

Risk assessment via layered mobile contact tracing for epidemiological intervention

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Abstract

There is strong interest globally amidst the current COVID-19 pandemic in tracing contacts of infectious patients using mobile technologies, both as a warning system to individuals and as a targeted intervention strategy for governments. Several governments, including India, have introduced mobile apps for this purpose, which give a warning when the individual's phone establishes bluetooth contact with the phone of an infected person. We present a methodology to probabilistically evaluate risk of infection given the network of contacts that individuals are likely to encounter in real life. Instead of binary "infected" or "uninfected" statuses, an infection risk probability is maintained which can be efficiently calculated based on probabilities of recent contacts, and updated when a recent contact is diagnosed with a disease. We demonstrate on realistic networks that this method sharply outperforms a naive immediate-contact method even in an ideal circumstance that all infected persons are known to the naive method. We demonstrate robustness to missing contact information (such as when phones fail to make bluetooth contact or the app is not installed). We show, within our model, a strong flattening of the infectious peak when even a small fraction of cases are identified, tested and isolated. In the real world, where most known-infected persons are isolated or quarantined and where many individuals may not carry their mobiles in public, we believe the improvement offered by our method warrants consideration. Importantly, in view of widespread concerns on privacy and contact-tracing, our method relies mainly on direct contact data that can be stored locally on users' phones, and uses limited communication via intermediary servers only upon testing, mitigating privacy concerns.

Introduction

The COVID-19 coronavirus pandemic, which has expanded from China in December 2019 to affect almost every country in the world by now (April 2020), has led to a strong interest in non-pharmacological interventions to curtail spread. Early efforts in China, Singapore, South Korea and other countries involved extensive testing as well as identification and isolation of contacts of infected individuals and mobile-based alerts [13]. Several governments have also experimented with mobile contact-tracing applications. At a basic level, these applications enable a mobile phone to communicate with other mobile phones via bluetooth, and warn the owner when contact has been made with an infected person. An example is India's Aarogya Setu ("health bridge") app ¹. Previously, such apps were developed in China, Singapore, and South Korea [10], and are under development in countries including France [7], the USA [10], and the UK [12] and elsewhere. Privacy concerns have been raised globally, and privacy-sensitive protocols have been proposed [4]. Meanwhile Google and Apple have announced a partnership to develop contact-tracing

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¹<https://www.mygov.in/aarogya-setu-app/>

40 infrastructure for inclusion in their basic mobile software stacks, promising to respect privacy and security.
41 [8]

42 Unfortunately most current apps are closed-source with opaque mechanisms, but as far as is docu-
43 mented by them, they rely on direct contacts with known infected individuals, and possibly on past direct
44 contacts with individuals who subsequently became diagnosed as infected.

45 Here we present a probabilistic framework to assess an infection risk based on risk factors of contacts,
46 whether positive or not. In this scheme, every individual has a risk factor based on their contact history.
47 We demonstrate via simulations that this method strongly outperforms a naive method based only on
48 direct contacts. Given some epidemiological assumptions and approximations, our calculation is rigorous
49 but can be performed locally on a mobile phone using only the owner’s risk factor and the risk factor of the
50 contact. Contact history, too, can be stored on the mobile phone and need not be shared with a server.
51 Only one crucial step, of updating risk factors of recent contacts based on subsequent diagnoses, may
52 require the use of a server. This too may probably be made secure, but we do not discuss security issues
53 here, only efficiency of identifying likely infected individuals (who could, for example, be asymptomatic
54 but spreaders).

55 Methods

56 Risk factor evaluation

57 We make the approximation that the person-to-person transmission probability is a constant per contact,
58 p_t . This parameter is related to the infection rate in the SEIR compartmental model, discussed in a later
59 subsection.

60 If individual A, who is uninfected, makes one contact with individual B, who is infected, then A gets
61 a probability p_t of being infected after that contact. But we can also consider the case when B has a
62 probability p_B of being infected; then the probability of A being infected after the contact is $p_B p_t$.

63 The most general scenario is that neither A nor B were uninfected, but had previous probabilities p_A
64 and p_B of being infected. Then A is now infected with a new probability $p'_A = 1 - (1 - p_A)(1 - p_B p_t)$. Note
65 that the bracketed terms are the probabilities of being originally uninfected, and of remaining uninfected
66 after the contact. Similarly, we update B’s probability too, to $p'_B = 1 - (1 - p_B)(1 - p_A p_t)$. Note that if
67 $p_B = 0$ then p_A is unchanged, while p_B updates to $p_A p_t$, as expected; and vice versa.

68 We therefore propose the following algorithm to update individual probabilities, or risk factors, of
69 being infected.

