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A CONTACT-NETWORK-BASED SIMULATION MODEL FOR EVALUATING INTERVENTIONS UNDER "WHAT-IF" SCENARIOS IN EPIDEMIC

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ABSTRACT

Infectious disease pandemics/epidemics have been serious concerns worldwide. Simulations for public health interventions are practically helpful in assisting policy makers to make wise decisions to control and mitigate the spread of infectious diseases. In this paper, we present our contact network based simulation model, which is designed to accommodate various "what-if" scenarios under single and combined interventions. With the incorporation of parallel computing and optimization techniques, our model is able to reflect the dynamics of disease spread in a realistic social contact network based on Singapore city, simulating combined intervention strategies as well as control effect at different levels of a social component. The framework of our model and experimental results show that it is a useful tool for epidemiological study and public health policy planning.

1 INTRODUCTION

Public health interventions are necessary for containing the spread of infectious diseases. In order to determine the most appropriate intervention, the evaluation of the effectiveness of interventions under different "what-if" scenarios is desirable. Simulation models, especially network-based models, have been widely used in epidemiological studies to discover insights of disease spreading and develop robust estimation. Among many simulation models, those incorporating intervention strategies are particularly helpful in assisting policy makers to make wise decisions for containing or mitigating the spread of infectious diseases. For example, school closure is a common social-distancing measure for influenza intervention. Its primary objective is to reduce infection attack to school children but with high costs in both economic and social aspects (Cauchemez et al. 2009; Sadique, Adams, and Edmunds 2008) and there is still open discussion on the effectiveness of school closure in the scientific community (Cowling et al. 2008). To investigate school closure under different settings, simulation approach is indispensible. According to Google Scholar as of 1 April 2012, there are 248 publications using simulation approaches to examine the effectiveness of school closure for the 2009 H1N1 Influenza pandemic alone, covering different factors and different communities around the world. For instance, Lee et al (Lee et al. 2010) discuss the scale of school closure as well as impact of the duration of school closure on the delay of epidemic peak in Allegheny County, US; Zhang et al (Zhang et al. 2010) systematically describe how starting threshold and duration of school closure affect the outcome of school closure in Singapore; Miller et al (Miller et al. 2010) and Halder et al (Halder, Kelso, and Milne 2010) elaborate the impact of compliance rate and disease severity to school closure in Boston U.S. and in Albany Australia respectively.

Simulating public health interventions is more complicated than a typical simulation of disease spreading. In addition to simulating stochastic propagation within a huge population, interventions introduce more volatility into the system by altering interconnecting contacts between persons in runtime. It often requires a number of iterations to explore the different settings for the particular factor of interest. It would become even more complex if simulating combined interventions, as the number of combinations of setting-pairs is the multiplication among all interventions. Furthermore, many social components like schools, hospitals and etc have their inherent hierarchical structure, e.g. classes in a school, wards/clinics in a hospital. It might lead to different outcomes when the interventions to the different level of social components are applied, raising the questions like "what happens if we close the class, close the school, or close the entire school system?" However, There is little mention on how to simulate a complex public health intervention, especially for combined interventions as well as multiple levels of social components could be targeted. Although there are a few infectious disease simulation models (Del Valle et al. 2006; Bisset et al. 2009; Barrett et al. 2008) in the literature. Most of the discussions about those models focus on discrete event simulation as well as parallelization in a distributed architecture.

There is no doubt that simulating complex interventions requires the extra effort and therefore a suitable tool or framework is desirable for facilitating the studies. In this paper, we discuss our contact network-based simulation model to present a systematic view of simulating complex interventions efficiently.

2 METHOD

There are four components in our simulation model: contact network generator (CAG), intervention measure builder (IMB), epidemic simulator (ES) and intervention evaluator (IE) as shown in Figure 1. CAG produces the contact network that represents persons (nodes) as well as the potential disease transmission paths among them (connecting edges) in a community. IMB creates the list of measures with the specifications defining how they can impact the disease transmission process. ES simulates both disease transmission between nodes (infection) under the effect of interventions and disease progression at host node (i.e. susceptible → exposed → infectious → …) within the generated contact network. Finally, IE monitors the state of individual nodes in ES, compute the statistics and performance scores of the interventions, and then report the prevalence rates back to ES for determining the trigger status of the interventions at the next time step.

As our contact network generator has been reported (Zhang et al. 2009), we shall focus the other three components in this paper.

