

Adaptive behaviors neutralize bistable explosive transitions in higher-order contagion

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During contagion phenomena, individuals perceiving a risk of infection commonly adapt their behavior and reduce their exposure. The effects of such adaptive mechanisms have been studied for processes in which pairwise interactions drive contagion. However, contagion and the perception of infection risk can also involve (“higher-order”) group interactions, leading potentially to new phenomenology. How adaptive behavior resulting from risk perception affects higher-order processes remains an open question. Here, we consider the impact of several risk-based adaptive behaviors on pairwise and higher-order contagion processes, using numerical simulations and an analytical mean-field approach. For pairwise contagion, adaptive mechanisms based on local (pairwise or group-based) risk perception impact only the endemic state, without affecting the epidemic phase transition. For higher-order contagion processes, instead, the adaptivity defuses the impact of nonlinear group interactions: This reduces or even completely suppresses the parameter range in which bistability is possible, effectively transforming a higher-order contagion process into a pairwise one.

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Many complex phenomena, such as the spread of epidemics, information, and opinions, can be represented by contagion processes on networks [1–4]. The description of the underlying infection mechanisms can involve either pairwise interactions (as in the modeling of epidemic spread, in which each transmission event corresponds to a single exposure to an infectious individual [1,2]) or (higher-order) group interactions. The latter description is often used for social contagion processes, where nonlinear reinforcement effects can occur in groups [3–6]. Recent research has shown how these higher-order interactions give rise to a rich phenomenology, including discontinuous transitions, multistability regimes, critical mass effects [4,5,7–18], and contagion paths differing from the ones due to pairwise interactions [19–21].

While the structure of interactions strongly impacts the unfolding of a contagion dynamics at both microscopic and macroscopic levels, the contagion process can also itself reshape the interactions, if individuals are aware of the contagion risk and adapt their behavior accordingly [22–30]. For instance, if a disease is spreading in a population, individuals can become aware of

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the spread through information available either globally at the population level [23,24,27] or locally as other individuals become infectious in their neighborhood [31–34]. Both healthy and infectious individuals may then modify their behavior to avoid being contaminated or infecting others [35–38]. These adaptive behaviors can consist either in the removal or rewiring of interactions or in the implementation of protective measures (masks, handwashing, etc.) that decrease the probability of infection [23,24,27].

In the context of pairwise contagion processes, many models have been proposed and studied to explore the effect of such adaptative mechanisms based on awareness, obtained also through pairwise interactions [31–34,38–44]. However, the impact of behavior change on higher-order contagion processes, including adaptive mechanisms triggered by group-driven information, remains largely unexplored [45–49].

Here and in a companion paper [47], we contribute to filling this gap by considering both pairwise and nonlinear higher-order contagion processes in the presence of adaptive strategies based on local information, driven either by pairwise or by group information. We focus in this Letter on the impact of these strategies on the epidemic phase transition. To this aim, we consider two specific mechanisms, where the adaptivity is driven by either the number of infectious neighbors of an individual (pairwise-based mechanism) or the number of infectious groups to which an individual belongs (group-based mechanism). Through numerical simulations and a mean-field analytical approach, we show that in pairwise contagion processes, these adaptive mechanisms impact the endemic state but do not influence the nature or the location of the epidemic phase transition, which remains continuous. For higher-order contagion processes, however, the adaptive interaction change has a strong impact on the epidemic transition itself: We show that it defuses the nonlinearity and explosiveness of group contagion events, which are responsible for the emergence of a discontinuous transition and of a bistable regime in the nonadaptive (NAD) case [9,14]. As a result, adaptive behaviors shrink the bistability region and decrease the discontinuity of the phase transition, possibly even completely suppressing it, reverting the transition to a continuous one as in pairwise contagion. In Ref. [47], we extend the results to a wide range of pairwise- and group-based adaptive mechanisms, providing a systematic investigation of their relative effectivity and cost.

Contagion models. We consider a hypergraph $\mathcal{H} = (\mathcal{V}, \mathcal{E})$, where $\mathcal{V}$ denotes the set of its nodes, $\mathcal{E}$ is the set of its hyperedges, and its weighted projected graph is $\mathcal{G} = (\mathcal{V}, \mathcal{L})$, obtained by projecting each hyperedge onto the corresponding clique (fully connected set of nodes) and assigning to each link $(i, j) \in \mathcal{L}$ a weight $w_{i,j}$ equal to the number of different hyperedges in $\mathcal{E}$ containing both i and j [see Fig. 1(a) and Appendix]. Note that, with this definition, the weights in $\mathcal{G}$ encode some of the higher-order information contained in $\mathcal{H}$, namely, regarding the overlap of hyperedges. $\mathcal{H}$ and $\mathcal{G}$ constitute the substrate for the spread of a susceptible-infectious-susceptible (SIS) model for epidemic spread in discrete time. Awareness of the spread (which drives adaptive mechanisms) is also sustained by $\mathcal{H}$ and $\mathcal{G}$. In the SIS model, each individual is at each time step in one of two states: Susceptible (S) individuals can become infectious (I) through contagion events, i.e., interactions with infectious individual(s), and infectious individuals recover spontaneously, with probability μ per time step. To describe in a phenomenological way both the risk of infection and the risk of transmission of the nodes, each node $i \in \mathcal{V}$ is assigned a parameter $\lambda_i(t)$. The probability for a susceptible node i to become infectious through interactions with a set of infectious nodes j in its neighborhood $\mathcal{N}(i)$ depends on $\lambda_i(t)$ and on $\{\lambda_j(t)\}_{j \in \mathcal{N}(i) \cap \mathcal{I}(t)}$ (where $\mathcal{I}(t)$ is the set of infectious nodes at time t). The parameters $\lambda_i(t)$ can be modulated in time to encode the effect of the nodes' awareness of the epidemic. Further, we assume that this parameter relaxes to a baseline value λ_0 for all the nodes in the absence of such awareness.

