A Generic Model of Contagious Disease and Its Application to Human-to-Human Transmission of Avian Influenza

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Abstract

Modeling contagious diseases has taken on greater importance over the past several years as diseases such as SARS and avian influenza have raised concern about worldwide pandemics. Most models developed to consider projected outbreaks have been specific to a single disease. This paper describes a generic System Dynamics contagious disease model and its application to human-to-human transmission of a mutant version of avian influenza. The model offers the option of calculating rates of new infections over time based either on a fixed "reproductive number" that is traditional in contagious disease models or on contact rates for different sub-populations and likelihood of transmission per contact. The paper reports on results with various types of interventions. These results suggest the potential importance of contact tracing, limited quarantine, and targeted vaccination strategies as methods for controlling outbreaks, especially when vaccine supplies may initially be limited and the efficacy of anti-viral drugs uncertain.

Introduction

Concerns about the use of contagious diseases for bioterrorism and naturally occurring diseases such as SARS and pandemic influenza have sparked recent interest in modeling these diseases and their effects on populations. Modeling efforts have included those focused on influenza (Meltzer, 1999) and SARS (Lipsitch, 2003). Smallpox has also attracted interest and there have been a number of models developed and reported on. (Meltzer, 2001; Kaplan, 2002; Halloran, 2002; Eubank, 2004) All of these models have some value for examining different response strategies, identifying the best ones in different circumstances, and thereby helping public health officials prepare in advance for what would otherwise be catastrophic events.

This paper reports on a generic contagious disease model and its application to human-to-human transmission of an H5N1-type virus widely referred to as avian influenza. It is a System Dynamics model developed with Vensim as part of a much larger emergency preparedness and infrastructure modeling effort being carried out by Sandia National Labs in cooperation with Los Alamos and Argonne National Labs for the US Department of Homeland Security. The model has been connected to modules representing other components of a community's infrastructure including energy supply, transportation, telecommunications, and health care. It can reflect how problems in these other sectors (e.g., breakdown in transportation affecting ability to obtain vaccines) can affect a community's ability to control an outbreak and how the spread of the disease will affect the availability of essential workers to those other sectors. The model focuses on contagious disease as a specific threat, following an earlier effort that examined the effects of an array of threats on a community's population and health care system. (See for example Hirsch, 2004)

Why a Generic Model?

With all of the work already being done in contagious disease modeling, why develop another model? There are several reasons:

• The other models tend to focus on individual diseases. There is value in a model that can simulate multiple contagious diseases in the same framework, by merely changing parameters, in order to identify public health capabilities that will help communities prepare for a wide range of threats from different diseases. This paper will describe the generic model's application to one disease and a version has been applied to another as well. (LeClaire, 2005) This approach is consistent with the "all hazards" approach being adopted in emergency preparedness planning. (FEMA, 2001)

• This model has modest computational requirements and can be made available to public health authorities to input their own communities' data for examining possible response strategies and doing sensitivity analyses in areas where there is uncertainty about how a particular disease will affect their population.

- As indicated above, the model can be linked to modules representing various infrastructure components and simulations can reflect how the spread of disease interacts with and affects those other components. It can also reflect the interaction between contagious disease and various other disasters or attacks.

- The model can address particular resource issues such as the size of temporary field hospitals that might be required to provide palliative care for victims of an outbreak if their number exceeds the normal capacity of a community's hospitals. It can also differentiate the effects of an outbreak on key population groups such as health care workers who are especially vulnerable and yet are crucial to an effective response.

The paper begins by describing the structure of the model. It then reviews results with different strategies for intervention in outbreaks of human-to-human transmission of avian influenza and sensitivity analyses that examine particular parameters.

Structure of the Generic Contagious Disease Model

1. Flows of Patients Among Stages

The model's flow structure and stages are similar to that in S-E-I-R models (Susceptible-Exposed-Infected-Recovered) often used in epidemiology. It represents the populations of two distinct regions and further segments those populations into five groups:
- Adults 18-64 who work in health care and emergency services
- Adults 18-64 who work in other industries
- Adults 18-64 who do not work
- Children 0-17
- Adults 65 and over
Simulations assume that an outbreak starts in one of the regions and spread to the other as people travel back and forth between the two.

Figure 1 provides an overview of the model's main flow of people through the various stages of contagious illness. People may contract the disease either though an initial release or through contact with someone who is infected as the disease spreads through the community. Vaccination and quarantine reduce the size of the unexposed population and reduce its rate of spread and ultimate penetration. The initial number of people unexposed is reduced by the fraction of people who have some immunity.   In the case of an H5N1-type influenza, it is assumed that no one has prior immunity.

Once exposed, people are assumed to incubate the disease for a day and show no symptoms during that time. (CDC, 2005) They begin to show vaguely flu-like symptoms during a one-day prodromal period during which there is a chance of transmitting the disease. More distinct symptoms break out after the prodromal period and persist for a five-day period during which the risk of transmission is greatest. The final stage is a four-day recovery period, during which transmission is unlikely.   Deaths may occur during the early or late symptomatic stages. The overall mortality rate of 2% is assumed to be distributed evenly between the two symptomatic stages.