- 70 1. Each individual has a unique ID (for example, mobile phone number).
- 71 2. Each individual’s infection probability is initialized in some way based on prior knowledge and self-
72 reporting. This could be done via a questionnaire upon installing the app. The vast majority will
73 be initialized to zero.
- 74 3. Each individual’s app maintains a list of ALL contacts in the past m days ($m \approx 14$, estimated upper
75 bound of the incubation time; contacts from earlier are unlikely to affect current infection status).
76 With each contact is included a list of all meeting times with that contact. This is required for a
77 thorough update of probabilities, as discussed below.
- 78 4. Every time two individuals A and B are in proximity, their mobile apps exchange their infection
79 statuses p_A and p_B . The update is made as above: $p'_A = 1 - (1 - p_A)(1 - p_B p_t)$ and $p'_B =$
80 $1 - (1 - p_B)(1 - p_A p_t)$
- 81 5. **Test update propagation:** If person A on B’s contact list tests positive, then p_A is updated from
82 its previous value to 1. But since A was probably already infected during their contact (given the
83 long incubation time of the virus), p_B needs to be updated too, to $p'_B = 1 - (1 - p_B) \frac{(1 - p_t)}{(1 - p_A p_t)}$ (where
84 p_A in the denominator is the previous value for A, and the new value for A is 1). But now we need
85 to update every C whom B met subsequent to meeting A (that is, B’s *last* meeting with C is more
86 recent than B’s *first* meeting with A). The formula for this is $p'_C = 1 - (1 - p_C) \frac{(1 - p'_B p_t)}{(1 - p_B p_t)}$. And, again,
87 every contact of C who was met subsequent to C’s first meeting with B, and so on. In our simulation

we accomplish this via a recursive function, avoiding cycling back to a previous contact by passing an “ignore list” of contacts in each function call. Additionally, if a contact was met multiple times in the relevant timeframe, an update is performed the same number of times, since each meeting carried a risk.

6. Each individual A’s contacts older than m days drop off the contact list, but this does not change p_A . That is, if B, who last met A more than m days ago, is diagnosed infected, this is unlikely to require updating p_A .

7. Individuals who are recovered are marked as as immune. They play no further part in our simulation. In the real world, some instances of re-infection within a short time frame have been noted ([5], and additional cases reported in news media).

Simulation

We simulate an agent-based model on a network, in which agents interact stochastically over time and are categorized as “susceptible”, “exposed”, “infectious”, and “recovered”. These are the categorizations of the compartmental SEIR model in epidemiology, discussed and compared in the next subsection. We implement the risk update algorithm on the same agent-based framework and compare the risk profile predicted by our algorithm with the actual infections of the agent-based simulations.

We initialize a population of size N , whose individuals are nodes on a weighted network. The network represents all *possible* contacts in this population: a link indicates two people who may make a contact, and the weight of the link is the probability of their making a contact at a given time. We consider random networks with uniform degree distribution and uniform link weights, Barabási-Albert-structured networks, and networks with family structures and small-world features. Our results are consistent across all these structures. A key parameter of the network, used below, is the average number of contacts per node, defined as

$$N_c = \frac{1}{N} \sum_{i=1}^N \sum_{j \in \text{neighbours of } i} w_{ij} \quad (1)$$

where w_{ij} is the weight of the link between i and j .

Individuals are marked as susceptible (S), exposed (E—infected but not yet infectious), infectious (I) and recovered (R, assumed immune to future infection). The simulation is initialized with all individuals being uninfected (susceptible) except a small number (eg, 10 out of 10,000) who are infectious. With each individual is associated a probability, which is initialized to 1 for infectious individuals and 0 for others.

In each pass of the simulation, which we call an “epoch”, every link on the graph is sampled once, and a contact is made with probability equal to the weight of the link. So links weighted 1 (such as family links) are always sampled, while other links may be rarely sampled. After each contact between a pair of individuals, if one is infectious and the other is susceptible, the other is marked “exposed” with a probability p_t . For contacts other than S-I, nothing is done.

At the end of each epoch, each individual is sampled for their status. Exposed individuals become infectious with a probability p_e and infectious individuals recover with a probability p_r .

In parallel with this, at each contact, the probability scores of individuals making contact are updated as described above in “Risk factor evaluation”. This is done for each sampled pair of contacts if at least one has a non-zero probability score.

An example of a possible simulation is in figure 1.

We also keep track of a “naive probability” for each individual, which consists simply of updating

$$p \leftarrow 1 - (1 - p)(1 - p_t) \quad (2)$$

every time a known susceptible individual meets a known infected individual. We call this the “naive oracle” approach, since this algorithm does not consider contact with people who have a risk factor, only with truly infectious people; but knows the true infectious status of the contacted person. In the real world, this is known for only a fraction of infectious people.

Thus, the parameters of the simulation are p_t , p_e and p_r . However, these are in turn derived from other parameters as follows: R_0 is the “basic reproduction number” (see next subsection); M_d is the number of