We consider two different contagion mechanisms [Fig. 1(b)], based either on pairwise transmission or on higher-order nonlinear effects. In the *pairwise contagion process*, the spreading is sustained by the weighted projected graph $\mathcal{G}$: A susceptible node i at time t has probability $w_{i,j}\lambda_i(t)\lambda_j(t)$ to get infected by each infectious node j in $\mathcal{N}(i)$ (each link $(i, j) \in \mathcal{L}$ can thus be source of infection with multiplicity equal to its weight $w_{i,j}$). In the *higher-order contagion process*,

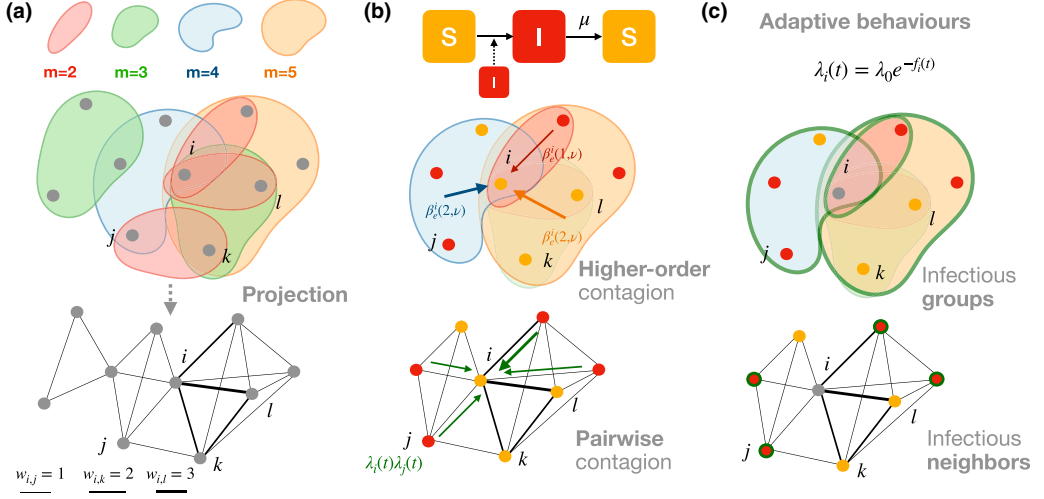


FIG. 1. Schematic representation of contagion and adaptation mechanisms. (a) Example of a hypergraph $\mathcal{H}$ and the corresponding weighted projected graph $\mathcal{G}$, obtained by projecting each hyperedge onto the corresponding clique (fully connected cluster) and assigning each link a weight equal to the number of distinct hyperedges containing both its extremities. For example, $w_{i,j} = 1$ in the sketched hypergraph, since only one hyperedge contains both i and j , while $w_{i,k} = 2$ and $w_{i,l} = 3$. (b) The SIS contagion mechanisms for the node i , with pairwise (bottom) and higher-order (top) processes. In the pairwise case, i can get infected by each infectious neighbor j with rate $\lambda_i(t)\lambda_j(t)$ and multiplicity equal to the weight $w_{i,j}$ of the link i, j in $\mathcal{G}$; in the higher-order case, i can get infected by each group e they participate in with rate $\beta_e^i(i_e, \nu)$, where i_e is the number of infectious nodes in the hyperedge e . (c) Adaptive strategies for node i , with group-based awareness (top, ng with $\theta = 0.3$: three hyperedges containing i are infectious, so $f_i^{\text{ng}}(t) = 3/\langle D \rangle$) or pairwise-based awareness (bottom, nn: four neighbors of i are infectious, so $f_i^{\text{nn}}(t) = 4/\langle k \rangle$).

the spreading is sustained by the hypergraph $\mathcal{H}$: A susceptible node i belonging to a hyperedge $e \in \mathcal{E}$, with i_e infected individuals at time t , is infected with probability $\beta_e^i(i_e, \nu) = \lambda_0 i_e^\nu \lambda_i(t) \langle \lambda_j(t) / \lambda_0 \rangle_{i_e}^\nu$. Here, $\langle \cdot \rangle_{i_e}$ indicates the average over the i_e infected nodes in e at time t and ν sets the nonlinearity of the process, modeling reinforcement group effects [14]. Note that in the $\nu = 1$ limit, the higher-order contagion process reduces to the pairwise one, since nonlinearities are removed and the weights of the projected graph take into account the overlap of hyperedges.

Adaptive mechanisms. We consider adaptive behaviors driven by risk perception due to local information (epidemic awareness) conveyed through the same interactions along which contagion can occur. We assume that these behaviors do not change the hypergraph topology, but act on the parameters $\lambda_i(t)$ of both susceptible and infectious nodes. This mechanism models a mild adaptation, where agents increase their attention during social interactions (e.g., through physical distance, use of masks, and increased hygiene), without changing the social structure but resulting in both a self-protective effect (for susceptible nodes) and an altruistic effect (for infectious ones) [40].

