Supplementary Information
Infection percolation:
A dynamic network model for disease spreading

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Size dependence. Our simulations occur on a fixed finite size network. All simulations presented are conducted on networks with $N = 10^4$ nodes. We verify that our results are not considerably influenced by finite-size effects by repeating simulations for five network sizes. Figure S1 shows that the critical growth of the total infected fraction ϕ_t above $\tilde{P}^* > 0.5$, corresponding to Fig. 1d in the main text, is insensitive the system size, even when the number of nodes is increased by two orders of magnitude ($N = 10^4$ to 10^6). We hence anticipate our system size $N = 10^4$ to be sufficiently large to capture the general dynamics of this spread.

Estimate of characteristic disease spreading timescales. For the case of a recovery-free population, we calculate the shortest possible time $\tilde{\tau}_{f,0}$ at which the infected fraction plateaus in the limit of high disease infectivity $\tilde{P}^*$. As time progresses, the disease spreads radially outward. Because we consider a square network comprising N_t nodes in total, $\sqrt{N_t}$ on a side, the leading edge of the circular infected region first reaches the boundary of the population when $\tilde{\tau} \approx \sqrt{N_t}/2$. However, the total infected fraction can continue to grow: it only plateaus when it spans the entire 2D network, including its corners. This occurs when the radius of the infected region is equal to half the diagonal of the square network, $\tilde{\tau}_{f,0} \approx (\sqrt{N_t}/2) \times \sqrt{2} = \sqrt{N_t/2}$.

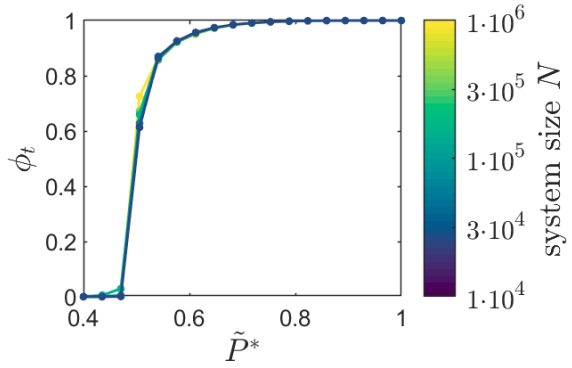

FIG. 1. Critical growth of the total infected fraction ϕ_t above a critical infectivity $\tilde{P}^* > 0.5$ for a recovery-free population. Network size is varied logarithmically from $N = 10^4$ to 10^6 with no discernible difference (some curves lie beneath $N = 10^4$).

For the case of a population with recovery duration $\tilde{\tau}_r$, we extend this calculation to estimate the time at which the infected fraction of the population will peak, $\tilde{\tau}_p$, as well as the time at which the infected fraction of the population reaches zero after all individuals recover, $\tilde{\tau}_f$, again in the limit of high disease infectivity. As time progresses, the disease spreads radially outward in a circular infected region, followed by an inner circular region of recovery that spreads at the same rate but is delayed by $\tilde{\tau}_r$. We consider two separate regimes: the "thin pulse" regime with $\tilde{\tau}_r < \sqrt{N_t}(1/\sqrt{2}-1/2)$, and the "thick pulse" regime with $\tilde{\tau}_r > \sqrt{N_t}(1/\sqrt{2}-1/2)$.

For a thin pulse, as in the recovery-free case, the leading edge of the infected region first reaches the boundary of the population when $\tilde{\tau} \approx \sqrt{N_t}/2$ (Fig. 2a). At this time, the total infected fraction is nearly maximal, and we therefore approximate $\tilde{\tau}_p \approx \sqrt{N_t}/2$. As time progresses, the leading edge of the region of recovery then first reaches the boundary of the population at a time $\tilde{\tau} \approx \sqrt{N_t}/2 + \tilde{\tau}_r$ (Fig. 2b). Both regions continue to spread into the corners of the square boundary, and the leading edge of the infected region eventually reaches the corners at a time $\tilde{\tau} \approx \sqrt{N_t/2}$ as in the recovery-free case (Fig. 2c). Subsequently, the region of recovery continues to grow; the total infected fraction continues to decrease, eventually reaching zero when the region of recovery has reached the corners of the square boundary, $\tilde{\tau}_f \approx \sqrt{N_t/2} + \tilde{\tau}_r$.