2. Two Different Methods of Projecting New Cases

In the next two views, variables are displayed selectively in different colors to highlight particular themes. Figure 2 highlights in red the factors that drive the rate of spread of an outbreak and can also slow it down. In this model, there are two different methods that can be used to calculate the rate of new infections. One is the approach traditionally used in S-E-I-R models (Susceptible-Exposed-Infected-Recovered) prevalent in epidemiology that are based on an assumed value of a "Reproductive Rate" (R0), the number of new people infected by each infected person over the course of their illness. The other is based on typical daily contact rates for different population groups and the fractions of those contacts by an infected person likely to result in transmission. This latter approach was inspired by the EPISIM/EPICAST agent-based modeling work developed at Los Alamos National Lab (LANL) and adapted for use in an SD model (Eubank, 2004).

The model contains a switch that allows one or the other of these approaches to be used. Given the uncertainty about the characteristics of a mutated form of avian influenza that can spread from person-to-person, having this option can provide a broader perspective about how the disease might be spread and how an outbreak could be stopped.

a. Traditional Approach of S-E-I-R Models

With the traditional approach, the key parameter in determining that rate of spread is the Reproductive Rate (R0), the number of new cases each infected person generates given normal patterns of contact and susceptibility. The value for R0 of 2.55 that was selected is consistent with a range of values derived for the 1918 pandemic (see Mills, 2004) and is based on calibration with a model derived from an agent-based approach.

The fraction of transmission occurring prior to clear symptoms distributes responsibility for the spread between people in the prodromal stage and the remainder that occurs during the early symptom phase. This is an important parameter for determining the extent to which a contagious disease outbreak can be brought under control by isolating symptomatic patients and whether more aggressive programs of vaccination and general quarantine are called for. The 35% value is close to the fraction of 0.4 suggested for influenza by (Fraser, 2004) and was adjusted based on calibration with the agent-based model.

b. Spread Based on Contacts

A model was developed separately by LANL that based the new infection rate on assumed numbers of contacts people in different demographic groups might have with others in their families, workplaces, and communities, and the potential infectiousness of contacts by infected people at each stage of the disease. The contact-based approach starts with normal contact rates for each demographic group that were derived from the EPISIM/EPICAST work and reduces those rates based on the fraction of people who are in quarantine (self- or imposed-). These normal rates are applied to people who are

Exposed and Incubating the illness and people in the Prodromal group since they have not yet developed distinct symptoms. Adults are assumed to have 20 contacts per day while children have 9 contacts per day, and older people have 7. Contact rates are reduced by 75% for the first day or so after people first become symptomatic and begin to restrict their activities (e.g., from 20 contacts per day to 5 for adults). After that first day, contacts are assumed to fall further to only 0.3 per day if people are isolated and one per day if they manage to evade isolation. These contact rates are multiplied by the number of people in each stage of the disease and a fraction of contacts for each stage that actually transmit the disease. The following fractions of contacts are assumed to transmit the disease.

Exposed and Incubating   0

Prodromal   0.04

Early Symptomatic   0.08

Late Symptomatic   0.015

Table 1: Fractions of Contacts Resulting in Transmission

Tests indicated that this formulation produces a "base run" comparable to one based on a fixed value of R0 as long as the circumstances being simulated are roughly the same. For this comparison, the simulation using the fixed value of R0 was also assumed to have the limited quarantine in effect. (See below for an explanation of Limited Quarantine). This is similar to the assumption in the contact-based formulation that people's contacts drop drastically as they become too sick to go out or are forced to isolate themselves. Results of the comparisons between the two methods are presented later in this paper.

3. *Spread Between Regions*

The rate of new infections based on the spread of the disease calculated for each region reflects spread within the region plus spread due to people traveling from the other region. For example, the rate of new infections based on the spread of the disease in Region A is the following:

Reproductive Rate (or Contact Rate x Fraction of Contacts Resulting in Transmission) x (People in Prodromal (A) x Fraction of Transmission Occurring Prior to Clear Symptoms + People with Early Symptoms (A) x (1- Fraction of Transmission Occurring Prior to Clear Symptoms) + Fraction Traveling to Other Region (B) x (People in Prodromal (B) x Fraction of Transmission Occurring Prior to Clear Symptoms + People with Early Symptoms (B) x (1- Fraction of Transmission Occurring Prior to Clear Symptoms)))

4. Limited Quarantine

The spread of the disease as reflected in the model is resisted to some extent by a mechanism referred to as limited quarantine in which patients are isolated once they display clear symptoms and no longer spread the infection to others. This mechanism is "switched in" once there are ten or more patients displaying symptoms. Limited quarantine is not assumed to be perfect, however, and 20% of patients with early symptoms are assumed to infect some others before being effectively isolated.

5. Strategies for Controlling Outbreaks

Figure 3 displays variables affecting the impact of vaccination and quarantine on the rate of new infection. The vaccination strategy in effect, rate of vaccinations, and vaccine effectiveness rate determine the rate of moving people from the unexposed to unexposed vaccinated populations. The model makes it possible to either vaccinate population groups based on their representation in the general population or assign priority to particular population groups (e.g., health and emergency service workers, children). Some of those vaccinated are people who have been infected, but are in the two day window during which vaccination might cut the number of people exposed who actually develop and transmit the disease and the fatality rate of those who do develop symptoms.

The fraction of infected likely to be vaccinated during the first two days calculated by the model is based on the
- program of vaccination in place (mass vs. targeted),
- hourly vaccination rate times 48 hours,
- number of people in the incubation stage,
- fraction of contacts identified, and
- vaccine effectiveness rate.

The fraction of contacts that can be identified is assumed to be 80% and represents an upper bound on the number of contacts successfully traced and vaccinated, regardless of the vaccination rate. People vaccinated during this two-day window go down a separate flow chain. 80% of them do not develop the symptoms. People identified as contacts during the course of targeted vaccination are also quarantined.