The baseline processes are represented by the *nonadaptive* case with $\lambda_i(t) = \lambda_0 \forall i \in \mathcal{V}, \forall t$, corresponding to the standard pairwise or higher-order SIS processes as in Ref. [14]. For the adaptive behaviors, we assume an exponential reduction $\lambda_i(t) = \lambda_0 e^{-\alpha f_i(t)}$ [32,34,40]. The parameter α modulates the reduction of λ_i and hence the intensity of adaptation: $\alpha = 0$ corresponds to the baseline NAD case; hereafter, we will consider $\alpha = 1$ for simplicity (in the companion paper [47], we investigate the impact of such parameter, showing that it does not alter the observed phenomenology). Instead, $f_i(t)$ encodes the local awareness of node i . Its specific form defines the adaptation strategy considered, i.e., how the node's risk perception depends on the health status of its neighborhood at time t .

We consider here two local adaptive strategies [see Fig. 1(c)]. In the “infectious neighbors” (nn) strategy, the awareness of a node is based on pairwise information, coming from its neighbors on the projected network $\mathcal{G}$:

$$f_i^{\text{nn}}(t) = |\mathcal{N}(i) \cap \mathcal{I}(t)| / \langle k \rangle, \quad (1)$$

where $\langle k \rangle$ is the average degree in $\mathcal{G}$ [50]. In the “infectious groups” (ng) strategy instead, the awareness of a node depends on the number of “infectious” hyperedges (groups) in which it participates: A group is defined as infectious for i if the number i_e of infected individuals within it *perceived by* i (i.e., distinct from i) is $i_e > \theta(|e| - 1)$, where θ is a fixed threshold (parameter of the strategy). In this case,

$$f_i^{\text{ng}}(t) = \sum_{e \in \mathcal{E}(i)} \mathbf{1}_\theta(e, i) / \langle D \rangle, \quad (2)$$

where $\langle D \rangle$ is the average hyperdegree in $\mathcal{H}$ (i.e., the average number of hyperedges of a node), $\mathcal{E}(i)$ is the set of hyperedges involving i , and $\mathbf{1}_\theta(e, i)$ is the group-indicator function, equal to 1 if $i_e > \theta(|e| - 1)$ (without counting i) and 0 otherwise. The parameter $\theta \in [0, 1]$ sets the level of alert: Hereafter, we will consider $\theta = 0.3$, while in Ref. [47] we investigate other values of θ . In Ref. [47], we also consider strategies involving mixed pairwise and higher-order information, or based on the fraction of infectious neighbors or groups rather than on their numbers.

Note that here we focus on purely local adaptive mechanisms; however, global mechanisms could also be implemented by introducing a node-independent phase in $\lambda_i(t)$: This would model information provided to the entire population through media and institutions [27,30,32].

Mean-field approach. To analyze the coupling between contagion dynamics and adaptive behaviors, we rely on an individual-based mean-field approach (IBMF), which describes the temporal evolution of the probability $P_i(t)$ that node i is infectious at time t . IBMF neglects dynamical correlations between the states of neighboring nodes but takes into account the actual structure of interactions (i.e., the graph or hypergraph on which contagion processes occur) [2]. For the pairwise contagion, $P_i(t)$ evolves according to

$$\partial_t P_i(t) = -\mu P_i(t) + (1 - P_i(t)) \sum_{j \in \mathcal{V}} w_{i,j} \lambda_j(t) \lambda_j(t) P_j(t), \quad (3)$$

where the first term accounts for the spontaneous recovery process and the second term describes the contagion of i , when it is susceptible, by one of its infectious neighbors (accounting for the link weights in $\mathcal{G}$).

For higher-order contagion, $P_i(t)$ evolves according to

$$\partial_t P_i(t) = -\mu P_i(t) + (1 - P_i(t)) \sum_{e \in \mathcal{E}(i)} \left[\sum_{i_e=1}^{|e|-1} Q_{e \setminus i}(i_e, t) \lambda_0 i_e^\nu \lambda_i(t) \overline{(\lambda_j(t) / \lambda_0)_{i_e}^\nu} \right], \quad (4)$$

where the second term on the right-hand side takes into account the probability that i is infected through a hyperedge in which it participates. In particular, $Q_{e \setminus i}(i_e, t)$ is the probability that in the hyperedge e at time t there are exactly i_e infectious individuals (distinct from i); $\overline{(\lambda_j(t) / \lambda_0)_{i_e}^\nu}$ denotes the average over these i_e infectious individuals and over all possible configurations of i_e infectious individuals in $e \setminus i$. Equations (3) and (4) constitute two closed sets of $N = |\mathcal{V}|$ coupled nonlinear differential equations. Indeed, we show in the Appendix how $\lambda_i(t)$, $Q_{e \setminus i}(i_e, t)$, and $\overline{(\lambda_j(t) / \lambda_0)_{i_e}^\nu}$ can be analytically obtained from the probabilities $P_j(t)$ of all nodes j .

