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A Social Structure Model for Evaluating the Effect of Response Measures on the Spread of Smallpox

by

Moshe Kress

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13. ABSTRACT ***(maximum 200 words)***

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A Social Structure Model for Evaluating the Effect of Response Measures on the Spread of Smallpox

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Abstract

Homogeneous mixing, where all instances of contacts between any two members of the population are equally likely, is a common assumption in modeling biodefense policies against smallpox. Such a mixing pattern is rather unlikely to represent population interaction in a modern urban setting, which typically is separated into households on one hand, and into daily meeting sites such as schools and offices, on the other hand. In this paper, we develop a dynamic two-level social interaction model where individuals move back and forth between home and daily meeting sites, possibly passing through a general meeting site such as a mass transit system or other crowded areas. Based on this difference-equations model, we evaluate the effect of situational awareness and response measures, such as vaccination, quarantining, and movement restrictions, on the spread of smallpox in the population.

1. Introduction

Responding to a bioterror attack of smallpox has become a major concern to governments, local public officials, and health authorities. This concern has been reflected in studies that model and evaluate possible response policies against smallpox [1]-[8]. A common assumption in these models (e.g., [1]-[5]) is homogeneous mixing, where all instances of contacts between any two members of the population are equally likely. In other words, interactions in the population are uniformly random. The main implication of this assumption is that interaction probabilities among individuals depend only on the relative size of the susceptible and infectious populations—a feature that simplifies the analysis.

Such a mixing pattern is quite unlikely to represent actual interactions in an urban setting where the population is typically divided into interconnecting subsets. Halloran et al. [6] present a heterogeneous mixing simulation model for smallpox where *ad hoc* social structure is considered. The model is applied to a small population of 2,000 people. An excellent review of recent smallpox models is by Furgeson et al [7]. A number of studies examine nonhomogeneous mixing in other epidemic settings. Some *ad hoc* social mixing patterns are studied in [9] and [10]. Ball and Lyne [11] consider a population partitioned into households, with local mixing within households and global mixing throughout the population, and develop a vaccination optimization model. The effects of a similar social structure are studied by Koopman et al [12]. Other heterogeneous mixing models have been studied by Kaplan with respect to the AIDS epidemic [13] [14].

The concept of small world networks [15] [16] is utilized by several researchers to model nonhomogeneous transmission in a population [17]-[19]. Eubank et al. [18] develop a detailed large-scale urban traffic simulation, and find that interactions among people form a strongly connected small-world-like graph. They examine several response policies and conclude that outbreaks can be contained by a combination of targeted vaccination and early detection.

In this paper we develop a two-level social interaction model that consists of households and other daily meeting sites such as schools, offices, and mass transit

systems. This SIR-based difference-equations model captures dynamic features of daily contacts among individuals in a major urban area. We apply this model to a large urban area (9 million people) and evaluate the effect of situational awareness (early detection and response) and several response measures, such as mass vaccination, quarantine, closure, mass-transit shutdown, and voluntary self-quarantine on the spread of the epidemic and on the total number of casualties.

The rest of the paper is organized as follows. Section 2 describes the social structure that forms the base for our model and analysis. Section 3 outlines the stages of the epidemic and discusses possible response actions. The two-level model is described in Section 4. In Section 5, we report the results of the analysis that is based on our model. Discussion and concluding remarks are presented in Section 6. A detailed description of the difference-equation model is given in the Appendix.

2. The Social Structure

We assume that during each time period (i.e., a day) a person interacts with other persons mainly in two places: in the *household* (HH) and in the *daily meeting site* (DMS), such as school or workplace. There may also be incidental contacts in public places such as mass transit systems, restaurants, or theaters. We consider these contacts as occurring in a *general meeting site* (GMS). During the course of a day, a person is in (close) contact people with a relatively small number of individuals in the HH, then she meets colleagues, fellow students, or coworkers in the DMS, and finally she may also contact (mostly strangers) in the GMS. Figure 1 presents this interaction pattern.

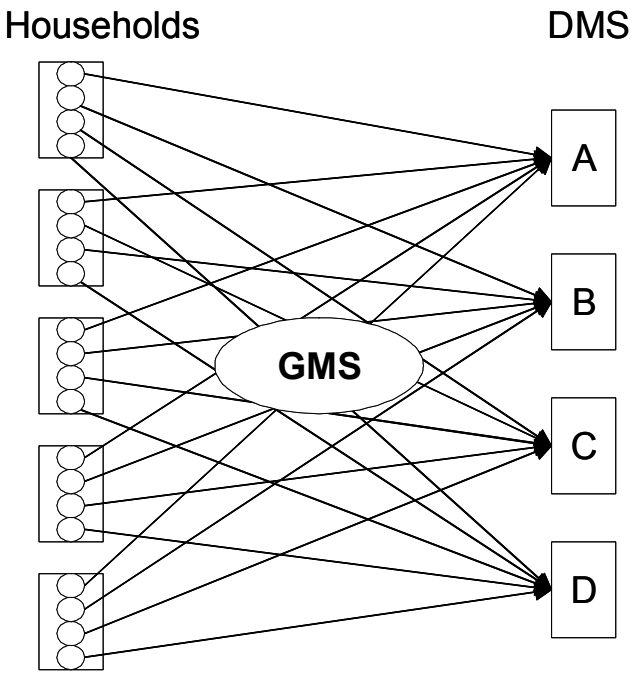

Figure 1: Interaction Pattern in a Two-Level Social Structure

Thus, we assume that the population is divided into m HHs of size h each. There are k DMSs, $k > h$, and one GMS. On each day, members of a HH visit certain DMSs. We assume a worst-case scenario, from the epidemic spread point of view, where the DMSs are chosen randomly and independently by HH members each day. We also assume that effectively no two members of a certain HH visit the same DMS, e.g., a mother does not visit the school (at least not for a significant length of time) and her child does not visit her workplace. Thus, members of the same HH do not interact in a DMS. They interact in the HH and possibly in the GMS (e.g., if both use the mass transit system on a certain day). Within each subset of population—HH, DMS, and GMS—we assume homogeneous mixing. However, the contact rates (and hence the transmission rates of the disease) are different in the three environments; the transmission rate is highest in a HH and lowest in the GMS.

The spread of the epidemic is observed at discrete time periods (days). Each time period is divided into two parts: the *HH subperiod* and the *DMS subperiod*. During the HH subperiod individuals stay in their respective HHs (homes), while during the DMS subperiod they are present in their respective DMSs (workplace, school, etc.). Some individuals may visit also the GMS in-between the two periods. The state of the epidemic in the HHs and DMSs is monitored at four points during a time period: at

the beginning and end of the HH subperiod, and at the beginning and end of the DMS subperiod. At the beginning of the HH subperiod, we observe the state of the members of a HH after they return from the DMSs and possibly GMS, and at the end of that period, we observe the transitions that have occurred in the HH during the HH subperiod. Similar observations apply to the DMS subperiod.

