# Epidemics with asymptomatic transmission: Subcritical phase from recursive contact tracing

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The challenges presented by the COVID-19 epidemic have created a renewed interest in the development of new methods to combat infectious diseases, and it has shown the importance of preparedness for possible future diseases. A prominent property of the SARS-CoV-2 transmission is the significant fraction of asymptomatic transmission. This may influence the effectiveness of the standard contact tracing procedure for quarantining potentially infected individuals. However, the effects of asymptomatic transmission on the epidemic threshold of epidemic spreading on networks have rarely been studied explicitly. Here we study the critical percolation transition for an arbitrary disease with a nonzero asymptomatic rate in a simple epidemic network model in the presence of a recursive contact tracing algorithm for instant quarantining. We find that, above a certain fraction of asymptomatic transmission, standard contact tracing loses its ability to suppress spreading below the epidemic threshold. However, we also find that recursive contact tracing opens a possibility to contain epidemics with a large fraction of asymptomatic or presymptomatic transmission. In particular, we calculate the required fraction of network nodes participating in the contact tracing for networks with arbitrary degree distributions and for varying recursion depths and discuss the influence of recursion depth and asymptomatic rate on the epidemic percolation phase transition. We anticipate recursive contact tracing to provide a basis for digital, app-based contact tracing tools that extend the efficiency of contact tracing to diseases with a large fraction of asymptomatic transmission.

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## I. INTRODUCTION

The methods used to fight the spread of the contemporary COVID-19 epidemic in its initial phase have largely been the same as 100 years ago during the Spanish flu [1,2]. In particular, contact tracing has been used as a standard procedure that is well understood, both analytically and in network modeling approaches [3–8]. Some early papers even already considered the concept of recursive contact tracing, i.e., not only tracing direct contacts but also contacts of contacts and so on [9,10].

However, the arrival of the SARS-CoV-2 epidemic, with its high asymptomatic transmission rate and the possibility of presymptomatic infections, presents new challenges that need addressing [11–14]. As such, a renewed interest in recursive contact tracing [15–21], as well as in digital contact tracing solutions [22–34] that could enable instantaneous recursive contact tracing, has emerged in an effort to surpass the methods of 100 years ago.

In this article, we introduce a simple model that considers an epidemic as a percolation problem, as is common in network epidemiology theory [35–44], in combination with a recursive contact tracing algorithm operating on the model. Throughout this paper, we assume this algorithm to be facilitated by a digital contact tracing app which enables contact tracing and quarantining to happen effectively instantly; however, similar results could be achieved using recursive manual contact tracing, provided that the time necessary to trace contacts is small compared to the time between a person being infected and being infectious themselves, and that the recursion depth is sufficiently small. Note that the model operates in the theoretical limit of an ideal world without reporting, communication, or quarantining delays and without noncompliance with quarantining instructions, and we aim not to make quantitative predictions but to create a base model to further theoretical understanding. We thus do not explicitly model the SARS-CoV-2 virus, but an arbitrary virus with finite asymptomatic rate. We will study the efficacy of recursive contact tracing and characterize the influence of a possible future disease's asymptomatic transmission rate on the model's critical transition. Our model allows for arbitrary instantaneous recursion depths, as has been done only in [16], and our results, to the best of our knowledge, are the first to discuss the relationship of recursion depth and asymptomatic infection rate with regard to the critical transition.

We find a critical value in the fraction of nodes participating in the contact tracing (corresponding to tracing app usage) which depends on the asymptomatic transmission rate of the disease. Further we find a critical (maximum allowed) asymptomatic transmission rate as a function of the algorithm's recursion depth. We show that any disease with arbitrary basic reproduction number and finite asymptomatic rate can be stopped by a sufficiently large recursion depth. Finally, we validate our calculations using simulations on infection trees and networks with different degree distributions, as degree distribution can have a significant impact on an epidemic [38,40,42,44–48]. Let us now start by defining the model.

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## II. THEORY

We consider an SIR (susceptible, infected, removed) model with $N$ nodes and an arbitrary degree distribution $p(k)$ in which a proportion $\Phi$ of nodes take part in contact tracing ("use a contact tracing app"). Nodes in the network are infected with a virus with symptomatic rate $\Theta$ and basic reproduction number $R_0$. It is known that in such a network, if we fix $R_0$, the disease has a transmissibility

$$T = R_0 \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle} \tag{1}$$

[40]. Carriers of the disease will be able to infect their susceptible neighbors with probability $T$ one time step after being infected themselves and be immune and noncontagious afterwards.

If an infectious agent is symptomatic and uses the contact tracing app, this will trigger an alarm on the app and warn neighboring nodes of the chance of being infected, sending them into quarantine for their one infectious time step and removing them from quarantine afterwards so they effectively skip the infectious state and jump directly to the recovered stage. An infectious, symptomatic node will, however, have the chance to infect its neighboring nodes before triggering an alarm, which can be interpreted as a presymptomatic period or a testing delay.

