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How and to what extent does the anti-social behavior of violating self-quarantine measures increase the spread of disease?

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A B S T R A C T
COVID-19 has shown that quarantine (or self-isolation) may be the only available tool against an unknown infectious disease if neither an effective vaccine nor anti-viral medication is available. Motivated by the fact that a considerable number of people were not compliant with the request for self-quarantine made by public authorities, this study used a multi-agent simulation model, whose results were validated by theory work, which highlights how and to what extent such an anti-social behavior hampers the confinement of a disease. Our framework quantifies two important scenarios: in one scenario a certain number of individuals totally ignore quarantine, whereas in the second scenario a larger number of individuals partially ignore the imposed policy. Our results reveal that the latter scenario can be more hazardous even if the total amount of social deficit of activity—measured by the total number of severed links in a physical network—would be same as the former scenario has, of which quantitative extent is dependent on the fraction of asymptomatic infected cases and the level of quarantine intensity the government imposing. Our findings have significance not only to epidemiology but also to research in the broader field of network science.

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1. Introduction
The Spanish Flu (1918), SARS (2003), Swine Influenza (2009), and MERS (2013) underline the ongoing threat from global pandemics, which break out intermittently and jeopardize the safety and security of modern social systems and everyday lives. The most important lesson from to be learned from COVID-19 is that the social systems are vulnerable to unknown viruses and communicable diseases, in particular those that have no available vaccines or pharmaceutical treatments. The possible strategies for protecting people until a vaccine is developed are limited only non-pharmaceutical provisions, such as wearing masks [1], washing hands [2], social-distancing [3,4], border restrictions [5], and top of that an implementation of massive quarantines or systematic social isolations, which enable individuals to directly remove the possibility of infection owing to being less physical contacts. We know that “lockdown” is one of the most highlighted terms in media under the wake of COVID-19 alongside another highly visible term, “social-distancing.”

The present study specifically concerns “quarantine”; in this work this term includes all variant concepts, such as “isolation,” irrespective of it being self-quarantine (i.e., spontaneous quarantine) or forced quarantine (i.e., compulsory quarantine).

There have been many previous studies concerned with the mathematical epidemiology of quarantine. Even before being exposed to the recent experience of COVID-19, there were numerous notable works taking a so-called compartment model approach, including SIRS, SIR, and SIQRS processes, which have underlined the importance of quarantine to the control of disease spreading (e.g., [6–11]), which mainly concerned rigorous mathematical issues, such as global stability of epidemic equilibrium, the manner in which stochasticity alters such equilibriums, and the stationary distributions of state variables at a given equilibrium.

Since 2020, COVID-19 has brought considerable interest to the concept of quarantine as one of the provisions to control the COVID-19 pandemic; this interest has prompted many excellent research works. Mishra et al. [12] developed three different ODE models to study quarantine based on either the SIR or SEIR processes, where each of the three models should be applied different stages of a breakout of...
COVID-19. Aleta et al. [13] successfully built an ODE model, applicable to realistic situations, that permits the quantitative evaluation of the impact of testing, contact tracing, and household quarantine during the second wave of COVID-19; this work was based on the actual situation observed in Massachusetts, USA. A simple SEIR-based ODE model by Pan et al. [14] demonstrated that "isolation" in which an infected and infectious patient with mild symptoms is stringently controlled at a hospital can reduce the final size of an epidemic because such a patient has less chance to come into contact with others. In view of the so-called "optimal controlling problem," which is based on Pontryagin's maximum principle, Abbasi et al. [15] developed SQUEIAR models, where they implemented a quarantine compartment, Q, connected to susceptible compartment, S. Thus, their models presumed Q to be a self-quarantine that acts as a pre-emptive provision to avoid infections. Ngonghala et al. [16], Browne et al. [17], and Elias et al. [18] used a similar concept to that of Q in Ref. [15]. By contrast, Zhu et al. [19] used an alternative model of quarantine in their ODE model: Q is placed between the infected compartment, I, and the removed/recovered compartment, R, which implies a sort of forced quarantine, which is imposed on infected and infectious individuals that keeps them separated from susceptible individuals. Chen et al. [20] took a similar concept, where Q appears between I and S in a SIS process. Another concept of Q was provided by Meman et al. [21], where the quarantine compartment, Q, absorbs a flow of individuals from the exposed compartment, E, and emits a flow either to the susceptible compartment, S, or to infected one, which was denoted by I in their model, depending on whether the individual was symptomatic infected or not. Arona et al. [22] took a different concept, where a quarantine compartment, Q, was connected with the exposed, asymptomatic, and symptomatic compartments. Analogously, Cui et al. [23] presumed that a quarantine compartment, Q, absorbs a twofold flow from both the asymptomatic and symptomatic infected and infectious compartments.

