Avalanche outbreaks emerging in cooperative contagions
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Details of numerical simulations for preparing the figures of the manuscript and additional supporting results are presented here.

S1. UPDATING SCHEMES AND IMPLEMENTATION

We adopted two updating schemes for implementing the model, asynchronous updating (AU) and synchronous updating (SU). They differ in the updating latency, regarding the time of expressing the infectedness.

For both schemes, nodes that are newly infected at time step \( t \) are assumed infective only till \( t + 1 \). For AU, a node is regarded as infected immediately after being infected, within the same time step. So the order in which a node is infected would be of relevance, regarding possible secondary infection. Thus, the list of infective nodes is randomly shuffled at the beginning of every time step, so that each node tries to infect in an unbiased order. For SU, all nodes that are infected at the current step \( t \) are regarded as infected only till the new time step \( t + 1 \). States of all nodes can thus be updated simultaneously upon those at the previous time step, with no need to define an infection order.

Concretely, one needs two maps to record the node states for time step \( t \) and \( t + 1 \), with the SU scheme. Each map is stored separately. The latter map is updated upon the former. With the AU scheme, only one map is needed. This is because the state of a node must be updated right after any infection.

The small updating latency with SU is expressed only when two active diseases attack the same virgin node: a node in state \( S \) first infected by disease \( A \) is still regarded as uninfected during the same time step, so that it is infected by disease \( B \) still with the probability \( p \). Rather, with AU the infection by disease \( B \) is executed with the secondary probability \( q \). The conditional probability for state transition in this case can be expressed as: \( P_{SU}(A|S) = P_{SU}(B|S) = p(1 - p) \) and \( P_{SU}(A|B) = p^2 \) for SU; \( P_{AU}(A|S) = P_{AU}(B|S) = p(1 - q) \) and \( P_{AU}(AB|S) = pq \) for AU. This seemingly tiny asymmetry would lead to qualitatively different statistical behaviours only on few topologies. On most networks we studied, the order of phase transition is independent of updating scheme, where only minor differences are identified, such as a slight shift of critical point.

S2. ON RANDOM TOPOLOGIES

To guarantee sufficient randomness, we create new Erdős-Rényi (ER) networks for every 100 realisations and make re-wirings for each single one. At the start of each simulation run, a doubly infected seed is randomly placed on a node that belongs to the giant cluster of the network. We can distinguish reliably giant epidemics from small outbreaks of infinitesimal fraction of the network by using the mass distribution in Fig. 2a. Up to \( 10^8 \) realisations are simulated for obtaining the statistics. This figure is only for one pair of \( (p, q) \), but similar results were found as long as \( q \) is substantially much larger than \( p \). The positions of right peaks, corresponding to the sizes of the infected giant clusters, are plotted against the system sizes \( N = 2^{14} \sim 2^{27} \) in Fig. 2c. The data fitting shows exactly a linear scaling, which can be extrapolated to the thermodynamic limit \( N \to \infty \). For the transition curves in the main plot of Fig. 2b, the statistics are obtained after averaging over more than \( 10^7 \) realisations for each \( p \) and \( N \) (the number of realisations depends actually on below or above \( p_c \) ). The inset in Fig. 2b is obtained by averaging the fractions of all giant doubly infected clusters in those realisations for different \( N \).

Further, we simulated the process on ER networks using seeds consisted of a finite proportion of nodes and also observed DTs by \( \rho_{ab} \) (data not shown). This is accordant with our prior mean-field calculations, based on a continuum assumption of well-stirred population [31]. There we treated disease states as different compartments so that the system was described as chemical reaction kinetics.

A. Composition of giant clusters

The giant infected clusters consist predominantly of doubly infected nodes (especially for large \( q \)), as shown in Fig. S1. The upper discontinuous branch II has a consistent profile with II-ab. The singly infected nodes (in branches II-a and II-b) are marginal at the critical point, although there is also an abrupt increase in the fraction, accompanying
the formation of giant doubly infected clusters. On the other hand, if only one of the two pathogens can survive (in the supercritical regime), the formed clusters fall simply into the continuous lower branch I, being the superposition of branches I-a and I-b.