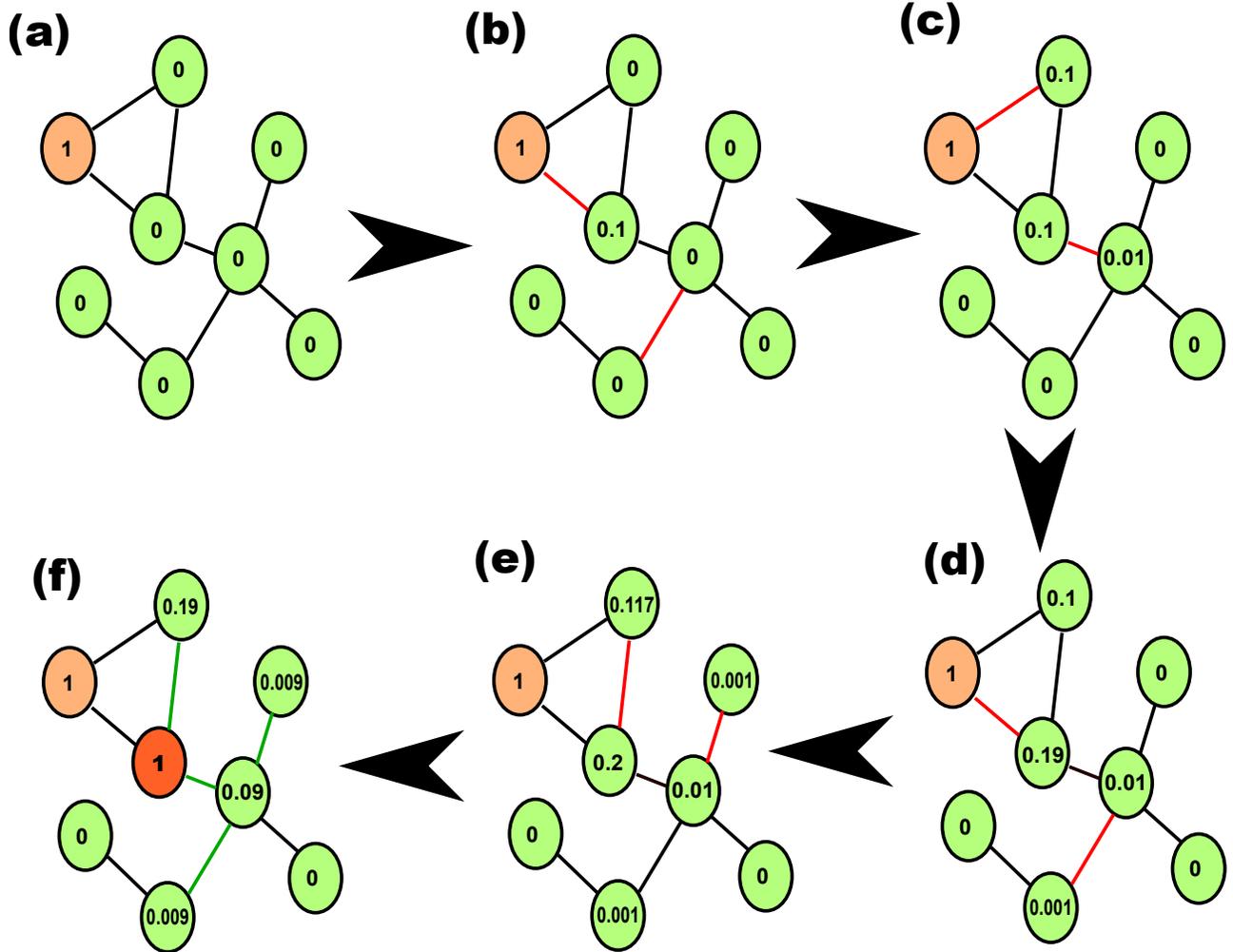


Figure 1: An example of a possible simulation, with $p_t = 0.1$. Numbers in ovals are probability values. The "infected" status values are not shown here but are updated stochastically in parallel with the probabilities. (a) Network with one individual initially infected. (b)–(e) Red links indicate contacts, and probabilities of respective nodes are updated. (f) A second individual is diagnosed infected (dark red) and that individual's probability value is changed to 1.0, and the chain of previous contacts is updated (green links).

134 epochs that constitute one day; d_e is the average exposure time in days (the average number of days to
 135 turn infectious); d_r is the average recovery time in days. From these, we get $p_e = \frac{1}{M_d d_e}$ and $p_r = \frac{1}{M_d d_r}$.
 136 Finally, p_t is determined from R_0 and N_c as in the next section (equation 9).

137 SEIR epidemiological model

138 The SEIR compartmental model in epidemiology, [16], without vital dynamics (i.e., without births and
 139 deaths in the population), is usually written as

$$\frac{dS}{dt} = -k_i SI \quad (3)$$

$$\frac{dE}{dt} = k_i SI - k_e E \quad (4)$$

$$\frac{dI}{dt} = k_e E - k_r I \quad (5)$$

$$\frac{dR}{dt} = k_r I. \quad (6)$$

140 Here S is the number of susceptible individuals in the population, E is the number of individuals exposed
 141 to infection but not yet infectious, I is the number of infectious individuals, R is the number of recovered
 142 individuals. The rate of infection (per person) is k_i , exposed individuals become infectious at rate k_e , and
 143 the recovery rate is k_r . These equations conserve the total population $S + E + I + R$. Within this model
 144 recovered individuals are permanently recovered (though deaths are not included here, individuals who
 145 die of the disease may also be counted in R since they are no longer infectious).

146 We seek to estimate parameters of our simulation from epidemiological measurements in the real
 147 world. A key epidemiological parameter is R_0 , the “basic reproduction number” or the average number
 148 of individuals infected by any individual while infectious. It can be shown easily[16] that

$$R_0 = k_i N / k_r \quad (7)$$

149 where N is the total population. This is valid for the SIR model as well as the SEIR model without births
 150 and deaths. This assumes a “well-mixed” system, but otherwise the same equation is commonly used with
 151 N being an “effective population”.

152 In terms of individual contacts and contact rates, we can alternatively write

$$R_0 = p_t \cdot C \quad (8)$$

153 where p_t = transmission probability per contact as above, and C is the total number of contacts while
 154 the patient is infectious. C is equal to the rate of contact R_c (per epoch, say) times the average recovery
 155 time (also in epochs). So if the contact rate is 100 contacts per epoch, and the recovery time is 10 epochs,
 156 then $C = 1000$, and if $R_0 = 2$, then $p_t = 0.002$.

