

Emergent Complex Contagion: Fast-Variable Reduction as a Unifying Principle for Epidemic and Social Spreading Models

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Abstract

Models of epidemics and social contagion acquire realism by adding mechanism: memory of past exposures, adaptive behavior, payoff-biased imitation, signed ties, co-circulating processes, or modular structure. Each mechanism enlarges the state space and typically destroys analytic tractability. We propose that a broad class of such models shares a hidden simplifying structure: the mechanism-specific degrees of freedom are *fast variables* that equilibrate on a timescale much shorter than the epidemic timescale. Adiabatic elimination of these fast variables then yields an *emergent complex contagion*: the macroscopic force of infection becomes a nonlinear, threshold-like function of prevalence even when every microscopic event is memoryless and simple. We formalize the reduction, prove an adiabatic error bound $\Delta_i \leq 1/\kappa_i$ in terms of the timescale separation κ_i , and work a fully explicit example — memory-induced exposure accumulation on a configuration network — deriving the reduced law $\dot{\rho} = c_2 \rho^3$ with $c_2 = 44 \text{ day}^{-1}$, the emergent threshold prevalence $\rho_c \approx 0.0674$, and the adiabatic error $\epsilon(\rho) = 44\rho^2$, equal to 1.76×10^{-2} at $\rho = 0.02$. The reduction predicts algebraic rather than exponential early growth, explaining why emergent complex contagions require seeding, and it organizes a reading of the complex-contagion literature in which macro-level reinforcement need not correspond to micro-level threshold rules.

1. Introduction

Epidemic and social-spreading modeling has fragmented into a zoo of mechanisms. Memory of past exposures [6], competition between simple and complex spreading [2], payoff-biased social learning [3], modular network structure [4], signed (trust/distrust) ties [8], behavioral vaccine decisions [10], and continuous-state financial contagion [11] each extend the basic susceptible–infected picture in a different direction. The price of realism is dimensionality: a model with per-agent memory, opinion states, or behavioral modes has many more variables than nodes, and closed-form analysis typically fails.

This paper advances a single organizing claim: in many of these extensions, the added degrees of freedom are dynamically *fast*. They relax to (quasi-)equilibrium on a timescale τ_{fast} that is short compared with the epidemic timescale τ_{epi} over which prevalence changes appreciably. When this two-timescale structure holds, the fast variables can be eliminated by adiabatic (quasi-static) approximation, and the slow epidemic dynamics inherits an effective force of infection that is a nonlinear function of the slow variables. We call this *emergent complex contagion*: the macroscopic spreading law exhibits the signature of complex contagion — a need for repeated or collective exposure before transmission — even though no microscopic rule encodes a response threshold.

The claim matters for three reasons. First, it offers a unified reading of otherwise disparate models: memory-induced reinforcement [6], behavioral feedback [10], and payoff-biased imitation [3] can all be viewed as fast variables whose quasi-equilibrium shapes the force of infection. Second, it gives a diagnostic: if a high-dimensional contagion model can be reduced, its macroscopic behavior should show the algebraic early growth and threshold-like dependence derived below, distinguishing it from simple contagion. Third, it connects to the empirical problem of telling simple from complex contagion in data, which is known to be difficult [9]: emergent complex contagion provides one mechanism by which complex-contagion phenomenology can arise without explicit thresholds.

Our contributions are: (i) a general two-timescale formulation of mechanism-augmented epidemic models and the adiabatic reduction procedure, with an explicit error bound; (ii) a fully worked analytic example — memory-induced exposure accumulation on a configuration network — with every numerical result derived step by step; and (iii) a discussion of how the reduction organizes the empirical and modeling literature on complex contagion in music listening [1], donations [5], information flow [7], and financial fire sales [11].

2. Background and Related Work

Simple versus complex contagion. Simple contagion transmits after a single exposure; complex contagion demands multiple exposures before transmission [2]. The distinction is empirically consequential but hard to settle from spreading data alone: [9] studies precisely the problem of distinguishing simple from complex contagion processes on networks and notes that empirical spreading data, even when available, do not easily allow one to uncover which of the two underlying mechanisms operates — a difficulty our framework speaks to directly, because it predicts that the two regimes can interconvert as parameters controlling the timescale separation vary.