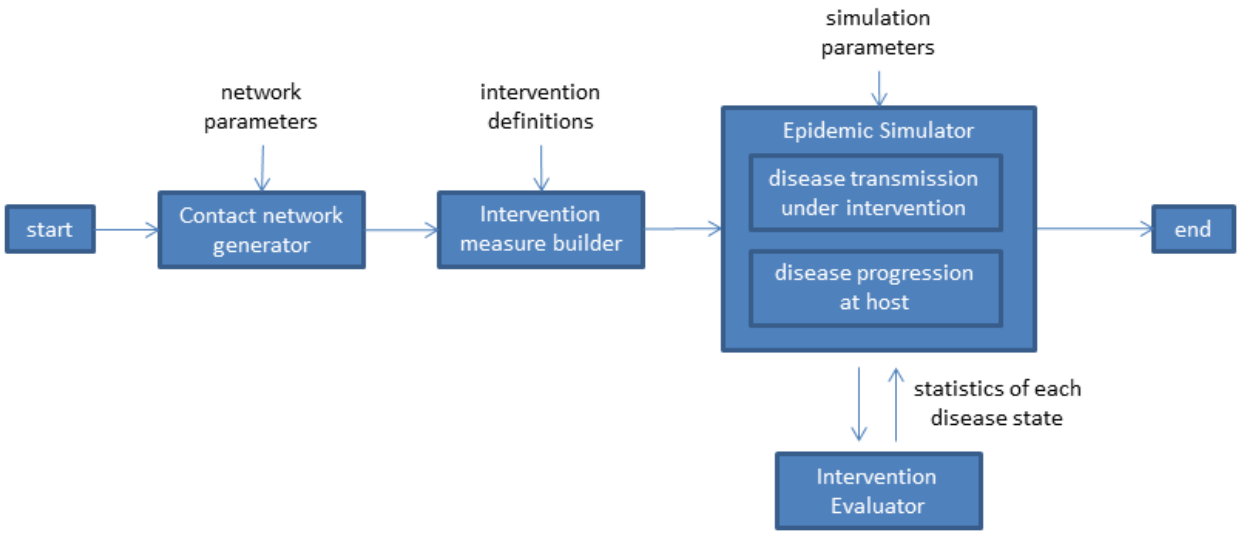

Figure 1: Structure of Simulation Model

2.1 Intervention Measure Builder

Public health interventions are comprised of pharmaceutical and non-pharmaceutical interventions. Pharmaceutical interventions refer to antiviral treatment and vaccination mostly; non-pharmaceutical interventions refer to social distancing as well as hand washing, face mask, etc those may reduce transmissibility of the disease. Both types of interventions can be defined by four types of parameters: *trigger threshold*, *duration*, *control target*, *efficacy*.

- Trigger threshold: when an intervention starts. e.g. 2 days from the date of patient-zero; 1% of whole population are infected; 5% of population in a class/school/office/etc show symptoms.
- Duration: how long an intervention lasts. e.g. school closure for 2 weeks; wearing face mask until epidemic ends; periodic duration like staying home in every other week.
- Control target: who an intervention affects. e.g. 80% of population is vaccinated; 100% of symptomatic patients are treated with antiviral drugs; schools/classes with more than 5 incidences are closed.
- Efficacy: the possibility that an intervention can produce the desired result. e.g. the vaccine has 80% efficacy; 10% of student contacts remain after school closure.

User can easily define the desired interventions by the above parameters and provide them in a format of text file to the simulation model. An internal parser is then used to interpret the user inputs and to create a list of measures. The builder also supports to create the special types of interventions. A combined intervention can be defined as the separate entries in a single text file. The levels of a hierarchical social component is indexed by 0, 1, 2, …, e.g. in the school component, level-0 refers to all-schools, level-1 refers to individual schools and level-2 refers to individual classes. So by specifying level-0/1/2 in *control target*, user can indicate the desired control level.

Giving a range instead of a specific value to a particular parameter can create a batch of interventions. For example, set *duration* to "2:1:5 weeks" is to create 4 interventions of 2, 3, 4 and 5 weeks respectively. This batch creation feature could be a great help in building up a mass of simulation scenarios for complex interventions.