157 The average recovery time is $1/k_r$, so comparing the two definitions of R_0 (equations 7 and 8), we
 158 can identify $k_i N = \text{rate of infection} = \text{rate of contacts} \times \text{probability of infection per contact} = R_c p_t$.
 159 Therefore, $p_t = R_0 k_r / R_c$. If, for example, R_0 is empirically estimated as 2, the recovery time is taken to
 160 be 10 days, and the average rate of contact per day is 100, then we estimate p_t as 0.002. More generally,
 161 we take the rate of contact per *epoch* to be exactly equal to the average number of contacts per link, N_c
 162 (equation 1). Then we have

$$p_t = \frac{R_0}{N_c M_d d_r} = \frac{R_0 p_r}{N_c} \quad (9)$$

163 which we use in the network simulation.

164 Gillespie simulations

165 To validate the accuracy of our sampling simulation, we also simulate the spread of the infection using
 166 the Gillespie algorithm. At any given time the state of the system is fully specified by the state of each
 167 individual; for the i th individual, its state $s_i \in \{S \text{ (susceptible), } E \text{ (exposed), } I \text{ (infectious), } R \text{ (recovered)}\}$.
 168 Let $N_S(t), N_E(t), N_I(t), N_R(t)$ be the number of individuals in each of these states at time t . Time is

again divided into epochs. At the beginning of each epoch the network links are sampled as above. Let $C_{ij}(t) = 1$ denote that there is a contact between individuals i and j in the epoch within which time t falls; $C_{ij}(t) = 0$ if there is no contact between these two in that epoch. Then the Gillespie algorithm is run until the time of the next epoch as follows:

1. Assign the start time of the epoch to the variable t
2. List all possible “events” that can occur which will change the state of the system: there will be $\sum_{\{i|s_i=S\}} \sum_{\{j|s_j=I\}} C_{ij}$ infection events, N_E exposed-becoming-infectious events, and N_I recovery events possible, for a total of n events. For each of these, compute the rate or probability per unit time for that event to occur, denoted a_j for event j , $j \in \{1, 2, \dots, n\}$.
3. Let $a_0 = \sum_{j=1}^n a_j$. Let Δt be a random number sampled from the exponential distribution with mean $1/a_0$.
4. If $t + \Delta t$ is larger than the time of the next epoch, set t equal to the time of the next epoch and end the Gillespie run, resample the network and run the Gillespie algorithm for the next epoch.
5. Otherwise, update t to be equal to $t + \Delta t$, and choose one event to occur at that time (event j is chosen with probability a_j/a_0). Update the state of each individual if the event has changed it, along with $N_S(t), N_E(t), N_I(t), N_R(t)$ (for example, if individual i infects individual j , then $s_j(t) = E$, N_E is incremented and N_S is decremented by one).
6. Go to step 2.

This process is repeated for as many epochs as desired, usually until the number of exposed and infected individuals has fallen to zero. This gives the distributions $N_S(t), N_E(t), N_I(t)$ and $N_R(t)$ as functions of time, for comparison with the network sampling simulation.

Results

Complete network: Comparison with well-mixed SEIR model

Figure 2 (a) shows a simulation on 2000 nodes, each node connected to all others with weight 1.0. For this “fully connected” or “complete” network, the SEIR model, with parameters determined as in the figure caption, shows excellent agreement with our simulation. The unrealistic assumption here is that each member of the population meets each other member exactly once per epoch; we exhibit it to demonstrate the agreement with a well-mixed SEIR model, but do not explore this network further.

Realistic network: Comparison with SEIR model and Gillespie simulation

We construct a more realistic network as follows. We initialize a random network with family links, such that every individual belongs to a family of size 1, 2, 3, 4, or 5 (with relative probabilities 0.2, 0.3, 0.3, 0.1, 0.1). Family networks are complete, i.e., all members are connected to all others with weight 1. Then, 1% of the individuals are randomly selected, and each is connected to 1000 other individuals, with weight 0.1. These are meant to indicate “spreaders”, that is, people such as shop attendants and receptionists who have a large number of daily contacts that vary every day. Further links are added via a modification of the Barabási-Albert (BA) algorithm[2], that is, each node is attached to a randomly selected other node with weight 0.1. In the BA algorithm the new node is selected from all other nodes with a probability proportional to their coordination numbers. For efficiency, we first select 1000 random other nodes, and then select from those using the BA method.

Figure 2 (b) shows the result of simulation on this network over 1500 epochs (150 days). The SEIR simulation shown is with an effective population size of 4,000, which has no justification but offers a better fit compared to either the total population (10,000) or the average contact number (37). It is known that the rate of spread of an infection on a network depends on its structure and not just on the average contact number [17], so it is not surprising that the best choice of effective population is different from the average contact number. In comparison, a Gillespie simulation on the same network agrees very well.

Figure 2 (c) shows a simulation on 100,000 nodes, with parameters as in the caption. The SEIR here is plotted for an effective population of 23,000, and again fits poorly while the Gillespie agrees well.