Vaccination can be subject to several limits due to:
- Stockpile limitations of available vaccine
- Refusal of people to be vaccinated
- Effectiveness of vaccine in terms of fraction vaccinated who are actually protected

The model applies the most stringent limit in determining the fraction that can ultimately be vaccinated.

Contact tracing and general quarantine (as opposed to the limited quarantine of symptomatic patients) are applied in a similar manner to vaccination. The fraction of infected likely to be quarantined during incubation period is determined by the ratio of the applicable contact tracing rate to the rate of people becoming newly infected. The fraction of newly infected people identified by contact tracing is a function of the

- total contact tracing rate for the region,
- fraction of contacts who have actually been exposed, and
- contact effectiveness rate.

The baseline assumption is that twelve contacts are identified for every symptomatic patient and only three of these have actually been exposed and are incubating the disease, yielding a fraction of contacts unexposed of 75%. As with targeted vaccination, the fraction of contact effectiveness (fraction of contacts identified) is 80%. Unexposed patients identified as contacts are still moved from the unexposed to unexposed in quarantine populations and remain there for a period to assure that they will not become symptomatic. People identified by contact tracing who have been exposed go down another chain shown in which they develop the disease, but are prevented from spreading it to others.

The model includes various mechanisms for implementing strategies to control outbreaks. These mechanisms can be switched in to examine the effects of different strategies. A vaccination switch determines whether vaccination is part of the response at all and a vaccination policy parameter determines the type of vaccination strategy:

- Mass vaccination
- Shifting vaccination in which targeted vaccination is used initially and then a shift occurs to mass vaccination after 28 days if the outbreak has not been controlled
- Targeted vaccination in which contact tracing is used in combination with selective vaccination of identified contacts

Vaccination programs selected are initiated after ten or more symptomatic patients appear. Once initiated, vaccination programs are implemented in a manner that enables them to reach their maximum hourly vaccination rate after a five-day third order delay. Maximum vaccination rates of 400 per hour for mass vaccination and 100 per hour for targeted vaccination (which is more labor-intensive due to the need for contact tracing).

Selecting targeted vaccination also automatically selects contact tracing. The effective targeted vaccination rate is the lesser of the contact-tracing rate, maximum targeted vaccination rate, or number of newly infected patients. Contact tracing by itself (solely to quarantine contacts) is initiated in the same manner as vaccination and is implemented after a similar delay before the maximum contact-tracing rate is achieved. As indicated earlier, the impact of both contact tracing and targeted vaccination is limited by the maximum fraction of contacts identified, assumed to be 80%.

The structure shown in Figure 4 simply reflects the flows of people in the community as the disease affects them. People move from the functioning to the disabled population as they develop the prodromal stage. They also begin to require treatment. The model tracks the number of people requiring hospitalization in order to be able to track bed requirements. A fraction of hospitalized patients die, determined by mortality rates elsewhere in the model, and the remainder go home to recover. After recovery, they rejoin the functioning population. A fraction could become permanently disabled, but this fraction is currently set to zero due to lack of the necessary data. The model also calculates the numbers of health care and emergency services workers and workers in other sectors unavailable due to deaths and disability caused by the disease.

The other significant piece of structure adopted from the LANL model is the tendency of people to self-quarantine once an epidemic has begun to spread in an area. This may be something people do spontaneously or in response to a government order. The maximum rates of people quarantining themselves range from 20-30% for adults who must go to work to 70-80% for young children and older people who can stay home more readily. The effect of self-quarantine with both formulations (fixed R0 and contact-based) is imposed on both the size of the susceptible population that can be affected and the rate of transmission that occurs between infected people and those who are susceptible. This pattern of self-quarantine was left as an option to be selected by the user rather than assumed to occur in each simulation.   As discussed later, the different formulations yield qualitatively different results that illustrate the value of taking these two perspectives.

Results of Simulations with Strategies for Controlling Outbreaks

1. Matching Baseline Simulations for Two Methods of Calculating New Infections

The first task, before exploring strategies for controlling outbreaks, was to assure that the two methods of calculating new infections produced roughly comparable results. Curves for cumulative cases from the two simulations are shown in Figure 5 below.   No other programs such as vaccination or contact tracing are in effect.