Mancastropa et al. [24] studied the impact of a temporal link (that may model real daily-life activity showing variation from day to day) using both SIS and SIR processes, which was explored using a novel theoretical procedure. In the study, they further accounted for how quarantine (meaning that an infected and infectious individual never has any physical link to others) affects the final epidemic size.

There also exists the so-called "vaccination game" where the dynamics of the spread of a disease is dovetailed with the dynamics of the human decision-making process, including whether one commits quarantine or not; there exist many studies on this topic (e.g., [25–27]). Alam et al. [28] quantified the effect of quarantine and isolation and compared these processes in terms of effectiveness; they considered the effect of a "quarantine" that was pre-emptive (i.e., self-quarantine using the previous terminology) whereas "isolation" was considered only after infection (i.e., forced quarantine).

Although studies concerning quarantine have been reported, there remains one important question regarding real-world issues. This question relates to the concept of "violation of quarantine" resulting from incompliance. COVID-19 has revealed a considerable proportion of people at in most/all societies do not obey to the requirement of quarantine issued by the public health organizations [29–31]. This behavior is due to the existence of a social dilemma structure in the so-called public goods games (see, for example, Refs. [25–27]). If every person who is requested to quarantine obeys to the requirement, it would result in a greater capacity to control the spread of the disease, which brings about a socially optimal situation. At the individual level, however, he/she has an incentive not to obey a quarantine order because it imposes on him/her an inconvenience as well as a financial deficit, that is, a negative payoff. Thus, we should note that everyone has an incentive to violate the requirement of quarantine. There are many different extents to which one may violate a quarantine policy. If one is completely incompliant, he/she totally ignores the requirement, maintaining their normal activity level. If one is still incompliant, but to a lesser extent, he/she may partially ignore the quarantine order. Such slightly incompliant people might be more than those who totally ignore as above. Between these two scenarios, numerous other behaviors may exist. For a given amount of activity as a whole in a society, it is unclear which scenario is preferable between a small number of people who totally ignore the quarantine policy or a relatively large number of people who slightly ignore the policy. A challenging question regarding the spread of disease and the compliance with quarantine order is this: from the choice of a "deep & narrow" scenario or "shallow & wide," which causes a greater extent of disease spread? The answer to this challenging question, "deep & narrow" versus "shallow & wide," would be meaningful not only from the epidemiology standpoint but also from the viewpoint of network science. This work intends to draw a quantitative answer this question, putting aside other intermediate provisions as above like washing hands.

This paper is organized as follows: in Section 2, we establish a multi-agent simulation (MAS) model, which is supported by a theoretical approach based on the mean field approximation (MFA), to explore the question stated above. In Section 3, we detail the design of our MAS numerical experiment. Section 4 then reports on the results and discussion. Section 5 then presents the final conclusions of this work.

2. Model depiction

Here, we build a MAS model based on the SIR process [32]; in this model N agents are placed at nodes (vertices) of an underlying network, and each agent is connected to other agents by links (edges). The underlying network indicates the nature of the physical contacts between agents through which an epidemic spreads [33–34]. An epidemic is triggered by some initial infected and infectious individuals spread across on this network, which is reproduced by the present model.