3. The Epidemic and Possible Response Actions

The stages of the epidemic are *S*, *A*, *B*, *I*, and *Q* for susceptible, infected and *immunable* (vaccine sensitive), infected and *nonimmunable*, infectious and quarantined. A HH is said to be *infected* if at least one member in the HH is infected, but no one is infectious (symptomatic). An infected HH may be *immunable* (denoted *type A*) if all of its infected members are immunable. Otherwise, it is *not-immunable* (*type B*). Clearly, some members in a nonimmunable infected HH (type *B*) may be susceptible (at stage *S*) or infected and immunable (stage *A*). A HH is said to be *infectious* (*type I*) if at least one member in the HH is or has been infectious, and it is said to be *quarantined* (*type Q*) if it has been put in quarantine. Otherwise, a HH is said to be *susceptible* (*type S*). We assume that vaccination and quarantine are applied to HHs and not to individuals. We assume perfect vaccination efficacy, therefore, susceptible or infected-immunable HHs that are vaccinated are removed from further consideration. Once an infectious individual is detected, his entire HH is quarantined. If that HH has not been previously vaccinated, all asymptomatic members are vaccinated upon entering the quarantine. Only infectious HHs are quarantined.

HHs of types *B* and *I* that are vaccinated are labeled *BV* and *IV*, respectively. Also, *QV* denotes a quarantined HH that has been previously vaccinated. Figure 2 presents the transitions among the stages.

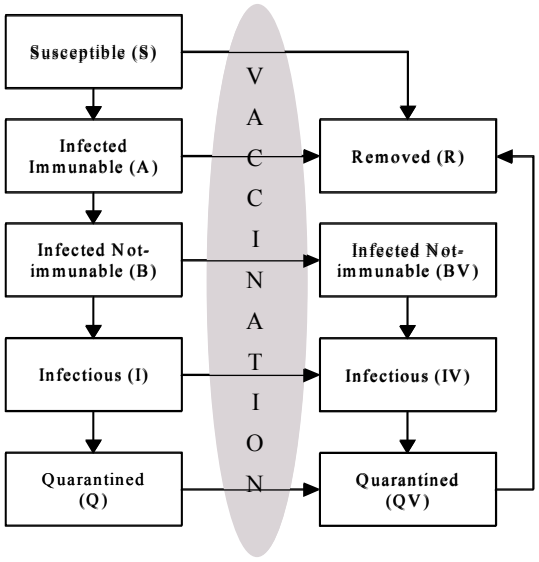

Figure 2: The Stages of the Epidemic

Since we assume perfect vaccination efficacy, vaccinated HHs of type *S* and *A* are immune and removed. Households of types *B* and *I* may be vaccinated, but only the individuals at stages *S* and *A* in those HHs become immune. The rest (those at stages *B* and *I*) are unaffected by the vaccination.

Without loss of generality we assume that transitions in the stage of a HH, including vaccination and quarantining, occur during the HH period. A DMS may be either *infectious*, if at least one member is infectious, or *noninfectious*. Infectious DMSs may generate new infected individuals.

We consider the following response actions:

- Mass vaccination;
- Quarantine;
- Shutdown of GMSs (e.g., shutdown of a mass transit system);
- Closure of DMSs (e.g., closing up schools); and
- Encouraging people to stay home.

The response actions are initiated after a certain number of individuals become infectious (Stage *I*). We assume that the vaccination and the DMS closure processes start after there are Δ_1 infectious individuals. This number is the *vaccination/DMS closure threshold*. The smaller the value of Δ_1, the better the situational awareness capabilities of the system. The shutdown of the GMS is also triggered by the number of infectious individuals in the population. The GMS is shut down if this number exceeds Δ_2. While the GMS is shut down instantaneously following a decision to that effect, the process of closing up the DMSs is gradual and takes time. Once the DMS closure process is initiated, it proceeds with a rate δ.

We assume that a fraction γ of the population passes through the GMS, and a proportion β of the (not yet isolated) infectious individuals complies with requests of the authorities and voluntarily stay at home. Note that in terms of the epidemic spread, the latter assumption represents a worst-case scenario; self-imposed quarantine does not apply to susceptible individuals—only to infectious ones. Finally, infectious HHs are isolated (removed) at a rate ρ.

4. The Model

The model comprises a set of difference-equations shown in the Appendix. It is essentially a deterministic model, but with embedded probabilities that capture the transition intensities between stages. The spread of the disease is observed at two levels: *high level*, at which we observe the transitions *between* sets (HHs and DMSs), and *low level*, at which we observe the transitions *within* sets. At any time period *t*, we record the number of sets of a certain type (e.g., *S*, *A*, etc.) at the high level, and the average *profile* (composition) of disease stages within a set, at the low level. For example, $B(t)$ is the number of HHs of type *B*, and $s_B(t), a_B(t), b_B(t)$ are the average numbers of individuals in such HHs that are at stages *S, A*, and *B*, respectively. The difference equations, shown in the Appendix, describe the transitions between sets of stages at the high level, and the changes in the average profiles within sets, at the low level. Recall that at each time period (day) the epidemic is observed four times: at the beginning and end of the HH subperiod, and the beginning and end of the DMS subperiod.

To simplify notation, we use capitals to denote both the type of a set and the number of sets of that type. For example, $I(t)$ denotes an infectious HH and the number of such HHs in the population. This double meaning should cause no confusion. Specifically, the symbol $X^j(t)$ denotes the number of sets (HHs or DMSs) of type X at time t. The index j is 0,1 where $j = 0$ indicates a beginning of a subperiod (HH or DMS), and $j = 1$ indicates the end of it. To summarize, X gets the following values:

S – Number of susceptible HHs.

A – Number of infective-immunable HHs.

B – Number of infective nonimmunable HHs.

BV – Number of infective nonimmunable HHs that have been vaccinated (Only individuals at stage B remain infective, the rest—S and A individuals—are vaccinated and removed).

I – Number of infectious HHs that have not been vaccinated yet. I_0 are newly infected HHs.

IV – Number of infectious HHs that have been vaccinated. VI_0 are newly infected HHs.

Q – Number of isolated HHs.

QV – Number of isolated, previously vaccinated, HHs.

D – Number of open DMSs.

ID – Number of open infectious DMSs.

The notation at the low level is of the form $y_X^j(t)$, where y indicates the stage of the epidemic, X is the type of HH or DMS, and j is a 0,1 parameter as before. Thus, for example:

$s_A^0(t)$ – Average number of susceptible individuals, at the beginning of the t-th HH period, in an infective-immunable HH that has not been vaccinated yet.

$s_{IV}^1(t)$ – Average number of susceptible individuals, at the end of the t-th HH period, in an infectious HH that has been vaccinated.

$a_A^0(t)$ – Average number of immunable infective individuals, at the beginning of the t-th HH period, in an infective HH that has not been vaccinated yet.

$b_{BV}^{1}(t)$ – Average number of nonimmunable infective individuals, at the end of the *t*-th HH period, in an infective nonimmunable HH that has been vaccinated.

$i_{I}^{1}(t)$ – Average number of infectious individuals, at the end of the *t*-th HH period, in an infectious HH that has not been vaccinated yet.

$s_{ID}^{0}(t)$ – Average number of susceptible individuals in an infectious DMS at the beginning of the DMS period.

