship is one of shared feedback architecture — self-sustaining correction gone awry — rather than biological equivalence.

Several structural parallels are worth noting. No continuing external adversary is required to sustain the cascade: the pathology arises from normal system operation under parameter conditions that permit bistability. The quiescent state remains locally stable: onset requires a perturbation exceeding a threshold. The pathological state is itself stable: the system does not spontaneously recover. Treatment requires parameter intervention — reducing R_0 below 4 — or a sufficiently large external correction to cross back below the

separatrix.

The analogy also suggests directions the present model does not capture. In biological systems, autoimmune pathology can involve molecular mimicry: a genuine external pathogen shares antigens with self-tissue, triggering a cross-reactive response that persists after the pathogen is eliminated. The analogous phenomenon in continuity systems — a genuine anomaly that shares features with legitimate state, triggering a verification response that persists after the anomaly is corrected — would require a model with memory or state-dependent thresholds, deferred to subsequent work.

9.2 The Verification Paradox

Verification is the corrective mechanism of a continuity system. At low intensity, it performs its intended function: detecting genuine anomalies and initiating correction. The paradox arises because verification itself generates signals — logs, cross-references, state transitions, metadata — that may be classified as anomalous by the same system. In the cascade regime, verification becomes the primary carrier of the anomaly signal it was designed to suppress.

The feedback structure is: Anomaly → Verification → Secondary signals → Anomaly classification → more Verification → ... In the sub-threshold regime, each cycle attenuates. In the super-threshold regime, each cycle amplifies. The system cannot determine from its internal state variables alone whether anomaly classifications correspond to independent ground-truth errors or to self-generated verification artifacts.

This is the verification paradox: the mechanism designed to detect corruption becomes its source. Increasing verification capacity (raising α or β) does not help; it worsens the cascade by increasing R_0. The system cannot verify its way out of a verification-induced cascade.

9.3 Threshold Dynamics and Perturbation Sensitivity

The always-stable quiescent equilibrium means the ACC is not a linear instability phenomenon. The cascade requires a finite perturbation — a transient fault, a configuration error, a burst of correlated noise — large enough to push the system past the separatrix. Small perturbations are self-correcting by design. The margin of safety is the distance from the current operating point to the separatrix, which shrinks as R_0 increases beyond 4. As R_0 grows, the saddle A_- moves toward the origin, reducing the perturbation size required to trigger the cascade. In the limit $R_0 \to \infty$, the saddle approaches the origin, and arbitrarily small perturbations trigger the cascade.

This threshold structure also means that the cascade may appear intermittently: a perturbation pushes the system past the separatrix, the cascade engages, and if external conditions subsequently change (reducing R_0), the system may recover — only to cascade again when the next perturbation arrives. Intermittent cascade behavior is a diagnostic signature of a system operating near $R_0 = 4$.

9.4 Implications for System Design

The model suggests several design principles for continuity-preserving systems. The cascade reproduction number should be tracked as a key operational metric; approach to $R_0 = 4$ should trigger alert, and approach to $R_0 = 1$ should trigger doctrinal review. Since β (verification-to-anomaly amplification) directly contributes to R_0, unbounded recursive verification is inherently dangerous. Verification depth should be capped, and recursive verification of verification artifacts should be suppressed. Alert correlation and deduplication can

reduce the effective β by preventing verification artifacts from being classified as anomalies — the most direct intervention, breaking the feedback loop at its source. Increasing γ (anomaly dissipation) through faster, more accurate correction raises the threshold for cascade onset.

The relevant diagnostic for anomaly amplification is not verification growth — $dV/dt > 0$ is a normal response to any nonzero A, occurring whenever the state lies above the V-nullcline — but rather anomaly growth: $dA/dt > 0$. From the A-nullcline, $dA/dt > 0$ when $V > \gamma/[\beta(1 - A)]$, i.e., when the state lies above the A-nullcline. Sustained growth of A in the absence of externally introduced anomalies indicates amplification. Actual cascade commitment, however, depends on the state being on the cascade side of the separatrix, not merely on $dA/dt > 0$. Proximity to the separatrix can be estimated from the state's position relative to both nullclines and the saddle equilibrium.

10. Bridge Doctrine (Expanded)

The two doctrinal maxims stated in BRD-WP-001 acquire precise meaning within the model.

"The absence of alert is not the absence of error."

The quiescent equilibrium (0, 0) is stable for all parameters, including those in the cascade regime. A system at quiescence may have arrived there by genuine correction or by the exhaustion of verification capacity without resolution. Since A measures classification output rather than ground-truth error, $A = 0$ certifies only that nothing is currently classified as anomalous. The model provides no mechanism for distinguishing quiescence from health without external validation.

"The presence of alert is not evidence of error."

In the cascade regime ($R_0 > 4$), alerts are generated by the verification process itself. The stable cascade attractor (A_+, V_+) sustains a non-zero anomaly level indefinitely, not because genuine ground-truth errors persist, but because verification generates the signals it classifies. An alerting system in the cascade regime produces alerts at a rate determined by the attractor, not by the underlying error rate.