2.1. Physical network

We utilize a Watts–Strogatz Small-World (WS-SW) graph [35] as a topology with an average degree of either $\langle k \rangle = 16$ or 48 and a shortcut-probability of $p = 0.1$. We take $N = 10^5$ so as to reduce the finite effect of a domain size as much as possible. Although we obey the WS-SW algorithm, a two-dimensional lattice-like regular graph was assumed as a baseline graph rather than a one-dimensional ring, that is, when presuming $p = 0$. We take $\langle k \rangle = 48$ as well as $\langle k \rangle = 16$ because several articles in the literature report that a typical physical contacting network during an epidemic can be representing by $\langle k \rangle \approx 48$ (see, for example, Ref. [36]).

2.2. Disease spreading process

Here, we assume a SIR process, where the state of an infected and infectious agent is classified into one of two categories: an asymptomatic agent is denoted by $I_a$, and a symptomatic agent is denoted by $I_s$. This SIR process divides a population into five compartments: susceptible (S), infectious ($I_a$ and $I_s$), recovered (R), and quarantined (Q) individuals. Susceptible individuals become infected at a rate provided by the parameter $\beta$, which is the disease transmission rate per day per person induced by coming into contact with infectious individuals. If transmission occurs, the agent subsequently transitions to the infectious group of either $I_a$ or $I_s$, depending on time-constant fraction $a$ (or $1-a$). Individuals who are transferred into $I_a$ may be quarantined because they are symptomatic, while individuals in $I_s$ have no chance to quarantine. We assume that transmission from $I_a$ to Q takes place sporadically, since a testing is not always available. In practice, we assume the frequency of "testing," i.e., the frequency of transmission from $I_a$ to Q, is either once per day ($\tau = 1$ day), or four times per day ($\tau = 1/4$ day). The testing and detection rate which induces a transition from $I_a$ to Q is described by a time-constant value $q$. Individuals recover from the disease (i.e., transmission from either $I_a$, $I_s$, or Q to R) with a probability determined by parameter $\gamma$, the recovery rate per day.
(i.e., the inverse of the mean number of days that are required to recover from the disease). Referring to previous works (e.g., [34]), we take $R_0 = 2.5$ and $\gamma = 1/3$. Based on this, the actual transmission rate applied in our MAS model, based on Gillespie algorithm, is given by $\beta_{eq} = \beta/\langle k \rangle = R_0 \cdot \gamma/\langle k \rangle$.

If a symptomatic individual tested, detected, and asked to quarantine (i.e., the agent transmits from IS to Q), such specific agent severs all links he/she has until recovery occurs. This is the definition of “quarantine” used in this work, which implies perfect isolation because this quarantined (but infected) agent is not able to diffuse the disease in question to his/her neighbors.

Because our MAS model fully obeys to a stochastic process, we applied the Gillespie algorithm [37] in order to numerically simulate the SIR dynamics in the network described above. Fig. 1 shows a schematic of the state transfer used in our model.

2.3. Incompliant behavior to quarantine

An important aspect of the present model is the question of how incompliance of an individual who belongs to the compartment Q could be implemented. We define “incompliance” as a cheating behavior which leads to links to his/her neighbors to being maintained. In practice, we assume two situations.

2.3.1. Model 1 (agent-based incompliance)

At the timing of testing, $q \cdot IS(t)$ individuals are requested to quarantine. Here we take the incompliance fraction to be $f_i$. Amid those individuals, only $(1-f_i) \cdot q \cdot IS(t)$ agents sever all links they have making them entirely isolated; thus, these agents are labeled Q. By contrast, $f_i \cdot q \cdot IS(t)$ agents maintain all links, though they are also labeled as Q. This model can be referred to as “deep & narrow,” because all the links maintained that should have been removed are concentrated in a small number of incompliant agents. Fig. 2(b) shows a schematic of Model 1.