2.2 Epidemic Simulator

2.2.1 Disease Progression at Host

Disease Progression refers to the process of infectivity or illness development of certain disease within the host person. Figure 2 shows a disease progression diagram for influenza. Any susceptible person has a chance (transmission probability) to be infected by his or her infectious contacts. If the person (denoted as *p*) is infected, *p* is firstly exposed but has no infectivity or any symptom yet. After the latent period, *p* becomes infectious. As we assume incubation period is equal to latent period for influenza, immediately after *p* is infectious, *p* has a chance (symptomatic rate) to develop the clinical symptoms of influenza and turn into symptomatic infectious; or turn into asymptomatic infectious otherwise. After the infectious period, *p* is finally removed, i.e. either recovered from influenza or dead.

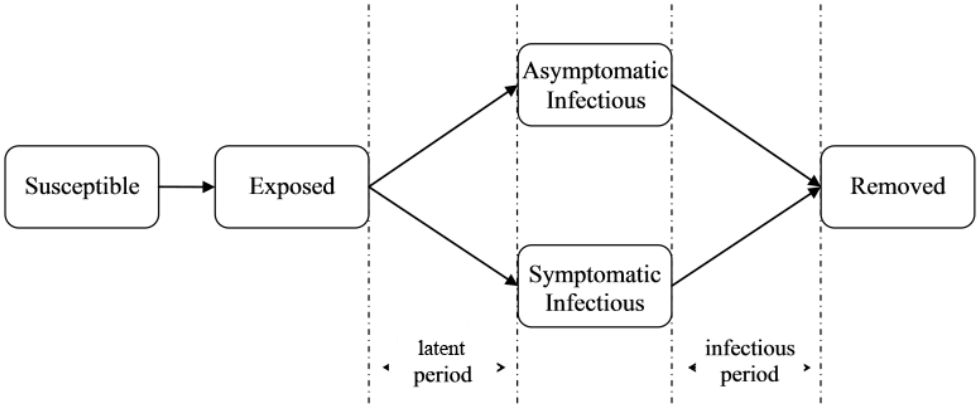

Figure 2: Disease Progression Model for Influenza

2.2.2 Disease Transmission under Intervention

Figure 3 illustrates the disease transmission process under interventions. A disease transmission process starts when an infectious node attempts to attack a susceptible node through their interconnecting edge. The first step is to check the state of the edge. The state is either *open* or *blocked*, where *blocked* means the edge stops transmitting disease due to intervention for a certain period of time; *open* refers to the state with no intervention. If the edge is *open*, the applicable interventions are checked against their trigger thresholds to determine the new state of the edge. If the new state is still *open*, the disease is passed to attack the susceptible node; otherwise the transmission stops and the *period of blocked* (*T*) of the edge is updated accordingly. A *blocked* edge will revert to the *open* state after a period of *T* elapses.

When the susceptible node is attacked, a transmission test is carried out by generating a random number *r* and compare *r* against the transmission probability *tp*. If $r < tp$, the susceptible node will be set to *exposed*; otherwise remains *susceptible*.