We can consider higher degrees of recursivity $r$ for the app, meaning how many time steps in the past the app will consider to guess who might currently be infected. For $r = 0$, only the node's direct neighbors are sent into quarantine. For $r = 1$ in addition to those nodes that are quarantined for $r = 0$, any node with a distance of exactly three to the symptomatic node is quarantined, for $r = 2$ any node with a distance of five is quarantined, and so on. This is illustrated in Fig. 1. The algorithm disregards any possible immunities due to nodes having already been infected previously, but it does consider breaks in the infection chain that are caused by the app's own quarantining algorithm, i.e., if a node was quarantined at time $t$, the app does not consider this node a possible infection spreader at that time step. We disregard possible immunities, although they are present in the underlying infection model because, for a new disease, the exact nature of immunity due to previous infection would likely not be immediately known, and it would thus be prudent to err on the side of caution and not assume immunity. Also note that it is nontrivial to determine which nodes' immunities would be known to the app (certainly the ones of nodes triggering alarms, but for other nodes it is unclear).

Given a vector $\vec{S}$ of symptomatically infected nodes at time $t_0$,

$$S_i = \begin{cases} 1 & \text{if node } i \text{ is symptomatically infected} \\ 0 & \text{otherwise} \end{cases},$$

the vector of nodes $\vec{U}$ using the app, the vectors $\vec{Q}(t)$ of quarantined nodes and $\vec{P}(t)$ of not quarantined nodes at time steps $t \leqslant t_0$, and the adjacency matrix $A$, the vector of quarantined nodes at $t = t_0 + 1$ can be calculated by

$$\vec{Q}(t_0+1) = \underbrace{\{A \cdot [\vec{S} \cdot \vec{U} \cdot \vec{P}(t_0)]\} \cdot \vec{P}(t_0) \cdot \vec{U}}_{r=0} + \underbrace{[A \cdot (A \cdot \{[A \cdot (\vec{S} \cdot \vec{U})] \cdot \vec{P}(t_0-1) \cdot \vec{P}(t_0-2) \cdot \vec{U}\}) \cdot \vec{P}(t_0-1) \cdot \vec{P}(t_0) \cdot \vec{U}] \cdot \vec{P}(t_0) \cdot \vec{U}}_{r=1} + \underbrace{\ldots}_{r>1}.$$

Multiplications with $\vec{P}(\cdot)$ ensure that a considered node in the backtracking chain was quarantined neither at its supposed time of infection nor at the time it could have infected its neighbors, and multiplications with $\vec{U}$ ensure that all nodes in the backtracking chain use the app. We now calculate the probability that an infected node is correctly put into quarantine by our algorithm. For this, we assume an infinitely large network, with a finite number of nodes being infected. In a network in which a finite fraction of nodes is infected, it is of course possible that a node will be in contact with multiple infected nodes in a single time step. With our assumption of the fraction of infected nodes being infinitely small, barring any nontrivial network structure, the chance of a node being in contact with more than one infected node also becomes infinitely small. We assume that the clustering in the network is negligible so that we can consider the infection chain effectively as a tree.

For $r = 0$, both the infected node and the infecting node must be part of the network and the infecting node needs to be symptomatic. Therefore, a first approximation of the probability $P_q^{r=0}$ of an infected node $i$ being correctly put into quarantine is simply

$$P_q^{r=0}(\Phi, \Theta) = \Phi^2 \Theta. \tag{2}$$

However, node $i$ has to have been infected by a different node $j$. For this infecting node $j$ to have been infectious in the previous time step, it cannot have been quarantined in that time step. There are two possible reasons why node $j$ would not have been quarantined despite being infected. Either it is not using the app, which happens with a probability $1 - \Phi$, or it is using the app, which has a probability of $\Phi$, but the algorithm did not quarantine it in the time step in which it was infectious, which happens, for a node using the app, with a probability $1 - \frac{P_q^r}{\Phi}$. Therefore, the node $j$'s probability of using the app, with the observation that it has not been quarantined despite being infectious, is

$$\Phi' = \frac{\Phi\left(1 - \frac{P_q^r}{\Phi}\right)}{\Phi\left(1 - \frac{P_q^r}{\Phi}\right) + (1 - \Phi)} = \frac{\Phi - P_q^r}{1 - P_q^r} \leqslant \Phi,$$

as the amount of nodes using the app with the ability to infect other nodes is reduced by a factor $(1 - P_q^{r=0})$, resulting in the