In addition, we denote

$a_{X}^{New}(t)$ – Average number of newly infected individuals in an infectious DMS, who belong to a HH of type *X*, *X*=*S*, *A*, *B*, *BV*, *I*, *IV*. $a_{ID}^{New}(t)$ is the average total number of newly infected individuals in an infectious DMS.

Note that while the number of individuals in a HH remains constant throughout the epidemic, the average number of individuals in a DMS changes over time as HHs are isolated and infectious persons stay home.

The parameters of the model are:

M – Number of HHs.

K – Number of DMSs.

h – Size of a HH.

V – Vaccination capacity (# of HHs/day).

V – Vaccination rate (percentage of population vaccinated).

p – Transition rate between stages *A* and *B*.

q – Transition rate between stages *B* and *I*.

α_H – Infection rate in a HH.

α_D – Infection rate in a DMS.

α_G – Infection rate in the GMS.

γ – Fraction of individuals that visit the GMS during a time-period.

β – Fraction of infectious people in a (not yet isolated) infectious HH that stay home because of personal choice (regardless if their corresponding DMSs are closed or open).

ρ – Isolation rate of infectious HHs.

θ – Recovery rate from isolation.

δ – Closure rate of (infectious) DMSs.

Δ_1 – Vaccination/DMS closure threshold.

Δ_2 – GMS shutdown threshold.

To demonstrate the basic idea of the model we present next a sample of three typical equations:

1. High level transition during the HH subperiod:

$$B^1(t) = [B^0(t)(1-q)^{b_B^0(t)} + A^0(t)(1-(1-p)^{a_A^0(t)})]Max\{1-v(t),0\}\,. \qquad (1)$$

Equation (1) gives the number of HHs of type *B* (not yet vaccinated) at the end of the HH subperiod.

2. Low level transition:

$$s_B^1(t) = \frac{1}{B^1(t)}\left[s_B^0(t)B^0(t)(1-q)^{b_B^0(t)} + s_A^0(t)A^0(t)(1-(1-p)^{a_A^0(t)} \right] Max\{1-v(t),0\}\,. \qquad (2)$$

Equation (2) gives the average number of susceptible individuals in HHs of type B.

3. Low level in a DMS.

$$a_{ID}^{New}(t) = \alpha_D s_{ID}^0 i_{ID}^0\,. \qquad (3)$$

Equation (3) gives the average number of newly infected in an infectious DMS.

The complete set of equations is presented in the Appendix.

5. Analysis

We consider two base cases. In Base Case 1, we assume that the response policy is based only on mass vaccination and quarantine of infectious HHs. Other response measures such as DMSs closure, GMS shutdown, and compliance with self-imposed quarantine are not implemented. Base case 2 is the complement of Base Case 1. There is no mass vaccination, only quarantine (and vaccination) of infectious (type *I*) HHs that are detected. However, DMSs are gradually closed up, the GMS is shut down after a while, and a certain proportion of infectious individuals (not yet quarantined) stay home. In both cases, we assume that the initial attack resulted in ten casualties (infected people) in each one of five DMSs plus ten infected in the GMS. Table 1 presents the parameters that are fixed for both base cases and in the subsequent sensitivity analysis.

Parameter	Description	Value
M	Number of HHs	3,000,000
h	Average size of a HH	3
k	Number of DMSs	10000
α_H	Infection rate in a HH	0.5
α_D	Infection rate in a DMS	0.001
α_G	Infection rate in the GMS	0.000001
p	Disease stage *A* rate	0.3
q	Disease stage *B* rate	0.12
ρ	Disease stage *I* rate (Isolation rate of infectious HHs)	0.3
θ	Disease stage *Q* rate (Recovery rate)	0.083
γ	Proportion of population that passes through the GMS	0.5

Table 1: Values of Fixed Parameters

Table 2 presents the values of the remaining parameters in the two base cases.

Parameter	Description	Base Case 1	Base Case 2
δ	Closure rate of DMSs	0	.03
β	Fraction of infectious individuals that stay home	0	.5
Δ_1	Vaccination/DMS Closure Threshold	20	20
Δ_2	GMS Shutdown Threshold	No Closure	250
V	Vaccination Capacity (HHs/Day)	157,000	0

Table 2: Values of Variable Parameters

Based on these parameters, Base Case 1 (vaccination, no public restrictions) results in 2,068 infectious individuals in addition to the casualties of the initial attack, while Base Case 2 (no vaccination, public restrictions) results in 2,070 additional casualties. Evidently, the total numbers of casualties are essentially equal. That is, preventive measures that include closure of DMSs at a rate of 3% per day, shutting down the GMS when there are 250 infectious cases, and 50% infectious "stay-home" compliance is equivalent to the mass vaccination of 157,000 HHs per day with no other social movement-control measures. However, the way the epidemic evolves over time in these two cases is significantly different, as shown in Figure 3.

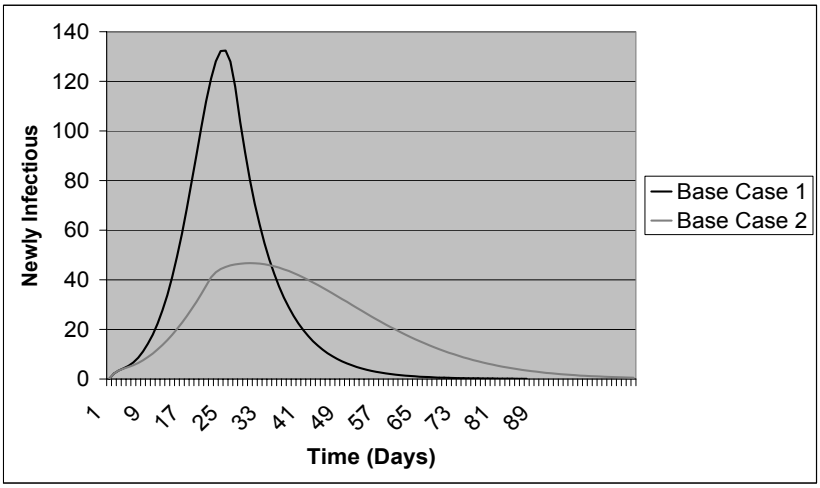

Figure 3: Daily Numbers of Newly Infectious

Figure 4 presents the daily number of people in quarantine in both base cases.

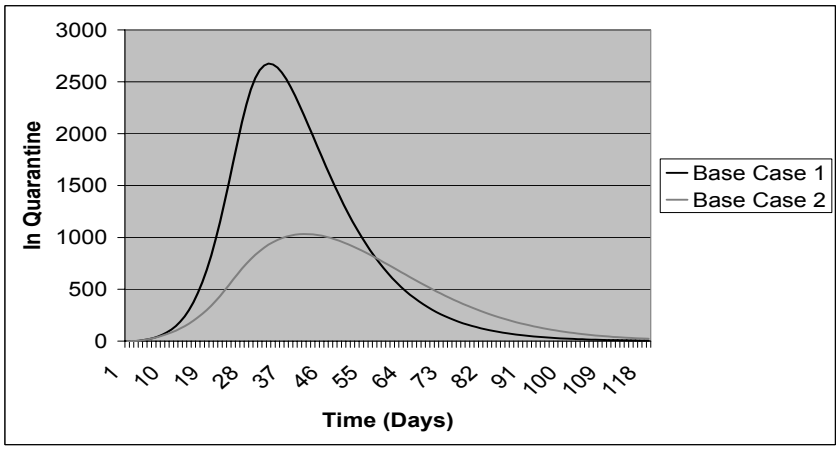

Figure 4: Daily Numbers of People in Quarantine

As shown in Figure 3, the epidemic in Base Case 1 is shorter, but with a higher daily peak than Base Case 2. Figure 4 shows the ramification of this effect, namely, higher demand in Base Case 1 for peak quarantine capacity than in Base Case 2.