Empirical complex contagion. Reference [1] constructs a dataset from Last.fm covering the listenership history of 1.3 million users over a two-month horizon and, using daily play counts and event attendance in a regression discontinuity design, finds evidence that live music events generate complex contagion in music streaming — but only for the most popular artists. Reference [5] studies the diffusion of campaign donations through a network of more than 50,000 elites and finds that connectivity among previous donors reinforces contagion. Both studies document reinforcement-style phenomenology whose microscopic origin is not directly observed, which is exactly the situation our emergent mechanism addresses: reinforcement at the macro level need not correspond to an explicit threshold rule at the micro level.

Mechanism-rich models. Reference [6] develops an infection mechanism endowed with memory of past exposures that simultaneously incorporates the joint effect of multiple infectious sources, providing analytic equations and simulations within susceptible–infected frameworks; our worked example in Sections 3–4 is a minimal analytic treatment of the same memory ingredient, recast as a fast variable. Reference [3] introduces a mathematical model combining complex contagion with payoff-biased imitation to describe how social behaviors spread, generalizing imitation beyond single-interaction adoption; in our language, the imitation kernel is a fast variable tracking the payoff environment. Reference [10] models the interplay of epidemic spreading and vaccine uptake, treating epidemic spread as simple contagion while vaccine uptake follows complex contagion dynamics in which individuals' decisions depend on multiple social contacts; this is a natural two-timescale candidate, with behavioral state as the fast variable, though a full reduction of that model is left here as a labeled projection (Section 6). Reference [8] studies complex contagion in social systems with both trust and distrust (signed) relations and group interactions, noting that very little is known about the effect of signed interactions on spreading; sign structure enters our framework as an additional fast-equilibrating layer that rescales the effective exposure accumulation rate. Reference [11] treats financial fire sales — where a bank failure causes asset liquidation, price falls, and further defaults — as a continuous-state complex contagion that cannot be modeled by standard binary-threshold approaches; this extends the relevance of complex contagion beyond binary adoption, and our continuous-state reduction (Section 3.3) is designed to cover such cases. Reference [2] studies competing contagion processes in which some agents follow simple contagion while others follow complex contagion, with complex contagion triggered by simple contagion; a triggering cascade is plausibly the fast variable that equilibrates ahead of the complex layer.

Structural drivers. Reference [4] applies a theoretical framework to study how the modular structure of the social network affects complex contagion, focusing on *optimal modularity* — the prediction that global cascades occur when the network exhibits just the right amount of modularity — and generalizing the findings under stated

assumptions. Modularity enters our reduction as a modifier of the degree moment $\langle k^\ell \rangle$ that controls the effective force of infection. Reference [7] shows that complex contagion features can arise in a model of social information flow *without* social reinforcement, i.e., without individuals requiring multiple exposures; this is the closest published antecedent to our thesis, since it demonstrates complex-contagion phenomenology from a mechanism that is not an explicit threshold, and our adiabatic elimination result supplies a general dynamical route to the same conclusion.

Framework documents. The QNFO corpus entries [12], [13], and [14] — a technical framework for network isomorphism, a resonant complexity framework, and a principia ontologica — are supplied with no summary text; the provided entries give no further detail, so we relate them to this work only at the level of their titles: network-structural and complexity-framework perspectives that are complementary to, but not derivational for, the reduction presented here.

In summary, the literature supplies rich mechanisms and rich phenomenology but treats each mechanism as a separate high-dimensional model. The reduction principle proposed here is not articulated as a general tool in any of the cited works, although [7] anticipates the spirit by producing complex features without stipulated reinforcement.

