

# Nested Hyperedges Promote the Onset of Collective Transitions but Suppress Explosive Behavior

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Higher-order interactions can induce abrupt collective transitions, yet the microscopic mechanisms controlling macroscopic critical behavior remain unclear. Here we show that nested hyperedges generate a dual effect on dynamical processes: they promote the onset of collective behavior while suppressing the explosive transitions driven by higher-order feedback. To uncover the mechanism, we develop an analytically tractable theory of contagion on hypergraphs that explicitly tracks nestedness between groups of different sizes, allowing us to identify the microscopic mechanism responsible for this dual behavior. By disentangling contagion pathways, we find that nestedness redirects transmission from external links to internal, group-embedded routes—boosting early activation but making dyadic and triadic channels increasingly redundant. This loss of structural independence quenches the nonlinear amplification required for bistability, progressively smoothing the transition as hyperedges become nested. The phenomenology holds for groups of any size and is not specific to spreading dynamics but also emerges in higher-order Ising and Kuramoto dynamics. Overall, our results identify nestedness between group interactions as a general structural mechanism governing critical transitions in complex systems.

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**Introduction**—Interactions involving groups of units, beyond pairwise ones, enrich the collective behavior of complex systems, giving rise to multistability, hysteresis, and discontinuous transitions [1–9]. Recent studies have shown that these phenomena depend not only on the presence of such higher-order interactions but also on their microscopic organization [10–18]. A particularly important structural property is *nestedness*, namely the tendency of smaller interactions to be embedded within larger ones. Such nested structures have been repeatedly observed in empirical systems [15,19–22], revealing that interactions of different sizes are often organized hierarchically rather than independently. Although nestedness is known to facilitate activation in higher-order contagion processes [12,15], whether and how it controls the nature of collective

transitions, in particular the emergence of backward bifurcations, leading to bistability and hysteresis, remains unknown.

In this Letter, we identify the microscopic mechanism by which nested hyperedges simultaneously promote collective activation and suppress explosive transitions. To uncover this mechanism analytically, we use higher-order contagion as a minimal yet tractable setting. We consider a homogeneous mean-field description that allows us to characterize the emergence of backward bifurcations while explicitly disentangling external and group-embedded transmission pathways. This, in turn, provides analytical access to the early-time dynamical correlations governing the onset of collective behavior through a fast-variable approach. We show that increasing nestedness promotes early activation but progressively reduces the structural independence between pairwise and higher-order transmission, weakening the nonlinear reinforcement responsible for bistability. As a consequence, nestedness simultaneously lowers the onset threshold while shrinking the bistable region. Finally, we show that this mechanism is neither specific to contagion dynamics nor restricted to pairwise-three-body interactions. The same phenomenology emerges across different interaction orders and in higher-order Ising [7,23] and Kuramoto dynamics [5],

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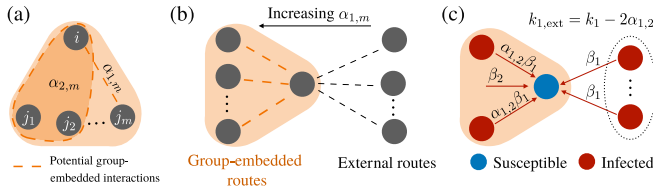


FIG. 1. Nestedness across interaction orders. (a) Inter-order overlap  $\alpha_{p,m}$  quantifies the fraction of  $p$  faces of higher-order groups that are realized as lower-order interactions. Dashed connections denote potential embedded interactions. (b) Increasing nestedness redistributes lower-order interaction routes from external neighborhoods to group-embedded ones. (c) In the SIS model, this structural reorganization is incorporated analytically by decomposing pairwise transmission into embedded and external routes. When the susceptible node is conditioned on belonging to a 2-hyperedge, nestedness reduces the number of available external routes to  $k_{1,ext} = k_1 - 2\alpha_{1,2}$ .

identifying nestedness between interaction orders as a general structural principle governing both the onset and the nature of collective transitions.

**Modeling nestedness of group interactions**—We consider hypergraphs  $\mathcal{H} = (\mathcal{N}, \mathcal{E})$ , where each hyperedge  $e \in \mathcal{E}$  has order  $m = |e| - 1$ , with  $m = 1$  denoting pairwise interactions,  $m = 2$  denoting three-body interactions, and so forth. To quantify structural correlations across interaction orders, we use the *inter-order hyperedge overlap* [11],  $\alpha_{p,m} = |\mathcal{E}_p \cap \mathcal{F}(\mathcal{E}_m)| / |\mathcal{F}(\mathcal{E}_m)|$ , with  $p < m$ , where  $\mathcal{F}(\mathcal{E}_m)$  is the set of  $p$  cliques contained within the  $m$ -hyperedges. This quantity measures the fraction of  $p$ -body interactions embedded within  $m$ -body groups. For example,  $\alpha_{1,2} = 0$  corresponds to independent pairwise and three-body interactions, whereas  $\alpha_{1,2} = 1$  denotes complete nesting. Although  $\alpha_{p,m}$  is defined as a global structural quantity, in homogeneous hypergraphs it can be interpreted as the probability that a randomly selected  $p$  face of an  $m$ -hyperedge is realized as a  $p$ -hyperedge [Fig. 1(a)]. Increasing nestedness redistributes lower-order interactions from external neighborhoods to group-embedded ones. For instance, larger  $\alpha_{1,m}$  reduces the number of external pairwise routes available within an  $m$ -body interaction while increasing the fraction of embedded ones [Fig. 1(b)]. Although illustrated for pairwise interactions, the same route redistribution applies to arbitrary orders  $p < m$ . Thus, nestedness tunes the structural independence between interaction orders, raising the question of how this reorganization shapes both the onset and the nature of collective transitions.