Cumulative Cases in Region

"Sddi: Total Cumulative Cases by Region"[Urg A] : base + infec sw=1 R0=255 base ▬ People
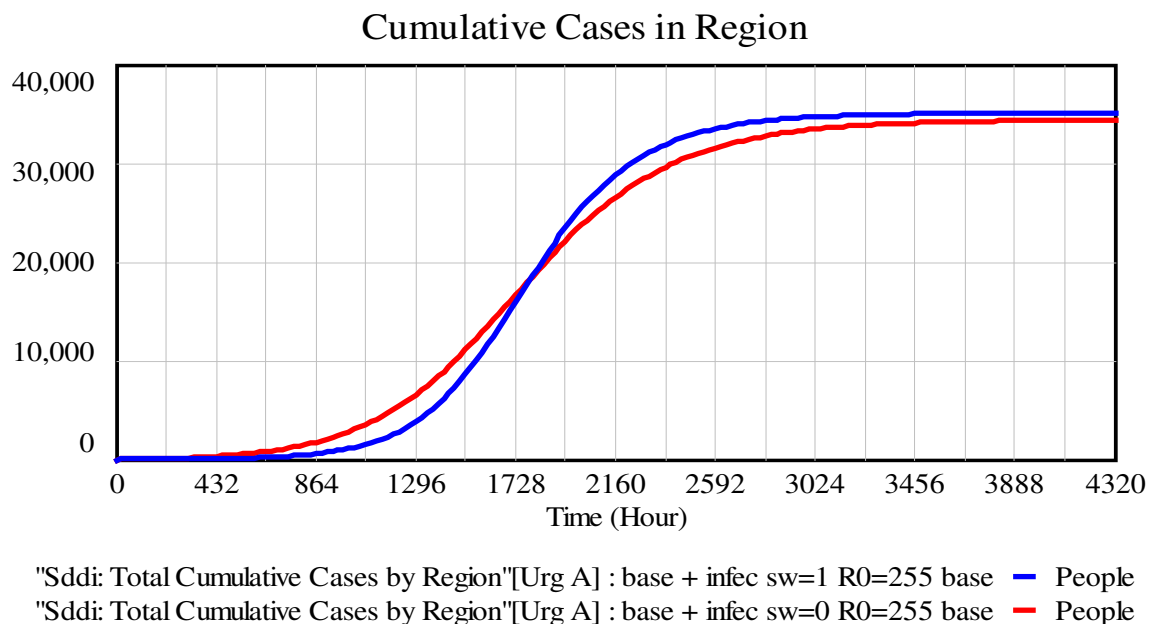"Sddi: Total Cumulative Cases by Region"[Urg A] : base + infec sw=0 R0=255 base ▬ People

Figure 5: Comparison of Baseline Simulations for Two Formulations of Infection Rates

The red line represents Cumulative Cases with the original formulation in the GID model (SW=0) based on an assumed R0 of 2.55. The blue line (SW=1) reflects the new formulation based on contacts. The two match reasonably well in terms of their timing and the ultimate attack rate experienced in the region. Further testing reveals some

interesting differences between how the two formulations react to particular interventions and sensitivity tests. These differences will be discussed later in this paper.

The small differences between the two simulations are easier to observe in the overall rates of new infections shown in Figure 6. The rate of new infections takes a bit longer to develop in the formulation based on contact rates (SW=1 in blue), but then has a narrower and taller peak.

New Case Comparison

"Sddi: Total New Infections"[Urg A] : base + infec sw=1 R0=255 base ──────
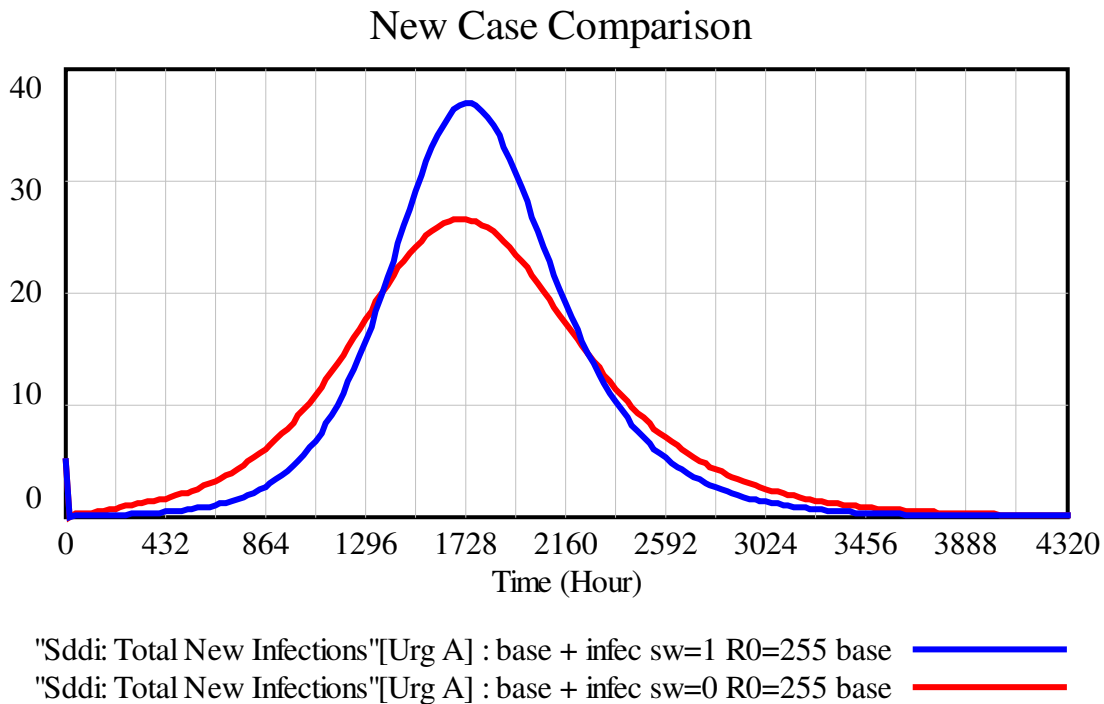"Sddi: Total New Infections"[Urg A] : base + infec sw=0 R0=255 base ──────

Figure 6: Comparison of New Infection Rates for the Two Formulations

In these baseline simulations and all of the others, the initial event that sets off the outbreak is five infected people arriving in Region A from elsewhere. Results shown in the following tables are for Region A only.