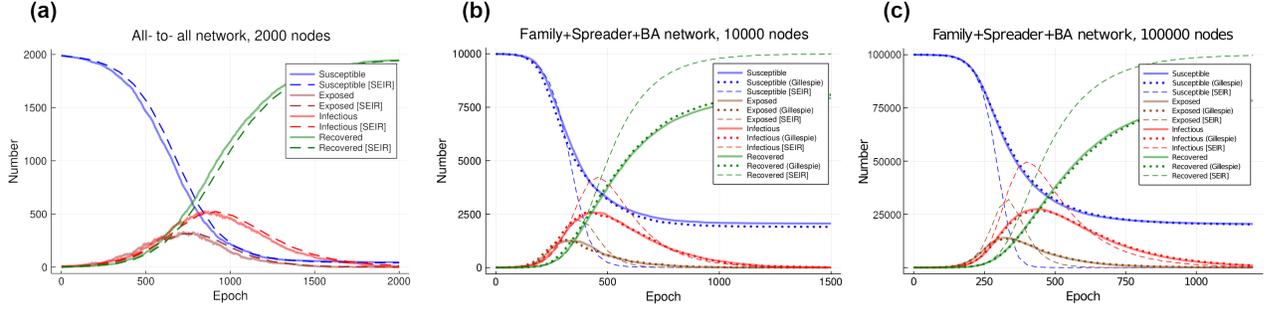


Figure 2: (a) A comparison of our network simulation on 2000 nodes, with every node connected to every other node with weight 1.0, with the compartmental SEIR model. The parameters taken for the network simulation were: $R_0 = 4.0$, $M_d = 10$, $p_e = 1/10$, $p_r = 1/20$, $p_t \approx 1.0 \times 10^{-5}$ (from equation 9). For the SEIR integration, since all rates are small, we use $k_e = p_e/M_d = 0.01$, $k_r = p_r/M_d = 0.005$, and $k_i = p_i$. The simulation over 2000 epochs (200 days) agrees very well with the SEIR solution. (b) Simulation on a 10000-node network that includes family units (size 1–5; link weight 1), spreaders (1% of total, 1000 links each, link weight 0.1 each) and links added via the Barabási-Albert method (link each node to a random new node with probability proportional to current coordination number of new node; link weight 0.1). This network has 183,599 links. We used $M_d = 10$ epochs. The average contact of each node is 3.77/epoch, ie 37/day. Parameters were: $R_0 = 3$, $d_e = 5$ days (5 epochs), $d_r = 15$ days (150 epochs), $p_t = 0.0053$ (calculated from above). The simulation over 1500 epochs (150 days) agrees well with an independently implemented Gillespie simulation, but disagrees with the SEIR prediction. Shown is SEIR for effective population size $N_c = 4000$, which gives the best fit but is hard to justify. (c) Simulation on a 100,000 node network with 1,948,709 links, layered similarly to the network in (b), except that the BA links have weight 0.05, with the same parameters except $p_t = 0.0069$ (calculated). The SEIR plotted is for effective population 23,000.

216 Growth of probabilities

217 While the object of this exercise is not to predict overall numbers in the population, but to identify in-
 218 dividuals most at risk, it is of interest to see how this probability varies with the growth in infectious
 219 individuals. Figure 3 (left) plots the "total probability" (the sum over all individuals of individual prob-
 220 abilities, which would crudely be the expected number of infected individuals), as well as the number of
 221 individuals with p exceeding 0.5, 0.6, 0.7, 0.8, 0.9. The total grows much faster than the epidemic, as do
 222 each of the fractional curves, but the latter start their growths at different times that roughly track the
 223 growth of infectious cases.

224 Figure 3 (right) does the same for the naive probability. Here the total probability tracks the infectious
 225 total more closely, but the fractional curves appear to increase at roughly the same epoch. This perhaps
 226 gives some intuition for the performance of our method compared to the naive method as measured by
 227 positive rate vs false positive rate, or precision vs recall, discussed below.

228 Notably, at this time we have no "recovery" for probabilities, other than the testing-and-resetting
 229 mechanism discussed further below, not used in this figure. The methodology is expected to be most
 230 useful in early stages of an epidemic.

231 Effectiveness of probabilistic prediction compared to naive methods

232 On the network exhibited in Figure 2 (c), we compare the predictions of our probabilistic method and
 233 the "naive oracle" method at epochs 200, 250 and 300 (representing 1815, 6479 and 14126 infections
 234 respectively out of 100,000). Subfigures 4 (a), (b), (c) show receiver operating characteristic (ROC)
 235 curves, and subfigures (d), (e), (f) show precision-recall curves (PRC). Also shown is the result for a
 236 random assignment of probability in $(0, 1)$ for each individual. In all cases probabilistic method clearly
 237 outperforms the naive oracle, even though the oracle has the strong advantage of knowing whether an
 238 individual in an encounter is truly infected or not. These are plotted by calculating true positive rates (also
 239 called recall), false positive rates, and precision varying the threshold p used to predict an individual's