**An analytically tractable framework**—To uncover the microscopic mechanism analytically, we use Susceptible-Infected-Susceptible (SIS) dynamics as a minimal yet tractable setting. We consider regular hypergraphs with pairwise ( $m = 1$ ) and three-body ( $m = 2$ ) interactions, where each node belongs to exactly  $k_1$  links and  $k_2$  three-body groups. Susceptible nodes become infected

through pairwise interactions at rate  $\beta_1$  or through three-body interactions at rate  $\beta_2$  when both other nodes in the group are infected, while infected nodes recover at rate  $\mu$ . The two transmission channels represent distinct dyadic and group-mediated mechanisms, as commonly assumed in models of social contagion [3,4,24], rather than a single transmission process whose rate depends on group size.

Our homogeneous mean-field framework explicitly retains the cross-order correlations induced by nestedness by tracking node, pair, and group motifs. The density of infected nodes evolves as

$$\dot{\rho}^I = -\mu\rho^I + \beta_1 k_1 \rho^{SI} + \beta_2 k_2 \rho^{ISI\Delta}, \quad (1)$$

where  $\rho^{SI}$  and  $\rho^{ISI\Delta}$  denote the densities of infected-susceptible links and mixed three-body groups, respectively. Together with the evolution of the remaining motif densities, Eq. (1) forms a closed dynamical system. Nestedness enters the dynamics through this probabilistic interpretation: pairwise transmission is statistically decomposed into group-embedded and external routes. For a node already belonging to a three-body interaction, increasing  $\alpha_{1,2}$  reduces the number of external routes in favor of internal ones, yielding  $k_{1,ext} = k_1 - 2\alpha_{1,2}$  [Fig. 1(c)], the only structural ingredient through which nestedness modifies the equations. The resulting motif hierarchy is then closed using standard homogeneous approximations [25,26], while preserving the cross-order correlations generated by nestedness. Details of the derivation are reported in the Appendix A.

**Nestedness reshapes critical transitions**—We now quantify, within this analytically tractable setting, how route redistribution induced by nestedness shapes the onset and nature of collective transitions. We use the rescaled infectivities  $\lambda_1 = k_1 \beta_1 / \mu$  and  $\lambda_2 = k_2 \beta_2 / \mu$  and denote by  $\lambda_1^*$  the transcritical threshold and by  $\hat{\lambda}_2$  the minimum group infectivity required for a backward bifurcation (bistability).

We linearize around the disease-free equilibrium  $\mathbf{x}^* = (0, 0, 1, 0, 0)$  and obtain the epidemic threshold  $\lambda_1^*$  analytically as the root of a quadratic equation from the linear stability condition (see the Appendix). Its dependence on model parameters is not transparent in closed form; to extract the leading structural dependence, we perform an asymptotic expansion for small  $\alpha$ , obtaining

$$\lambda_1^* \approx \frac{k_1}{k_1 - 1} - \alpha_{1,2} \lambda_2 \frac{k_1^2}{(k_1 - 1)^3}. \quad (2)$$

This expression shows that nestedness anticipates the epidemic onset through the combined control  $\alpha_{1,2} \lambda_2$ , while recovering the standard SIS threshold on networks,  $\lambda_1^{*,(0)} = k_1 / (k_1 - 1)$ , in the non-nested limit  $\alpha_{1,2} = 0$  [25]. This behavior is consistent with previous results on higher-order contagion [12,15]. To determine the *type* of transition, we perform a center-manifold reduction [27,28].

Near the epidemic threshold, the dynamics reduces to the normal form,

$$\dot{u} = hu^2 + z\phi u + \mathcal{O}(u^3, \phi u^2), \quad (3)$$

where  $\phi = \lambda_1 - \lambda_1^*$  measures the distance from criticality. The coefficients  $h$  and  $z$  are given by standard projections of the nonlinear vector field onto the critical eigenspace of the Jacobian (see Appendix B), and quantify, respectively, the leading nonlinear self-interaction and the linear unfolding of the instability. We find that  $z > 0$  at criticality; therefore, the direction of the bifurcation is entirely controlled by the sign of  $h$ :  $h < 0$  yields a supercritical (continuous) transition, while  $h > 0$  implies a subcritical (backward) bifurcation with bistability and explosive onset. Although the nonlinear coefficient  $h$  can be obtained analytically as a rational function implicitly depending on the critical point  $\lambda_1^*$  and the model parameters, its dependence on the key control parameters is not immediately obvious. Consequently, the bistability threshold  $\hat{\lambda}_2$  is determined numerically. Nevertheless, inspecting the full expansion of the nonlinear coefficient shows that  $h$  depends nonlinearly on both the overlap  $\alpha_{1,2}$  and the critical pairwise infectivity  $\lambda_1^*$ , with contributions up to fourth order in  $\alpha_{1,2}$ . These terms encode competing reinforcing and suppressing effects arising from pairwise and group interactions.

Figure 2(a) shows that  $h$  decreases monotonically with  $\alpha_{1,2}$  for  $k_1 = 5$ ,  $k_2 = 2$  and  $\lambda_2 \in \{1, 2, 3, 4\}$ , identifying a critical overlap  $\alpha_{1,2}^{(c)}$  where  $h = 0$  (dashed): increasing nestedness weakens the nonlinear amplification needed to sustain bistability and eventually turns a backward bifurcation into a continuous onset. For  $\lambda_2 = 4$ ,  $h$  remains positive over  $\alpha_{1,2} \in [0, 1]$ , indicating that bistability survives even at strong overlap, although it is progressively weakened. Together with Eq. (2), this already reveals the dual role of overlap: it lowers  $\lambda_1^*$  while pushing the system away from the subcritical regime.

For  $\alpha_{1,2} = 0$ , the condition for bistability simplifies to

$$\hat{\lambda}_2^{(0)} = \frac{(k_1 - 1)^2}{k_1^2}. \quad (4)$$

Thus, when dyadic and triadic interactions are uncorrelated, the onset of the backward bifurcation is controlled solely by the pairwise connectivity  $k_1$ . Notably, sparse pairwise layers favor explosive behavior at smaller  $\lambda_2$ .