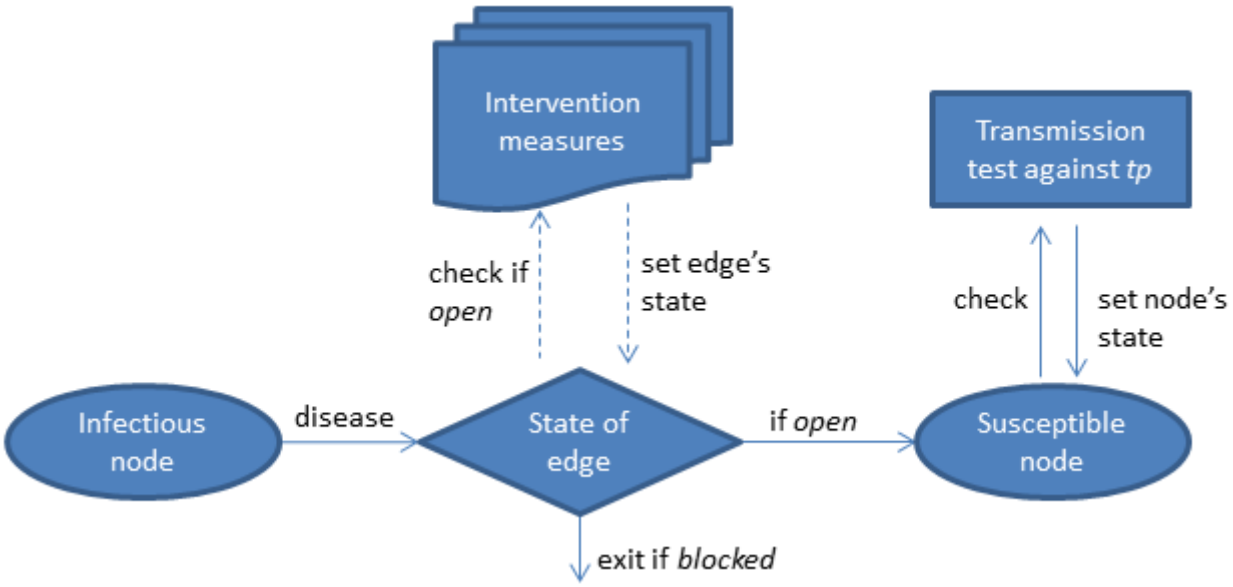

Figure 3: Disease Transmission Process

2.2.3 Weekend Effect, Self-Quarantine and Self-Protection in Epidemic Dynamic

It is known that the structure of contact network might change significantly during the weekends. According to our survey conducted in Singapore in 2009, during the weekend, 26.4% more people visit shopping malls; 50.4% of students meet their classmates/schoolmates; 30.1% of working adults contact their colleagues, including those working in the weekend. We model the weekend effect by adding 3 additional intervention measures to school, workplace and shopping-mall components respectively to simulate its impact to network structure. Those interventions are triggered at the beginning of simulation, and operate in a recurring manner with a period of 7 days.

Self-quarantine or self-protection is another phenomenon that should not be neglected in the epidemic simulations. When a person turns symptomatic, he or she might stay home voluntarily to avoid the unnecessary contacts with others because of social responsibility. It is called as "self-quarantine". Or many people intend to avoid the contacts with a sick person (symptomatic) for "self-protection". We simulate both self-quarantine and self-protection in our model by randomly cutting off the number of contacts by a specific rate for any symptomatic nodes.

2.3 Intervention Evaluator

The evaluator monitors the state of contact network nodes during the process of epidemic simulation. At each time step, the new and cumulative headcounts of each disease state either globally or within some social component at different levels (depends on control target of interventions) are updated. Those headcounts are used to compute the disease prevalence rates for testing if trigger thresholds of interventions are reached.

The headcounts are also used to compute the performance scores of the interventions. The goal of our simulations is to evaluate the effectiveness on mitigating the spread of disease. In order to measure and compare, we need to define the measurement for effectiveness in public health intervention. In our model, we adopt the common metrics in the literature (Ferguson et al. 2006): attack rate (AR), peak incidence (PI), and peak day (PD). Attack rate refers to the cumulative proportion of symptomatic cases in the overall population; peak incidence refers to the highest number of the daily incidence of symptomatic cases;

peak day refers to the day when the peak incidence happens. In the public health perspective, attack rate indicates the size of epidemic and the overall burden on the public health system due to an epidemic; and peak incidence and peak day display the challenge to an effective response to patient surges in public health system.

There is an additional feature in the model to allow users to aggregate AR, PI and PD to a single score by user's weighting to the three measurement. The single score is useful in evolutionary search for the optimal intervention in a fixed setting.

2.4 Parallelization and Optimization

As we mentioned, intervention simulations are more computationally intensive than standard epidemic simulations as there are a number of intervention scenarios to be simulated and the computation workload is multiplied by the number of scenarios. As a result, parallelization and optimization techniques that improve the speed of simulation are indispensible to ensure reasonable turnaround time.

We adopt two-level parallelization in our system. The first level is multi-threading within a single simulation (Figure 4). Multiple threads can be created to exploit the advantage of modern multi-core processors by running the simulations concurrently. The underlying population is clustered by their interconnections and distributed to individual threads to reduce cross-thread synchronization. Each thread is responsible for simulating the disease transmission and progression within the assigned subpopulation. A system-wide synchronization happens at the beginning of each round of time-step update.

The theoretical speedup of multi-threading is n, where n is the number of simulation threads. The value of n is usually limited by the number of CPU cores available. Moreover, there is the overhead for cross-thread synchronization. For example, thread A requests to access people P while thread B is working on P as well, A will have to wait until B finishes. The wait time and all other related cost of computation time are the overhead for cross-thread synchronization.