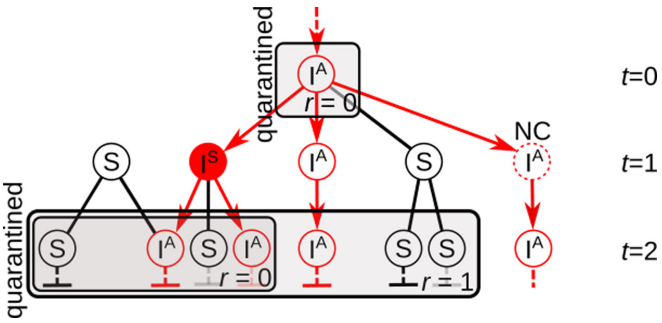


FIG. 1. Illustration of the quarantining algorithm with an infection spreading from top to bottom. Nodes with a black outline are not infected (susceptible = S), nodes with a red outline are infected (I), either symptomatically (filled nodes, $I^S$) or asymptomatically (unfilled nodes, $I^A$), and nodes with a dashed outline are not using the contact tracing app (noncompliant, NC). Red arrows indicate the spread of the infection, while black lines indicate noninfectious connections between nodes. The time $t$ indicated on the right-hand side marks the time at which infected nodes are infectious—or, in the case of the uninfected nodes, the latest time at which the app would consider them to be possibly infectious. While nodes could reappear in later time steps, e.g., the node in the $t = 0$ row could also be shown in the $t = 2$ row as it is connected to (most of) the nodes in the $t = 1$ row, we show nodes only once for visual clarity. At time $t = 1$ a symptomatic node triggers an alarm on the app. For recursion depth $r = 0$, only its nearest neighbors are quarantined. These quarantined nodes cannot infect any other nodes, as indicated by the blocked outgoing connections. For $r = 1$, the app considers every nearest neighbor of the symptomatic node as a possible origin of the symptomatic node's infection and therefore quarantines all nodes that the infection could have spread to within two time steps from these nearest neighbors. This results in every node with a distance of exactly three to the symptomatic node being quarantined, so long as the connection is not interrupted by a node not using the app or by a node that was in quarantine itself at its time of infection or in the time step after infection, as shown on the right-hand side. This can, of course, also include nodes which have not yet actually been in contact with any infected nodes, as shown by the leftmost nodes in the $t = 1$ and $t = 2$ rows. Note that, although the infection chain is shown in a treelike structure for visual clarity, these nodes can be part of a network of arbitrary structure, so that two nodes might be connected via multiple different paths and therefore also have multiple possible distances to each other.

numerator, which is normalized by the total fraction of nodes that are not being quarantined, which is the denominator; and therefore

$$P_q^{r=0} = \Phi\Phi'\Theta. \tag{3}$$

For higher degrees of recursion, the chance of being quarantined is increased:

$$P_q^{r>0} = P_q^{r=0} + \underbrace{\left(1 - P_q^{r=0}\right)\Phi'' P_1}_{\text{r=1}} + \underbrace{\cdots}_{r>1} \tag{4}$$

$$= \Phi\Phi'[P_0 + (1 - P_0)\Phi''\{P_1 + (1 - P_1)\Phi''(\cdots)\}] \tag{5}$$

$$\text{with } P_0 = \Theta. \tag{6}$$

Here, in every part of the sum, the chance of a node having already been quarantined due to a lower recursion level is excluded via $(1 - P_i)$, and a factor $\Phi''$ is added for the chance of the next upstream node using the app. The factor $\Phi''$ represents the chance of a node using the app if the next downstream node has not been quarantined, and needs to be used for nodes that are two or more levels above the currently regarded node in the infection tree. The chance of such a node using the app regardless of the behavior of its downstream nodes is $\Phi'$. The chance of a downstream node, which is using the app, of a node that is also using the app not being quarantined is approximately $(1 - \frac{P_q^r}{\Phi\Phi'})$. Since we assume both infecting node and infected node to be using the app, the factor $\Phi\Phi'$ is removed from $P_q^r$. This approximation disregards that the upstream node not being quarantined also influences the chance of its downstream node being quarantined. Then the chance of an upstream node using the app, given that its downstream node is using the app and has not been quarantined is

$$\Phi'' = \frac{\Phi'\left(1 - \frac{P_q^r}{\Phi\Phi'}\right)}{\Phi'\left(1 - \frac{P_q^r}{\Phi\Phi'}\right) + (1 - \Phi')} \tag{7}$$

$$= \frac{\Phi\Phi' - P_q^r}{\Phi - P_q^r}. \tag{8}$$

Next, we need to calculate the chance $P_i$ of a node being quarantined due to the $i$th recursion step, given that its $r$ nearest upstream nodes are using the app. For simplicity's sake, we start with $P_1$. Here a leaf node $i$ is quarantined due to the first recursion step if any of the downstream nodes of $i$'s second degree upstream node, which we call $j$, have been infected, use the app, and are symptomatic. The chance of one node fulfilling these conditions is $\Phi'\Theta T$. Since just one node needs to cause an alarm on the app, the chance of being quarantined is

$$P_1 = 1 - (1 - \Phi\Theta T)^n, \tag{9}$$

where $n$ is the average number of $j$'s downstream nodes minus one. We subtract one, since one of $j$'s downstream nodes is $i$'s direct upstream node and would already have caused $i$ to be quarantined in the zeroth recursion step, if it were symptomatic. Since the chance of a node of degree $k$ being infected is proportional to $kp(k)$ [46], the average number of downstream nodes minus one is

$$n = \frac{\sum_{k=2}^{\infty} k(k-2)p(k)}{\sum_{k=2}^{\infty} kp(k)}, \tag{10}$$

where we subtract two from $k$ because of the one downstream node that is not considered and $j$'s upstream node. Therefore,

$$P_1 = 1 - (1 - \Theta\Phi T)^{\frac{\sum_{k=2}^{\infty} k(k-2)p(k)}{\sum_{k=2}^{\infty} kp(k)}} \tag{11}$$

$$= P_1(x)|_{x=2} = 1 - (1 - \Theta\Phi T)^{\frac{\sum_{k=x}^{\infty} k(k-x)p(k)}{\sum_{k=x}^{\infty} kp(k)}}\Big|_{x=2}. \tag{12}$$

We indicate how many connections are removed when calculating $n$ via the variable $x$.