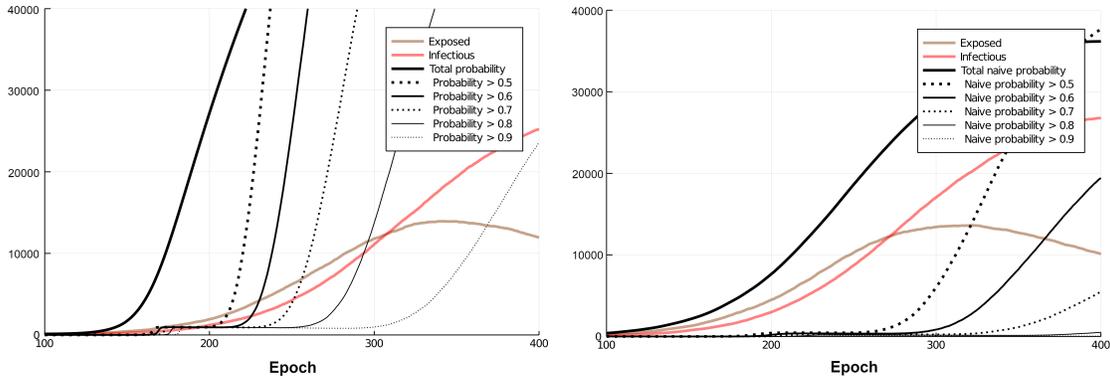


Figure 3: (left) The “total probability” (p summed over all 100,000 individuals) as well as the fraction of individuals over 0.5, 0.6, 0.7, 0.8, 0.9 in probability. (right) Similar plot for the naive probability.

240 infective state. A true positive is an infected patient who is predicted to be infected (p above threshold); a
 241 false positive is an uninfected patient predicted infected; a true negative is an uninfected patient predicted
 242 uninfected; a false negative is an infected patient predicted uninfected. If the total numbers of these are
 243 respectively TP , TN , FP and FN then

$$\begin{aligned} \text{True positive rate} \equiv \text{Recall} &= \frac{TP}{TP + FN} \\ \text{False positive rate} &= \frac{FP}{FP + TN} \\ \text{Precision} &= \frac{TP}{TP + FP} \end{aligned}$$

244 So, at all epochs shown, the probabilistic method has a TPR of about 67% at a FPR of 50%; the naive
 245 oracle performs at less than 50% TPR at 50% FPR in all cases, and its performance grows worse at later
 246 epochs (as the infection spreads.) Since suspected individuals will be tested via accurate RT-PCR tests,
 247 we feel this FPR rate is acceptable, especially given the effectiveness of a testing+isolation strategy that
 248 tests even a small fraction of risky individuals (next section)

249 Effectiveness of testing and isolation of patients

250 The naive oracle above is assumed to know the status of every covid-19 positive patient. Also, we update
 251 naive probabilities only on contact between infectious and uninfected patients.

252 For a more realistic comparison with the real world operation of these methods, we can simulate “test-
 253 ing” of patients, after which they are marked “tested positive” (known infectious) or negative (susceptible).

254 We implement testing at each epoch by selecting a predetermined fraction of all individuals with
 255 a high probability to be tested; the test simply looks at their true infected status. If truly infected,
 256 they are marked “tested positive”, their probabilities and naive probabilities are set to 1, and non-naive
 257 probability updates are propagated to their contacts (as in figure 1 (f)). If they test negative, they are
 258 marked susceptible, and their probabilities are set to zero and their contact updates propagated.

259 We modify the naive probability tracing to only consider contacts with known-infectious (tested) cases,
 260 and to update as in equation (2) for each such contact (regardless of the status of the contactor.) The
 261 sophisticated tracing goes on as before, but is aware of known-infectious contacts because their probabilities
 262 are set to 1 (but does not deal with them in any special way).

263 The results are shown in figure 5, for a test threshold of $p = 0.9$ and rates of 5% and 1% of above-
 264 threshold cases tested. Interestingly, it appears that this non-oracular naive method is only able to achieve
 265 a very small recall (TPR) regardless of precision. Also, overall performance of the probabilistic method is
 266 reduced in the presence of testing, perhaps because probabilities after negative tests are being set to zero
 267 even though they could be in risky populations and liable to be infected.