The following corollaries extend the doctrine:

"Verification that generates more verification is not verification."

Sustained growth of verification activity in the absence of externally introduced anomalies warrants investigation, though verification growth alone ($dV/dt > 0$) is a normal response to any nonzero A. The more diagnostic indicator is sustained anomaly growth ($dA/dt > 0$): verification that produces more anomaly classification than it resolves is not correcting the system but amplifying its pathology.

"The system that corrects itself corrupts itself."

The fundamental paradox of autonomous self-correction: the corrective mechanism, operating on its own outputs, can become the source of the cascade. Self-correction without external ground truth is structurally vulnerable to the ACC. The system can observe its own state variables A and V, but it cannot determine from

them alone whether anomaly classifications correspond to independent ground-truth errors or to self-generated verification artifacts. The distinction between signal and noise is not available from internal measurements.

11. Limitations and Future Work

11.1 Limitations

Two-variable minimalism. The model captures only aggregate anomaly fraction and verification intensity. Real systems exhibit heterogeneous anomaly distributions, multiple verification modalities, and coupled state domains. The scalar model cannot represent phenomena such as spatially localized cascades or domain-specific verification failures.

Constant parameters. The rates α, β, γ, δ are treated as fixed. In practice, these may depend on system state, load, configuration, or time. State-dependent parameters could produce richer bifurcation structures, potentially including Hopf bifurcations and oscillatory dynamics not present in the current model.

Deterministic framework. The model does not account for stochastic perturbations. In systems operating near $R_0 = 4$, noise may drive threshold crossing with non-negligible probability, making the cascade a stochastic rather than purely deterministic phenomenon.

No oscillatory dynamics. The present two-variable model with constant parameters does not produce oscillations: the upper equilibrium is always a stable node, and the Bendixson-Dulac criterion excludes periodic orbits. Oscillatory behavior requires extensions such as delays, state-dependent parameters, or additional degrees of freedom.

Well-mixed assumption. The model assumes uniform interaction between anomalies and verification. Spatially distributed or network-coupled systems may exhibit propagating cascade fronts, synchronization effects, and spatial pattern formation not captured by the scalar model.

11.2 Future Directions

Stochastic ACC. Model noise-induced threshold crossing and estimate first-passage times to the separatrix as a function of noise amplitude and R_0. This would quantify the probability of cascade onset in systems operating near the threshold.

Networked ACC. Extend to coupled continuity domains on a network topology, investigating cascade propagation, synchronization, and the conditions under which a local cascade becomes global.

Delayed verification. Introduce verification latency, $dV/dt = \alpha A(t - \tau) - \delta V$, and analyze the resulting delay-induced dynamics. Delays can destabilize otherwise stable equilibria, potentially producing the oscillatory behavior absent from the present model and lowering the effective cascade threshold.

Control and intervention. Develop control strategies for driving a cascaded system back below the separatrix, including parameter modulation (reducing α or β, increasing γ or δ) and external ground-truth correction — the introduction of independently verified state information to displace the system from the cascade basin without relying on the corrupted verification channel.

State-dependent parameters. Allow α, β, γ, δ to vary with A or V, potentially producing Hopf bifurcations (persistent oscillations) and routes to chaos through period-doubling cascades — none of which arise in the present constant-parameter model.

Multi-domain cascade propagation. Model ACC propagation across coupled subsystems, identifying conditions for cascade contagion and designing inter-domain isolation mechanisms.

12. Conclusion

The Autoimmune Continuity Cascade model demonstrates that continuity-preserving systems can fail not despite their corrective mechanisms but because of them. The two-dimensional dynamical system reveals a saddle-node bifurcation at $R_0 = 4$ that separates the monostable quiescent regime from the bistable cascade-supporting regime. The cascade is a threshold phenomenon: the quiescent equilibrium is always locally stable, but a sufficiently large perturbation can push the system past a separatrix into a stable cascade attractor from which it cannot recover without parameter intervention. The upper equilibrium is a stable node for all parameter values, and the Bendixson-Dulac criterion excludes periodic orbits: the present model produces monotonic convergence, not oscillation.

The doctrinal criterion $R_0 > 1$ identifies the necessary condition for vulnerability; the bistability threshold $R_0 > 4$ identifies the condition under which cascade-supporting structure emerges. Between these thresholds lies a warning zone where the system is doctrinally vulnerable but structurally safe. Even above $R_0 = 4$, cascade onset is not guaranteed: it requires a perturbation sufficient to cross the separatrix.

The Bridge Doctrine's maxims — that quiescence does not prove health and alert does not prove error — find their mathematical expression in the model's equilibrium structure. The stable quiescent equilibrium guarantees silence; the stable cascade attractor guarantees sustained alert. The system can observe its own state variables, but it cannot determine from them alone whether anomaly classifications correspond to independent ground-truth errors or to self-generated verification artifacts. The model's contribution is to show that these two stable states coexist in the same system, separated by a threshold that shrinks as amplification grows, and that the distinction between genuine anomaly and verification-induced noise is not available from internal measurements alone.

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