Results. We perform direct Monte Carlo numerical simulations of the pairwise and higher-order contagion processes in discrete time, as well as a numerical integration of the mean-field equations, obtaining in each case the average epidemic prevalence in the asymptotic stationary state $I_\infty \equiv |\mathcal{I}|(t \rightarrow \infty)$ (see the Appendix). As substrate for the spread, we consider several empirical hypergraphs describing human face-to-face interactions in different settings [51–56], and also

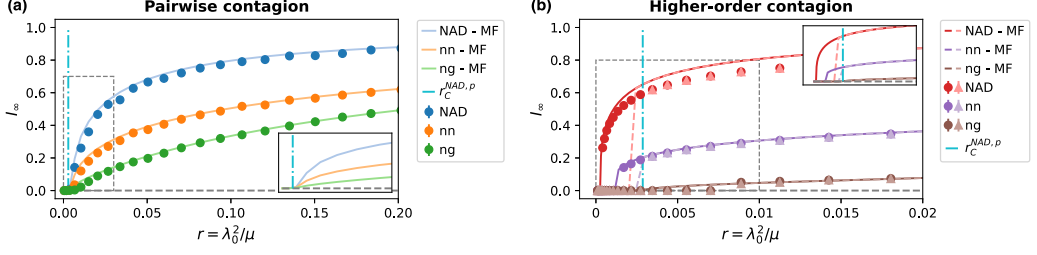


FIG. 2. Phase diagram for pairwise and higher-order contagion processes. (a), (b) The epidemic prevalence in the asymptotic steady state, I_∞ , as a function of the effective infection rate r , for the nonadaptive case and for the two adaptation strategies (nn, ng), respectively, for the pairwise and higher-order contagion processes. Lines give the results of the numerical integration of the IBMF equations, and markers show the results of numerical simulations. For the higher-order processes, we consider simulations and numerical integration starting either with a low (triangles and dashed lines) or with a high (dots and solid lines) fraction of infected nodes. We consider as substrate a dataset describing interactions in a hospital and the numerical results are averaged over 300 simulations. Here, $\nu = 4$, $\theta = 0.3$. The insets represent a close-up on the mean-field results in the area marked by a dashed rectangle, and the vertical line indicates $r_C^{\text{NAD},p}$. Note that the error bars on numerical simulations are smaller than the corresponding markers (considering one standard deviation).

synthetic hypergraphs (see the Appendix and Supplemental Material [57]). Here, we present results for a dataset describing interactions in a hospital [51,52], and we refer to the Supplemental Material for similar results on other hypergraphs [57]. Figure 2 displays I_∞ as a function of the reduced infection rate $r = \lambda_0^2/\mu$ for pairwise and higher-order processes in the nonadaptive case as well as for the two adaptive strategies considered: The IBMF framework and the numerical simulations show a very good agreement in all cases.

We refer to Ref. [47] for a detailed investigation of the ability of a wide set of adaptive strategies (including ng and nn) to reduce the asymptotic epidemic prevalence of pairwise and higher-order processes in the active phase of the epidemics, as well as a study of the associated societal costs (in terms of reduction of interactions). In Fig. 2, we simply highlight how both nn and ng strategies drastically reduce I_∞ for pairwise and higher-order processes, with a stronger effect for the group-based awareness (ng). Here, we focus on a more fundamental aspect, namely, how adaptive strategies impact the epidemic phase transition separating the disease-free absorbing phase ($I_\infty = 0$) from the active phase with $I_\infty > 0$ [2].

In the pairwise contagion process, the nonadaptive case presents the well-known continuous phase transition [2]. Interestingly, the adaptive mechanisms do not alter the phase transition, which remains continuous and presents the same epidemic threshold [see Fig. 2(a)]. We show in the Appendix that it is actually possible to obtain analytically the equality between the epidemic threshold of adaptive and nonadaptive cases:

$$r_C^{\text{NAD},p} = r_C^{\text{nn},p} = r_C^{\text{ng},p} = 1/\Lambda_w, \quad (5)$$

where Λ_w is the maximum eigenvalue of the weight matrix $\{w_{i,j}\}$. This can be understood intuitively quite simply. Indeed, as the epidemic transition is continuous, the fraction of infected nodes tends to 0 in the active phase close to the critical point. Thus, $f_i^{\text{nn}}, f_i^{\text{ng}} \ll 1$ and $\lambda_i \sim \lambda_0 \forall i$ for r close to $r_C^{\text{NAD},p}$. The effect of the adaptation strategies becomes of second order and cannot shift the transition point.

The higher-order contagion process presents a different and more interesting phenomenology. Indeed, the nonadaptive case presents a discontinuous phase transition, with a bistability region where both the endemic and absorbing states are stable [14]. However, as shown in Fig. 2(b), both adaptive strategies shrink the bistability region. Specifically, the nn strategy increases the value of the lower bound of this region. Strikingly, the ng strategy completely neutralizes the bistability