Next, we investigate the sensitivity of these base cases to changes in the values of the operational parameters. The measure of effectiveness is the number of casualties, which are the total number of infectious individuals. Figure 5 shows the impact of situational awareness and responsiveness of the public-health system. The delay in the vaccination process, which is represented by the number of infectious individuals that trigger the initiation of the process, affects the number of casualties. If the public-health system has excellent situational awareness and is quick to respond, then the minimum possible number of casualties is about 850. If, because of poor situational awareness and/or slow response, there are, say, 80 infectious people in the population before vaccination starts, then the number of casualties is higher in an order of magnitude.

Figures 6-8 depict the consequences of Base Case 1 (with V = 150,000 HHs/day) when additional social movement-control measures are imposed. Figure 6 investigates the effect of DMS closure on the number of casualties. With no DMS closure, the number of casualties is close to 2,400. A closure process at a rate of 10% a day decreases the number of casualties to about 500. The effect of the delay in shutting down the GMS is shown in Figure 7. If, theoretically, the GMS (e.g., mass transit system) is shut down immediately following the attack, then there are only 282 casualties. If it is shut down when there are already 500 infectious individuals, then the consequence is 1,987 casualties.

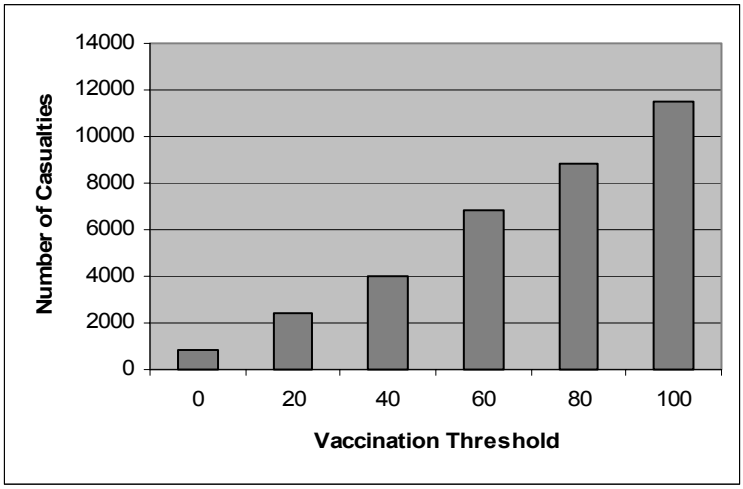

Figure 5: The Effect of Vaccination Threshold – Base Case 1

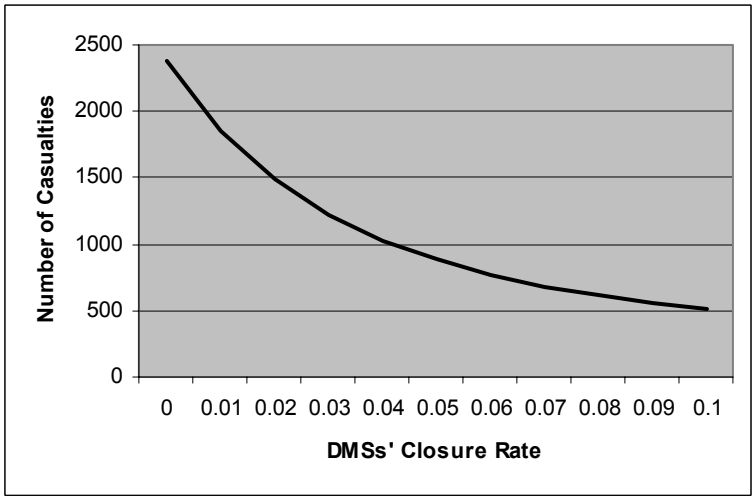

Figure 6: The Effect of DMSs Closure Rate – Base Case 1

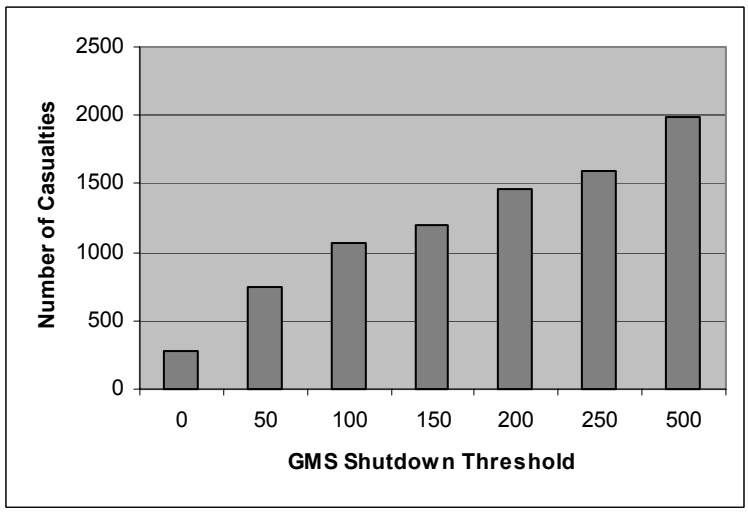

Figure 7: The Effect of GMS Shutdown Threshold – Base Case 1

The effect of voluntary self-quarantine is demonstrated in Figure 8. Note that if 60% of the infectious individuals stay home, then the number of casualties is reduced by almost 80% compared to no self-quarantine.

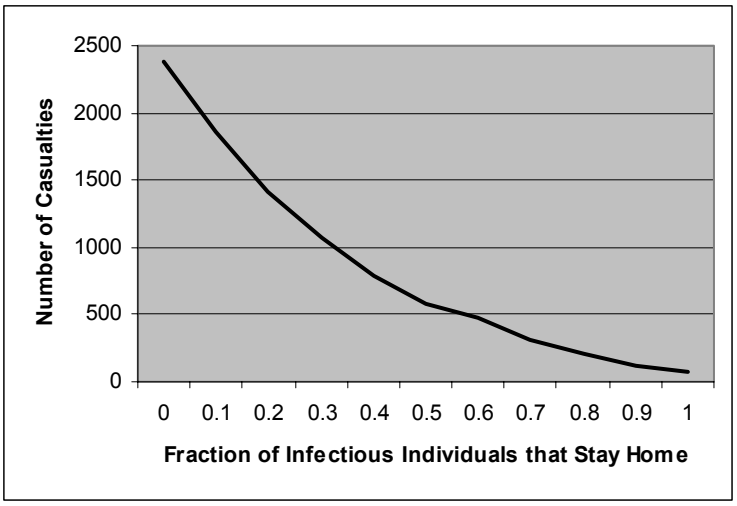

Figure 8: The Effect of Self-Quarantine – Base Case 1

Let us consider now Base Case 2 and investigate the effect of adding to the social movement-control measures mass vaccination. Figures 9-12 depict the effect of vaccination capacity, with respect to DMSs' closure rate (δ), vaccination/DMS closure threshold (Δ_1), GMS shutdown threshold (Δ_2), and self-quarantine compliance rate (β), respectively. Each chart represents the base case and two additional possible scenarios, worse case (darker shade) and better case (lighter shade).