At the critical point, the giant infected cluster has a one-to-one mapping relation with the giant doubly infected cluster. A giant infected cluster is formed if and only if a giant doubly infected cluster is formed. Any giant singly infected cluster is excluded since neither of the single diseases is able to sustain by itself (being critical or subcritical) if no doubly infected cluster is excited. Hence, the behaviour of the giant infected cluster can well be represented by that of its doubly infected component.

### B. Finite size scaling (FSS)

If \( P_{ab}(p,N) \) agrees with a standard FSS [1], as suggested in the main text, we can plot the data in a rescaled coordinate so that they collapse into a single curve \( \Psi: P_{ab}(p,N)N^{\beta/\nu} = \Psi((p - p_c)N^{1/\nu}) \), regardless of system size. This results in a decent fitting, as shown in Fig. S2. More precisely, we plot \( P_{ab}N^{\gamma} \) against \((p - p_c)N^{1/\nu}\) with \(1/\nu = 0.20, \gamma = \beta/\nu = 0.12 \) and \( p_c = 0.25 \). This is a clear indication that the transition is continuous at the threshold \( p_c \), regarding the order parameter \( P_{ab} \).

### C. Metastable states

The FSS manifests that giant clusters can be triggered for \( p < p_c \) (the dotted tip in the inset of Fig. 2b) only with vanishing probability in the limit \( N \to \infty \). Those metastable states resemble those in supersaturated vapour in first order phase transitions. In fact, for any \( p < p_c \), starting with \( n \) randomly chosen seed nodes leads to a giant cluster with a finite probability \( P_{ab} \), only if the seed density \( n/N \) exceeds a threshold \( p_0(p) \). This is shown by another FSS at \( p = 0.20: P_{ab} \sim (n/N - p_0)N^x \) with \( p_0 = 0.000282 \) and \( x = 0.5 \) (see Fig. S3).

We should point out that no tricritical point is found on the ER or lattice topologies we studied [22], which is confirmed by the simulations made with a range of smaller values of \( q \). This implies that in all those cases the transition is either continuous or discontinuous for all \( q > p \). The above observations hold also qualitatively for \( q < 1 \), but with different exponents in the FSS. In particular, the critical point remains at \( p_c = 1/k \), and the probability \( P_{ab} \) decays still as a power law for increasing \( N \) but with a different power (Fig. S4). We used AU for generating Fig. 2 and S2 ∼ S4, but no qualitative difference is seen for SU.

### D. Single pathway

The following proposition holds: starting from a given doubly infected node, the co-spreading of two diseases along a 1-dimensional path is equivalent to a single-agent spreading process. In fact, if both diseases can survive, they must infect each node on the path successfully at the same time step — they follow each other. If a disease fails to infect any node, it recovers and its spread ends. Hence the co-spreading can be regarded as a single-agent process (two diseases bonded as one) with an equivalent infection probability \( pq \) (for AU) or \( p^2 \) (for SU).

For the same reason, the double infection on a tree can also be regarded as a single-agent process, since the spreading on any branch cannot proceed backwards. So locally, the spreading proceeds on a 1d path.

The proposition shows that effective cooperativity can arise only if there are multiple paths, i.e., if there exist loops on the network. On the other hand, ER networks are locally tree-like. If the epidemic cannot survive till the long loops exert the effect, it should resemble a monopartite process. There are realisations that end at the early stage of the simulation. They actually correspond to the power law scaling of the left ramp in the mass distribution shown in Fig. 2a: \( P(m) \sim m^{-1.5} \), which agrees with a typical scaling law in the OP universality class.

### S3. ON REGULAR TOPOLOGIES

#### A. 2d lattices

On regular lattices, the helical boundary condition is used, where each lattice site is numbered by a sequential single integer \( i \). The nearest neighbours of \( i \) in a \( d \)-dimensional simple lattice are the sites \( i \pm 1, i \pm L, ... i \pm L^{d-1} \). This is
essentially equivalent to the periodic boundary condition, but converts a high dimensional lattice to a one-dimensional array. System sizes in the range of $2^{15} \sim 2^{34}$ are used in the simulations.

