

EPIDEMIC MODELLING USING SARS AS A CASE STUDY

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ABSTRACT

The recent Severe Acute Respiratory Syndrome (SARS) epidemic has highlighted a new dimension to the risks confronting insurance companies. Conventional approaches to insurance pricing take an almost exclusively retrospective view of future mortality experience, extrapolating past mortality trends into the future. Such an approach fails to take account of mortality shocks such as epidemics, which may arise spontaneously and that are not reflected in past experience. If actuaries are to maintain their position as risk experts in an ever-changing world, it is important for the actuarial profession to adopt a more comprehensive approach to assessing risks that goes beyond past experience.

This paper will take a look at the modelling of epidemics, using SARS as a case study, and will examine the potential impact of SARS and similar epidemics on insurance companies.

1. EPIDEMICS AND EPIDEMIC MODELLING

Infectious diseases have always played a leading role in the death and disability of humans. The influenza pandemic of 1918–19 is generally acknowledged to have accounted for 20 to 50 million worldwide (see World Health Organization [WHO] 2005), substantially more than the number of people who died in World War I. With the 2003 outbreak of SARS and subsequent isolated outbreaks of Avian influenza, research activity among medical and healthcare professionals has accelerated rapidly. Current epidemiological models from the U.S. Centers for Disease Control and Prevention (CDC) predict that, even in the best-case scenario of a new influenza pandemic evolving out of existing Avian influenza strains, 2 to 7 million people would die and tens of millions would require medical attention. In any such scenario, it is undoubted that epidemics will have an impact on insurance industry. For actuaries to play a part in assessing the potential impact of

SARS and other epidemics, further understanding in the characteristics, spread, and control of epidemics is crucial.

This paper firstly introduces some fundamental concepts and techniques in the mathematical modelling of epidemics (Section 2) and uses these concepts and techniques to develop a model for the 2003 SARS outbreak in Hong Kong (Section 3). Using this case study, we then demonstrate how mathematical models can be used to develop a deeper understanding of epidemics (Sections 4 and 5) with a particular focus on the three main aims espoused by Daley and Gani (1999):

1. to understand the mechanisms through which infectious diseases spread;
2. to determine how the spread of an epidemic can be controlled; and
3. to predict the future course of an epidemic.

2. CONVENTIONAL APPROACHES TO EPIDEMIC MODELLING

Almost all modern epidemic models (see Anderson and May [1991], Daley and Gani [1999], and Hethcote [2000]) make use of a multiple-state approach, segmenting the modelled population into a set of distinct classes, each exhibiting

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different characteristics with respect to the disease. Such an approach should be familiar to actuaries as very similar principles underpin the modelling of most health and disability insurance products.

The states that are modelled would typically include those described in Table 1. The existence (or otherwise) of each of these five states, together with the links between the respective states, is usually sufficient to provide a broad outline of the particular epidemic model being used, particularly in describing acute epidemics, which develop and spread over a short period of time. However, models for more chronic and long-term epidemics would also need to consider births and deaths. A further enhancement for models dealing with diseases for which vaccines have been developed is to include a further state for those lives that have been immunized against a particular disease.

Making use of the states outlined above, it is conventional to name epidemic models after the states considered in the modelling process and the possible transitions between the various states. For example, one of the simplest epidemic models is called the Susceptible, Infective, and Removed (SIR) model (see Section 2.1) after the 3 states considered in the modelling. Various other epidemic models, considering combinations of the above five states are shown in Table 2.

2.1 The Classic SIR Model

One of the most basic epidemic models is the SIR model. The states underlying the SIR model are

illustrated in Figure 1. The total population (N) in the Basic SIR model is fixed such that, at all times, $N = S + I + R$. The movement of lives into and out of each state are then described using the equations outlined in the paragraphs below.

Firstly, we consider movements from the Susceptible (S) state to the Infective (I) state. In order to determine the number of new exposed lives in any one period of time, the classic SIR model uses a relationship known as the *Law of Mass Action*. Applied to epidemics, this law states that:

“If the individuals in a population mix homogeneously, the rate of interaction between two different subsets of the population is proportional to the product of the numbers in each of the subsets concerned.”

Noting that the mixing within the total population (N) is assumed to be homogeneous, the number of newly exposed lives in any period of time is assumed to be proportional to the product of the number of lives in the Susceptible (S) and Infective (I) states. By convention, the constant of proportionality is denoted by β or β/N and hence, the number of new infections per unit time is:

$$\frac{\beta IS}{N}$$

Next, we consider movements from the Infective (I) state to the Removed (R) state. A much simpler process is assumed in this case, such that lives recover at a constant rate, conventionally de-

Table 1
States Used in Conventional Epidemic Modelling

Symbol	Epidemic State	Description
M	Passively Immune	Lives who have acquired temporary immunity to a particular disease without having ever been infected. An example of this state would be newborn infants with antibodies against the disease passed from their mother. These antibodies eventually disappear from the body at which time the infant moves into the Susceptible state.
S	Susceptible	Lives who are healthy, but who could potentially develop the disease.
E	Exposed	Lives who have been infected and with the disease, but who are still in the latent period (with or without symptoms of the disease) and who cannot transmit the disease to others.
I	Infective	Lives who are infected with the disease (with or without symptoms of the disease) and who are capable of transmitting the infection to others.
R	Removed	Lives who have either died or recovered from infection thereby acquiring immunity (temporary or permanent) from infection.