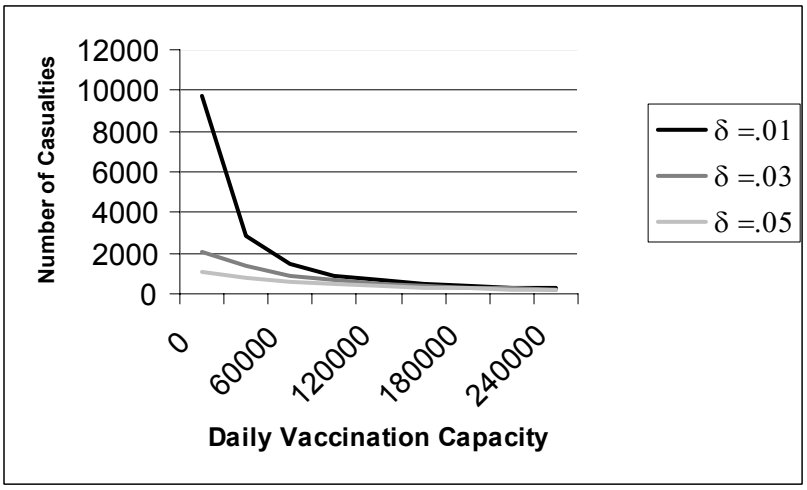

Figure 9: Effect of DMSs' Closure Rate

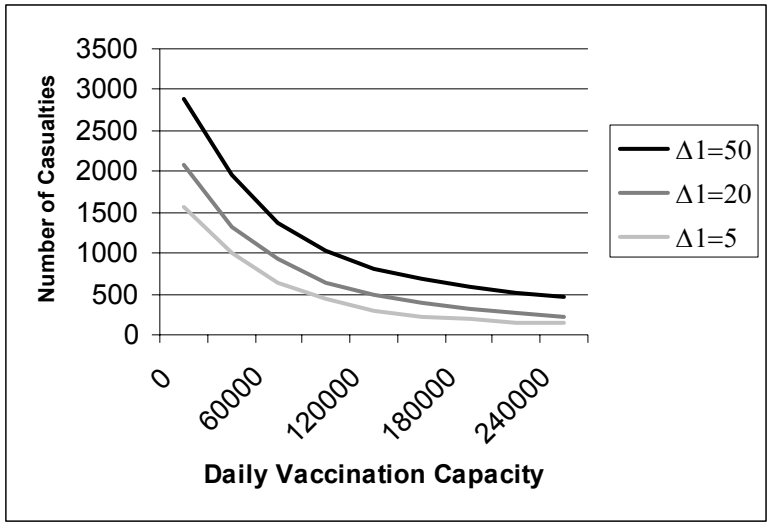

Figure 10: Effect of Vaccination/DMS Closure Threshold

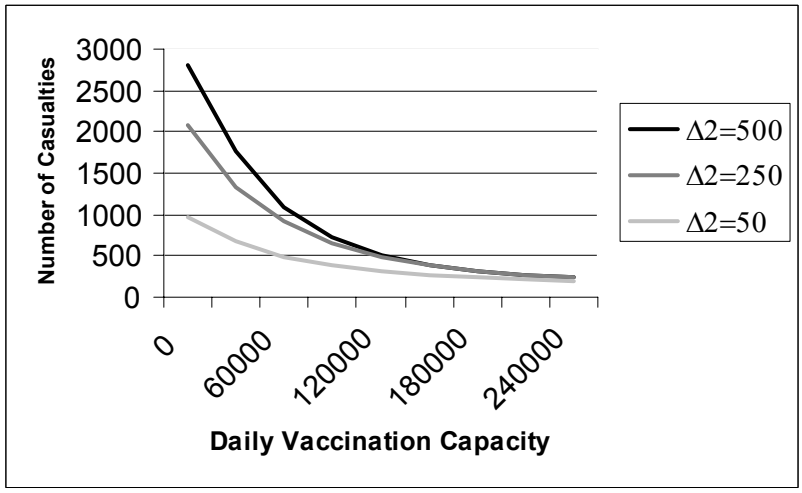

Figure 11: Effect of GMS Shutdown Threshold

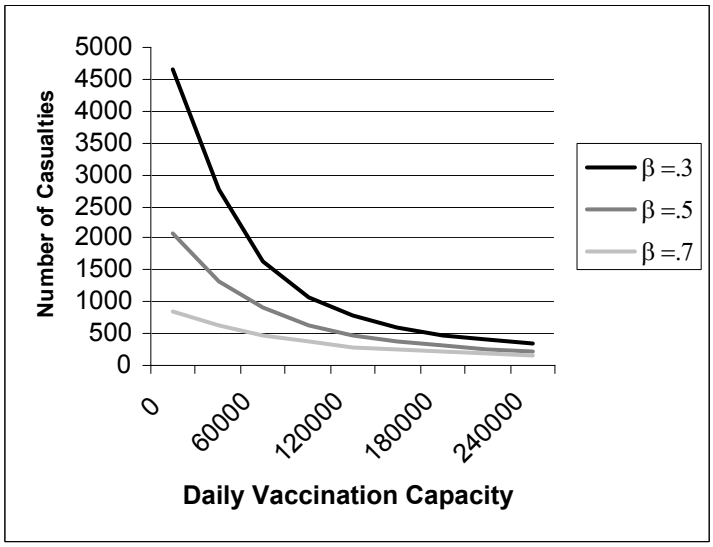

Figure 12: Effect of Self-quarantine

Figures 9-12 reveal a consistent picture, namely, that the impact of situational awareness and social movement-control measures can be significant, but it decreases as the vaccination capacity increases. One small exception is the vaccination/closure threshold Δ_1. Even if the vaccination capacity is 240,000 HHs/day, the number of casualties in the worse case scenario $\Delta_1 = 50$ is almost 3.5 times higher than in the case where $\Delta_1 = 5$.

6. Discussion and Conclusions

Figures 3 and 4 demonstrate that (mass) vaccination-free policies, where the response relies entirely on social movement-control measures, may be compatible to a policy that is based on mass vaccination. Moreover, while the latter policy eradicates the epidemic faster than the former, the required peak quarantine capacity is significantly higher. Figures 5-8 show what happens if a mass vaccination policy is augmented with social movement-control measures and situational awareness is considered. The effects are quite significant. For example, if one-third of the infectious population withdraws to their home, the number of casualties is reduced by 55% compared with no self-quarantine. A similar effect is recorded when the GMS is shutdown early in the epidemic. If the trigger for a shutdown is 250 infectious people in the population, then the number of casualties is 1,594. If the trigger is 50 people, then the result is only 742 casualties.