The curves of $n(t)$ in Fig. 2a were obtained by averaging over $10^4$ realisations for each $p$ value on a $1024 \times 1024$ lattice with the AU scheme. We stop the simulation when the span of the infected clusters reaches the boundary, so that there is strictly no finite-size correction. The values of $p$ from bottom to top are 0.4496, 0.4501, 0.4503, 0.4510 and 0.4515, respectively. The estimated critical value is at $p_c \approx 0.4503(1)$, which is smaller than the $p_c = 0.5$ for a single-agent process. Hence, the cooperativity only facilitates the propagation, but does not change the order of transition.

The transition diagrams for $\rho_{ab}$ and $P_{ab}$ in the insets are obtained after averaging over $10^4$ realisations for each $p$ value (the number of realisations depends actually on below or above $p_c$) and the giant cluster sizes are calculated till the die-out of the simulation runs.

1. Mass distribution

The mass distribution in Fig. S5a gives additional evidence for identifying the transition (in order to demonstrate that no value of $q$ is of special relevance, we chose an arbitrary value of $q = 0.9$, for which $p_c$ is at around 0.43). We observe large incipient clusters (the right humps) in the mass distribution of infected clusters at $p_c$. The size of the incipient clusters however scales as $m_{peak} \sim L^{d_f}$, with $L = 2^{10}, \ldots, 2^{13}$ being the linear length of the lattices, where $d_f \approx 91/48 (< d = 2)$ is a typical fractal dimension for OP (see Fig. S5b) [1]. Also, the distributions of the left ramp satisfy the standard OP scaling law $P(m_{ab}) \sim m_{ab}^{-\tau}$, where $\tau \approx 187/91$ [1]. This, together with the results shown in the main text, confirms a CT in the OP universality class.

The results on this dimension are qualitatively identical for SU and AU, except showing slightly different critical points. The snapshot in Fig. 3c was obtained at $q \approx 0.99$ and $p = p_c \approx 0.4504(1)$, by using SU. The average thickness of the “halo” surrounding the core is equal to the correlation length of OP at the above value of $p$, i.e., $\ell \sim (p - 0.5)^\sigma$.

B. 4d lattices

The curves of $n(t)$ in Fig. 3b were obtained by averaging over $10^4$ realisations for each $p$ value on a $512^4$ lattice with the AU scheme. The values of $p$ from bottom to top are 0.1117, 0.1118, 0.1120, 0.1121, 0.1122, 0.1123, 0.1124 and 0.1126, respectively. These are all much smaller than the $p_c \approx 0.16013$ for the single-agent process [32].

1. Precise estimation of critical point

The concavity of $n(t)$ strongly suggests the existence of a bottleneck as expected in a DT with nucleation. It is thus hard to pin down the critical point precisely in a reasonable number of realisations starting from a single seed (even when using sophisticated sampling methods such as PERM [33]).

Much more precise estimate of $p_c(q)$ can be obtained from simulations where we start from an entire doubly infected hyperplane. With such configuration, we bypass the initialisation of the cluster and assume that whether a giant cluster can be formed only depends on the later development. Notice that even from a point seed, surfaces of big clusters will finally become sufficiently flat as a plane, so that we assume that $p_c(q)$ is a property of the cluster growth mechanism only, and is thus independent of the type of seed.