For a thick pulse, the leading edge of the infected region again first reaches the boundary of the population when $\tilde{\tau} \approx \sqrt{N_t}/2$ (Fig. 2d). The leading edge of the infected region then reaches the corners of the square boundary at a time $\tilde{\tau} \approx \sqrt{N_t/2}$ as in the recovery-free case (Fig. 2e). Thus, the time at which the infected fraction is maximal is between these two times: $\sqrt{N_t}/2 \lesssim \tilde{\tau}_p \lesssim \sqrt{N_t/2}$. As time progresses, the region of recovery then continues to grow, eventually first reaching the boundary of the population at $\tilde{\tau} \approx \sqrt{N_t}/2 + \tilde{\tau}_r$ (Fig. 2f). Subsequently, the region of recovery continues to grow; the total infected fraction continues to decrease, eventually reaching zero when the region of recovery has reached the corners of the square boundary, $\tilde{\tau}_f \approx \sqrt{N_t/2} + \tilde{\tau}_r$.

For our simulations with $N_t = 10^4$, the transition between the thin and thick pulse regimes occurs at $\tilde{\tau}_r \approx 21$;

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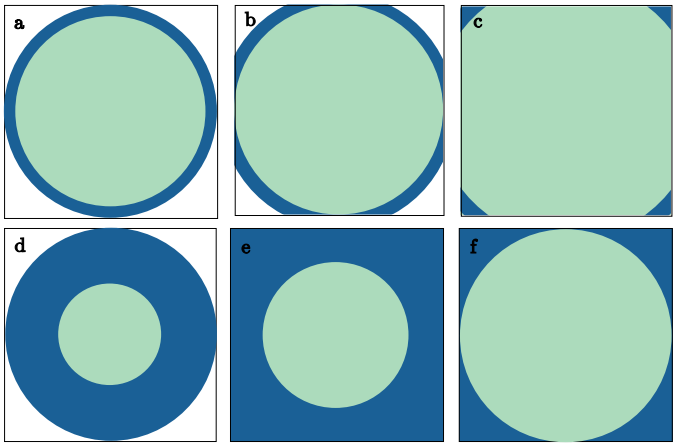

FIG. 2. Schematics showing the growth of the regions of infection (dark blue) and recovery (light green) over time. Top row shows the thin pulse regime with low $\tilde{\tau}_r$ while bottom row shows the thick pulse regime with high $\tilde{\tau}_r$. In the thin pulse regime, **(a)** the leading edge of the infected population reaches the boundary first, **(b)** followed by the leading edge of the recovered population, **(c)** followed by the leading edge of the region of infection circumscribing the entire population. However, in the thick pulse regime, **(d)** while the leading edge of the infected population again reaches the boundary first, **(e)** the region of infection reaches the corners of the square lattice before **(g)** the leading edge of the recovered population reaches the boundary.

therefore, our analysis of the example system with $\tilde{\tau}_r = 4$ presented in the main text is in the thin pulse regime, with $\tilde{\tau}_p \approx \sqrt{N_t}/2 \approx 50$ and $\tilde{\tau}_f \approx \sqrt{N_t/2} + \tilde{\tau}_r \approx 75$ as reported in the main text. Together with Eq. 2, these estimates provide a universal scaling for the peak infection time, $\phi_p = \phi(\tilde{\tau}_p)$ (Figs. 3d-f insets).

SUPPORTING MOVIE CAPTIONS

Movie S1. Sequence of infection for a disease with low infectivity $\tilde{P}^* = 0.3$, showing that disease spreading is quickly localized. This simulation is without recovery.

Movie S2. Sequence of infection for a disease with intermediate infectivity $\tilde{P}^* = 0.6$, showing that the disease spreads in a spatially heterogeneous, ramified pattern, leading to the formation of discrete clusters of bypassed individuals who remain uninfected. Infected individuals are shown in dark blue, uninfected susceptible individuals are shown in white. This simulation is without recovery.

Movie S3. Sequence of infection for a disease with higher infectivity $\tilde{P}^* = 0.7$, showing that the disease spreads in a more compact pattern, with a smoother leading edge, leading to the formation of fewer and smaller clusters of bypassed individuals. Infected individuals are shown in dark blue, uninfected susceptible individuals are shown in white. This simulation is without recovery.

Movie S4. Sequence of infection for a disease with intermediate infectivity $\tilde{P}^* = 0.6$, showing that recovery causes disease spreading to be quickly localized. Infected individuals are shown in dark blue, recovered individuals are shown in light green, uninfected susceptible individuals are shown in white. This simulation is with $\tilde{\tau}_r = 4$.

Movie S5. Sequence of infection for a disease with higher infectivity $\tilde{P}^* = 0.7$, showing that the disease spreads continually, but recovery causes the disease to spread in a spatially heterogeneous, ramified pattern, leading to the formation of discrete clusters of bypassed individuals who remain uninfected. Infected individuals are shown in dark blue, recovered individuals are shown in light green, uninfected susceptible individuals are shown in white. This simulation is with $\tilde{\tau}_r = 4$.