These results indicate that a hybrid policy that combines moderate rate of mass-vaccination with moderate application of social movement-control measures may be an efficient response policy. Table 3 presents the parameters of a third base-case, which is a combination of the two approaches: vaccination and social movement-control.

Parameter	Description	Base Case 3
δ	Closure rate of DMSs	.01
β	Fraction of infectious individuals that stay home	.3
Δ_1	Vaccination/DMS Closure Threshold	10
Δ_2	GMS Shutdown Threshold	100
V	Vaccination Capacity (HHs/Day)	75,000

Table 3: Base Case 3

The total number of casualties in Base Case 3 is 1,358—about 35% less casualties than in Base Cases 1 and 2. Similar to Figure 3, Figure 13 compares the three base cases over the period of the epidemic.

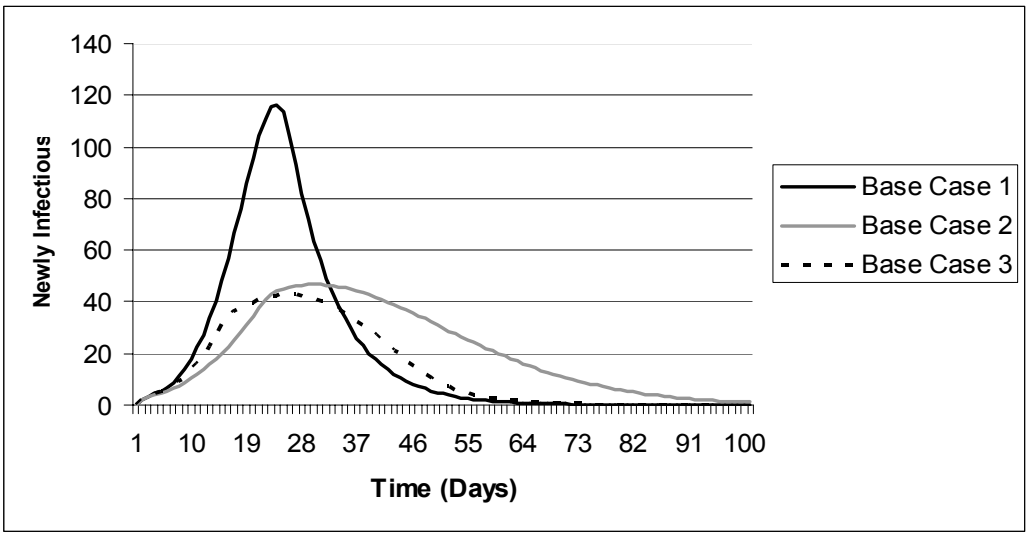

Figure 13: Daily Numbers of Newly Infectious – Base Cases 1, 2, and 3

While the peak of the epidemic is about the same as in Base Case 2 (47 casualties in Base Case 2, 43 casualties in Base Case 3), the epidemic is eradicated much faster: 76 days compared with 113 days.

Appendix: Difference-Equations Model

A. HH Subperiod

Let

$$v(t) = \frac{V(t)}{S^0(t) + A^0(t) + I^0(t)} \tag{4}$$

$v(t)$ is the vaccination rate.

High Level

$$S^1(t) = S^0(t)Max\{1 - v(t), 0\} \tag{5}$$

$$A^1(t) = A^0(t)(1-p)^{a_A^0(t)}Max\{1 - v(t), 0\} \tag{6}$$

$$B^1(t) = [B^0(t)(1-q)^{b_B^0(t)} + A^0(t)(1-(1-p)^{a_A^0(t)})]Max\{1 - v(t), 0\} \tag{7}$$

$$BV^1(t) = BV^0(t)(1-q)^{b_{BV}^0(t)} + [B^0(t)(1-q)^{b_B^0(t)} + A^0(t)(1-(1-p)^{a_A^0(t)})]Min\{v(t), 1\} \tag{8}$$

$$I_0^1(t) = B^0(t)[1-(1-q)^{b_B^0(t)}]Max\{1 - v(t), 0\} \tag{9}$$

$$IV_0^1(t) = BV^0[1-(1-q)^{b_{BV}^0(t)}] + B^0(t)[1-(1-q)^{b_B^0(t)}]Min\{1, v(t)\} \tag{10}$$

$$I^1(t) = I^0(t)Max\{1 - v(t), 0\}(1-\rho) \tag{11}$$

$$IV^1(t) = (IV^0(t) + I^0(t)Min\{1, v(t)\})(1-\rho) \tag{12}$$

$$Q(t) = I^0(t)\rho + Q(t-1) \tag{13}$$

$$QV(t) = IV^0(t)\rho + QV(t-1)\,. \tag{14}$$

Low Level

Susceptible HH (S)

$$s_S^1(t) = s_S^0(t) = h \tag{15}$$

$$a_S^1(t) = b_S^1(t) = i_S^1(t) = a_S^0(t) = b_S^0(t) = i_S^0(t) = 0\,. \tag{16}$$

Infective Immunable HH (*A*)

$$s_A^1(t) = s_A^0(t) \tag{17}$$

$$a_A^1(t) = a_A^0(t) \tag{18}$$

$$b_A^1(t) = b_A^0(t) = 0 \tag{19}$$

$$i_A^1(t) = i_A^0(t) = 0\,. \tag{20}$$

Infective Not-immunable HH (*B*)

$$s_B^1(t) = \frac{1}{B^1(t)}\left[s_B^0(t)B^0(t)(1-q)^{b_B^0(t)} + s_A^0(t)A^0(t)(1-(1-p)^{a_A^0(t)} \right] Max\{1-v(t),0\} \tag{21}$$

$$\begin{aligned} a_B^1(t) = \frac{1}{B^1(t)}\Bigg[& a_B^0(t)(1-p)B^0(t)(1-q)^{b_B^0(t)} + \\ & a_A^0(t)\left(1-\frac{p}{1-(1-p)^{a_A^0(t)}} \right) A^0(t)(1-(1-p)^{a_A^0(t)}) \Bigg] Max\{1-v(t),0\} \end{aligned} \tag{22}$$

$$\begin{aligned} b_B^1(t) = \frac{1}{B^1(t)}\Bigg[& (b_B^0(t) + a_B^0 p)B^0(t)(1-q)^{b_B^0(t)} + \\ & a_A^0(t)\frac{p}{1-(1-p)^{a_A^0(t)}} A^0(t)(1-(1-p)^{a_A^0(t)}) \Bigg] Max\{1-v(t),0\} \end{aligned} \tag{23}$$

$$i_B^1(t) = i_B^0(t) = 0\,. \tag{24}$$

Vaccinated Infective Not-immunable HH (*BV*)

$$s_{BV}^1(t) = s_{BV}^0(t) = a_{BV}^1(t) = a_{BV}^0(t) = i_{BV}^0(t) = i_{BV}^1(t) = 0 \tag{25}$$

$$\begin{aligned} b_{BV}^1(t) = \frac{1}{BV^1(t)}\Bigg[& b_{BV}^0(t)BV^0(t)(1-q)^{b_{BV}^0(t)} + \\ & \Bigg[(b_B^0(t) + a_B^0 p)B^0(t)(1-q)^{b_B^0(t)} + \\ & a_A^0(t)\frac{p}{1-(1-p)^{a_A^0(t)}} A^0(t)(1-(1-p)^{a_A^0(t)}) \Bigg] Min\{v(t),1\} \Bigg] \end{aligned} \tag{26}$$