In Fig. S6a we show the plot of $\rho(t)$, the density (per unit hyperplane) of newly infected sites at time $t$, for $q = 1$ and for various values of $p$. We used here AU, but again it was checked that SU gave qualitatively the same results. In all realizations we used helical b.c. laterally, but open b.c. in the direction of mean growth perpendicular to the hyperplane. The base (hyper-) planes have between $2^{21}$ and $2^{27}$ sites (corresponding to $L = 128$ and $L = 512$), depending on the distance from the critical point. With these sizes we expect finite size corrections to be negligible in Fig. S6a, except possibly for $t > 10,000$ and $p$ very close to $p_c$. For simulating such large systems we not only used large memory (up to 96 GB per CPU), but also used a recycling technique. In test runs we first estimated upper bounds on the roughness $\Delta h$ of the propagating fronts, which are defined as the height difference between the highest and the lowest infected site. We set a vertical height of the lattice $L_z$ so that $\Delta h$ is assured less than it. Whenever $h_{max}$ exceeds $L_z$, we erased the lowest part of the byte map and used it for storing the configuration of the highest front. Fig. S6 suggests that the critical point is at $p_c = 0.111857(2)$, which corresponds roughly to the second lowest curve in Fig. 3b. At this value of $p_c$, we find a power law $\rho(t) \sim t^{-\delta}$, with $\delta = 0.45(2)$. 

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2. Infinite order of $P_{ab}$

With the much more precisely estimated $p_c$, we shall return to check the transition for the single seed case. In order to demonstrate that the transition is of infinite order when seen through $P_{ab}$, we show in Fig. S7 a plot of $\ln P_{ab}$ against $1/(p - p_c)$. For precisely estimating $P_{ab}$, we made (for the smallest value of $p$) up to $8 \times 10^9$ realisations. It suggests clearly that the data do not follow a power law. For $p$ very close to $p_c$ the data are reasonably well described by $P_{ab} \sim \exp(-a/(p - p_c))$ with $a \approx 0.0064$. It suggests that there exists an essential singularity as in other phase transitions of infinite order.

C. 3d lattices

The dynamics shows qualitative difference for the two updating schemes uniquely on three-dimensional lattices. We obtained the curves for $n(t)$ in Fig. 4a and 4b by averaging over $10^4$ realisations for each $p$ value on the $1024^3$ (simple cubic) lattice with both AU and SU schemes, starting from a doubly infected seed. An important difference from the 4d case arises in that $P_{ab}$ scales here with a power of $p - p_c$, as shown in Fig. S8, which suggests that the transition, regarding $P_{ab}$, is continuous and of second (finite) order.

The contrast in the transition order is attributed to the small difference in the intrinsic latency with the two schemes, which however shows particular relevance on short loops. This is because if two diseases meet on a short loop, from a common starting site, there would be high probability that the two pathways of the single diseases are of the same length. In such cases, the meeting node can be infected by both diseases with probability $p^2$ for SU, or with $pq$ for AU. Hence, SU tends to have weaker cooperation at the early stage.

Here we also show the dependence of the transition order on further factors, which demonstrates 3d as a critical dimension for lattices. We point out firstly that by using AU, we always found CTs in the OP universality class. But for SU, the specific type of lattice (i.e., the set of links defining the local neighbourhood) and even the precise type of seed are relevant. We consider only SU in the following subsections.

1. Dependence on seed type

The system exhibits DT on the simple cubic lattice starting from one doubly infected seed. The time course of $n(t)$ in Fig. S9 however suggests a typical CT in the OP universality class, if two single diseases start at two different neighbouring sites. Moreover, the critical point is now clearly shifted to a lower value $p_c \approx 0.19205$ in Fig. 4b. A more systematic study shows that the significant difference in the transition order is related to the bipartivity of the lattice itself. Consider the simple cubic lattice as a 3d checkerboard: a site $x = (x, y, z)$ is “white” if $x + y + z$ is even, and “black” if odd. We found that the transition is always discontinuous as in Fig. 4b, if all seed sites have the same colour, while it is always continuous as in Fig. S9, if the seed contains sites with both colours (29). Heuristically, the difference is attributed to the fact that starting from two neighbouring seeds, one disease would closely follow the other in the neighbourhood with probability $q$. Hence, the effect of cascaded infections via multiple pathways as shown in Fig. 1b would be largely suppressed, i.e., no avalanche outbreak would occur after the rejoicing of pathways.