2. *Mass Vaccination*

Results with the two formulations for mass vaccination differ only in terms of the thresholds at which mass vaccination fails to be effective when certain key parameters are changed.   The effects of having a very small stockpile of vaccine available or having to use anti-viral drugs rather than vaccines are of special interest because of potential delays in producing enough vaccine if avian flu mutates into a form that can be transmitted between people. Table 2 shows results for mass vaccination, using each of the two formulations, in terms of Cumulative Cases in one region of 100,000 at the end of a six-month period. References to stockpiles represent the availability of vaccines or anti-viral drugs expressed as a percentage of the population that is initially susceptible.

	Based on Fixed R0	Based on Contacts
Base, No Interventions	34,373	35,088
Mass Vaccination with Stockpile =		
10% of Population	15,390	24,195
20%	1,312	9,451
25%	503	2,205
30%		420
25% Stockpile with		
71% Vaccine Effectiveness	547	
50% Vaccine Effectiveness	609	
10% Stockpile with		
50% Vaccine Effectiveness	16,051	24,228
3x Delay in Phase-In of Program (15 days vs. 5 days) and		
30% Stockpile		717
20% Stockpile	1,925	10,347
10% Stockpile	16,421	

Table 2: Results with Mass Vaccination Programs (Cumulative Cases)

As shown in Table 2, the size of the stockpile of vaccine available appears to make the biggest difference in whether it's possible to limit the attack rate experienced by the community. Though even a stockpile equal to 10% of the population can have a significant impact on the number of cases, compared to the "do-nothing" baseline, a stockpile of 20-30% is required to bring the attack rate (cumulative percentage of people infected) down below 1% of the population. Longer delays in starting the program don't seem to make much of a difference whether stockpiles are too small or large.

Levels of vaccine effectiveness that were simulated (percentage of people rendered immune) reflect the 71% estimated in one paper for anti-viral drugs (Balicer, 2005) and 50% which may be more realistic in light of recent news reports about the declining effectiveness of anti-virals. Changes in the effectiveness assumed for vaccines also appear to have little impact in making the program any less effective. However, vaccine effectiveness' apparent lack of impact is partly the result of the parameters chosen for these simulations. Experiments with a low level of effectiveness and large stockpile have the same result as the simulations with a small stockpile and higher level of assumed effectiveness. The smaller of the two constraints, size of the stockpile or vaccine effectiveness, appears to determine whether a mass vaccination program can stop an outbreak

The impacts of mass vaccination are similar for both formulations, but are achieved at a lower level of stockpile available with the formulation based on the fixed R0. Given the degree of uncertainty in the numbers used in both formulations, it's difficult to say that either is likely to be more accurate. Instead, they together represent a range of possible thresholds for effective mass vaccination. The more important point is that severe stockpile limitations early in an outbreak may compromise the effectiveness of mass vaccination and not be the best use of limited supplies. It may be necessary to commit enough vaccine to the initial communities that experience outbreaks instead of following a politically more expedient course of spreading limited supplies over many states.

3. *Targeted Vaccination*

Another, more efficient use of limited supplies may be targeted vaccination which combines contact tracing, vaccination of contacts of symptomatic patients, and quarantine of those contacts until it's clear they're not going to develop the disease. Table 3 shows the results of a number of simulations using targeted vaccination strategies with different assumptions about effectiveness and potential flows of infected people into the community after an outbreak has begun. Unless otherwise noted, only a 10% stockpile is assumed along with 98% vaccine effectiveness and 80% contact effectiveness (people are able to remember and/or contact tracing is able locate 80% of actual contacts). Again, results shown are Cumulative Cases after six months.

	Based on Fixed R0	Based on Contacts
Base, No Interventions	34,373	35,088
Targeted Vaccination with		
80% Contact Effectiveness	68	
50%	115	50
30%		404
20%	1,106	6,171
20% Contact Effectiveness and		
36% Vaccine Effectiveness	1,184	10,162
10% Contact Effectiveness and		
36% Vaccine Effectiveness	11,091	25,314
Inflow of 20 Infected People/Day and		
30% Contact Effectiveness	14,032	15,583
50%	8,475	8,367
80%	5,159	4,782

Table 3: Results with Targeted Vaccination Programs (Cumulative Cases)

	Based on Fixed R0	Based on Contacts
R0=4, 50% Contact Effectiveness	1,649	
R0=4, 50% Contact Effectiveness, Inflow of 20 People/Day	23,970	

Table 3 (Cont'd): Results with Targeted Vaccination Programs (Cumulative Cases)

The striking thing about the results shown in Table 3 is that targeted vaccination programs appear to be effective even when only 20-30% of contacts can be found and treated effectively. This is because the "effective R0" with both formulations is low enough that even a 20-30% reduction will bring its value below one. (The assumptions that symptomatic people are isolated or otherwise drastically reduce their contacts helps to reduce this effective R0.) Again, the effect of targeted vaccination is apparent with both formulations for new infection rate, but is evident at a lower threshold of contact effectiveness with the first formulation than the second.

These results suggest that targeted vaccination is a much more efficient use of limited vaccines since they were achieved with a stockpile of only 10%. (An even smaller amount of vaccine was actually needed.) The power of this approach is in its name, targeting those contacts who are most likely to transmit the disease, and in the simultaneous use of quarantine in addition to vaccination. However, the very brief incubation period of influenza makes speed critical if this strategy is to be effective. Carrying out this strategy will require preparation in the form of

- enough well-trained people (on whom it might be a good idea to use some of the limited vaccine supplies),
- very good communications, and
- a support system to bring food and other necessities to people who must remain quarantined for a number of days.