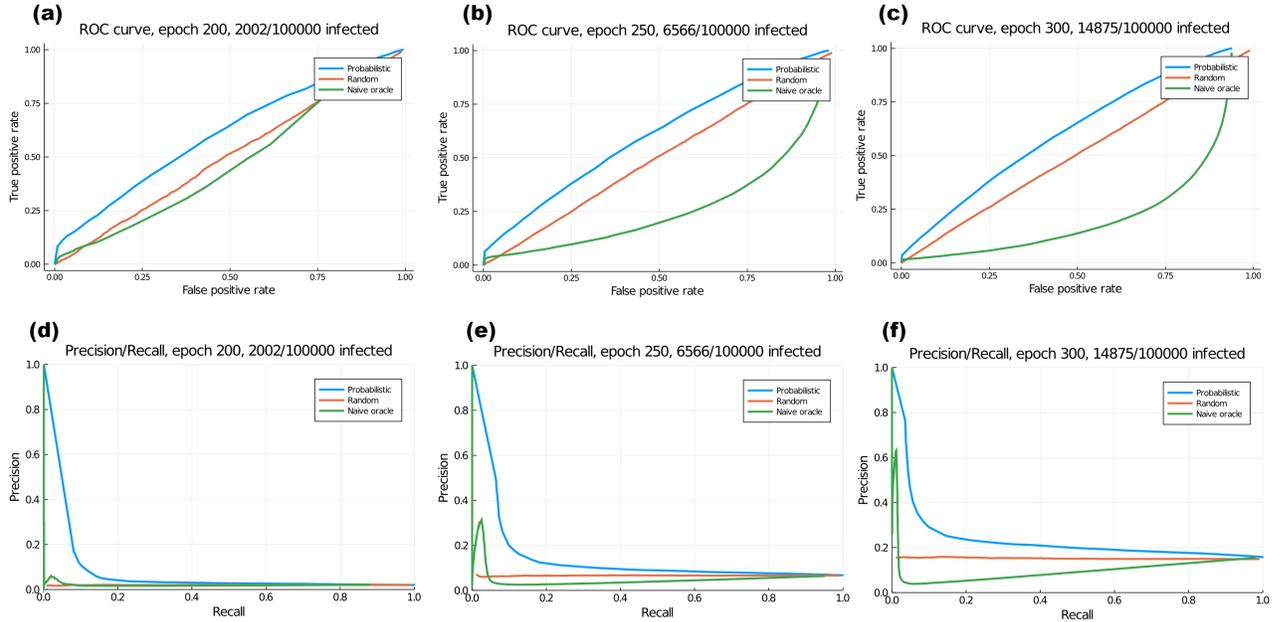


Figure 4: (a), (b), (c): ROC curves for our probabilistic prediction of infections, a random allocation of probabilities, and the naive oracle method, at epochs 200, 250 and 300 on the network in figure 2 (c). (d), (e), (f): Precision recall curves at the same epochs.

268 We can also isolate tested-positive patients, by weakening their links to all their contacts. Figure 6 (a)
 269 shows the effect of this, for a testing threshold of 0.8 (individuals whose probability exceeds 0.8 are tested),
 270 for testing percentages of 10% and 20% of risky individuals, and with a weakening of links to 10% of their
 271 previous value. (For a population of 100,000, at epoch 300 in our simulation, about 2,200 individuals have
 272 $p > 0.8$, so 10% testing here means testing 220/100,000 individuals, or 0.2% of the full population.) This
 273 suggests that a test rate of even 10% has a very strong effect in flattening the curve. However, though
 274 this suggests the effectiveness of testing and isolation (which has been widely noted [1, 14, 3] and is being
 275 practised by most countries), we caution against drawing quantitative conclusions from our model.

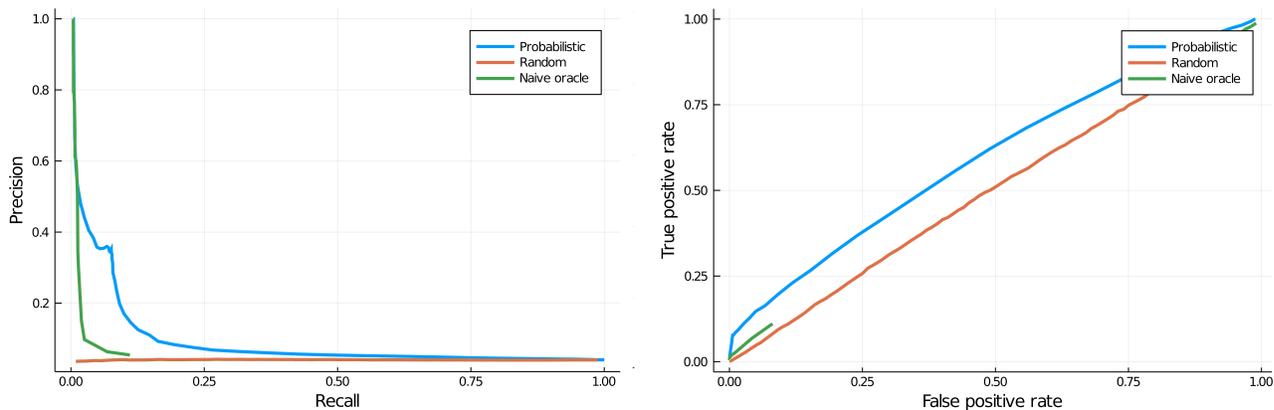
276 Lossy data

277 With mobile tracking, it is likely that several individuals will not be carrying their mobile or will not have
 278 the app installed, therefore the probabilistic updates will not occur. Figure 6 (b) shows the effect of such
 279 missing contacts, implemented by randomly ignoring updates with a given probability. This appears to
 280 have negligible effect for up to 60% loss in contacting (40% successfully recorded contacts).

281 Discussion

282 Several authors have discussed the possibility of tracing contacts of recently infected patients via mobile
 283 phones, with the goal of isolating them. This is argued[9, 3] to be an effective way to control the outbreak
 284 and build “digital herd immunity”. We demonstrate in an agent-based simulation on a network that our
 285 method is a better predictor, based on TPR/FPR or precision/recall, of truly infected patients compared
 286 to a naive first-contact-based prediction, even in an ideal case where the naive method is an “oracle”
 287 that always knows the true status of the contact. Our results are robust to loss in detection of contacts,
 288 which is expected to be significant in real life. Our simulations show that testing only the most probable
 289 individuals ($p > 0.8$) and isolating them (reducing link strength by a factor of 10) strongly flattens the
 290 curve of infection. Though we have tried to make our network structure realistic, the real world has several
 291 complications over a simulation; nevertheless we expect these results to hold qualitatively.