For the second recursion step, at least one of the downstream nodes of $j$'s upstream node, which we call $l$, must fulfill the condition of $P_1$, meaning that at least one of their downstream nodes must be infected, using the app, and

symptomatic. This chance is given by

$$P_2 = 1 - [1 - P_1(1)\tilde{\Phi}]^{\frac{\sum_{k=2}^{\infty} k(k-2)p(k)}{\sum_{k=2}^{\infty} kp(k)}} \tag{13}$$

$$= P_2(x)|_{x=2} = 1 - [1 - P_1(1)\tilde{\Phi}]^{\frac{\sum_{k=x}^{\infty} k(k-x)p(k)}{\sum_{k=x}^{\infty} kp(k)}}\Big|_{x=2} \tag{14}$$

$$\text{with } \tilde{\Phi} = \frac{\Phi T(1-\Theta)}{\Phi T(1-\Theta) + (1-\Phi T)}. \tag{15}$$

Here, in $P_1(x)$, we do not discount one of each node's downstream nodes, since these nodes are not upstream nodes of node $i$, and therefore all of their downstream nodes need to be considered. Thus, we use $P_1(1)$ instead of $P_1(2)$. Also, we use $\tilde{\Phi}$, because nodes that are using the app and symptomatically infected would have already caused a quarantine in a previous time step and can therefore not be part of the considered tree. Similarly, the equation for following recursion steps is

$$P_i(x) = 1 - [1 - P_{i-1}(1)\tilde{\Phi}]^{\frac{\sum_{k=x}^{\infty} k(k-x)p(k)}{\sum_{k=x}^{\infty} kp(k)}}. \tag{16}$$

Summarizing these calculations, the chance of a leaf node being quarantined with a recursion degree of $r$ is

$$P_q^r \approx \Phi\Phi' \sum_{i=0}^{r} \left( \left\{ \prod_{j=0}^{i-1} [1 - P_j(2)]\Phi'' \right\} P_i(2) \right) \tag{17}$$

$$\text{with } P_i(x) = \begin{cases} \Theta & \text{if } i = 0 \\ 1 - (1 - P_0(1)\Phi T)^{n(x)} & \text{if } i = 1 \\ 1 - (1 - P_{i-1}(1)\tilde{\Phi})^{n(x)} & \text{otherwise} \end{cases} \tag{18}$$

$$\text{and } n(x) = \frac{\sum_{k=x}^{\infty} k(k-x)p(k)}{\sum_{k=x}^{\infty} kp(k)}. \tag{19}$$

Note that (17) is a self-consistent equation, since $\Phi'$ and $\Phi''$ contain $P_q^r$.

## III. THEORETICAL RESULTS

It is easy to see that the upper limit of $P_q^r$ is

$$P_q^r \leqslant \Phi\Phi' < \Phi \text{ if } \Phi < 1, \tag{20}$$

so contact tracing by recursive backtracking is strictly worse than vaccinating a fraction $\Phi$ of the population. Since such a vaccination strategy is already insufficient to stop an epidemic on an infinitely large scale-free network with a degree distribution $p(k) \propto k^{-\gamma}$ with $\gamma \leqslant 3$ [46], recursive backtracking can also not stop such an epidemic for $\Phi < 1$.

However, there is still something that can be learned from taking a closer look at scale-free networks. For $\gamma \leqslant 3$, the sum $\sum_{k=2}^{k} k^2 p(k)$ in the exponent of the $P_i$'s diverges, therefore $P_1 \to 1$ (if $\Phi\Theta T > 0$), and $P_q^r$ becomes

$$P_q^r = \Phi\Phi'[\Theta + (1-\Theta)\Phi'']. \tag{21}$$

We can see that all infected nodes that can be caught by the algorithm will already be detected in the first recursion step.

Luckily, real-world networks are not infinitely large, so the sum mentioned previously will not diverge, so recursive backtracking will be able to stop epidemics for $\Phi < 1$. For such networks, we expect the observation made for infinitely large scale-free networks to be still be relevant, i.e., the closer a real-world network is to an infinitely large scale-free network, the less will the epidemic threshold $\Phi_c$ be affected by recursion depths past $r = 1$.