Figure 2(b) reports  $\hat{\lambda}_2(\alpha_{1,2})$  (from  $h = 0$ ) together with the corresponding  $\lambda_1^*(\alpha_{1,2})$  (from the Jacobian), showing that  $\hat{\lambda}_2$  increases while  $\lambda_1^*$  decreases with overlap. Figure 2(c) summarizes the stationary infected density  $\rho^* \equiv \rho^I$  in the  $(\lambda_1, \alpha_{1,2})$  space, at  $\lambda_2 = 3$ : the bistable region shrinks with  $\alpha_{1,2}$  and vanishes at  $\alpha_{1,2}^{(c)}$ . To validate these predictions,

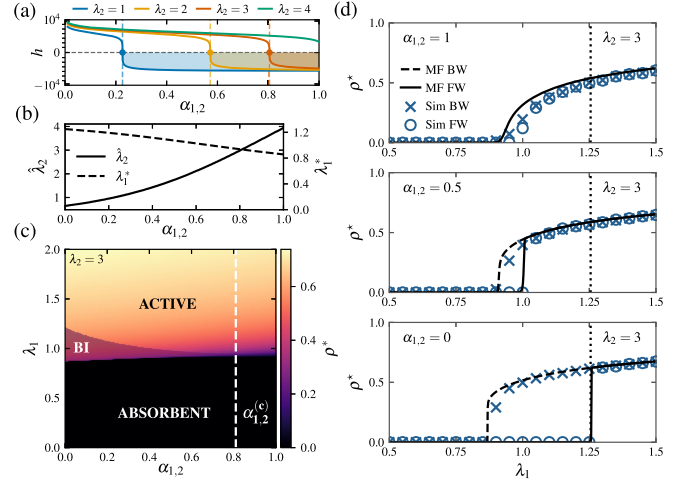


FIG. 2. Nestedness shapes critical transitions. (a) Nonlinear coefficient  $h$  from center-manifold theory as a function of  $\alpha_{1,2}$ , for  $\lambda_2 \in \{1, 2, 3, 4\}$  (with  $k_1 = 5$ ,  $k_2 = 2$ ). Vertical dashed lines mark the critical overlap  $\alpha_{1,2}^{(c)}$  at which  $h = 0$  and the transition changes from subcritical (bistable) to supercritical (continuous). (b) Critical three-body infectivity  $\hat{\lambda}_2$  (solid) required for bistability and the corresponding epidemic threshold  $\lambda_1^*$  (dashed), both obtained numerically as functions of  $\alpha_{1,2}$  for  $k_1 = 5$ ,  $k_2 = 2$ . (c) Phase diagram in the  $(\alpha_{1,2}, \lambda_1)$  plane predicted by the model for  $k_1 = 5$ ,  $k_2 = 2$ , and  $\lambda_2 = 3$ , showing that increasing  $\alpha_{1,2}$  lowers  $\lambda_1^*$  while shrinking the bistable region, which disappears for  $\alpha_{1,2} > \alpha_{1,2}^{(c)}$ , yielding continuous transitions. (d) Stationary infected density  $\rho^*$  from theory (lines) and Gillespie simulations (markers) on random regular hypergraphs with  $N = 3000$ ,  $k_1 = 5$ , and  $k_2 = 2$ , for three representative values of  $\alpha_{1,2}$  at  $\lambda_2 = 3$ . For  $\alpha_{1,2} = 1$ , forward (FW) and backward (BW) branches coincide (continuous transition); for  $\alpha_{1,2} = 0.5$ , bistability emerges; for  $\alpha_{1,2} = 0$ , bistability is maximal and the forward threshold occurs at  $\lambda_1^{*,(0)}$  (vertical dotted lines).

we perform Gillespie simulations on random regular hypergraphs with tunable  $\alpha_{1,2}$ . Details of the rewiring procedure are provided in Supplemental Material (SM) [29]. Figure 2(d) shows excellent agreement between theory and simulations. Small deviations at large  $\alpha_{1,2}$  are consistent with nestedness-induced pairwise clustering in sparse hypergraphs, which is neglected by our theory [30,31].

*Microscopic mechanisms underlying the anticipated onset and suppressed bistability*—Having established the macroscopic effects of nestedness, we now exploit the analytical tractability of our SIS framework to uncover their microscopic origin. Specifically, we use early-time dynamical correlations to characterize how nestedness reshapes contagion pathways already near the disease-free state. From Eq. (1), the infected population grows whenever

$$\left( \lambda_1 + \lambda_2 \frac{\rho^{\text{ISI}_\Delta}}{\rho^{\text{SI}}} \right) \frac{\rho^{\text{SI}}}{\rho^I} > 1. \quad (5)$$

The ratios  $\Pi = \rho^{\text{SI}}/\rho^{\text{I}}$  and  $\delta = \rho^{\text{ISI}_\Delta}/\rho^{\text{SI}}$  act as *fast variables* [32,33]: they relax much faster than  $\rho^{\text{I}}$  and rapidly reach quasistationary values, denoted by  $\bar{\Pi}$  and  $\bar{\delta}$  (see SM [29]).

Near the disease-free state, the epidemic threshold is entirely determined by these early-time correlations,

$$\lambda_1^* = \frac{1}{\bar{\Pi}} - \lambda_2 \bar{\delta}. \quad (6)$$

The quasistationary fast variables can be obtained analytically as implicit functions of  $\lambda_1^*$  (see SM [29]). In particular,  $\bar{\delta} = 0$  when  $\alpha_{1,2} = 0$ , showing that higher-order contagion does not contribute to the epidemic onset in the absence of nestedness. Substituting the analytical fast-variable solutions into Eq. (6) exactly recovers the epidemic threshold obtained from the linear stability analysis of the full system, revealing that the onset of contagion is completely encoded in the early-time dynamical correlations generated by nestedness.

To further uncover the microscopic origin of this effect, we decompose the infected density into pairwise- and group-mediated contributions,

$$\begin{aligned} \dot{\rho}_1^{\text{I}} &= -\mu \rho_1^{\text{I}} + \beta_1 k_1 \rho^{\text{SI}}, \\ \dot{\rho}_2^{\text{I}} &= -\mu \rho_2^{\text{I}} + \beta_2 k_2 \rho^{\text{ISI}_\Delta}, \end{aligned} \quad (7)$$

where  $\rho_1^{\text{I}}$  and  $\rho_2^{\text{I}}$  denote the pairwise- and three-body-mediated contributions to the infected population, respectively. The same decomposition can be applied to all motif variables. Applying the chain rule to the corresponding ratios then yields a closed early-time description in terms of disentangled fast variables, which naturally split into pairwise and higher-order contributions,  $\Pi = \Pi_1 + \Pi_2$  and  $\delta = \delta_1 + \delta_2$ , respectively.