Newly Infectious HH (I_0)

$$s^1_{I_0}(t) = s^0_B(t) \tag{27}$$

$$a^1_{I_0}(t) = a^0_B(t)(1-p) \tag{28}$$

$$b^1_{I_0}(t) = b^0_B(t)\left(1-\frac{q}{1-(1-q)^{b^0_B(t)}}\right)+a^0_B(t)p \tag{29}$$

$$i^1_{I_0}(t) = b^0_B(t)\frac{q}{1-(1-q)^{b^0_B(t)}}. \tag{30}$$

Vaccinated Newly Infectious HH (IV_0)

$$s^1_{IV_0}(t) = s^0_{IV_0}(t) = a^1_{IV_0}(t) = a^0_{IV_0}(t) = 0 \tag{31}$$

$$\begin{aligned} b^1_{IV_0}(t) = \frac{1}{IV^1_0(t)}\Bigg\{ & b^0_{BV}(t)\left(1-\frac{q}{1-(1-q)^{b^0_{BV}(t)}}\right)BV^0(t)(1-(1-q)^{b^0_{BV}(t)}) \\ & + b^{\,0}_{\,B}(t)\left(1-\frac{q}{1-(1-q)^{b^0_B(t)}}\right)B^0(t)(1-(1-q)^{b^0_B(t)})Min\{v(t),1\}\Bigg\} \end{aligned} \tag{32}$$

$$i^1_{IV_0}(t) = \frac{1}{IV^1_0(t)}\left\{qb^0_{BV}(t)BV^0(t)+qb^{\,0}_{\,B}(t)B^0(t)Min\{v(t),1\}\right\}. \tag{33}$$

Infectious HH (I)

$$s^1_I = s^0_I(1-\alpha_H i^0_I) \tag{34}$$

$$a^1_I(t) = a^0_I(t)(1-p)+\alpha_H s^0_I i^0_I \tag{35}$$

$$b^1_I(t) = b^0_I(t)(1-q)+a^0_I(t)p \tag{36}$$

$$i^1_I(t) = i^0_I(t)+b^0_I(t)q\,. \tag{37}$$

Vaccinated Infectious HH (IV)

$$s^1_{IV}(t) = s^0_{IV}(t) = a^1_{IV}(t) = a^0_{IV}(t) = 0 \tag{38}$$

$$b^1_{IV}(t) = b^0_{IV}(t)(1-q) \tag{39}$$

$$i^1_{IV}(t) = i^0_{IV}(t)+b^0_{IV}(t)q\,. \tag{40}$$

Isolated Not Previously Vaccinated HH (Q)
(Assumption: Individuals not previously vaccinated are vaccinated immediately upon arrival at the quarantine).

$$s_Q^1(t) = s_Q^0(t) = a_Q^1(t) = a_Q^0(t) = 0 \tag{41}$$

$$b_Q(t) = \frac{1}{Q(t)}\left\{(1-q)\,b_Q(t-1)Q(t-1) + (1-q)\,b_I^0(t)I^0(t)\rho\right\} \tag{42}$$

$$i_Q(t) = i_Q(t-1) + \frac{1}{Q(t)}\left\{qb_Q(t-1)Q(t-1) + (qb_I^0(t) + i_I^0(t))I^0(t)\rho\right\}. \tag{43}$$

Isolated Previously Vaccinated HH (QV)

$$s_{QV}^1(t) = s_{QV}^0(t) = a_{QV}^1(t) = a_{QV}^0(t) = 0 \tag{44}$$

$$b_{QV}(t) = \frac{1}{QV(t)}\left\{b_{QV}(t-1)(1-q)QV(t-1) + b_{IV}^0(t)(1-q)IV^0(t)\rho\right\} \tag{45}$$

$$i_{QV}(t) = i_{QV}(t-1) + \frac{1}{QV(t)}\left\{qb_{QV}(t-1)QV(t-1) + (qb_{IV}^0(t) + i_{IV}^0(t))IV^0(t)\rho\right\}. \tag{46}$$

At the end of the HH-cycle, the total number of susceptibles and immunable infected are:

$$s_{Total}^1(t) = S^1(t)h + A^1(t)s_A^1(t) + B^1(t)s_B^1(t) + I_0^1(t)s_{I_0}^1(t) + I^1(t)s_I^1(t) \tag{47}$$

$$a_{Total}^1(t) = A^1(t)a_A^1(t) + B^1(t)a_B^1(t) + I_0^1(t)a_{I_0}^1(t) + I^1(t)a_I^1(t). \tag{48}$$

And the total number of infectious individuals is:

$$i_{Total}^1(t) = I_0^1(t)i_{I_0}^1(t)) + I^1(t)i_I^1(t)) + IV_0^1(t)i_{IV_0}^1(t)) + IV^1(t)i_{IV}^1(t)). \tag{49}$$

The number of commuting infectious individuals from an infectious HH is

$$ic_I^1(t) = (1-\beta)i_I^1(t). \tag{50}$$

$ic_{I_0}^1(t), ic_{IV}^1(t)$ and $ic_{IV_0}^1(t)$ are defined similarly.

The total commuting infectious individuals is:

$$ic_{Total}^1(t) = I_0^1(t)ic_{I_0}^1(t)) + I^1(t)ic_I^1(t)) + IV_0^1(t)ic_{IV_0}^1(t)) + IV^1(t)ic_{IV}^1(t). \tag{51}$$

B. Transition HH → GMS →DMS

DMS

$$D(t) = (1-\delta)D(t-1) \tag{52}$$

$$ID(t) = D(t)\left(1-\left(1-\frac{ic_I^1(t)}{K}\right)^{I^1(t)}\left(1-\frac{ic_{I_0}^1(t)}{K}\right)^{I_0^1(t)}\left(1-\frac{ic_{IV}^1(t)}{K}\right)^{IV^1(t)}\left(1-\frac{ic_{IV_0}^1(t)}{K}\right)^{IV_0^1(t)}\right) \tag{53}$$

Explanation: Suppose D(t) = uK. Only a proportion u of the DMSs are open and therefore only a proportion u of the population leaves home. Since each member of a HH goes to a different DMS, this means that only *ui* leave home. *ui/uK = i/K.*

Individuals

We assume that the contacts in the GMS occur between the HH cycle and the DMS cycle.