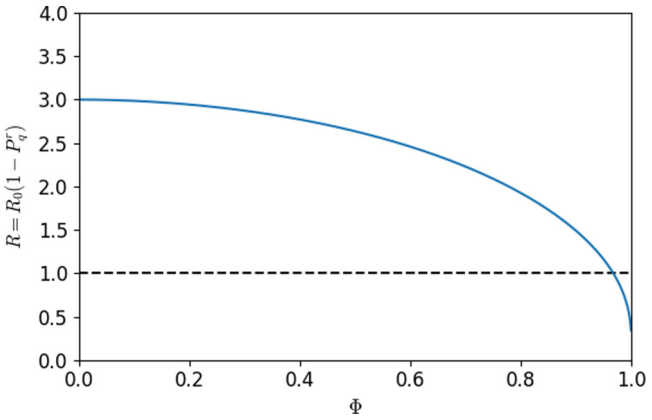


FIG. 2. Reduction of the reproduction number $R$ as a function of the app-usage rate $\Phi$ for a Barabási-Albert (BA) network with a cutoff $\kappa = 1000$, recursion depth $r = 1$, $R_0 = 3$, and $\Theta = 0.5$. The dashed line shows the critical value $R_0(1 - P_q^r) = 1$.

In Fig. 2 we show the reduction of the reproduction number $R = R_0(1 - P_q^r)$ as a function of $\Phi$ for a Barabási-Albert (BA) network with average degree $\langle k \rangle = 4$ and a cutoff at $\kappa = 1000$ and recursion depth $r = 1$. We also tested this for a simple Erdős-Rényi (ER) network with average degree $\langle k \rangle = 4$, a scale-free network with exponential cutoff $p(k) \propto k^{-2} \exp(\frac{k}{94.2})$ that produces an epidemic threshold comparable to that of urban networks for SARS [49] and higher recursion depths. Since all of the resulting graphs are nearly indistinguishable [except that $R(\Phi = 1) \to 0$ for $r \to \infty$ while $R(\Phi = 1) \neq 0$ for $r = 1$], we chose to show only the BA network. We can also calculate the critical value $\Phi_c$ as a function of the symptomatic rate $\Theta$, as is shown in Fig 3. There is a large visible difference between the classic contract tracing method with $r = 0$ and recursive contact tracing, even for relatively large values of $\Theta$. While for $r > 0$ the

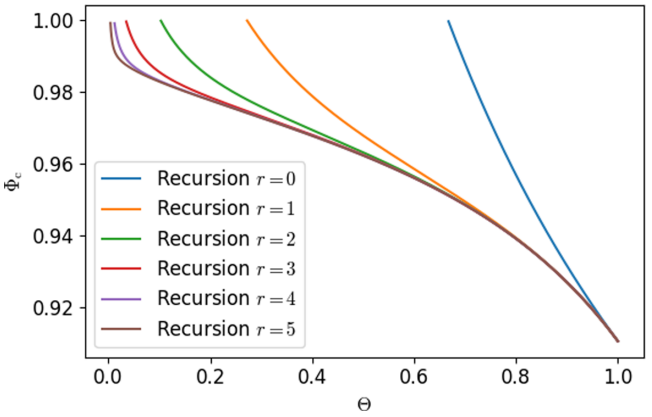


FIG. 3. Critical value $\Phi_c$ as a function of the symptomatic rate $\Theta$ for different recursion depths $r$ with $R_0 = 3$. Since the ER distribution and the scale-free distribution with an exponential cutoff again yield almost the same results, we plot $\Phi_c$ only for the Barabási-Albert distribution with average degree $\langle k \rangle = 4$ and cutoff $\kappa = 1000$.

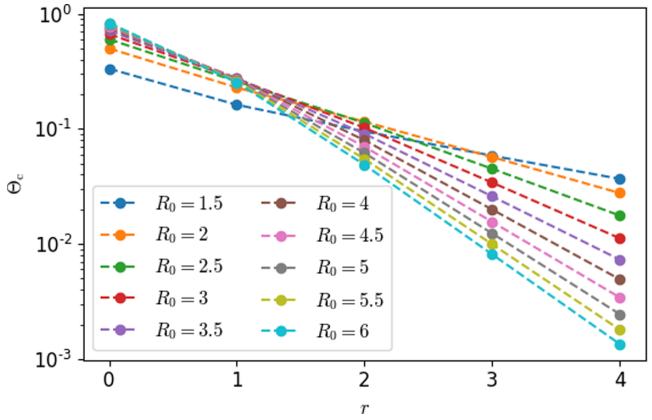


FIG. 4. Critical symptomatic rate $\Theta_c$ below which an epidemic cannot be stopped even for $\Phi = 1$ as a function of the recursion depth $r$ for different basic reproduction numbers $R_0$ using a Barabási-Albert distribution with average degree $\langle k \rangle = 4$ and cutoff $\kappa = 1000$. For large recursion depths, the critical value $\Theta_c \to 0$ for all basic reproduction numbers, whereas for $r = 1$ there is a maximum $\Theta_c^{\max} \approx 0.28$ at $R_0 \approx 3.6$.

recursion depth has little influence on $\Phi_c$ for large values of the symptomatic rate $\Theta$, we see that there is a critical value $\Theta_c$, depending on the recursion depth, below which, even with $\Phi = 1$, an epidemic cannot be stopped. This critical value is approximately halved when going from the classical method $r = 0$ to $r = 1$, meaning that recursive contact tracing is an effective method to combat diseases with high asymptomatic rates which would not have been able to be stopped by previous contact tracing methods.