Other than multi-threading, Message Passing Interface (MPI) is an alternative option for parallelization. MPI takes advantage of workstation clusters and its parallel processes are not constrained by an upper bound as long as there are adequate workstations to be connected to the cluster. However, MPI suffers more significant overhead as its communication between parallel processes is subject to the speed of cluster network and IO interface, which are much slower than memory bus that is used in cross-thread synchronization. Therefore, MPI-based epidemic simulations often require more sophisticated network partitioning scheme to minimize the overhead; however, the complicated partitioning itself is a non-trivial overhead too.

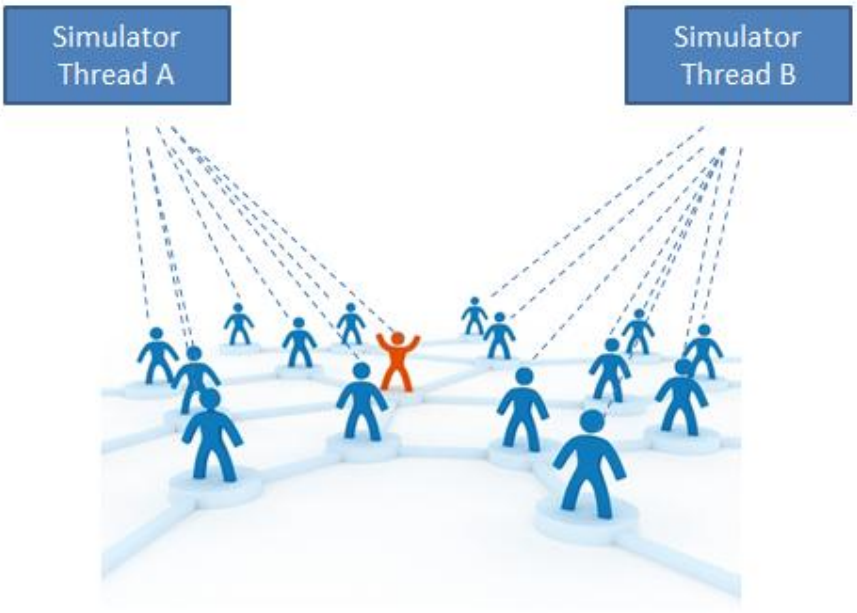

Figure 4: Network partition for multiple threading

In order to overcome the constraints of multi-threading, we implement the application-level parallelism. As multiple intervention scenarios are to be simulated and those tasks are highly independent to each other, it is intuitive to run the simulations for individual scenarios simultaneously. Instead of using MPI, we take advantage of the job-scheduling feature which is commonly available in large workstation clusters, for example, Platform LSF scheduler. Our simulator is able to submit the jobs of simulating individual intervention scenario to the scheduler. The scheduler will allocate those jobs to separate multi-core machines within the cluster by specifying number of cores required by each job. The simulator will monitor the file system and collect the result files back upon the simulations finish.

In addition to parallelization, we also adopt two optimization techniques to speed up the simulation. The first approach is active node switching (Jin et al. 2010). There are two ways in which to execute the infection process: take an infectious node as the active node, and attack all of the susceptible nodes in its contacts; or take a susceptible node as the active node, and acquire the disease from all of the infectious nodes in its contacts. Assume n infectious nodes with average degree k_n and m susceptible nodes with average degree k_m in a contact network, and constant cost c of visiting a contact, the cost of simulating disease propagation is either $n \times k_n \times c$ or $m \times k_m \times c$. So when $n \times k_n > m \times k_m$, it is wise to make susceptible nodes as the active nodes; and vice versa. The simulator is able to monitor n, m, k_n and k_m and switch the active nodes for lower computation cost.

Fixed fan-out (D'Angelo and Ferretti 2009) is another optimization technique to reduce computation cost for a large number of random number generation. Random numbers are essential to simulate a stochastic process of disease transmission. If a disease has the transmissibility of tp, to simulate the outcome of infection, a random number will be generated and compared with tp: infected if less than tp and uninfected otherwise. Take an example to illustrate how fixed fan-out works. Assume the cost of generating a random number is c, an infectious node is active and has n susceptible nodes in its contacts. Then $n \times c$ is the total cost of random number generation. For fixed fan-out, first we determine the number of successful infections (fan-out) by $\| n \times p \|$, then iteratively select one from the list of susceptible nodes and remove it from the list until the fan-out number is reached. The total cost of fixed fan-out is $(\| n \times p \| + 1) \times c$ and its speedup is $\frac{n}{\|n \times p\| + 1}$ compared to the standard approach.