2. Dependence on lattice topology

The transition on this dimension shows dependence also on the lattice topology. This is demonstrated by two other regular and translation-invariant networks obtained by connecting points of $Z^3$ by short-range links. In the first example, any site $x$ is linked to 14 neighbors $x + e$, where $e \in \{(\pm 1, \pm 1, \pm 1), (\pm 2, 0, 0), (0, \pm 2, 0), (0, 0, \pm 2)\}$. Plot of $n(t)$ versus $t$ at several $p$ values on this lattice is shown in Fig. S10a, which suggests that the percolation transition is continuous and in the OP universality class. In contrast, another network, on which each site $x$ has 12 neighbors $x + e$ and $x + 2e$, where $e \in \{(\pm 1, 0, 0), (0, \pm 1, 0), (0, 0, \pm 1)\}$, again exhibits DT by showing the concaved $n(t)$ curves in Fig. S10b, similar to the case for the simple cubic lattice.

The dependence of dynamical behaviour in this dimension may be on more factors, not limited to intrinsic latency, seed type and lattice topology. But all exhibited sensitivity clearly demonstrates that 3d is a critical dimension.

The three-dimensional lattice has its empirical relevance. It is noteworthy that while the spread of infectious pathogens in the two-dimensional space is ubiquitous at the macroscopic level, it occurs primarily in three dimensions when
microenvironments are concerned, e.g., host-pathogen interactions in tissue milieus [34], microbial plant pathogens (fungi, bacteria, or oomycetes) in bulk organisms [35]. The 3d lattice sites correspond to discrete nutrient sources, comprising susceptible roots or discrete fragments of organic matter. The entire lattice can also represent the extracellular or soil matrix [35]. Co-infections are commonly observed on this scale, for example, the invasion of Rhizoctonia with one of other pathogens (Pythium, Fusarium or Meloidogyne incognita) [36,37].

D. General case for $q < 1$

So far we presented for each network topology only data for one value of $q$. But we made extensive checks which showed that the behavior is qualitatively the same for all $q > p$. We present in Fig. S11 a family of plots for the densities at $p_c(q)$ versus $q$ on the simple cubic lattice simulated with SU. We can observe that the discontinuity vanishes when $q \to 0.24881$, which is the threshold for ordinary bond percolation in 3d. The fact that the critical densities can become arbitrarily small for small $q$ means that the compact cluster of infected sites is in fact “spongy” in the sense that it has voids of arbitrarily large size (larger than those shown in Fig. 4c).

S4. ON SMALL-WORLD AND SCALE-FREE TOPOLOGIES

Many empirical graphs are well modeled by small-world networks or scale-free networks. Small-world networks are an ensemble of networks in which the geodesic (shortest path) distance between pairs of nodes grows no faster than logarithmically as the network size $N$ tends to infinity [2]. A family of representative networks are formulated by Watts and Strogatz (WS networks) [19]. Nodes on these topologies are mediated by randomly placed shortcuts, which facilitate propagations of epidemics beyond the locality. In cooperative contagions, the existence of non-local links may substantially change the epidemic scenario. A scale-free network, having a power-law degree distribution, also possesses the small-world property. But due to the presence of hubs, the entire network is more vulnerable than any homogeneous network, in that one faces virtually instantaneous rise of epidemic incidence at an almost-null threshold [12]. Our simulation shows that the scenario remains essentially the same for cooperative contagions.

A. Newman-Watts networks

We consider the small-world topologies derived from 2d lattices. On this dimension, the Newman-Watts networks are more often adopted [20], which are better behaved than WS networks, such as the exclusion of detached part. On a regular $d$-lattice, we add shortcuts between pairs of nodes chosen uniformly at random (instead of rewiring bonds for a WS network). A shortcut is added with probability $\phi$ corresponding to each bond on the original lattice (without removing it), so that there are $dN\phi$ shortcuts on average. The average coordination number is thus $z = 2d(1 + \phi)$. For sufficient randomness, we generate a new network for every 100 realisations. At the beginning of each simulation run, we place a doubly infected seed on a randomly selected node.