3. Methods

3.1 General two-timescale form

Let $\rho(t) = I(t)/N$ denote prevalence (the slow variable) and let $\mathbf{m}(t)$ collect mechanism-specific fast variables (memory levels, behavioral states, imitation pressures, trigger intensities). We consider systems of the form

$$\dot{\rho} = F(\rho, \mathbf{m}), \quad \dot{m}_i = \frac{1}{\tau_{\text{fast}}} G_i(\rho, \mathbf{m}),$$

where τ_{fast} is the fast relaxation time and F, G_i are smooth. The fast subsystem $G_i = 0$ defines a quasi-equilibrium manifold $\mathbf{m} = \mathbf{m}^*(\rho)$. Substitution gives the reduced one-dimensional system

$$\dot{\rho} = F(\rho, \mathbf{m}^*(\rho)) \equiv f(\rho).$$

3.2 Adiabatic error bound

Let γ_i be the linear relaxation rate of m_i near its quasi-steady state and $\kappa_i = \gamma_i/\mu$ the separation from the epidemic recovery rate μ . To first order in $1/\kappa_i$, the slaved solution is $m_i = m_i^*(\rho) - \dot{\rho}(\partial m_i^*/\partial \rho)/\gamma_i + \mathcal{O}(\kappa_i^{-2})$. Since prevalence changes by at most $\mathcal{O}(1)$ over the time $1/\mu$, the relative adiabatic error in m_i is bounded by

$$\Delta_i \leq \frac{\mu}{\gamma_i} = \frac{1}{\kappa_i}.$$

Thus a one-order-of-magnitude separation ($\kappa_i = 10$) guarantees errors of at most $\mathcal{O}(10^{-1})$ in the fast variables, and the error decreases linearly in κ_i . Equivalently, the reduction is valid to leading order when $\epsilon(\rho) \equiv \tau_{\text{fast}} |f'(\rho)| \ll 1$, i.e., when prevalence changes little over one fast relaxation time; Section 4.4 quantifies this for the worked example.

3.3 Worked model: memory-induced emergent complex contagion

Individuals sit on a configuration-model network with degree distribution p_k ; infected individuals recover at rate γ (units day^{-1}). Each susceptible individual of degree k carries a scalar memory (exposure) variable m obeying

$$\dot{m} = \beta k \rho - \gamma_m m,$$

where β is the per-edge exposure rate from a unit-infected population and γ_m is the memory decay rate. Infection occurs at hazard αm^ℓ with reinforcement exponent $\ell \geq 1$; $\ell = 1$ recovers simple contagion, $\ell = 2$ is the minimal emergent-complex case. Note that the *microscopic* rules are memoryless in the Markov sense: m is a mechanistic accumulator, not a response threshold, yet the macroscopic hazard αm^ℓ is threshold-like for $\ell > 1$.

Because γ_m^{-1} is short compared with the epidemic timescale (verified a posteriori in Section 4.4), we set $\dot{m} \approx 0$:

$$m^*(\rho, k) = \frac{\beta k \rho}{\gamma_m}.$$

The reduced prevalence equation, with susceptible fraction ≈ 1 in the early phase, is

$$\dot{\rho} = \sum_k p_k \alpha [m^*(\rho, k)]^\ell \rho = \frac{\alpha \beta^\ell \langle k^\ell \rangle}{\gamma_m^\ell} \rho^{\ell+1} \equiv c_\ell \rho^{\ell+1}.$$

The instantaneous (per-capita) reproduction number is

$$R_{\text{eff}}(\rho) = \frac{c_\ell \rho^\ell}{\gamma},$$

and the emergent threshold prevalence solves $R_{\text{eff}}(\rho_c) = 1$:

$$\rho_c = \left(\frac{\gamma \gamma_m^\ell}{\alpha \beta^\ell \langle k^\ell \rangle} \right)^{1/\ell}.$$

For $\ell > 1$ the early growth is algebraic, not exponential: the system has no autonomous takeoff from an infinitesimal seed, the hallmark of complex contagion.

3.4 Continuous-state extension

For continuous-state contagions such as fire-sale dynamics [11], the same reduction applies with m a continuous distress level and ℓ effectively set by the local scaling of the hazard near equilibrium; the threshold ρ_c becomes a continuous instability boundary rather than a binary cascade condition.

4. Analysis

Every input number below is stated with its origin: all parameter values are *reference choices made in this paper* for the worked model of Section 3.3; all derived numbers follow from the formulas of Section 3.3 by explicit arithmetic. None are empirical measurements.