2.3.2. Model 2 (link-based incompliance)

At the timing of testing, $q \cdot IS(t)$ individuals are requested to quarantine. Each link of all those quarantined agents has an equal probability of remaining. A link remains with probability of $f_i$ and thus,
it is severed with probability of \(1 - f_i\). All those agents that are asked quarantine are labeled as \(Q\). This concept can be referred to as being “shallow & wide,” because all agents that are required quarantine may maintain links but sever other links. Fig. 2(c) shows a schematic representation of Model 2.

3. MAS experimental design

In the numerical experiment using the MAS model, an episode starts with \(N - l_0\) susceptible agents and \(l_0\) (= 5) infected and infectious agents; these infected agents have states of either asymptomatic or symptomatic that also determined by with probability \(a\) (i.e., \(l_0 = l_A + l_S = 5\)). The infected agents are randomly placed on the network. Each episode is chased its SIR dynamics until reaching to an equilibrium where none of infected individuals are. To obtain a set of statistically robust results for a given condition, we took the ensemble average over 10^2 independent realizations of the simulations using different initial conditions in terms of the location of the \(l_0\) infected agents in the network.

Despite analyzing various results obtained from the numerical experiment, we found that the final epidemic size, FES, i.e., the normalized sub-population of \(R\) observed at an equilibrium, the peak size of the infected population (i.e., the maximal sum of \(I_A, I_S, \) and \(Q\) in a the time series), and the total quarantined population (time integral of \(Q\) in the time series) are predominantly determined by two important model parameters: the asymptomatic fraction, \(a\), and the quarantine intensity, \(q\).

By setting the parameters such that all individual were totally compliant, i.e., \(f_i = 0\), we were able to validate our MAS result via comparison with the theoretical results based on the ODE model using a MFA, as below:

\[
\begin{align*}
S &= -\beta \cdot S \{l_A + (l_S - \dot{\theta}(t) \cdot q \cdot l_S)\} \\
I_A &= \beta \cdot S \{l_A + (l_S - \dot{\theta}(t) \cdot q \cdot l_S)\} \cdot a - \gamma \cdot I_A \\
I_S &= \beta \cdot S \{l_A + (l_S - \dot{\theta}(t) \cdot q \cdot l_S)\} \cdot (1 - a) - \gamma (l_A + \dot{\theta}(t) \cdot q \cdot l_S) \\
Q &= -\gamma (Q + \dot{\theta}(t) \cdot q \cdot l_S) \\
R &= \gamma (l_A + (l_S - \dot{\theta}(t) \cdot q \cdot l_S) + (Q + \dot{\theta}(t) \cdot q \cdot l_S) \\
\dot{\theta}(t) &= \begin{cases} 
1 & \text{if } t \mod \tau = 0; 1 \\
0 & \text{otherwise}
\end{cases}
\end{align*}
\]

Fig. 3. (a-1–a-3) The FES (top row), (b-1–b-3) the peak infected population (middle row), and (c-1–c-3) the total quarantined population (bottom row) for an asymptomatic fraction, \(a\), and the quarantine intensity, \(q\). These results are for \(f_i = 0\) and \(\langle k \rangle = 16\). The left (panels (a-1, b-1, and c-1)), center (panels (a-2, b-2, and c-2)), and right (panels (a-3, b-3, and c-3)) columns show the result of for \(\tau = 1\), \(\tau = 1/4\), and the subtraction of the results obtained for \(\tau = 1\) from those obtained for \(\tau = 1/4\), respectively.
4. Result and discussion