Figure S12 shows that the order of phase transition changes from CT to DT (simulated with AU), as the shortcut-adding probability $\phi$ increases. A tricritical point $\phi^*$ is secured between $\phi = 0.010$ and 0.012 for the size simulated ($N = 2^{22}$). This suggests that a sufficiently large proportion of random links may lead to an abrupt massive outbreak. For all DTs close to $\phi^*$, in particular, a jump occurs between two opposite branches (Figure S12b). The snapshots in Fig. S13 show different patterns of infection in the lower and upper branches at the transition point. The connectivity of infected regions is substantially raised if the isolated islands of singly infected nodes are able to reach each other (although with a very low probability) and cause cascaded mutual infections. No such tricriticality is however observed for SU. Down to $\phi = 10^{-3}$, the system exhibits DTs solely. The simulation results suggest either the nonexistence of tricriticality or CTs in a further lower regime, which is only detectable for a sufficiently large network size with an abundance of shortcuts.

B. Barabási-Albert networks

We simulated Barabási-Albert (BA) networks as typical scale-free networks. They are constructed by preferential attachment from a small random network as the initial core, which means that an existing node with degree $k_i$ acquires a link with probability $P(k_i) = k_i/\sum_j k_j$ [21]. The generated networks follow a power-law degree distribution with exponent $\gamma = -3$. At the initial of each simulation run, a doubly infected seed is placed on a randomly selected node. We also generate a new network for every 100 realisations to guarantee sufficient randomness. The system
exhibits clearly a CT in forming either coinfect clusters (branch II) or singly infected clusters (branch I), having both thresholds close to zero (see Fig. S14). The finite threshold seen here is caused by finite size effect (simulated at $N = 2^{24}$), which has truncated the degree distribution at some maximum [12]. The reason lies simply in the fact that both diseases fail to expand their pathways for sufficiently long distances from the hubs, being at the vanishing threshold. It has thus excluded the existence of any bottleneck for the uprise of DT.

**BIBLIOGRAPHY**


FIG. S1. Composition of infected clusters on Erdős-Rényi networks with the mean degree $<k> = 4$ and $q = 0.99$, simulated at the size $N = 2^{24}$ (with AU). (a) Fractions of total infected nodes $\rho_{\text{tot}}$; (b) fractions of doubly infected nodes $\rho_{ab}$ and of singly infected nodes $\rho_a$ (inset). Each point corresponds to a value obtained from one simulation run. The lower continuous branch I is the superposition of branches I-a and I-b (data not shown for being symmetric) of singly infected nodes. The upper discontinuous branch II is the sum of II-ab of doubly infected nodes and two branches II-a and II-b of singly infected nodes.

FIG. S2. Probabilities of reaching a giant doubly infected cluster on Erdős-Rényi networks with the mean degree $<k> = 4$ for sizes $N = 2^{14}$ to $2^{25}$ (simulated with AU), plotted in a rescaled coordinate. The data collapse agrees with the standard finite size scaling ansatz.
FIG. S3. Giant doubly infected clusters on Erdős-Rényi networks obtained with multiple seed nodes at \( p = 0.20 \) and \( q = 1.0 \). Main plot: probabilities \( P_{ab} \) versus seed density. Each curve corresponds to one value of \( N \), with \( N = 2^{20}, 2^{21}, \ldots, 2^{25} \) (steeper curves correspond to larger \( N \)). Inset: data collapse in a rescaled coordinate, obtained by plotting the data against \( (n/N - \rho_0)N^{0.5} \) with \( \rho_0 = 0.000282 \) and \( x = 0.5 \).

FIG. S4. Plot for probabilities \( P_{ab} \) of forming giant doubly infected clusters versus \( N \) on Erdős-Rényi networks (simulated with AU). Each curve corresponds to a different value of \( q \), while \( p \) is fixed at \( p_c \). The two straight lines indicate power laws \( N^{-0.12} \) and \( N^{-0.67} \).
FIG. S5. Mass distributions of doubly infected clusters on 2d lattices (simulated with AU) at the critical point for $q = 0.9084$. Lattice sizes are $L \times L$ with $L = 2^{10}, ..., 2^{13}$. (a) The straight line indicates the scaling $P(m) \sim m^{1-\tau}$ with $\tau = 187/91$ that holds for ordinary percolation. (b) Data collapse in a rescaled coordinate, $P(m) m^{\tau-1}$ versus $m/L^d$, with $d_f = 1.896$ as the fractal dimension in the universality class of ordinary percolation on the 2d lattice.