Targeted vaccination seems more vulnerable if one assumes a steady, small stream of infected people coming into the region from other areas. The inflow of infected people from other areas may be likely if people flee areas with outbreaks and bring new infections to regions that have been relatively unaffected. This may require government to urge people to "quarantine in place" rather than moving around the country to "safer" areas and bringing their germs with them. A more rapid rate of spread (e.g., due to a more easily transmitted virus), reflected in the simulations where R0 is set to 4, may also undermine the effectiveness of targeted vaccination. The combination of more rapid spread and steady inflow of infected people would produce a significant number of cases at a rate of contact effectiveness (50%) that might otherwise stop an outbreak.

4. Transmission by Asymptomatic People

The simulations reported on up to this point assume that transmission only occurs in the case of people who also become symptomatic. Transmission by asymptomatic people is suggested by the literature as a distinct possibility, but no one appears to have estimates of the magnitude of this threat. The model was used to do some sensitivity analysis to gauge the potential effects of asymptomatic people transmitting the disease at some fraction of the rate typical of symptomatic patients. Table 4 shows the results of these simulations. The basic assumptions are that the additional number of people who are asymptomatic is equal to 50% of the number showing symptoms and those people are 25% as likely to transmit the disease as someone who is symptomatic.

	Based on Fixed R0	Based on Contacts
Base, No Interventions (Without the effect of asymptomatic transmission)	34,373	35,088
Asymptomatic People = 50% of Symptomatic; Transmission 25% as Likely with		
No Interventions	60,046	62,442
Targeted Vaccination with		
30% Contact Effectiveness	4,528	
50%	238	16,266
60%	150	1,076
80%		94
Mass Vaccination with		
10% Stockpile	44,773	
25%	19,830	
30%	9,901	
40%	831	23,492
50%	353	12,804
60%		1,633
70%		320

Table 4: Results of Simulations Assuming Transmission by Asymptomatic People

These results indicate a significant increase in the number of Cumulative Cases if there is transmission by asymptomatic people, even if it is at a much lower rate than for symptomatic patients. With transmission by asymptomatic patients included, targeted vaccination requires a higher degree of contact effectiveness and mass vaccination needs larger stockpiles in order to bring an outbreak under control. There is again the same

pattern where simulations with the formulation based on contacts indicate a need for higher rates of contact effectiveness or larger vaccine stockpiles in order to limit the spread. These results suggest that the issue of transmission by asymptomatic people is an important one and should be a focus for research.

5. *Self-Quarantining*

The last set of tests of the model involves the self-quarantining feature that was added. As indicated earlier, setting a switch can cause different fractions of the population to self-quarantine in response to the beginning of an outbreak. Self-quarantining is assumed to cause a reduction in both the size of the susceptible population and rates of contacts and transmission. The maximum fractions that self-quarantine are shown in Table 5.

Population Group	Maximum Tendency to Self-Quarantine
Health and Emergency Workers	10%
Other Employed 18-64	20
Non-Employed 18-64	40
Under 18	70
Over 65	70

Table 5: Maximum Tendency (%) to Self-Quarantine

Table 6 shows the results of simulations with the self-quarantining function switched on.

	Based on Fixed R0	Based on Contacts
Base, No Interventions	34,373	35,088
With Self-Quarantining	16,146 (34,117 at the end of one year)	52
With Self-Quarantining and Mass Vaccination with 10% Stockpile	319	

Table 6: Effect of Self-Quarantining on Cumulative Number of Cases at Six Months

The results of assuming self-quarantining are significant with both formulations, but strikingly different between the two. The difference is even more striking in that the number of Cumulative Cases in the simulation based on the fixed R0 is growing at the

end of six months. The final value of 34,117 if the simulation is allowed to run out to the end of a year is close to the value in the base run (without self-quarantining).   Evidently, self-quarantining has the effect with both formulations of suppressing the growth of new cases. In the contact-based formulation, this is sufficient to end the outbreak. With the other formulation based on a fixed R0, there are evidently enough cases left that the numbers start growing again after people stop self-quarantining. Without another round of self-quarantining or any other interventions, the numbers eventually reach the level they would have in the no intervention base case. The outbreak is merely delayed rather than stopped.

This disparity between results of the two formulations shows the potential value of having the option to use both. Looking at the result with the contact-based formulation, one might hope that self-quarantining by a significant fraction of the population is enough to stop an outbreak. However, the results with the other formulation suggests that there might be a real danger of merely reducing the number of cases to a low level and then having the outbreak spring up again once people come out of self-quarantine. This further suggests a need to combine self-quarantining with other interventions and to prepare the public for more than one wave of self-quarantining if an outbreak springs up again.   Table 6 shows that combining even a weak mass vaccination program (that would have resulted in 15,390 cases by itself) with self-quarantining might be sufficient to stop an outbreak. A stronger effort at self-quarantine (e.g., making it mandatory for a larger fraction of the population (e.g., those not in essential occupations) might also have a greater likelihood of stopping the outbreak.