4.1 Network moments

We take a Poisson (configuration) degree distribution with mean $\langle k \rangle = 10$. For a Poisson distribution the second moment is $\langle k^2 \rangle = \langle k \rangle^2 + \langle k \rangle$:

$$\langle k^2 \rangle = 10^2 + 10 = 100 + 10 = 110.$$

Since $\ell = 2$ in the emergent case, the required moment is $\langle k^\ell \rangle = \langle k^2 \rangle = 110$.

4.2 Reduced growth coefficient

Reference parameters: $\beta = 0.1 \text{ day}^{-1}$, $\gamma_m = 1.0 \text{ day}^{-1}$, $\alpha = 40 \text{ day}^{-1}$, $\gamma = 0.2 \text{ day}^{-1}$, $\ell = 2$. Then

$$c_2 = \frac{\alpha \beta^2 \langle k^2 \rangle}{\gamma_m^2} = \frac{40 \times 0.1^2 \times 110}{1.0^2} = \frac{40 \times 0.01 \times 110}{1} = \frac{44}{1} = 44 \text{ day}^{-1},$$

so the reduced dynamics is $\dot{\rho} = 44 \rho^3$.

4.3 Emergent threshold and effective reproduction number

$$\rho_c = \sqrt{\frac{\gamma}{c_2}} = \sqrt{\frac{0.2}{44}} = \sqrt{4.5454545 \times 10^{-3}} = 0.0674200$$

($\approx 6.74\%$ of the population). The instantaneous reproduction number is

$$R_{\text{eff}}(\rho) = \frac{c_2 \rho^2}{\gamma} = \frac{44 \rho^2}{0.2} = 220 \rho^2.$$

Checks:

- At $\rho = 0.05$: $R_{\text{eff}} = 220 \times (0.05)^2 = 220 \times 2.5 \times 10^{-3} = 0.55 < 1$.
- At $\rho = 0.10$: $R_{\text{eff}} = 220 \times 0.01 = 2.2 > 1$.
- At $\rho = \rho_c$: $R_{\text{eff}} = 220 \times \rho_c^2 = 220 \times (\gamma/c_2) = 220 \times (0.2/44) = 220 \times (1/220) = 1$ exactly.

For contrast, the $\ell = 1$ (simple-contagion) baseline with the same parameters gives

$$R_0^{(\ell=1)} = \frac{\alpha \beta \langle k^2 \rangle}{\gamma_m \gamma} = \frac{40 \times 0.1 \times 110}{1.0 \times 0.2} = \frac{440}{0.2} = 2200,$$

i.e., the same microscopic rules with linear hazard are massively supercritical from an infinitesimal seed, whereas the quadratic-hazard (memory-reinforced) system is subcritical below $\rho_c \approx 0.0674$.

4.4 Adiabatic error

The leading correction to the quasi-static memory is $m \approx m^* - \dot{m}^*/\gamma_m$, with $\dot{m}^* = \beta k \dot{\rho}/\gamma_m$. The relative correction is

$$\frac{\dot{m}^*}{\gamma_m m^*} = \frac{\dot{\rho}}{\gamma_m \rho} = \frac{c_2 \rho^2}{\gamma_m} = 44 \rho^2 \quad (\gamma_m = 1.0).$$

This equals $\tau_{\text{fast}} |f'(\rho)|$ up to order-unity factors, with $\tau_{\text{fast}} = 1/\gamma_m = 1 \text{ day}$ and $f'(\rho) = 3c_2 \rho^2$; we quote the conservative value $\epsilon(\rho) = c_2 \rho^2/\gamma_m$:

- At $\rho = 0.02$: $\epsilon = 44 \times (0.02)^2 = 44 \times 4 \times 10^{-4} = 1.76 \times 10^{-2}$.
- At $\rho = 0.05$: $\epsilon = 44 \times 2.5 \times 10^{-3} = 0.11$.
- At $\rho = \rho_c = 0.0674$: $\epsilon = 44 \times 4.5455 \times 10^{-3} = 0.20$.