3 CASE STUDY

We develop the intervention simulation model in JAVA for its built-in support for multi-threading and platform independence. The hardware platform is a workstation cluster of 32 octa-core machines with the installed Platform LSF scheduler.

In our previous work (Zhang et al. 2012), we applied the simulation model to investigate the impact of temporal factors (i.e. trigger threshold and duration) in the combined interventions of school closure and workforce shift for mitigating the spread of influenza in Singapore. We created a synthetic population of 480,000, one tenth of real Singapore population, with the approximate demographic composition and individual contact patterns. Six types of community structures were modeled, including household, hospital, school, workplace, shopping mall and public transport. While accessing those community structures, people come close to each other in a short physical distance, and form the potential transmission channel (contacts) for airborne diseases like influenza.

School closure and workforce shift are both social distancing interventions, whose primary objective is to reduce people-to-people contacts so as to mitigate the spread of disease. Compared to school closure, workforce shift intervention is a new scheme. It requires a portion (work team) of workforce is scheduled away from workplaces for a certain time span and then return by shifting with others.

Table 1 lists the parameters used in those simulations (Zhang et al. 2012). "Shift length" is a special parameter designed to support periodic interventions. For example, 4-week workforce shift with 7-day length of shift means team *A* works in the 1st and 3rd week; and team *B* works in the 2nd and 4th week (two work-teams are assumed). For influenza transmissibility, by using Longini's approach (Longini et al. 2005), we approximated the $R_0 \approx 1.9$ and picks two more values 1.5 and 2.3 for sensitivity test.

Table 1: Intervention Scenario Parameters

Parameters	**School Closure**	**Workforce Shift**
Trigger Threshold	0.02%, 0.25%, 1.5%, 5%	0.02%, 0.25%, 1.5%, 5%
Duration	2,4,6,8,10 weeks	2,4,6,8,10 weeks
Target	all school contacts	all workplace contacts
Efficacy	100%	100%
Shift length	NA	7 days

For each simulation scenario, the simulation starts from day 0 with 10 infectious persons seeded into a susceptible population without prior immunity to the influenza virus. The simulation is repeated 100 times to produce a robust result. In our experiments, there are 4 different trigger thresholds and 5 different durations. Hence there are a total of 121 scenarios per R_0: 1 control scenario (without any intervention); 20 scenarios for school closure and workforce shift each; and 80 scenarios for the combined interventions (we assume that the individual interventions in each combination scenario share the same length of duration). In total, there are 363 scenarios in which 36,300 epidemic simulations are included in that study.

The results of that study have been reported (Zhang et al. 2012). By using contact-network-based simulations, we discover that there exists an upper bound of the duration of either school closure and workforce shift, further extension beyond which will not bring additional benefits to suppressing the attack rate and etc. We also found the combination do not always outperform individual interventions and are more effective only when the duration is longer than 6 weeks or school closure is triggered at the 5% threshold; combined interventions may be more effective if school closure starts first when the duration is less than 4 weeks or workforce shift starts first when the duration is longer than 4 weeks.

Furthermore, using the same configurations and experimental setup, we study the different effectiveness of school closures at different levels. There are three types of school closures included in this study:

class closure (close individual class in which the trigger threshold is met); school closure (close individual school in which the trigger threshold is met); all-schools closure (close the entire school system when population-wise trigger threshold is met).