For group-state variables, the pairwise contribution can be further decomposed into external and group-embedded transmission events. For instance,  $\rho_1^{\text{ISI}_\Delta} = \rho_{1,\text{ext}}^{\text{ISI}_\Delta} + \rho_{1,\text{int}}^{\text{ISI}_\Delta}$ , which leads to the corresponding evolution equations. Consequently, the pairwise fast variable can also be decomposed as  $\delta_1 = \delta_{1,\text{ext}} + \delta_{1,\text{int}}$ , allowing us to quantify separately the contributions of external and embedded transmission routes at early times (see Appendix D).

Figure 3(a) compares the theoretical predictions obtained from this fast-variable framework with Gillespie simulations. Fast variables are measured by initializing the system from a single infected node near  $\lambda_1^*$  and averaging over 5000 realizations within the early-time window  $0.005 < \rho^{\text{I}}(t) < 0.01$ . Theory and simulations show excellent agreement, particularly for the higher-order contribution  $\bar{\Pi}_2$  and the internal pairwise contribution  $\bar{\delta}_{1,\text{int}}$ . Most importantly, the disentangled dynamics reveals that the only nonzero contribution to  $\bar{\delta}$  originates from *internal* pairwise transmission within higher-order groups and

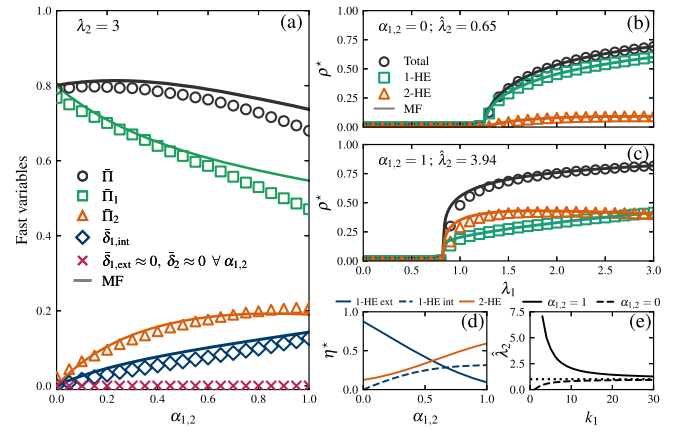


FIG. 3. Microscopic mechanisms underlying the anticipated onset and suppressed bistability. (a) Quasistationary states for the fast variables  $\Pi$  and  $\delta$  as functions of  $\alpha_{1,2}$ , obtained from theory (lines) and Gillespie simulations (symbols) on random regular hypergraphs with  $N = 3000$ ,  $k_1 = 5$ ,  $k_2 = 2$ , and  $\lambda_2 = 3$ . Each point is evaluated close to  $\lambda_1^*$  for each  $\alpha_{1,2}$ . The contribution to  $\bar{\delta}$  originates entirely from the internal pairwise component  $\delta_{1,\text{int}}$  and increases with  $\alpha_{1,2}$ . (b),(c) Decomposition of the stationary infected density  $\rho^*$  into total, pairwise (1-hyperedge), and group-based (2-hyperedge) contributions for  $\alpha_{1,2} = 0$  and  $\alpha_{1,2} = 1$  on hypergraphs with the same  $N$ ,  $k_1$ , and  $k_2$ , evaluated at the corresponding critical values  $\hat{\lambda}_2$ , before bistability emerges. In both cases the transition remains continuous, although nested structures display a sharper onset. (d) Fractions of accumulated infection events  $\eta_1^*$  (pairwise, internal and external) and  $\eta_2^*$  (higher-order) near  $\lambda_1^*$  as functions of  $\alpha_{1,2}$ , showing a progressive shift from external pairwise to internal and higher-order transmission channels. (e) Critical group infectivity  $\hat{\lambda}_2$  versus  $k_1$  (with  $k_2 = 2$ ) for  $\alpha_{1,2} = 0$  (dashed, analytical) and  $\alpha_{1,2} = 1$  (solid, numerical), illustrating the strong suppression of explosive behavior induced by nestedness in sparse networks; both curves converge to the mean-field limit  $\hat{\lambda}_2 = 1$  (dotted line) for large  $k_1$ .

increases monotonically with nestedness. Combined with Eq. (6), this demonstrates that nestedness anticipates the epidemic threshold by promoting mixed group configurations already at the earliest stages of the dynamics, thereby increasing the ratio  $\rho^{\text{ISI}_\Delta}/\rho^{\text{SI}}$ .

We now connect this early-time picture to the stationary regime by decomposing  $\rho^*$  into total, pairwise, and group-based contributions. Figures 3(b) and 3(c) show this decomposition for  $\alpha_{1,2} = 0$  and  $\alpha_{1,2} = 1$  at their respective  $\hat{\lambda}_2$  values. While pairwise transmission dominates for  $\alpha_{1,2} = 0$ , increasing nestedness enhances internal pairwise transmission, activating more higher-order interactions and shifting the dominant transmission channel without inducing bistability. To quantify this route redistribution, we define  $\eta_1^*$  and  $\eta_2^*$  as the fractions of accumulated pairwise and higher-order infection events up to stationarity, with  $\eta_1^* = \eta_{1,\text{ext}}^* + \eta_{1,\text{int}}^*$ . Figure 3(d) completes the microscopic picture by showing these quantities, averaged over 1000 Gillespie realizations on random regular

hypergraphs with  $N = 3000$ ,  $k_1 = 5$ , and  $k_2 = 2$ , evaluated at  $\lambda_1 = \lambda_1^* + 0.05$  and the corresponding bistability threshold  $\hat{\lambda}_2$ . As nestedness increases, infection events are progressively redirected from external pairwise links to internal pairwise and higher-order transmission. This redistribution enhances higher-order activity but at the same time concentrates pairwise and higher-order contagion within the same groups, making the two transmission channels increasingly redundant. Consistently, center-manifold theory predicts an increasing  $\hat{\lambda}_2(\alpha_{1,2})$ , showing that progressively stronger higher-order transmission is required to compensate for this loss of independent nonlinear reinforcement and sustain bistability.