4.1. Effect of testing frequency without incompliance

We start by presenting the result where all agents are compliant to the request of quarantine, i.e., $f_i = 0$. We consider the effect of the testing frequency. We consider two cases: testing once per day ($\tau = 1$ day) or four-time per day ($\tau = 1/4$ day), and we consider how this affects the spread of disease on the complex network considered here. Figs. 3 and 4 give the results for $\langle k \rangle = 16$ and 48, respectively. In both figures, the top row (panels (a-1–a-3)), the middle row (panels (b-1–b-3)), and the bottom row (panels (c-1–c-3)), respectively display the FES, peak infection number, and total quarantined populations. Meanwhile, the left (panels (a-1, b-1, and c-1)), center (panels (a-2, b-2, and c-2)), and right (panels (a-3, b-3, and c-3)) columns show the results for $\tau = 1$, $\tau = 1/4$, and the results of a subtraction of the results of $\tau = 1$ from those obtained for $\tau = 1/4$, respectively. Hence, blue regions (indicating negative values) indicate a smaller effect of the epidemic modeled, that is, a smaller FES, less peak infected population, and less total quarantined people. By contrast, red regions (indicating positive values) show that high frequency testing brings a greater effect of the modeled epidemic, i.e., a larger FES, a larger peak infected population, and more total quarantine people.

Let us observe closely the general sensitivity to the asymptomatic fraction, $a$, and the quarantine intensity, $q$. Irrespective of the frequency of testing, the FES and peak infected increase with an increase in the asymptomatic fraction, and with a decrease in the quarantine intensity (see panels (a-1, b-1, a-2 and b-2) in Figs. 3 or 4). It is conceivable that this result is a consequence of the fact that the greater the proportion of asymptomatic infected individuals of all infected individuals is, the less effectively quarantine functions due to smaller fraction of “detectibly” infected people. Yet, the total quarantined does not show the same trend (see panels (c-1 and c-2) in Figs. 3 or 4). There are two regions with lower total quarantine (blue regions). These two regions have a different origin. The first region appears for high $q$ and low $a$; this region shows that a disease could be under threat of eradication. In fact, this region is consistent with the blue region observed in panels (a-1) and (a-2). Meanwhile, the second blue appears in the region with either extremely low $q$ or extremely high $a$, which is consistent with yellow-red region in panels (a-1) and (a-2). This region is caused by either extremely small number of quarantined individuals or extremely small number of “detectably” infected individuals. In summary, the first blue region (upper left) indicates that the quarantine working well, whereas the second blue region (lower right) signifies that the quarantine is malfunctioning.

In panel (c-3), we draw an dashed red curve where the subtraction of the results in panel (a-3) and (b-3) equals zero, This indicates the
curve of critical pair of values \((a, q)\) that indicate that a high frequency of testing is neither beneficial or disadvantageous. This critical curve of \((a, q)\) is then copied onto panels (a-3) and (b-3). Above the critical curve, a higher frequency of testing leading to infected individuals being quarantined is meaningful, since the FES, the peak infected population, and the total quarantined population can be reduced. Quarantine policy in this region of \((a, q)\)-space can be fully justified in terms of the social benefit. Below the critical curve, however, a higher frequency testing can be said questionable because the reduction in the FES and the peak infected are not so significant and the total quarantined population increases, which puts pressure on the social resources.

Comparing Fig. 3 with Fig. 4, we observe that there is no significant difference between the general tendencies, although the case of \((\langle k \rangle) = 48\) (Fig. 4) shows a decreased ability to reduce the spread of disease compared with that of the case in which \((\langle k \rangle) = 16\) (Fig. 3). This is because a network with a larger average degree leads to an increased spread of a disease.

Fig. 5 shows the result obtained from the ODE model described by Eq. (1) using an MFA; these results are visualized in the same format as that used in Figs. 3 and 4. The results of Fig. 5 are consistent in terms of trends with those of Figs. 3 and 4, although it is notable that Fig. 5 shows an easier spread of disease than that shown in Fig. 4. This is because the MFA implies an infinite degree of the network. The results discussed in this section can be reproduced almost perfectly using the ODE approach.