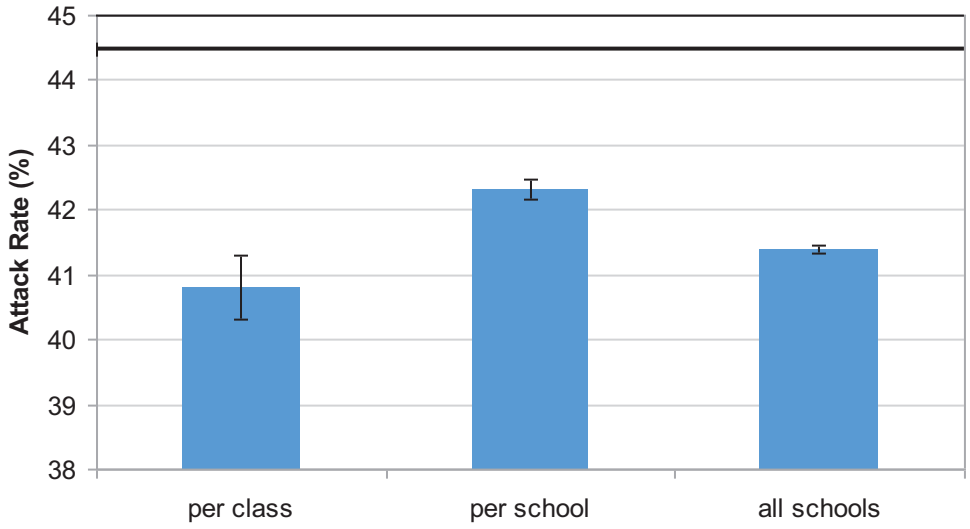

Figure 5: Attack Rates for 6-Week School Closure at Different Levels (5% trigger threshold; black horizontal line is the baseline without any intervention; error bars indicate 95% confidential intervals)

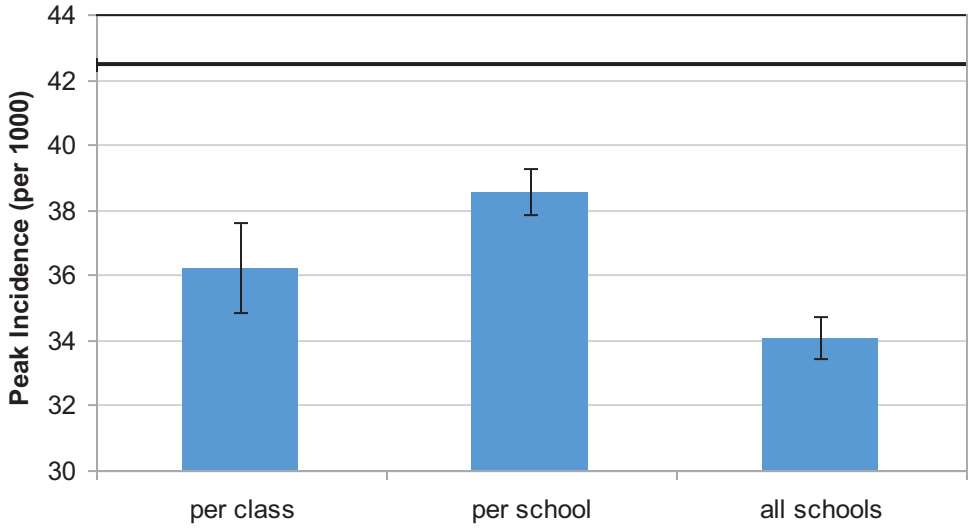

Figure 6: Peak Incidences for 6-Week School Closure at Different Levels (5% trigger threshold; black horizontal line is the baseline without any intervention; error bars indicate 95% confidential intervals)

As shown in Figure 5 and 6, class closure is the most effective measure on reducing attack rate (8.24% of reduction); whereas all-schools closure is the most effective on reducing peak incidence (19.71% of reduction). It is interesting to observe the tradeoff between the scale and frequency of the intervention. In the model, there are total 33 schools and 1531 classes. For all-schools closure, it closes all 33 schools at once for 6 weeks, which significantly cut down the peak incidence by massive removal of

contacts but there is no intervention after 6 weeks in the system. For school closure, it removes a smaller number of contacts but it may be triggered up to 33 times. Although some schools may trigger the closure on the same day, the aggregated length (from the start of first closure to the end of last closure) is more than 6 weeks. Apparently, the extended period of closure cannot compensate the loss of intervention scale for school closure. But it works for class closure which may be triggered up to 1531 times. Such higher frequency of closure in a very limited scale results in a lower final attack rate and less economic and social impact; however, class closure tends to prolong the epidemic period despite with lower prevalence, i.e. the epidemic period last over 150 days under class closure compared to 73 days in baseline without any intervention.

Although the above results of single simulation scenario are not comprehensive to draw any conclusion on multiple-level school closure, they demonstrated the capability of our model to simulate the closure of community structures at their different hierarchical levels. Incorporating this feature with temporal variations (trigger/duration) and combination of interventions, our model is able to simulate more complicated scenarios for epidemiological studies.

4 CONCLUSION

Our epidemic simulation model is designed to evaluate the effectiveness of complex interventions including combined measures as well as interventions to different levels of social components in an efficient manner. It may serve a helpful tool to carry out epidemic experiments with different intervention settings, leading to time-saving and simplicity in epidemiological studies and ultimately providing scientific evidences for making wiser decisions in public health policies.

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