Figure 3(e) illustrates how nestedness fundamentally changes the role of pairwise connectivity. For  $\alpha_{1,2} = 0$ , the analytical predictions of Eqs. (2) and (4) explain the dashed curve: low  $k_1$  delays the epidemic onset while lowering the critical value  $\hat{\lambda}_2$  required for bistability, consistently with the general picture of discontinuous transitions driven by nonlinear feedback [34]. Nestedness reverses this dependence. Equation (2) predicts that its correction to  $\lambda_1^*$  is strongest for small  $k_1$ . Together with the fast-variable result that group-embedded pairwise routes are the only contribution anticipating activation, this explains why the solid curve is shifted upward at low  $k_1$ : sparse systems contain fewer external routes, making nestedness most effective at anticipating activation while simultaneously suppressing the independent nonlinear feedback. As  $k_1$  increases, external routes are progressively restored and  $\hat{\lambda}_2$  decreases, with both curves approaching  $\hat{\lambda}_2 \rightarrow 1$  for large  $k_1$  [3]. Together, these results show that the onset and the nature of the transition are complementary signatures of the same microscopic route redistribution.

*A general mechanism across interaction orders and dynamical processes*—Having established the mechanism for contagion with pairwise and three-body groups, we now investigate its generality across interaction orders and dynamical processes. In direct analogy with Fig. 2(b), for each system we compute the corresponding stationary order parameter and use its forward and backward branches to estimate the lower-order coupling marking collective onset and the higher-order coupling at which a robust hysteresis loop first emerges, according to a common operational criterion (see SM [29]). We first consider SIS dynamics on random regular hypergraphs with pairwise and four-body interactions ( $N = 2000$ ,  $k_1 = 7$ ,  $k_3 = 2$ ), with tunable nestedness  $\alpha_{1,3}$  (see SM [29]). Susceptible nodes are infected through pairwise interactions at rate  $\beta_1$  or through four-body interactions at rate  $\beta_3$  when the other three nodes are infected, with  $\lambda_3 = k_3\beta_3/\mu$ . Figure 4(a) shows that increasing  $\alpha_{1,3}$  lowers the epidemic threshold  $\lambda_1^*$  while increasing the critical four-body infectivity  $\hat{\lambda}_3$  required for a backward bifurcation, demonstrating that the dual effect persists beyond three-body contagion.

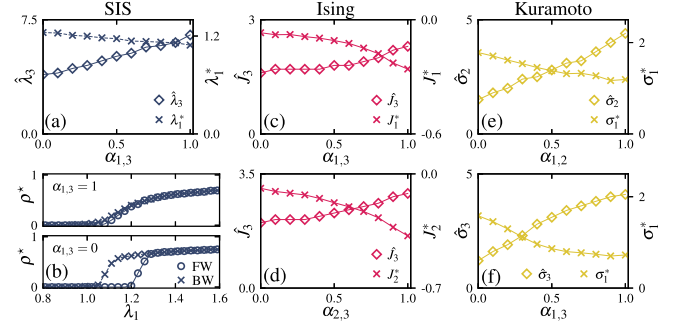


FIG. 4. Generality across interaction orders and dynamical processes. Critical onset thresholds and higher-order parameters marking the emergence of robust hysteresis as functions of nestedness, extracted from the forward and backward branches of the corresponding stationary order parameters. Results are shown for (a) SIS dynamics with pairwise and four-body interactions; (b) representative forward (FW) and backward (BW) SIS branches for  $\alpha_{1,3} = 0$  and  $\alpha_{1,3} = 1$  at  $\lambda_3 = 6$ ; (c) Ising dynamics with pairwise and four-body interactions; (d) Ising dynamics with three- and four-body interactions only; and Kuramoto dynamics with (e) pairwise-three-body and (f) pairwise-four-body interactions. In all cases, nestedness shifts the onset of collective behavior to lower values of the corresponding control parameter while increasing the critical higher-order parameter required for discontinuous behavior.

We next consider the higher-order Ising model of Ref. [7], where pairwise, three-body, and four-body interactions favor aligned spin configurations through couplings  $J_1$ ,  $J_2$ , and  $J_3$ . The system undergoes a transition from a paramagnetic (disordered) to a ferromagnetic (ordered) phase, quantified by the stationary absolute magnetization  $|\langle \mathcal{M} \rangle|$  (see Appendix E). Since discontinuous transitions arise only for  $m > 2$ , this model also allows us to probe nestedness entirely within the higher-order sector. We therefore consider (i) pairwise and four-body interactions, estimating  $J_1^*$  and  $\hat{J}_3$ , and (ii) three- and four-body interactions, estimating  $J_2^*$  and  $\hat{J}_3$ . For hypergraphs with pairwise and four-body interactions ( $N = 2000$ ,  $k_1 = 7$ ,  $k_3 = 2$ ), Fig. 4(c) shows that increasing  $\alpha_{1,3}$  lowers  $J_1^*$  while increasing the critical four-body coupling  $\hat{J}_3$  required for hysteresis. Furthermore, for hypergraphs with three- and four-body interactions only, with an empty set of dyadic interactions ( $N = 2000$ ,  $k_2 = 7$ ,  $k_3 = 2$ ) and tunable nestedness  $\alpha_{2,3}$  (see SM [29]), increasing  $\alpha_{2,3}$  again lowers the transition point while increasing  $\hat{J}_3$  [Fig. 4(d)]. The same mechanism therefore persists even without pairwise interactions.