The reduction is therefore accurate to about 2% at $\rho = 0.02$ and degrades to $\mathcal{O}(1)$ near the threshold, where the intrinsic epidemic timescale $\tau_{\text{epi}} = 1/|f'(\rho)|$ becomes comparable to τ_{fast} . This defines the validity domain of the reduction: deep in the early-growth regime and, with weaker accuracy, through the threshold crossing. Consistency with the general bound of Section 3.2: here $\kappa_m = \gamma_m/\mu$ -type separation is replaced by the prevalence-based ratio, and the bound $\Delta \leq 1/\kappa$ is respected since $\epsilon(\rho_c) = 0.20 < 1$.

4.5 Growth times

Solving $\dot{\rho} = c_2 \rho^3$ by separation: $d(1/\rho^2)/dt = -2c_2$, so the time to grow from ρ_0 to ρ_1 is

$$t = \frac{1/\rho_0^2 - 1/\rho_1^2}{2c_2} = \frac{1/\rho_0^2 - 1/\rho_1^2}{88}.$$

From $\rho_0 = 10^{-2}$ to $\rho_1 = \rho_c$ (using $1/\rho_c^2 = c_2/\gamma = 44/0.2 = 220$):

$$t = \frac{10^4 - 220}{88} = \frac{9780}{88} = 111.13636 \text{ days} \approx 111.1 \text{ days}.$$

This long seeding time — more than a hundred days to climb two orders of magnitude in prevalence from a 1% seed — is the operational content of the statement that emergent complex contagions require seeding: an infinitesimal seed effectively never takes off, while a seed above ρ_c invades directly.

5. Results

All numbers below are computed in Section 4 from the stated parameter values; none are empirical measurements or simulations.

1. **Network moment.** For the Poisson reference network with $\langle k \rangle = 10$, $\langle k^2 \rangle = 110$ (Section 4.1).
2. **Reduced growth coefficient.** $c_2 = 44 \text{ day}^{-1}$, giving $\dot{\rho} = 44\rho^3$ (Section 4.2).
3. **Emergent prevalence threshold.** $\rho_c = \sqrt{0.2/44} = 0.0674200 \approx 6.74\%$ of the population (Section 4.3).
4. **Reproduction numbers.** $R_{\text{eff}}(\rho) = 220\rho^2$; $R_{\text{eff}} = 0.55$ at $\rho = 0.05$, 2.2 at $\rho = 0.10$, and 1 exactly at ρ_c . The $\ell = 1$ baseline with the same parameters gives $R_0^{(\ell=1)} = 2200$ (Section 4.3).
5. **Adiabatic error.** $\epsilon(\rho) = 44\rho^2$: 1.76×10^{-2} at $\rho = 0.02$, 0.11 at $\rho = 0.05$, 0.20 at ρ_c (Section 4.4).
6. **Seeding time.** Growth from $\rho_0 = 10^{-2}$ to ρ_c takes $t = 9780/88 \approx 111.1$ days (Section 4.5).

Projection (labeled, not computed). Applying the reduction to coupled behavior–epidemic systems of the kind in [10] — vaccine uptake as a fast behavioral variable coupled to a simple-contagion pathogen — should yield an effective force of infection modulated by a quasi-steady uptake state, with error bounded by the behavioral relaxation ratio $\kappa_v = \gamma_v/\mu$. We project qualitatively that such reductions will produce threshold phenomena in the *behavioral* variable analogous to ρ_c above; we do not report numerical values for this class, since no parameter set for it is specified here.

6. Discussion

What the reduction buys. A single principle — fast auxiliary variables equilibrate and can be eliminated — converts a family of high-dimensional contagion models into one-dimensional effective systems with nonlinear forces of infection. The worked example shows that the hallmark of complex contagion (a finite prevalence threshold, algebraic early growth, seeding dependence) emerges from simple contagion plus a fast memory loop, with all quantities obtainable in closed form. This complements [7], which demonstrated complex features without stipulated reinforcement in a specific information-flow model, by giving a general dynamical route with an explicit error bound. It also sharpens the inference problem of [9]: if emergent complex contagion is generic under fast equilibration, then observing threshold-like macrodynamics does *not* certify microlevel multiple-exposure rules.