The critical value $\Theta_c$ is shown in Fig. 4 as a function of the recursion depth for different values of $R_0$. The critical value $\Theta_c$ exponentially decreases with $r$, with $\Theta_c \to 0$ for $r \to \infty$. Therefore, any disease with a symptomatic rate $\Theta > 0$ and arbitrarily large basic reproduction number $R_0$ can be stopped via recursive contact tracing, given a sufficiently large recursion depth and app usage rate.

## IV. SIMULATIONS

To test the accuracy of our calculations in Sec. II, we simulate infection trees with recursive backtracking. The simulation starts with a single infected node, and each time step for each infected, unquarantined leaf node $k - 1$ downstream nodes are added, with $k$ proportional to $kp(k)$. These new leaf nodes are infected with probability $T$ and symptomatic with probability $\Theta$. Then, according to the rules described in Sec. II, infected leaf nodes may be quarantined, causing them to not receive any downstream nodes. We let these dynamics run for 100 time steps or until there were 10 000 new infected leaf nodes added in a time step, at which point we consider the epidemic out of control. In Fig. 5 we show the fraction of trees in which the epidemic is not stopped within 100 time steps, the fraction of quarantined nodes, and the average reproduction number $R$ for trees using an ER degree distribution

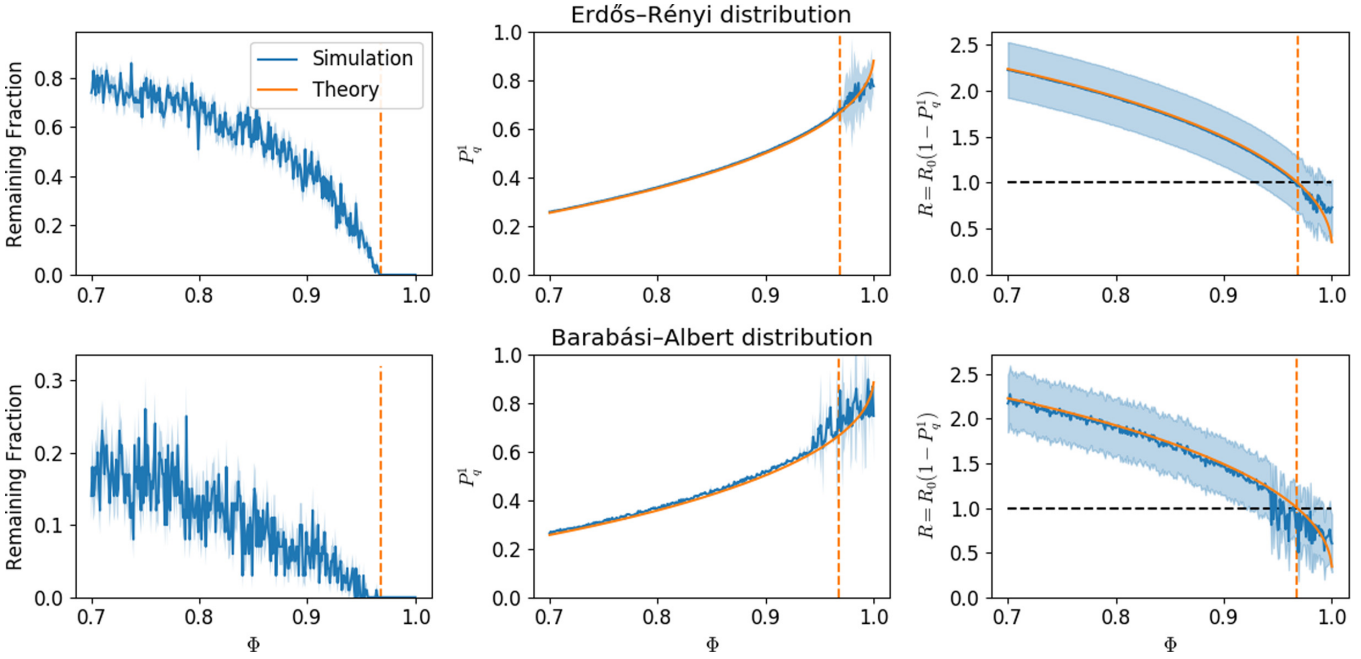


FIG. 5. Fraction of trees in which the epidemic survives 100 time steps (left column), probability of an infected node being quarantined $P_q^1$ (center column), and reproduction number $R$ (right column) for trees built with an ER degree distribution (upper row) or a BA degree distribution with cutoff $\kappa = 1000$ (lower row), with $r = 1$, $R_0 = 3$, and $\Theta = 0.5$ where the shaded areas show the standard deviation. Blue lines show the averages of 100 trees per data point, unbroken orange lines show the theoretical results for $P_q^1$ and $R$, and dashed orange lines show the theoretical critical value $\Phi_c$. The dashed black lines in the reproduction number diagrams show the critical value of $R$. Note that the measurement for the reproduction number $R$ and the quarantined fraction $P_q^1$ are skewed near or past the critical point, because the measurements here are dominated by just the beginning of the tree where the quarantining algorithm does not have enough history yet to quarantine nodes.