$$i_{GMS}(t) = \gamma\frac{D(t)}{K}ic_{Total}^1(t) \tag{54}$$

$$s_{GMS}(t) = \gamma s_{Total}^1(t) \tag{55}$$

The number of newly infected individuals at the GMS is:

$$a_{GMS}^{New}(t) = \alpha_G s_{GMS} i_{GMS}\,. \tag{56}$$

Let
$a_{GS}^{New}(t), a_{GA}^{New}(t),\ a_{GB}^{New}(t)$ and $a_{GI}^{New}(t),\ a_{GS}^{New}(t)+a_{GA}^{New}(t)+a_{GB}^{New}(t)+a_{GS}^{New}(t)=a_{GMS}^{New}(t),$
denote the number of newly infected at the GMS that belong to *S*, *A*, *B*, and *I* HH, respectively. Since the probability that a newly infective belongs to a certain type of a HH is proportional to the number of susceptibles in such a HH, we have:

$$a_{GS}^{New}(t) = a_{GMS}^{New}(t)\frac{S^1(t)h}{s_{Total}^1(t)} \tag{57}$$

$$a_{GA}^{New}(t) = a_{GMS}^{New}(t)\frac{A^1(t)s_A^1(t)}{s_{Total}^1(t)} \tag{58}$$

$$a_{GB}^{New}(t) = a_{GMS}^{New}(t)\frac{B^1(t)s_B^1(t)}{s_{Total}^1(t)} \tag{59}$$

$$a_{GI}^{New}(t) = a_{GMS}^{New}(t)\frac{I_0^1(t)s_{I_0}^1(t)+I^1(t)s_I^1(t)}{s_{Total}^1(t)}\,. \tag{60}$$

Infectious DMS (*ID*)

$$i_{ID}^{0}(t) = \frac{D(t)}{K}\frac{ic_{Total}^{1}(t)}{ID(t)} \tag{61}$$

$$s_{ID}^{0}(t) = \frac{s_{Total}^{1}(t) - a_{GMS}^{New}(t)}{K}. \tag{62}$$

Since response actions and transitions between disease stages are assumed to take place during the HHs cycle, we do not need to track either the noninfectious open DMSs or the individuals who are at the latent stages (immunable and nonimmunable) of the disease.

C. DMS Cycle

Since transitions between disease stages are assumed to take place only in the HHs, the DMSs do not change their status during this cycle. The only parameter of interest during the DMS cycle is the number of newly infected.

Individuals

Infectious Open DMS (*ID*)

The number of newly infected individuals in an open DMS is:

$$a_{ID}^{New}(t) = \alpha_D s_{ID}^{0} i_{ID}^{0}. \tag{63}$$

A newly infected individual may belong to a susceptible HH (S), an infective HH (A), or an infectious HH (I).

Let
$a_{DS}^{New}(t), a_{DA}^{New}(t), a_{DB}^{New}(t)$ and $a_{DI}^{New}(t)$, $a_{DS}^{New}(t) + a_{DA}^{New}(t) + a_{DB}^{New}(t) + a_{DS}^{New}(t) = a_{ID}^{New}(t)$, denote the number of newly infectives that belong to S, A, and I HH, respectively. Since the probability that a newly infective belongs to a certain type of HH is proportional to the number of susceptibles in such a HH, we have:

$$a_{DS}^{New}(t) = a_{ID}^{New}(t)\frac{S^{1}(t)h}{s_{Total}^{1}(t)} \tag{64}$$

$$a_{DB}^{New}(t) = a_{ID}^{New}(t)\frac{B^{1}(t)s_{B}^{1}(t)}{s_{Total}^{1}(t)} \tag{65}$$

$$a_{DB}^{New}(t) = a_{ID}^{New}(t)\frac{B^{1}(t)s_{B}^{1}(t)}{s_{Total}^{1}(t)} \tag{66}$$

$$a_{DI}^{New}(t) = a_{ID}^{New}(t)\frac{I_{0}^{1}(t)s_{I_0}^{1}(t) + I^{1}(t)s_{I}^{1}(t)}{s_{Total}^{1}(t)}. \tag{67}$$

D. DMS – HH Transition

HH

Let

$$S^0(t) = S^1(t-1)\left(1-\frac{a_{DS}^{New}(t-1)}{S^1(t-1)}\right)^{ID(t-1)}\left(1-\frac{a_{GS}^{New}(t-1)}{S^1(t-1)}\right) \tag{68}$$

$$A^0(t) = A^1(t-1) + S^1(t-1) - S^0(t) \tag{69}$$

$$B^0(t) = B^1(t-1) \tag{70}$$

$$BV^0(t) = BV^1(t-1) \tag{71}$$

$$I^0(t) = I^1(t-1) + I_0^1(t-1) \tag{72}$$

$$IV^0(t) = IV^1(t-1) + IV_0^1(t-1)\,. \tag{73}$$

Individuals

Susceptible HH (*S*)

$$s_S^0(t) = h \tag{74}$$

$$a_S^0(t) = b_S^0(t) = i_S^0(t) = 0\,. \tag{75}$$

Infective Immunable HH (A)

$$\begin{aligned} s_A^0(t) = \frac{1}{A^0(t)}\Big[\, & A^1(t-1)s_A^1(t-1) - ID(t-1)(a_{DA}^{New}(t-1) + a_{DS}^{New}(t-1)) \\ & -(a_{GA}^{New}(t-1) + a_{GS}^{New}(t-1)) + (S^1(t-1) - S^0(t))h \,\Big] \end{aligned} \tag{76}$$

$$\begin{aligned} a_A^0(t) = \frac{1}{A^0(t)}\Big[\, & A^1(t-1)a_A^1(t-1) + ID(t-1)(a_{DA}^{New}(t-1) + a_{DS}^{New}(t-1)) \\ & + a_{GA}^{New}(t-1) + a_{GS}^{New}(t-1)\,\Big] \end{aligned} \tag{77}$$

$$b_A^0(t) = 0 \tag{78}$$

$$i_A^0(t) = 0\,. \tag{79}$$

Infective Not-immunable HH (B)

$$s_B^0(t) = \frac{1}{B^0(t)}\Big[B^1(t-1)s_B^1(t-1) - ID(t-1)a_{DB}^{New}(t-1) - a_{GB}^{New}(t-1)\Big] \tag{80}$$

$$a_B^0(t) = \frac{1}{B^0(t)}\Big[B^1(t-1)a_B^1(t-1) + ID(t-1)a_{DB}^{New}(t-1) + a_{GB}^{New}(t-1)\Big] \tag{81}$$

$$b_B^0(t) = b_B^1(t-1) \tag{82}$$

$$i_B^0(t) = 0\,. \tag{83}$$

Infectious HH (*I*)

$$s_I^0(t) = \frac{1}{I^0(t)}\Big[I^1(t-1)s_I^1(t-1) + I_0^1(t-1)s_{I_0}^1(t-1) - ID(t-1)a_{DI}^{New}(t-1) - a_{GI}^{New}(t-1)\Big] \tag{84}$$

$$a_I^0(t) = \frac{1}{I^0(t)}\Big[I^1(t-1)a_I^1(t-1) + I_0^1(t-1)a_{I_0}^1(t-1) + ID(t-1)a_{DI}^{New}(t-1) + a_{GI}^{New}(t-1)\Big] \tag{85}$$

$$b_I^0(t) = \frac{I^1(t-1)b_I^1(t-1) + I_0^1(t-1)b_{I_0}^1(t-1)}{I^0(t)} \tag{86}$$

$$i_I^0(t) = \frac{I^1(t-1)i_I^1(t-1) + I_0^1(t-1)i_{I_0}^1(t-1)}{I^0(t)}\,. \tag{87}$$

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