100,000 nodes, testing above 0.9, 5% tested



100,000 nodes, testing above 0.9, 1% tested

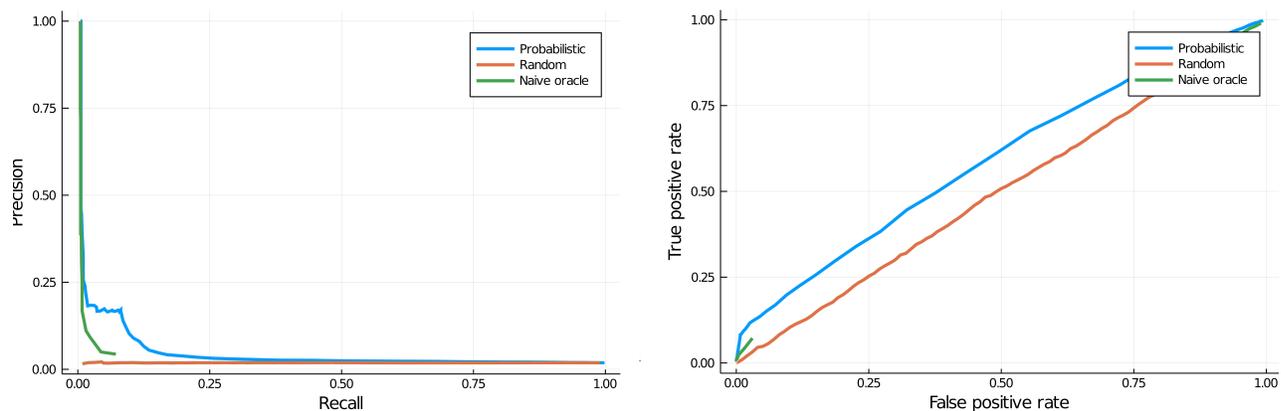


Figure 5: ROC and Precision-Recall curves with testing for a non-oracle naive method, which is aware only of tested-positive individuals, and the probabilistic method.

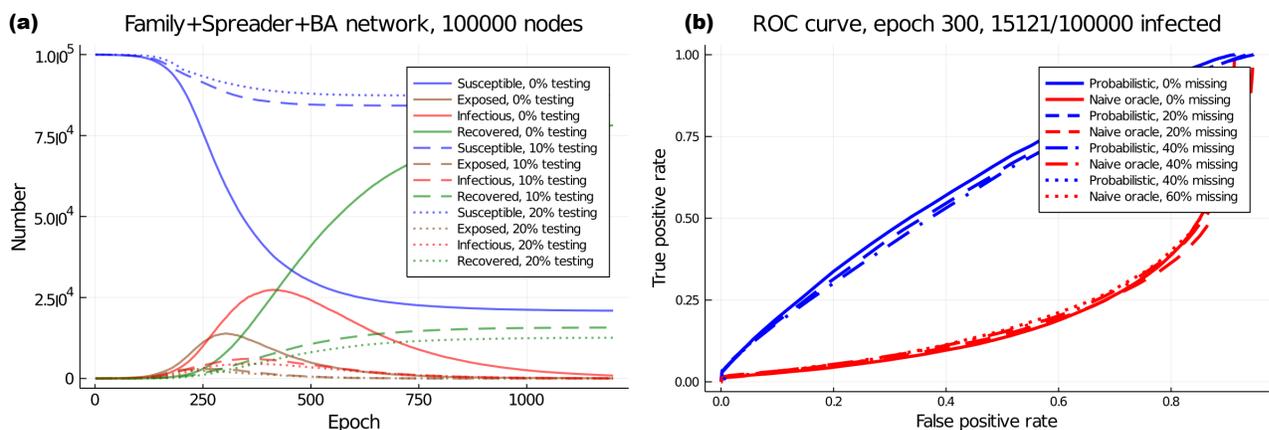


Figure 6: Effect of testing, and of missing contacts

292 Several authors have also raised privacy concerns [11, 6] over mobile contact tracing, and proposed
293 privacy protocols to handle this [4]. We do not directly address privacy concerns here. However, our
294 method requires most contact information to be stored only on the users’ own mobile phone. This
295 information only consists of the contact’s mobile number, number of meetings, and times of first meeting
296 and last meeting. Infection probability information is exchanged via bluetooth at the time of contact,
297 but is used only to update one’s own probability and need not be stored. Only one step, the “update
298 contacts” procedure that propagates the change in diagnosis of an individual to the individual’s contacts,
299 and the contacts’ contacts, recursively, requires the means for one mobile phone to communicate to another
300 post-contact. This likely requires the use of an intermediary server, but this use is limited and privacy
301 concerns can be mitigated by using an encrypted protocol and deleting communication request data once
302 the request is carried out.

303 Overall, our probabilistic contact tracing framework appears to outperform the naive method signif-
304 icantly, whether implemented as an “oracle” that knows all truly infected individuals, or implemented
305 with a testing framework to recognize only positively-tested individuals. While it can be used to identify
306 immediate contacts of a tested individual, it can go further to identify at-risk individuals in the wider
307 population, while also substantially taking care of privacy concerns.

308 While we focus on the SEIR disease model, more complex models featuring asymptotic individu-
309 als, different levels of symptomatic individuals, limited recovery period (recovered individuals becoming
310 susceptible after a time), etc, can be considered and are being considered for COVID-19 [15]². Our sim-
311 ulation framework can be modified easily for such cases too, as well as for bigger and more geographically
312 structured networks. We plan to explore such complexities in future work.

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320 Availability

321 The network generation and simulation code is available at <https://github.com/rsidd120/EpiTracSim>.

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²Ongoing study at <https://indscicov.in/indscisim>

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