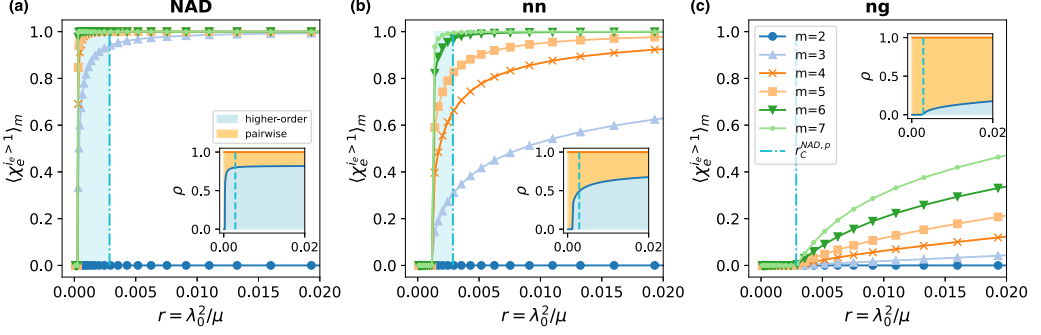


FIG. 3. Neutralization of higher-order contagion. Probability $\langle \chi_e^{ie>1} \rangle_m$ that a group of size m with at least one susceptible node has more than one infected individual in the asymptotic steady state, as a function of the effective infection rate r , for different values of m . $\langle \chi_e^{ie>1} \rangle_m$ is estimated through numerical integration of the mean-field equations, with initial conditions having high fraction of infected nodes. Panel (a) corresponds to the nonadaptive case, and panels (b) and (c) each to an adaptive strategy. We use $\nu = 4$ and $\theta = 0.3$, the vertical line indicates $r_C^{NAD,p}$, and the light-blue colored area indicates the bistability region. The inset shows the probability ρ that a group of arbitrary size supports higher-order contagion (light-blue area), i.e., a contagion event triggered by more than one infectious, as a function of r , and the complementary probability that it supports pairwise contagion (orange area); see the Appendix for how to compute ρ .

regime and the explosiveness of the transition, making it continuous with the same epidemic threshold as the pairwise contagion, $r_C^{NAD,p}$.

Although the adaptive strategies are based on the same mechanisms in pairwise and higher-order contagion, they have drastically different effects. This can be understood both intuitively and at a more fundamental level as follows. As the transition in the higher-order case is discontinuous, the prevalence in the NAD case takes a large value right above the transition point. Therefore, f_i^{nn} and f_i^{ng} are not necessarily small as in the pairwise case. Thus, λ_i can become locally much smaller than λ_0 , bringing the prevalence down and making the high-prevalence state unstable for r not large enough.

We can investigate this point in a more quantitative manner by recalling that the discontinuity of the transition and the bistability regime are due to the nonlinearity in the contagion probability [14], which are relevant only in hyperedges where more than one infectious node is present. We thus consider the probability (in the stationary state) $\chi_e^{ie>1}$ that a hyperedge e with at least one susceptible individual (i.e., where a contagion can occur) has more than one infectious individual. We show in the Appendix how this probability can be computed from P_i in the IBMF framework and we present the results in Fig. 3. In the NAD case, the average of this quantity over hyperedges of size m obeys $\langle \chi_e^{ie>1} \rangle_m \sim 1$ for all $m > 2$ in the bistability region. The contagion events involving the nonlinear higher-order mechanism are thus sustained in this region. In fact, the propagation is predominantly driven by group contagions, as measured by the fraction ρ of potential infections driven by nonlinear group processes [see the Appendix and Fig. 3(a)].

Adaptive behaviors reduce the probability of contagion within groups in which the infectious nodes are located, by locally increasing awareness (see the Supplemental Material [57]). Figures 3(b) and 3(c) show how both strategies lead to a strong decrease of $\chi_e^{ie>1}$ for all hyperedge sizes, leading to a reduction of the fraction ρ of contagions driven by nonlinear group mechanisms. Higher-order effects are thus suppressed by the strategies, which make it more difficult to have several infectious nodes at the same time in the same hyperedge, replacing group contagion events by pairwise ones. As a result, the discontinuous transition at the lower bound of the bistability region is pushed toward higher values of r , and the amplitude of the discontinuity is reduced. Instead, the upper bound of the bistability region, which corresponds to the pairwise epidemic threshold, is not modified, as the stability of the absorbing state with $I_\infty = 0$ is determined solely

by pairwise interactions (see the Appendix) [10]. As a result, the width of the bistability region decreases. For adaptive strategies having a mild effect, such as the nn one, $\chi_e^{i_e>1}$ and ρ are decreased for all r but go to zero only for r small enough: The bistability region shrinks but the transition remains discontinuous [Fig. 3(b)]. On the other hand, for adaptive strategies efficient enough to make $\chi_e^{i_e>1} = 0 \forall e$ for all $r < r_C^{\text{NAD},p}$, such as the ng strategy, only pairwise contagions can be sustained in this regime [Fig. 3(c)]. As a result, since only the absorbing state is stable for pairwise contagion when $r < r_C^{\text{NAD},p}$, the bistability region completely disappears and the phase transition changes from discontinuous to continuous. This can also be shown analytically at the mean-field level: If we assume that $\chi_e^{i_e>1} \sim 0$, the mean-field equations for higher-order contagion reduce to those of pairwise contagion around the critical point (see the Appendix), thus leading to a continuous transition at the same epidemic threshold. Note that these considerations apply only for $r \leq r_C^{\text{NAD},p}$: For $r > r_C^{\text{NAD},p}$, the pairwise contagion by itself leads to a nonzero I_∞ , and the prevalence increases with r , so that $\chi_e^{i_e>1}$ also increases and nonlinear group contagion events become sustainable again, hence altering the dynamics compared to the pairwise case.

We show in the Supplemental Material that these findings are robust for processes unfolding over other empirical and synthetic hypergraphs, and we highlight the role of the underlying structure [12,13,47,57].