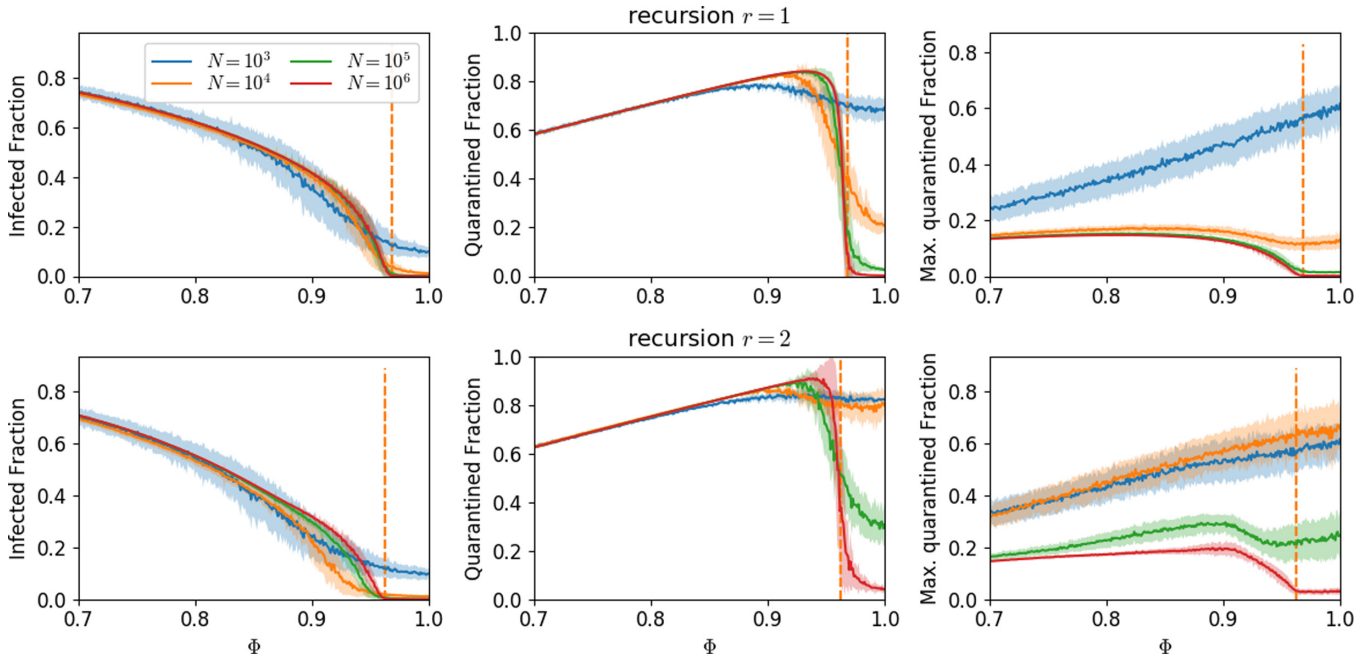


FIG. 6. Fraction of infected nodes (left), fraction of nodes that have ever been quarantined (center), and maximum number of nodes that have been quarantined at one time (right) for ER networks with recursion depth $r = 1$ (top row) and $r = 2$ (bottom row) as a function of the app-usage rate $\Phi$. Different color graphs show networks of different sizes $N$, and orange dashed lines show the theoretical critical value $\Phi_c$. All data points are the average of 100 simulation runs, and shaded areas show the standard deviation.

or a BA degree distribution with a cutoff $\kappa = 1000$. We see a very good agreement between our calculation and simulations for recursions $r = 1$; see Fig. 5. We have also verified that our calculations and simulations agree very well for larger recursion depths.

Next, we move away from the tree structure and use networks instead. In these networks, we start with ten initially infected nodes, which are chosen with a probability proportional to $kp(k)$, and we let the dynamics run until no new nodes are infected within a time step. Figure 6 shows the fraction of infected nodes, the fraction of nodes that have ever been quarantined, and the maximum fraction of nodes that has been quarantined at one point in time for ER networks with different recursion depths.

For the network size $N \to \infty$, we see that the fractions of infected and quarantined nodes drop to zero at the theoretical critical value $\Phi_c$. For higher recursion depths and relatively small networks, the infected fraction is already kept quite low below the theoretical critical value because a large fraction of nodes is being quarantined and therefore the assumption we made in Sec. II that nodes are not coincidentally swept up in unrelated infection trees does not hold anymore; however, this lower infected fraction comes at the cost of wrongly quarantining a relatively large fraction of nodes. Also, this effect is mitigated for larger network sizes $N$.