Finally, we consider the higher-order Kuramoto model of Ref. [5], where oscillators interact through order-dependent couplings  $\sigma_m$ . Synchronization is quantified by the stationary time-averaged Kuramoto order parameter  $\langle r \rangle$  (see Appendix F). We consider (i) pairwise and three-body interactions, estimating  $\sigma_1^*$  and  $\hat{\sigma}_2$ , and (ii) pairwise and four-body interactions, estimating  $\sigma_1^*$  and  $\hat{\sigma}_3$ . In both cases,

increasing nestedness lowers the synchronization threshold while shifting the onset of bistability toward larger higher-order couplings [Figs. 4(e) and 4(f)]. SM [29] provides an extensive analysis across interaction orders and dynamical processes and further shows that the Kuramoto results are robust to alternative higher-order coupling functions. Overall, these results show that nestedness promotes collective onset while suppressing discontinuous transitions across fundamentally different dynamical systems.

**Conclusions**—In this Letter, we showed that nestedness between interaction orders plays a dual role in collective dynamics: it promotes the onset of collective behavior while suppressing the nonlinear feedback responsible for abrupt transitions. Using higher-order contagion as an analytically tractable setting, we developed a microscopic theory revealing that nestedness redistributes dynamical routes from external neighborhoods to group-embedded ones, reducing the structural independence between interaction orders. This route redistribution lowers activation thresholds but progressively weakens the independent reinforcement required for bistability, thereby smoothing discontinuous transitions. Beyond contagion dynamics, we demonstrated that the same mechanism persists across multiple interaction orders and dynamical processes, including higher-order Ising and Kuramoto dynamics, and even in the absence of pairwise interactions. Taken together, our results identify nestedness as a general structural mechanism governing both the onset and the nature of collective transitions in higher-order systems.

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**Data availability**—The data that support the findings of this study are available from the corresponding author upon reasonable request.

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## End Matter

*Appendix A: Homogeneous mean-field model*—To analytically characterize the impact of nestedness on SIS dynamics, we develop a homogeneous mean-field description for regular hypergraphs with pairwise (1-hyperedges) and three-body (2-hyperedges) interactions. Susceptible nodes become infected through pairwise interactions at rate  $\beta_1$  or through a three-body interaction containing two infected neighbors at rate  $\beta_2$ , while infected nodes recover at rate  $\mu$ . Throughout this appendix, we denote the nestedness parameter by  $\alpha \equiv \alpha_{1,2}$ .

The model explicitly retains the cross-order dynamical correlations induced by nestedness by tracking the state vector  $\mathbf{x} = (\rho^I, \rho^{SI}, \rho^{SSS_\Delta}, \rho^{SSI_\Delta}, \rho^{ISI_\Delta})$ , where  $\rho^A = [A]/N$ ,  $\rho^{AB} = [AB]/(Nk_1)$ , and  $\rho^{ABC_\Delta} = [ABC_\Delta]/(2Nk_2)$  denote normalized node, link, and three-body motif densities, respectively. Homogeneity implies  $\rho^{SI} = \rho^{IS}$  and  $\rho^{ISI_\Delta} = \rho^{IIS_\Delta} = \rho^{II\Delta}$ . The evolution equations naturally generate higher-order composite motifs coupling links and three-body interactions. Their explicit definitions are reported in SM [29].

The resulting dynamical system reads as

$$\begin{aligned}
 \dot{\rho}^I &= -\mu\rho^I + \beta_1 k_1 \rho^{SI} + \beta_2 k_2 \rho^{ISI_\Delta}; \\
 \dot{\rho}^{SI} &= \beta_1 [(k_1 - 1)(\rho^{SSI} - \rho^{SI}) - \rho^{SI}] \\
 &\quad + \beta_2 \frac{k_2}{k_1} [(k_1 - 2\alpha)(\rho^{IIS_\Delta} - \rho^{IIS_\Delta I}) - 2\alpha\rho^{ISI_\Delta}] \\
 &\quad + \mu(\rho^{II} - \rho^{SI}); \\
 \dot{\rho}^{SSS_\Delta} &= -3\beta_1 (k_1 - 2\alpha)\rho^{SSS_\Delta I} - 3\beta_2 (k_2 - 1)\rho^{SSS_{II\Delta}} \\
 &\quad + 3\mu\rho^{SSI_\Delta}; \\
 \dot{\rho}^{SSI_\Delta} &= \beta_1 [(k_1 - 2\alpha)(\rho^{SSS_\Delta I} - 2\rho^{ISS_\Delta I}) - 2\alpha\rho^{SSI_\Delta}] \\
 &\quad + \beta_2 (k_2 - 1)(\rho^{SSS_{II\Delta}} - 2\rho^{ISS_{II\Delta}}) + \mu(2\rho^{ISI_\Delta} - \rho^{SSI_\Delta}); \\
 \dot{\rho}^{ISI_\Delta} &= \beta_1 [(k_1 - 2\alpha)(2\rho^{ISS_\Delta I} - \rho^{IIS_\Delta I})] \\
 &\quad + 2\alpha\beta_1 (\rho^{SSI_\Delta} - \rho^{ISI_\Delta}) \\
 &\quad + \beta_2 [(k_2 - 1)(2\rho^{ISS_{II\Delta}} - \rho^{IIS_{II\Delta}}) - \rho^{ISI_\Delta}] \\
 &\quad + \mu(\rho^{III_\Delta} - 2\rho^{ISI_\Delta}). \tag{A1}
 \end{aligned}$$

The remaining motif densities are obtained from the conservation identities  $\rho^S = 1 - \rho^I$ ,  $\rho^{II} = \rho^I - \rho^{SI}$ ,  $\rho^{SS} = \rho^S - \rho^{SI}$ , and  $\rho^{III\Delta} = 1 - \rho^{SS\Delta} - 3\rho^{SSI\Delta} - 3\rho^{ISI\Delta}$ .

Nestedness enters the model solely through the availability of external pairwise transmission routes. For a node already belonging to a three-body interaction, increasing nestedness progressively replaces external links by group-embedded ones, yielding an effective number of external links  $k_{1,\text{ext}} = (1 - \alpha_{1,2})k_1 + \alpha_{1,2}(k_1 - 2) = k_1 - 2\alpha_{1,2}$ , which appears whenever transmission requires an external infected neighbor while the focal susceptible node is already conditioned on belonging to a three-body interaction.