Discussion. In this Letter, we characterized in detail the impact of adaptive behaviors based on local awareness, driven by either pairwise or group interactions, on pairwise and higher-order contagion processes. The adaptive mechanisms are based on the same principle in all cases: Infectious nodes trigger awareness of risk in their neighborhoods, locally reducing the parameters determining contagion events and thus the transmission rates. We have analyzed their effect through an analytical mean-field approach and numerical simulations, applied on a series of empirical and synthetic hypergraph substrates. In the active phase of the epidemic, in which the disease-free equilibrium is unstable, adaptive behaviors reduce the epidemic prevalence for both types of contagion, with awareness based on group information having a stronger effect. However, their impact on the epidemic transition is very different for pairwise and higher-order processes. The former present a continuous phase transition in the nonadaptive case, and adaptive strategies do not change it, preserving its continuous nature and the epidemic threshold. Indeed, the triggering of adaptive behaviors tends to vanish when approaching the transition from above, as the epidemic prevalence itself goes to 0. The higher-order contagion process instead presents a discontinuous phase transition with a bistability regime, driven by nonlinear group infection mechanisms. Because of the discontinuity, the epidemic prevalence is large enough close to the transition, so that awareness mechanisms can be triggered and have an impact on the transition. As a consequence, adaptive behaviors are able to make the high-prevalence state unstable for r not large enough, contracting the bistability region. If the strategy is efficient enough, it can even completely suppress higher-order effects and make the discontinuous transition and the bistability region disappear. As adaptivity reduces the prevalence indeed, it decreases the possibility of occurrence of nonlinear contagion events, defusing the explosiveness of group contagions that drive the discontinuous transition.

Our work allows for a deeper understanding of higher-order processes on hypergraphs in the presence of adaptive behaviors and makes possible a systematic comparison of the performance of different adaptive strategies on hypergraphs. In particular, we show that strategies resulting from group-based awareness are more effective than those based on pairwise information. We explore both performance and costs of strategies in detail in Ref. [47], considering a wide set of strategies based on local information of different nature, which can be pairwise, group, or hybrid. Finally, our results can be the starting point for studying the impact of adaptive behaviors on other higher-order processes such as opinion formation and voter models [49,58–60], for investigating the role of heterogeneity in higher-order adaptive mechanisms' adoption [41,43,44], and for understanding the nontrivial impact of global adaptive mechanisms in hypergraphs [27,30,32].

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Data availability. The data that support the findings of this study are publicly available. The SocioPatterns datasets are available in Refs. [51,61].

Appendix: Empirical and synthetic hypergraphs. We consider several empirical and synthetic hypergraphs $\mathcal{H}$, and their corresponding weighted projected graphs $\mathcal{G}$. Projected graphs were obtained by projecting each hyperedge onto the corresponding clique and assigning each link a weight equal to the number of distinct hyperedges containing the two extremities of the link.

We built static hypergraphs from empirical datasets describing face-to-face interactions in diverse environments: a hospital [51,52], conferences [54], and schools [51,55,56] (see the Supplemental Material for details on each dataset and preprocessing [57]). These hypergraphs cover a wide spectrum of properties (see the Supplemental Material [57]), ranging from 76 to 327 nodes and from 315 to 4795 hyperedges of sizes up to 7. For example, the hospital dataset (considered in the main text) features 76 nodes and 1102 hyperedges of size $m \in [2, 7]$.

Synthetic hypergraphs are obtained by both (1) applying randomization procedures to the empirical ones, with different reshuffling methods [19,62], and (2) setting specific features and mechanisms for hyperedge formation (such as specific hyperdegree distributions or overlap level). We refer to the Supplemental Material for more details [57].

Numerical simulations and integration of mean-field equations. We performed stochastic numerical simulations of the epidemic processes in discrete time: Infected individuals recover with probability μ per unit of time; a susceptible individual i becomes infectious at time t with probability $p_i(t)$. In the pairwise contagion process,

$$p_i^p(t) = 1 - \prod_{j \in \mathcal{V}} (1 - \lambda_i(t) \lambda_j(t))^{w_{i,j}}, \quad (\text{A1})$$

which takes into account all possible contagion events from a neighbor j of i toward i , and the multiplicity $w_{i,j}$ of the corresponding links. In the higher-order contagion process,

$$p_i^{\text{ho}}(t) = 1 - \prod_{e \in \mathcal{E}(i)} (1 - \beta_e^i(i_e, v)), \quad (\text{A2})$$

which takes into account all possible contagion events. In both processes, the simulations stop when the system reaches the endemic steady state or the absorbing state. If the system does not reach the absorbing state, the simulation is run for a minimum of T time steps. We denote by $\mathbf{I}_{a,b} = [|\mathcal{I}|(a), |\mathcal{I}|(a+1), \dots, |\mathcal{I}|(b-1), |\mathcal{I}|(b)]$ the time series of the epidemic prevalence from time a to b , $\bar{\mathbf{I}}_{a,b}$ indicates its average, and $\sigma(\mathbf{I}_{a,b})$ indicates its standard deviation. The system is then assumed to have reached the stationary state at the time step $t > T$ if (1) $\sigma(\mathbf{I}_{0.8t,t}) < 10^{-2}$ and (2) $|\bar{\mathbf{I}}_{0.9t,t} - \bar{\mathbf{I}}_{0.8t,0.9t}| < 10^{-2}$. The asymptotic epidemic prevalence is then calculated as $I_\infty = \bar{\mathbf{I}}_{0.8t,t}$.