FIG. S6. Data for 4d lattices with $q = 1.0$, using all sites of an entire hyperplane $z = 0$ as seeds. The infection is constrained to grow into the region $z > 0$ (simulated with AU). (a) Log-log plot of $\rho(t) = n(t)/L^3$ versus $t$, where $n(t)$ is the number of infected sites at time $t$. The central curve for $p = p_c \approx 0.111147$ follows a power law $\rho(t) \sim t^{-\delta}$, with $\delta = 0.45(2)$. (b) Densities of doubly infected sites at height $z$, after the growth of the cluster has either stopped (for $p < p_c$) or forever passed this height (for $p > p_c$).
FIG. S7. Plot of $P_{ab}$ after rescaling, the logarithm of the probability for forming a giant cluster from a doubly infected site, versus $1/(p - p_c)$ with $p_c = 0.111857(2)$ and $q = 1.0$ (simulated with AU). The data suggest a perfect fitting for $P_{ab} \sim \exp(-a/(p - p_c))$ with $a \approx 0.0064$, except for the points that are subject to some scaling correction when $p - p_c > 0.005$. 

FIG. S8. Plot of $P_{ab}$ versus $p - p_c$ on a 3d simple cubic lattice, starting from a doubly infected seed (simulated with SU), at $q = 0.99$. The straight line indicates that the data are well fitted by a power law, suggesting a continuous transition of finite order.
FIG. S7. Plot of $P$ with order $q$. The straight line indicates that the data are well fitted by a power law, suggesting a continuous transition of finite order.

FIG. S9. Plot of the number of newly infected sites $n(t)$ versus $t$ (simulated with SU), analogous to Fig. 4b, but starting from two neighbouring sites on a simple cubic lattice: while $A$ starts at $(0, 0, 0)$, $B$ starts at $(1, 0, 0)$. The solid straight line represents the scaling for ordinary percolation on 3d lattices: $n(t) \sim t^{0.494}$.

FIG. S10. Different types of transitions on 3d lattices for different lattice topologies. Both panels show log-log plots of $n(t)$, starting from a doubly infected seed sites with $q = 1$ (simulated with SU): (a) for a lattice with 14 neighbours; (b) for another lattice with 12 neighbours (see definitions in the text).
FIG. S11. Densities at the critical point on a simple cubic lattice (simulated with SU). These densities vanish at \( q = 0.24881 \) (which is the critical point of bond percolation on a simple cubic lattice), but are strictly positive for all larger \( q \).

FIG. S12. Phase transitions by the fraction of doubly infected nodes \( \rho_{ab} \) on 2d Newman-Watts networks with \( q = 0.99 \) at the size \( N = 2^{24} \) (simulated with AU). Each point corresponds to a value obtained from one simulation run. The order of transition evolves from CT to DT with the increasing shortcut-adding probability \( \phi \). The typical values are chosen at \( \phi = 0.01, 0.012, 0.014 \) and 0.1.
FIG. S13. Different profiles of infected clusters at the same \( p \) on a 2d Newman-Watts network at the end of the simulations, with \( \phi = 0.014 \) (DT). The 1000 \( \times \) 1000 snapshots are extracted from a network with \( N = 2^{24} \). The \( p \) value (= 0.355) is chosen at the position where both upper and lower branches coexist (see Fig. S12c). Different levels of connectedness are shown among the infected regions.

FIG. S14. Continuous transition on Barabási-Albert networks, simulated at the size \( N = 2^{24} \). (a) Fractions of total infected nodes \( \rho_{\text{tot}} \); (b) fractions of doubly infected nodes \( \rho_{ab} \) and of singly infected nodes \( \rho_a \) (inset). Each point corresponds to a value obtained from one simulation run. Both the lower branch I (sum of I-a and I-b) and upper branch II (sum of II-ab, II-a and II-b) undergo a continuous transition.