The hierarchy is closed through standard homogeneous approximations [25,26], neglecting pairwise clustering and intra-order overlap among distinct three-body interactions [17]. Consequently, higher-order motifs are expressed in terms of lower-order densities while explicitly retaining the cross-order correlations generated by nestedness. The complete set of closure relations and their derivation are reported in SM [29].

To determine the onset of spreading, we introduce the rescaled infectivities  $\lambda_1 = k_1\beta_1/\mu$  and  $\lambda_2 = k_2\beta_2/\mu$  and linearize Eq. (A1) around the disease-free equilibrium  $\mathbf{x}^* = (0, 0, 1, 0, 0)$ . The epidemic threshold (transcritical bifurcation) is obtained by imposing that the dominant eigenvalue of the Jacobian crosses zero. For compactness, we define  $\Gamma(\lambda_1) \equiv k_1 - (k_1 - 1)\lambda_1$  and  $\Theta(\alpha, \lambda_1) \equiv k_1k_2 + (k_1 + k_2 - 1)\lambda_1 - k_1 - 4\alpha k_2\lambda_1$ , which yields the exact condition

$$\alpha\lambda_1\lambda_2 \left[ 4\alpha^2 k_2\lambda_1 + k_1\Theta(\alpha, \lambda_1) \right] = k_1^2 k_2 \Gamma(\lambda_1). \quad (\text{A2})$$

*Appendix B: Center-manifold reduction*—To characterize the *type* of transition at the epidemic threshold, we perform a center-manifold reduction of the closed system  $\dot{\mathbf{x}} = \mathbf{F}(\mathbf{x}; \lambda_1, \lambda_2, \alpha)$  about the disease-free equilibrium  $\mathbf{x}^* = (0, 0, 1, 0, 0)$ . The critical point is defined implicitly by Eq. (A2); throughout, all quantities below are evaluated on this critical manifold. Let  $J = D_{\mathbf{x}}\mathbf{F}(\mathbf{x}^*; \lambda_1, \lambda_2, \alpha)|_{\text{crit}}$  be the Jacobian at criticality, and let  $\mathbf{w}$  and  $\mathbf{v}$  denote the right and left eigenvectors associated with the simple zero eigenvalue, normalized by  $\mathbf{v}^T \mathbf{w} = 1$ . Introducing the unfolding parameter  $\phi = \lambda_1 - \lambda_1^*$  [with  $\lambda_1^*$  defined implicitly by Eq. (A2)], the dynamics on the center manifold reduces to the scalar normal form  $\dot{u} = hu^2 + \phi u + \mathcal{O}(u^3, \phi u^2)$ .

In terms of the vector field  $\mathbf{F}$ , the quadratic coefficient is

$$h = \sum_{i,j,k=1}^b v_i \frac{\partial^2 F_i}{\partial x_j \partial x_k}(\mathbf{x}^*; \lambda_1, \lambda_2, \alpha) \Big|_{\phi=0} w_j w_k, \quad (\text{B1})$$

and the parameter-dependent coefficient is

$$z = \sum_{i,j=1}^b v_i \frac{\partial^2 F_i}{\partial x_j \partial \lambda_1}(\mathbf{x}^*; \lambda_1, \lambda_2, \alpha) \Big|_{\phi=0} w_j, \quad (\text{B2})$$

with  $b = \dim(\mathbf{x}) = 5$ .

*Appendix C: Fast-variable system*—In the early stage ( $\rho^I \rightarrow 0$ ), ratios of motif densities relax on a fast time scale. We define the fast variables  $\Pi \equiv \rho^{SI}/\rho^I$  and  $\Psi \equiv \rho^{ISI\Delta}/\rho^I$ , so  $\delta \equiv \rho^{ISI\Delta}/\rho^{SI} = \Psi/\Pi$ . To close the fast-variable dynamics we introduce two additional ratios,  $\Omega \equiv \rho^{SS\Delta}/\rho^I$  and  $\Upsilon \equiv \rho^{SSI\Delta}/\rho^I$ . The remaining pair fast variable is eliminated using the identity  $\rho^{SI} + \rho^{II} = \rho^I$ , i.e.,  $\rho^{II}/\rho^I = 1 - \Pi$ . Fast-variable equations follow by differentiating ratios via the chain rule; e.g.,  $\dot{\Pi} = (\dot{\rho}^{SI}/\rho^I) - \Pi(\dot{\rho}^I/\rho^I)$  and similarly for  $\Upsilon, \Psi, \Omega$ .

Substituting the motif equations and closures into the chain-rule expressions yields

$$\begin{aligned} \dot{\Pi} &= \mu(1 - \Pi) + \beta_1(k_1 - 2)\Pi + \beta_2 \frac{k_2}{k_1}(k_1 - 4\alpha)\Psi \\ &\quad - \beta_1 k_1 \Pi^2 - \beta_2 k_2 \Pi\Psi, \\ \dot{\Upsilon} &= \beta_1(k_1 - 2\alpha)\Pi - 2\beta_1\alpha\Upsilon + [2\mu + \beta_2(k_2 - 1)]\Psi \\ &\quad - \beta_1 k_1 \Pi\Upsilon - \beta_2 k_2 \Upsilon\Psi, \\ \dot{\Omega} &= \mu(2\Upsilon + \Omega) - 3\beta_1(k_1 - 2\alpha)\Pi - 3\beta_2(k_2 - 1)\Psi \\ &\quad - \beta_1 k_1 \Pi\Omega - \beta_2 k_2 \Omega\Psi, \\ \dot{\Psi} &= 2\beta_1\alpha\Upsilon + (-2\beta_1\alpha - \mu - \beta_2)\Psi + \mu(1 - \Omega \\ &\quad - 3\Upsilon - 3\Psi) - \beta_1 k_1 \Pi\Psi - \beta_2 k_2 \Psi^2. \end{aligned} \quad (\text{C1})$$

This system directly provides the early-time evolution of  $\Psi = \rho^{ISI\Delta}/\rho^I$  and hence of  $\delta = \Psi/\Pi = \rho^{ISI\Delta}/\rho^{SI}$ .