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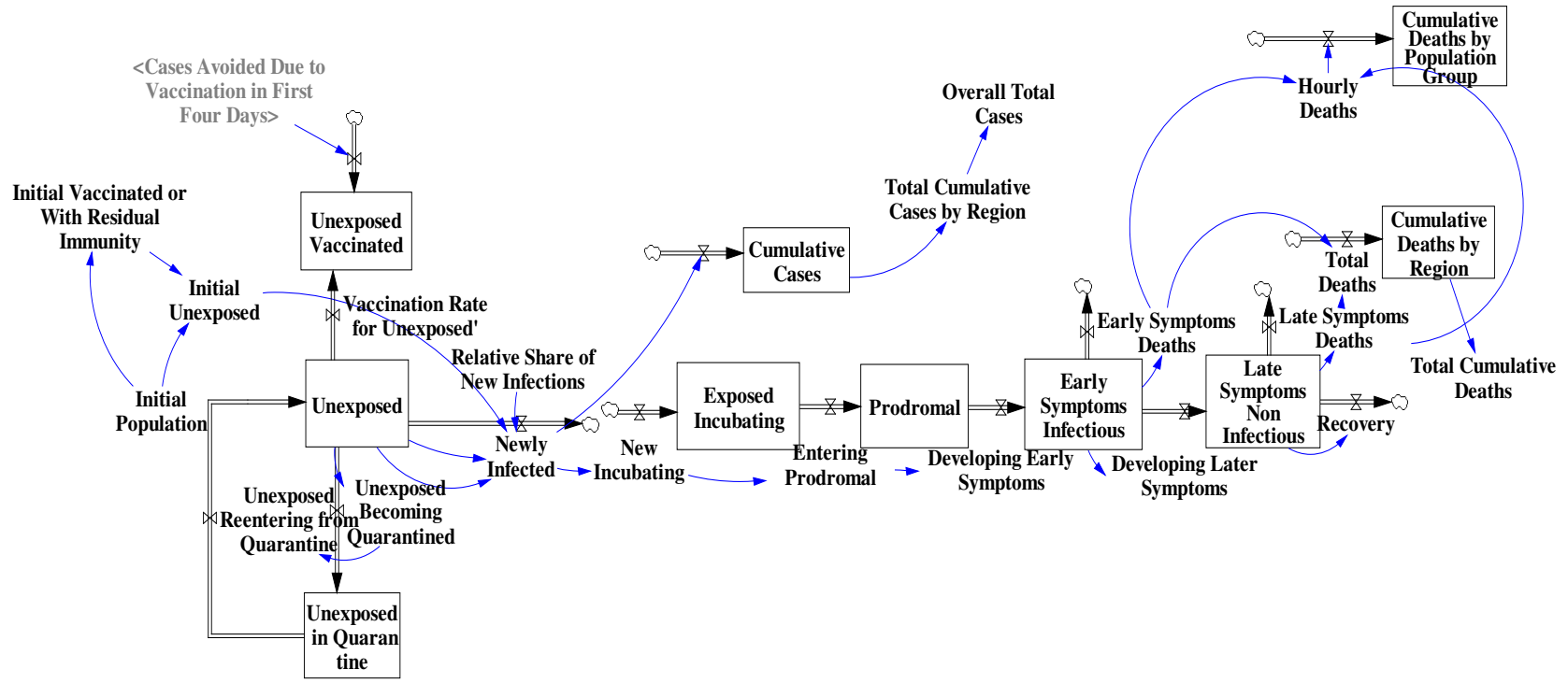
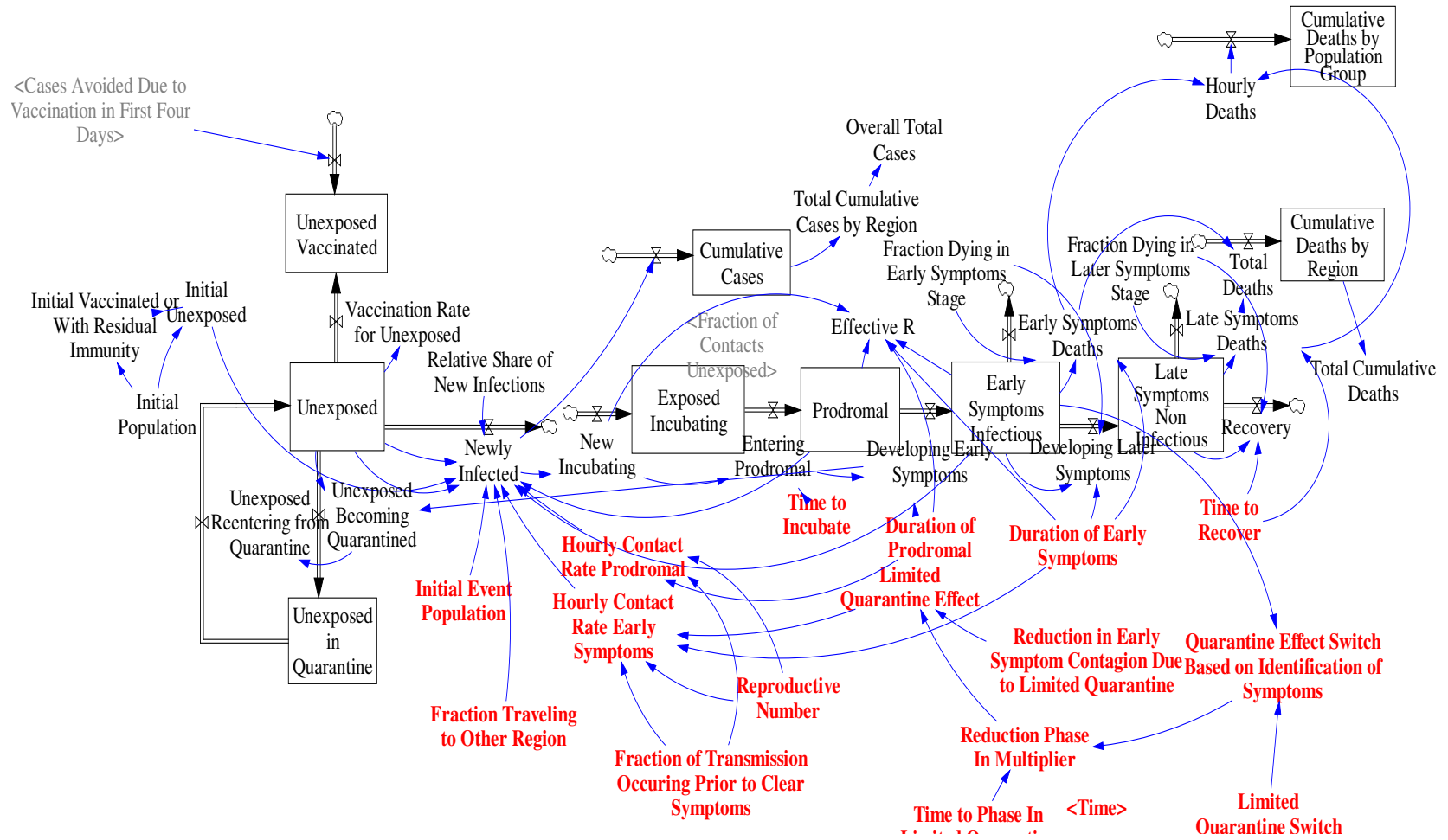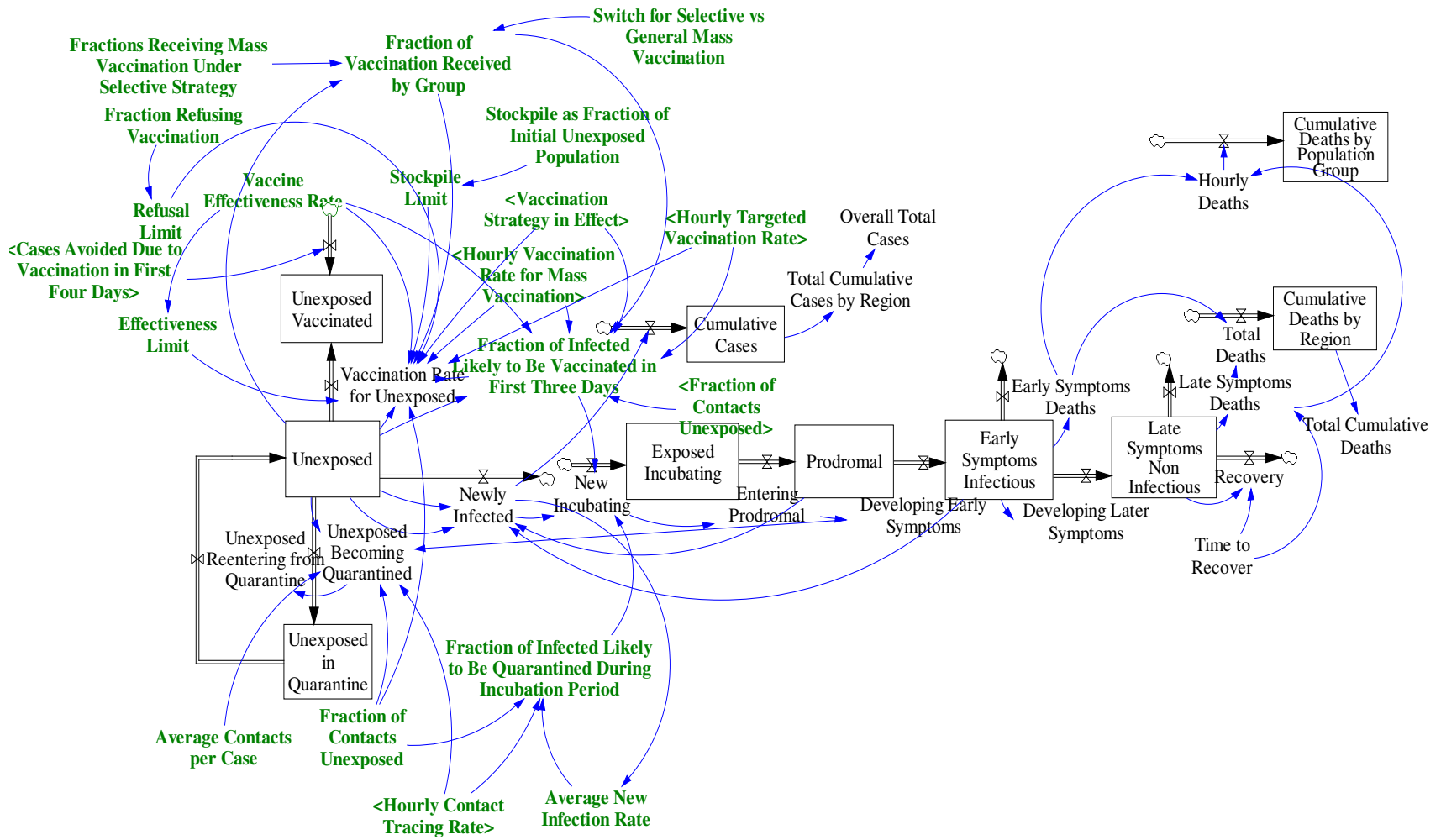Figure 1: Flows of People Through Disease Stages

Figure 2: Factors Affecting Rate of Spread

Figure 3: Impacts of Quarantine and Vaccination