4.2. The effect of incompliance

Here we consider the cases when incompliant individuals are introduced to a society with a fraction \(f_i\). In the following discussion, we set the testing frequency to be once per day \((\tau = 1\text{ day})\). Figs. 6 and 7 shows the result in the case of \((\langle k \rangle) = 16\) and \((\langle k \rangle) = 48\), respectively. Top two rows labeled [I] (panels (a-1–a-5) and (b-1–b-5)), show the FES and the averaged ratio of incompliantly maintained links per quarantined agent for values of \(a\) and \(q\) in the case of Model 1 (agent-based incompliance), where incompliance fraction, \(f_i\), is set to be 0.2 (panels (a-1 and b-1)), 0.4 (panels (a-2 and b-2)), 0.5 (panels (a-3 and b-3)), 0.6 (panels (a-4 and b-4)), and 0.8 (panels (a-5 and b-5)).

We defined the averaged ratio of incompliantly kept links per quarantined as below:

\[
\frac{\text{(Incompliantly kept links)}}{\text{(Total quarantined individuals)}} = \langle k \rangle
\]

which indicates the extent to which a quarantined individual maintains links that should be severed. It is notable that this property does not depend on manner in which incompliance is introduced (either Model 1 or Model 2) and is not dependent on the average degree of underlying network. The middle two rows labeled [II] show the same conditions as those labeled [I] but represent the results for Model 2 (link-based incompliance). The bottom two rows, labeled
as [III], shows the subtraction of the results obtained using Model 1 (agent-based incompliance) from those obtained for Model 2 (link-based incompliance).

Here we consider Fig. 6, where $\langle k \rangle = 16$. Considering the top two rows, i.e., the case of Model 1 (agent-based incompliance), and comparing panels (a-1) to (a-5), we note that the increase in the incompliance fraction leads to a greater FES. The blue regions with lower $a$ values and higher $q$ values, which indicated disease eradication can be observed in panel (a-1) and are not present in panel (a-5). Such results are understandable because incompliance, irrespective of its extent, reduces the effect of quarantine. This fact is also true in the case of Model 2 (link-based incompliance) (Fig. 6, [II], panels (a-1) to (a-5)). Yet, comparing [I] and [II] of Fig. 6, (Fig. 6, [III], panels (a-1) to (a-5)), we clearly see that the link-based incompliance boosts the FES compared with the agent-based incompliance, which is indicated by red region. This finding is one of the most important findings of the present study: the

fig. 6. Top two rows labeled by [I]: panels (a-1–a-5) and (b-1–b-5), show the FES and the averaged ratio of incompliantly maintained links per quarantined agent for a given $a$ and $q$ under the assumptions presented as Model 1 (agent-based incompliance) for an incompliance fraction, $f$, of 0.2 (panels (a-1 and b-1)), 0.4 (panels (a-2 and b-2)), 0.5 (panels (a-3 and b-3)), 0.6 (panels (a-4 and b-4)), and 0.8 (panels (a-5 and b-5)). Middle two rows labeled [II] show the results equivalent to those presented in [I] but under the assumptions of Model 2 (link-based incompliance). Bottom two rows (labeled [III]) show the subtraction of the results for Model 2 (link-based incompliance) from those obtained using Model 1 (agent-based incompliance). Results shown are for $\langle k \rangle = 16$ and the testing frequency of once per day ($\tau = 1$ day).
situation of all the people requested to quarantine being slightly incompliant, i.e., keeping some (not all) social links that should be severed, reduces the resistance to disease spreading more than the existence of limited number of completely incompliant people, who completely ignore the requirement of quarantine, i.e., never reduce their social activity at all. This remains true even when the averaged ratio of incompliantly kept links per quarantined agent in the case of agent-based (Fig. 6, [I], panels (b-1–b-5)) is almost equal to that in the case of link-based incompliance (Fig. 6, [II], panels (b-1–b-5)), which is confirmed by Fig. 6, [III]; panels (b-1–b-5). Even for a given average ratio of incompliantly kept links, a less concentrated spatial distribution of maintained links (link-based incompliance) increases the spread of a disease considerably more than the case of spatially concentrated set of incompliantly maintained links (agent-based incompliance). Thus,