For BA networks, especially for large networks, the infection dies out quickly even for low values of $\Phi$, because the infection dynamics are dominated by the strongly connected hub nodes, which, after some time, will be in the recovered state, and therefore the effective degree distribution for the infection is quickly cut off for larger $k$. Additionally, in a BA network the first few nodes which are added to the network and later are likely to grow into the strongest connected nodes are likely to connect to each other and have common neighbors, meaning that the assumption we made in Sec. II of low clustering does not hold, which reduces the number of susceptible nodes adjacent to an infected large spreader $i$ because its neighbors are likely to have already been infected by $i$'s own infecting node. Both these effects lower the basic reproduction number $R_0$ below the theoretical value given by Eq. (1).

## V. CONCLUSION

Considering the problem of epidemic spreading of an infectious disease with a finite asymptomatic transmission rate, such as the current epidemics caused by the SARS-CoV-2, we have introduced a combined infection model of nodes taking susceptible, infected, or recovered states with a recursive contact tracing algorithm for quarantining, equivalent to an app used by a network's nodes to stop a pandemic in our model. The contact tracing algorithm changes the percolation phase transition of the epidemic spreading model, and we here studied the interplay of these two processes in a minimal statistical mechanics model.

We have calculated the odds of an infected node being quarantined by the contact tracing algorithm, as well as the resulting theoretical critical values for the app usage rate above which an infection does not percolate through the network, and the minimum symptomatic rate beneath which a disease cannot be stopped, depending on the algorithm's recursion depth, the disease's basic reproduction number, and the contact network's underlying degree distribution.

We found that the critical app adoption rate and critical symptomatic rate are both significantly lower for an algorithm using recursive contact tracing, even with a low recursion

depth, than for the classically employed, nonrecursive method of direct contact tracing. In fact, any disease with a finite symptomatic rate and arbitrary basic reproduction number can be stopped if the app usage rate and recursion depth are large enough, meaning that recursive contact tracing can be an effective method for controlling diseases with large asymptomatic transmission rates which could not have been stopped with previous contact tracing methods.

Our critical app adoption rate of over 95% may seem unusually high at first glance compared to some other results [5,23,27,29], with other estimates generally lying between 56% and 95% [50]. However, this is simply caused by our model's harsh assumptions, such as a very high basic reproduction number $R_0 = 3$, a relatively high asymptomatic rate of 50%. Furthermore, keep in mind that we here study an idealized statistical mechanics model without further infection prevention measures, such as random testing or social distancing, apart from contact tracing, not distinguishing between the infectivity of symptomatic and asymptomatic disease carriers (symptomatic carriers are often assumed to self-quarantine and therefore infect fewer people), and a lack of manual contact tracing even for symptomatic infected individuals who are not using the app. Our results are comparable to those of other models making harsh assumptions [16,17,26]. We stress once again, however, that our model's goal is not to make quantitative predictions, but to provide a theoretical basis for understanding the limits of recursive contact tracing and further work.

Further, we found that, while higher recursion depths can stop diseases with a high asymptomatic rate, for low asymptomatic rates, recursion depths higher than one show very little improvement in the critical app usage rate while falsely quarantining more uninfected nodes, implying that for such diseases recursion depths larger than one are mostly not useful.

Also, the contact network's degree distribution was shown to have little impact on these critical values, so recursive contact tracing is not only viable for Erdős-Rényi graphs, as tested in previous studies, but also for more realistic scale-free-like networks, i.e., scale-free networks with a cutoff.

We have ensured the accuracy of our theoretical calculations using simulations on infection trees and networks with different degree distributions. We found very good agreement between our calculations and simulations for any degree distribution on infection trees and for Erdős-Rényi networks. For Barabási-Albert networks, the simulation's critical values lie below the calculated ones because quarantining the most connected nodes quickly changes the network's degree distribution and because the effect of clustering, as highly connected nodes in Barabási-Albert networks are likely to be connected to each other, was not considered in the calculations.

The calculations presented here are viable for a simple model, but we believe that the qualitative conclusions should be applicable to the real world as well. Future research should expand this simple model to be more realistic and possibly fit the infection profiles of real diseases, as well as consider the effect of clustering on the model's critical values.

The presented model could easily be extended to more closely model real-life processes, for example, by introducing parameters for presymptomatic durations, delays in testing or communication, using different reproduction numbers for symptomatic and asymptomatic individuals, or studying real-life networks that model household structures. Further, in the real world, an asymptomatic node who is considered by the algorithm to have potentially been infected could be tested and then be used as a new index case for further contact tracing.

Also, as the exact nature of immunity due to previous infection would not be immediately known for any new disease, we erred on the side of caution and assumed the possibility of reinfection when considering who should be quarantined, although the underlying infection model does not allow this. Should the existence of such immunities be known, one could instead remove previously infected individuals from consideration when determining possible infection chains and thereby lower the false positive rate of quarantining. Conversely, the model could also be modified to take into consideration the possibility of recovered or vaccinated agents still possibly becoming infectious disease carriers, despite being immune themselves.

Finally, while digital contact tracing has been the underlying case for our model, it could also be extended to simulate and explore the theoretical limits of recursive manual contact tracing, with the inherent difficulties and unavoidable delays therein.

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