The pairwise contagion process is simulated starting from a single infectious individual (selected uniformly at random) in a completely susceptible population. We set $\mu = 10^{-2}$ and $T = 1000$ without loss of generality and average the results over different initial conditions and over all outcomes of the epidemic (as long as at least one transmission event has occurred during the simulation). The higher-order process is simulated starting with either 2% or 80% of the population in the infectious state (infectious nodes are selected uniformly at random), in order to explore both branches of the phase diagram in the bistability region. In this case, we use $\mu = 10^{-1}$, $T = 300$, and the results are averaged over different initial conditions and only over the realizations where the epidemic reaches an endemic state. For both type of processes, the results are averaged over 300 numerical simulations.

The mean-field equations are integrated numerically, considering the same epidemiological parameters as the numerical simulations. The pairwise process is integrated starting from

$P_i(0) = 0.01 \forall i$, while the higher-order process is integrated either from $P_i(0) = 0.01 \forall i$ or from $P_i(0) = 0.8 \forall i$, to explore the two branches of the phase diagram.

Individual-based mean-field approach. We show here that we obtain two closed sets of mean-field equations for the two contagion processes, by expressing all quantities as functions of $P_i(t)$. We first need to evaluate the probability $Q_{e \setminus i}(i_e, t)$ that in the hyperedge e at time t there are exactly i_e infected individuals (other than i):

$$Q_{e \setminus i}(i_e, t) = \sum_{A \in \mathcal{C}_{e \setminus i}(i_e)} \prod_{j \in A} P_j(t) \prod_{k \in e \setminus \{i, A\}} (1 - P_k(t)), \quad (\text{A3})$$

where $\mathcal{C}_{e \setminus i}(i_e)$ is the set of all possible sets of i_e nodes in $e \setminus i$.

Analogously, we need to evaluate the term $\langle \lambda_j(t) / \lambda_0 \rangle_{i_e}^v$, which involves a first average over the i_e infected individuals in e , and then a second average over all possible configurations of i_e infected individuals in $e \setminus i$:

$$\overline{\langle \lambda_j(t) / \lambda_0 \rangle_{i_e}^v} = \sum_{A \in \mathcal{C}_{e \setminus i}(i_e)} H_A(t) \left(\frac{1}{i_e} \sum_{j \in A} e^{-f_j(t)} \right)^v, \quad (\text{A4})$$

where

$$H_A(t) = \frac{\prod_{j \in A} P_j(t) \prod_{k \in e \setminus \{i, A\}} (1 - P_k(t))}{\sum_{B \in \mathcal{C}_{e \setminus i}(i_e)} \prod_{j \in B} P_j(t) \prod_{k \in e \setminus \{i, B\}} (1 - P_k(t))}. \quad (\text{A5})$$

To integrate the equations when adaptive strategies are taken into account, we need to evaluate the awareness function $f_i(t)$. For the nn strategy,

$$f_i^{\text{nn}}(t) = \sum_{n=0}^{|\mathcal{N}(i)|} n Q_{\mathcal{N}(i)}(n, t) / \langle k \rangle, \quad (\text{A6})$$

where $Q_{\mathcal{N}(i)}(n, t)$ is the probability that i has exactly n infected neighbors at time t , which is calculated analogously to Eq. (A3). For the ng strategy,

$$f_i^{\text{ng}}(t) = \sum_{g=0}^{D(i)} g Z_{\mathcal{E}(i)}(g, t) / \langle D \rangle, \quad (\text{A7})$$

where $D(i)$ is the hyperdegree of node i , $\mathcal{E}(i)$ is the set of hyperedges in which i is involved, and $Z_{\mathcal{E}(i)}(g, t)$ is the probability of having g infectious hyperedges at time t among the hyperedges in $\mathcal{E}(i)$. We can write

$$Z_{\mathcal{E}(i)}(g, t) = \sum_{A \in \mathcal{C}_{\mathcal{E}(i)}(g)} \prod_{e \in A} \xi_{e,i}(t) \prod_{h \in \mathcal{E}(i) \setminus A} (1 - \xi_{h,i}(t)), \quad (\text{A8})$$

where $\mathcal{C}_{\mathcal{E}(i)}(g)$ is the set of all possible sets of g hyperedges in $\mathcal{E}(i)$ and $\xi_{e,i}(t)$ is the probability that the hyperedge e is infectious at time t for node i . In particular,

$$\xi_{e,i}(t) = \sum_{i_e = \phi_e(\theta)}^{|e|-1} Q_{e \setminus i}(i_e, t), \quad (\text{A9})$$

where $\phi_e(\theta) = \lfloor \theta(|e| - 1) \rfloor + 1$ is the minimum number of infectious individuals in e (distinct from i) so that i perceives e as at-risk.

Stability of the absorbing state. By applying a linear stability analysis to Eq. (3), we obtain a mean-field estimation of the epidemic threshold for the pairwise contagion case [2]. In the NAD

case, Eq. (3) reduces to

$$\partial_t P_i(t) = -\mu P_i(t) + \lambda_0^2 (1 - P_i(t)) \sum_{j \in \mathcal{V}} w_{i,j} P_j(t). \quad (\text{A10})$$

Linearizing Eq. (A10) around the absorbing state $\mathbf{P} = [0, 0, \dots, 0]$ leads to the Jacobian matrix with element $J_{i,j} = -\mu \delta_{i,j} + \lambda_0^2 w_{i,j}$. The absorbing state is thus stable if and only if the largest eigenvalue of $\{J_{i,j}\}$ is negative, i.e., if

$$r = \lambda_0^2 / \mu < 1 / \Lambda_w \equiv r_C^{\text{NAD}, p}, \quad (\text{A11})$$

where Λ_w is the largest eigenvalue of $\{w_{i,j}\}$.