Limitations and failure modes. (i) *Chosen parameters.* All parameter values (β , γ_m , α , γ , $\langle k \rangle$, ℓ) are reference choices, not empirical calibrations; the specific numbers ($c_2 = 44$, $\rho_c \approx 0.0674$, $t \approx 111.1$ days) illustrate the structure, not any particular real system. (ii) *Error near threshold.* The adiabatic error reaches 0.20 at ρ_c and grows as prevalence rises further; the reduction is quantitative only in the early-growth regime. (iii) *Degree distribution.* The Poisson moment computation is exact only for that distribution; heavy-tailed networks change $\langle k^2 \rangle$ drastically and with it ρ_c , and modularity as studied in [4] modifies the effective moment in ways not computed here. (iv) *Homogeneous mixing of memory.* The model assumes a single population-average memory variable per degree class; correlated memory and reinfection loops could violate the fast-equilibration hypothesis. (v) *Signed ties.* For distrust relations [8], the supplied summary gives no quantitative detail, so the claim that sign structure acts as a fast layer is a hypothesis, not a derivation.

What would falsify the claims. The central falsifiable prediction is algebraic early growth $\dot{\rho} \propto \rho^{\ell+1}$ with a finite ρ_c , in contrast to exponential growth from infinitesimal seeds in the $\ell = 1$ baseline (where $R_0^{(\ell=1)} = 2200$ under the same parameters). Empirical spreading cascades that take off from arbitrarily small seeds with no threshold prevalence, in systems independently established to have fast memory-like variables, would contradict the reduction. Conversely, direct measurement of fast-variable relaxation rates γ_i comparable to or slower than the epidemic rate μ would remove the systems from the reduction's validity domain.

Open questions. How does the reduction interact with optimal modularity [4], where cascade geometry is itself the object of design? Does distrust [8] introduce additional slow variables that break the separation? Can the vaccine-uptake coupling [10] be reduced with empirically measured κ_v ? These require parameterized empirical studies that the supplied literature summaries do not provide.

7. Conclusion

Fast-variable reduction offers a unifying analytical framework for complex contagion models across biological, social, and financial systems. By exploiting the separation between rapidly equilibrating auxiliary processes and the slower epidemic core, we obtain low-dimensional equations that retain the influence of memory, reinforcement, and higher-order exposure structure, with an explicit error bound $\Delta_i \leq 1/\kappa_i$. The worked example demonstrates the full phenomenology of complex contagion — threshold prevalence $\rho_c \approx 0.0674$, algebraic growth, and a seeding requirement of order 111 days from a 1% seed — arising from entirely memoryless microscopic rules. This provides both a modeling tool and a caution for inference: complex-contagion phenomenology at the macro level does not uniquely identify multiple-exposure rules at the micro level.

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Appendix A. Divergence report

The independent drafts agreed on the core reduction principle, the adiabatic error bound, and the readings of the literature, but **diverged on the choice of worked example and its quantitative consequences**:

- **Draft B** worked a well-mixed susceptible–infected–susceptible model with a fast memory variable entering the force of infection multiplicatively, $\lambda_{\text{eff}} = \beta_0(1 + cI)$, with chosen parameters $\beta_0 = 0.09$, $\mu = 0.1$, $c = 2$. It derived a bare reproduction number $\mathcal{R}_0^{(0)} = 0.9$, an unstable emergent threshold $I_c = 1/6 \approx 0.1667$, a stable endemic state $I_e = 1/3 \approx 0.3333$, and a peak effective reproduction number $\mathcal{R}_{\text{eff}}^{\text{max}} = 1.0125$ at $I = 0.25$ — i.e., bistability between a disease-free and an endemic state.
- **Draft C** worked a configuration-network model with a fast memory accumulator and hazard αm^ℓ , deriving algebraic growth $\dot{\rho} = c_2 \rho^3$ with $c_2 = 44 \text{ day}^{-1}$ and a threshold prevalence $\rho_c \approx 0.0674$.

Convention behind the disagreement. The two examples differ in both microscopic hazard structure (multiplicative reinforcement of the transmission rate versus power-law hazard in a memory accumulator) and population structure (well-mixed versus degree-heterogeneous network). Both are legitimate instantiations of the same reduction principle; they are not competing measurements of the same