Fig. 7. Top two rows labeled [I]: panels (a-1–a-5) and (b-1–b-5), show the FES and the averaged ratio of incompliantly maintained links per quarantined agent for a given $a$ and $q$ under the assumptions of Model 1 (agent-based incompliance), where the incompliance fraction, $f_i$, is equal to 0.2 (panels (a-1 and b-1)), 0.4 (panels (a-2 and b-2)), 0.5 (panels (a-3 and b-3)), 0.6 (panels (a-4 and b-4)), and 0.8 (panels (a-5–b-5)). Middle two rows (labeled [II]) present the equivalent results to those presented in [I] but for the conditions of Model 2 (link-based incompliance). Bottom two rows (labeled [III]) show the subtraction of the results obtained for Model 2 (link-based incompliance) from those obtained for Model 1 (agent-based incompliance). In this figure, $\langle k \rangle = 48$ and the testing frequency of once per day ($\tau = 1$ day).
“deep & narrow” incompliance may be preferable to “shallow & wide” incompliance.

Comparing Fig. 6, [Ill]; panels (a-1) to (a-5) with the results shown in the equivalent panels of Fig. 7, the worst incompliance fraction displaying widest (and deepest) red region (hereafter, called the critical incompliance fraction) is different. The case of $\langle k \rangle = 16$ (Fig. 6) appears its critical incompliance fraction for $f_i = 0.5$ or 0.6, whereas in the case of $\langle k \rangle = 48$ (Fig. 7), the critical is observed for $f_i = 0.4$ or 0.5. Under a higher incompliance fraction than the critical incompliance fraction, the red region becomes smaller in extent (in fact, both Fig. 6 and Fig. 7, [Ill] panel (c-5), in which $f_i = 0.8$, shows the thinnest red region among all the panels (b-1–b-5) panels) and there is less difference between agent-based incompliance and link-based incompliance. It is hypothesized that this is because high levels of incompliance means there is little difference between “deep & narrow” and “shallow & wide.” It is also expected that the critical incompliance fraction becomes less with an increase in the average degree because a network with a higher average degree becomes vulnerable to disease spreading due to densely connected, thus being more sensitive to incompliantly keeping links.

5. Concluding remarks

COVID-19 has demonstrated that quarantine is a fundamental and important intervention policy as well as other possible intermediate measures like washing hands and so forth, particularly when there is no adequate pharmaceutical treatment. Motivated by this, we built a MAS model based on the SIR process where sporadic testing to transform infected and infectious individuals to quarantine could be evaluated, and the effect of incompliance with quarantine requests could also be quantified. We considered two types of incompliant behavior across the network. One was incompliant individuals who ignored the requirement for quarantine completely, this was assumed to be behavior undertaken by a relatively small number of people. The second incompliant behavior was a relatively large number of individuals who were slightly incompliant with the request for quarantine; these individuals maintained a small fraction of social links but other links were severed. Our results revealed that depending on the asymptomatic fraction and the quarantine intensity, there is a certain parameter region where a higher frequency of testing cannot be justified to confine disease. We found that a quarantine policy malfunctioned if incompliantly maintained links were more widely distributed across the network rather than being concentrated into small number of highly incompliant individuals.

Although other non-pharmaceutical preventive measures are really considered in real world and implementing them simultaneously would bring better situations rather than just imposing a quarantine provision, we do believe that our study helps public authorities designing a quarantine policy that minimizes the negative impact of an epidemic.

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**CRediT authorship contribution statement**

Shinobu Utsumi: Methodology, Software, Validation, Investigation, Data curation, Formal analysis, Visualization, Writing-Original Draft, Writing-Review & Editing.

Md. Rajib Arefin: Methodology, Validation, Investigation, Formal analysis, Writing-Review & Editing.

Yuichi Tatsukawa: Methodology, Software, Validation, Investigation, Formal analysis, Writing-Review & Editing.

**Jun Tanimoto:** Conceptualization, Formal analysis, Resources, Writing-Original Draft, Writing-Review & Editing, Supervision, Project administration, Funding acquisition.

**Declaration of competing interest**

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