The adaptive strategies we considered feature the same Jacobian matrix as the NAD case. Indeed, expanding around the absorbing state and keeping only the linear leading order yields $Q_{\mathcal{N}(i)}(n, t) \sim \delta_{n,1} \sum_{j \in \mathcal{N}(i)} P_j(t)$ and $Q_{e \setminus i}(i_e, t) \sim \delta_{i_e,1} \sum_{j \in e \setminus i} P_j(t)$; hence,

$$f_i^{\text{nn}}(t) \sim \sum_{j \in \mathcal{N}(i)} P_j(t) / \langle k \rangle, \quad (\text{A12})$$

$$f_i^{\text{ng}}(t) \sim \sum_{e \in \mathcal{E}(i) | \phi_e(\theta) \leq 1} \sum_{j \in e \setminus i} P_j(t) / \langle D \rangle. \quad (\text{A13})$$

Therefore, for both strategies,

$$\lambda_i(t) = \lambda_0 e^{-f_i(t)} \sim \lambda_0 (1 + o(\mathbf{P})) \forall i, \forall t. \quad (\text{A14})$$

Inserting in Eq. (3), we recover the NAD case of Eq. (A10), which implies that the epidemic thresholds for the pairwise contagion in the adaptive cases are the same as for the NAD case.

Moreover, the conditions for the absorbing state to be stable in the higher-order contagion process are fully determined by the pairwise interactions only [10], and are the same of the pairwise process [Eq. (A11)] independently of the adaptive strategy. Indeed, substituting the expansions around the absorbing state in Eq. (4), we obtain

$$\partial_t P_i(t) \sim -\mu P_i(t) + (1 - P_i(t)) \lambda_0^2 \sum_{e \in \mathcal{E}(i)} \sum_{j \in e \setminus i} P_j(t), \quad (\text{A15})$$

which correspond exactly to Eq. (A10), since $\sum_{e \in \mathcal{E}(i)} \sum_{j \in e \setminus i}$ can be rewritten as $\sum_{j \in \mathcal{V}} w_{i,j}$.

Quantifying the presence of nonlinear contagion. Finally, we define the probability $\chi_e^{i_e > 1}$ that a group e , containing at least a susceptible node, contains more than one infected individual in the asymptotic steady state:

$$\chi_e^{i_e > 1} = \sum_{i_e=2}^{|e|-1} Q_e(i_e, t \rightarrow \infty), \quad (\text{A16})$$

with $Q_e(i_e, t \rightarrow \infty)$ calculated analogously to Eq. (A3). Then, we can define the average probability $\langle \chi_e^{i_e > 1} \rangle_m$ for groups of size m and we can estimate the fraction of potential infections driven by nonlinear group phenomena:

$$\rho = \sum_{m=2}^M \Psi(m) \frac{\langle \chi_e^{i_e > 1} \rangle_m}{\langle \chi_e^{i_e > 1} \rangle_m + \langle \chi_e^{i_e = 1} \rangle_m}, \quad (\text{A17})$$

where $\chi_e^{i_e = 1} = Q_e(1, t \rightarrow \infty)$, $M = \max_{e \in \mathcal{E}} \{|e|\}$, and $\Psi(m)$ is the hyperedge size distribution.

From higher-order transition to a pairwise one. We can analytically show, through the mean-field equations, that the higher-order contagion dynamics becomes equivalent to the pairwise one near the critical point, when the probability of having more than one infected agent in a hyperedge becomes

close to zero. Let us indeed assume that $\chi_e^{i_e > 1} \sim 0$, i.e., that the probability of having i_e infected nodes in a hyperedge e with one susceptible node at time t is zero for $i_e > 1$:

$$Q_{e \setminus i}(i_e, t) \sim \delta_{i_e, 1} \sum_{j \in e \setminus i} P_j(t) \prod_{k \in e \setminus \{i, j\}} (1 - P_k(t)). \quad (\text{A18})$$

In this case, the epidemic dynamics reduces to

$$\partial_t P_i(t) = -\mu P_i(t) + (1 - P_i(t)) \sum_{e \in \mathcal{E}(i)} \sum_{j \in e \setminus i} P_j(t) \lambda_0 \lambda_i(t) (\lambda_j(t) / \lambda_0)^v \prod_{k \in e \setminus \{i, j\}} (1 - P_k(t)). \quad (\text{A19})$$

We replace $\sum_{e \in \mathcal{E}(i)} \sum_{j \in e \setminus i}$ with $\sum_{j \in \mathcal{V}} w_{i,j}$ and linearize the infection term around the absorbing state, using Eq. (A14):

$$\lambda_0 \lambda_i(t) (\lambda_j(t) / \lambda_0)^v \prod_{k \in e \setminus \{i, j\}} (1 - P_k(t)) \sim \lambda_0^2. \quad (\text{A20})$$

Therefore, by substituting in Eq. (A19), if $\chi_e^{i_e > 1} \sim 0$, the contagion dynamics near the critical point is governed by

$$\partial_t P_i(t) \sim -\mu P_i(t) + (1 - P_i(t)) \sum_{j \in \mathcal{V}} w_{i,j} \lambda_0^2 P_j(t), \quad (\text{A21})$$

which reproduces that of the NAD pairwise case [see Eq. (3)], implying the continuous nature of the phase transition and the epidemic threshold $r_C = 1/\Lambda_w$.

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