*Appendix D: Disentangling by interaction order and by internal and external pairwise channels*—We decompose the evolution equations for the state densities in Eq. (A1) by explicitly disentangling the microscopic processes contributing to each transition. In particular, we distinguish between (i) infections generated by pairwise interactions (subscript 1) and (ii) infections mediated by three-body interactions (subscript 2). For group-state densities, such as  $\rho^{ISI\Delta}$ , the pairwise contribution can be further separated into transmission along interactions embedded within three-body groups and transmission along external pairwise interactions (subscripts 1, int and 1, ext, respectively), yielding an expanded system of differential equations. For example, for the state variable  $\rho^{ISI\Delta}$ , we obtain

$$\begin{aligned}
 \dot{\rho}_{1,\text{ext}}^{\text{ISI}\Delta} &= +\mu(\rho_{1,\text{ext}}^{\text{III}\Delta} - 2\rho_{1,\text{ext}}^{\text{ISI}\Delta}) \\
 &\quad + \beta_1(k_1 - 2\alpha)(2\rho^{\text{ISS}\Delta\text{I}} - \rho^{\text{IS}\Delta\text{I}}); \\
 \dot{\rho}_{1,\text{int}}^{\text{ISI}\Delta} &= +\mu(\rho_{1,\text{int}}^{\text{III}\Delta} - 2\rho_{1,\text{int}}^{\text{ISI}\Delta}) + 2\alpha\beta_1(\rho^{\text{SSI}\Delta} - 2\rho^{\text{ISI}\Delta}); \\
 \dot{\rho}_2^{\text{ISI}\Delta} &= +\mu(\rho_2^{\text{III}\Delta} - 2\rho_2^{\text{ISI}\Delta}) \\
 &\quad + \beta_2[(k_2 - 1)(\rho^{\text{ISSII}\Delta} - \rho^{\text{ISII}\Delta}) - \rho^{\text{ISI}\Delta}]. \quad (\text{D1})
 \end{aligned}$$

The full disentangled system of equations can be found in SM [29]. Furthermore, the fast variables can be consistently decomposed into the sum of their higher-order, internal pairwise, and external pairwise contributions as

$$\begin{aligned}
 \Upsilon &= \Upsilon_2 + \Upsilon_{1,\text{int}} + \Upsilon_{1,\text{ext}}, & \Psi &= \Psi_2 + \Psi_{1,\text{int}} + \Psi_{1,\text{ext}}, \\
 \Omega &= \Omega_2 + \Omega_{1,\text{int}} + \Omega_{1,\text{ext}}, \quad (\text{D2})
 \end{aligned}$$

respectively. Their corresponding differential equations are also displayed in SM [29]. This disentangled system provides a closed early-time description of the fast variables, capturing the contributions by interaction order and by internal-external link channel within groups.

*Appendix E: Higher-order Ising model*—To demonstrate the generality of our results beyond spreading dynamics, we consider the higher-order Ising model introduced in Refs. [7,23]. Each node carries a binary spin  $s_i \in \{-1, +1\}$ , and an energetic contribution is assigned whenever all spins belonging to the same hyperedge are aligned. The Hamiltonian reads as

$$H = - \sum_{\ell=1}^{\ell_{\max}} J_{\ell} \sum_{\{\sigma: |\sigma|=\ell+1\}} \delta(\{s_i\}_{i \in \sigma}), \quad (\text{E1})$$

where  $J_{\ell}$  is the coupling associated with  $(\ell + 1)$ -body interactions and

$$\delta(s_1, \dots, s_n) = \begin{cases} 1 & \text{if } s_1 = s_2 = \dots = s_n, \\ 0 & \text{otherwise} \end{cases} \quad (\text{E2})$$

is the generalized Kronecker delta. We characterize the state of the system via the absolute value of the stationary magnetization,

$$|\langle \mathcal{M} \rangle|, \quad \mathcal{M} = \frac{1}{N} \sum_{i=1}^N s_i, \quad (\text{E3})$$

where  $\langle \cdot \rangle$  denotes the temporal average over Monte Carlo samples collected in the stationary regime. Details on the stochastic simulations are provided in SM [29].

*Appendix F: Kuramoto dynamics*—We consider Kuramoto oscillator dynamics with group interactions up to order  $M = 3$  [5], where  $\theta_i$  is the phase of oscillator  $i$ ,  $\omega_i$  is its natural frequency,  $\sigma_m$  is the coupling strength of interactions of order  $m$ , and  $k_m$  is the corresponding regular degree. The adjacency tensors  $a_{ij}^{(1)}$ ,  $a_{ijl}^{(2)}$ , and  $a_{ijln}^{(3)}$  encode pairwise, three-body, and four-body interactions, respectively. The dynamics is given by

$$\begin{aligned}
 \dot{\theta}_i &= \omega_i + \frac{\sigma_1}{k_1} \sum_{j=1}^N a_{ij}^{(1)} \sin(\theta_j - \theta_i) \\
 &\quad + \frac{\sigma_2}{k_2} \sum_{j=1}^N \sum_{l=1}^N a_{ijl}^{(2)} \sin(2\theta_j - \theta_l - \theta_i) \\
 &\quad + \frac{\sigma_3}{k_3} \sum_{j=1}^N \sum_{l=1}^N \sum_{n=1}^N a_{ijln}^{(3)} \sin(\theta_j + \theta_l - \theta_n - \theta_i). \quad (\text{F1})
 \end{aligned}$$

Synchronization is quantified by the Kuramoto order parameter  $r(t) = |N^{-1} \sum_{j=1}^N e^{i\theta_j(t)}|$ , ranging from 0 (incoherence) to 1 (phase synchronization). In the main text, we vary the nestedness between interaction orders and identify the critical pairwise coupling  $\sigma_1^*$  for the onset of synchronization and the critical higher-order coupling  $\hat{\sigma}_m$  ( $m = 2, 3$ ) required for explosive synchronization from the stationary time average  $\langle r \rangle$  of  $r(t)$  (SM [29]).