Table 2
Conventional Epidemic Models

Model	Characteristics
<i>SI</i>	Once a susceptible member of the population (<i>S</i>) has been infected with the disease, he or she is immediately infective (<i>I</i>) and capable of transmitting the infection to others. No recovery from the disease is possible.
<i>SIS</i>	Same as the <i>SI</i> model, except that recovery from the disease is possible. However, upon recovery, a life is immediately susceptible (<i>S</i>) once again. That is, recovery from the disease does not confer any immunity against future infection.
<i>SEI</i>	Same as the <i>SI</i> model, except that, following initial exposure (<i>E</i>) to the disease leading to infection, there is a latent or incubation period during which the disease cannot be passed on to others.
<i>SEIS</i>	Same as the <i>SIS</i> model, except that, following initial exposure (<i>E</i>) to the disease leading to infection, there is a latent or incubation period during which the disease cannot be passed on to others.
<i>SIR</i>	Same as the <i>SI</i> model, except that recovery (<i>R</i>) from the disease is possible. Once recovered from the disease, there is lifelong immunity from reinfection.
<i>SIRS</i>	Same as the <i>SIR</i> model, except that postrecovery immunity is only temporary. Following a period of immunity, a life may become susceptible (<i>S</i>) once again.
<i>SEIR</i>	Same as the <i>SIR</i> model, except that following initial exposure (<i>E</i>) to the disease leading to infection, there is a latent or incubation period during which the disease cannot be passed on to others.
<i>SEIRS</i>	Same as the <i>SEIR</i> model, except that postrecovery immunity is only temporary.
<i>MSEIR</i>	Same as the <i>SEIR</i> model, with the addition of lives who are passively immune (<i>M</i>) from infection when they enter the population.
<i>MSEIRS</i>	Same as the <i>MSEIR</i> model, except that postrecovery immunity is only temporary.

noted by γ . Thus, the number of movements from the Infective (*I*) to the Removed (*R*) state per unit of time is γI .

Putting the transitions together gives the following system of differential equations:

$$N = S + I + R$$

$$\frac{dS}{dt} = -\frac{\beta IS}{N}$$

$$\frac{dI}{dt} = \frac{\beta IS}{N} - \gamma I$$

$$\frac{dR}{dt} = \gamma I.$$

Solving this system of differential equations to give the number of Infective (*I*) cases over the course of an epidemic yields the typical epidemic curves shown in Figures 2a and 2b.

2.2 Extensions to the Classic SIR Model

From the description above, it is evident that the classic SIR model represents a gross simplifica-

tion of the spread of most modern infectious diseases. In terms of practical application to the real world, the SIR model is generally used only as a foundation for more complex epidemic models and as an educational tool to illustrate the principles underlying the mathematical modelling of epidemics. The extensions to the modelling of epidemics are numerous and some of these are described in Table 3.

2.3 The Basic Reproductive Ratio

In line with the literature on the mathematical modelling of infectious diseases, we will also introduce a further key parameter that provides a fundamental basis for comparing the “infectiousness” of different infectious diseases.

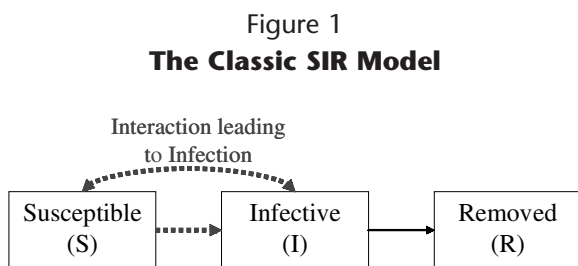


Figure 2a
Typical Epidemic Curves
 Proportion of Lives in Infective (*I*) State

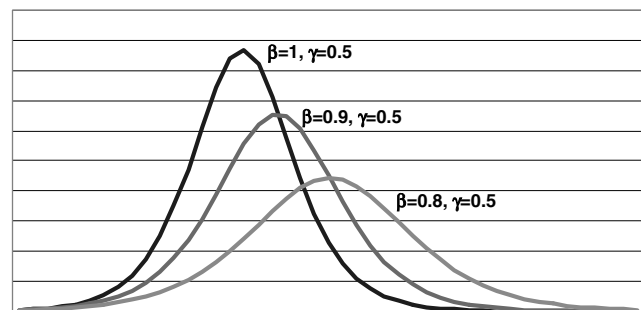


Figure 2b
Typical Epidemic Curves

Proportion of lives in Susceptible (S) and Removed (R) States

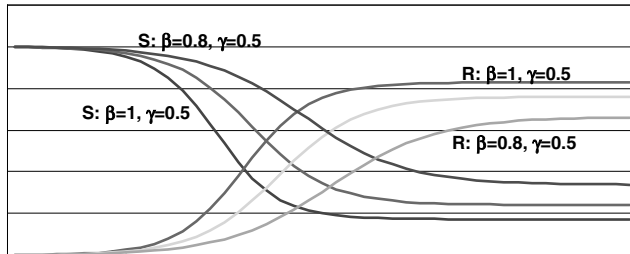


Table 4
Estimates of R_0 for some Common Infectious Diseases

Disease	R_0 Estimate
Measles	16
Chicken Pox (Varicella)	11
Mumps	12
Rubella (German Measles)	7
Poliomyelitis	5
Smallpox	8
AIDS	2 to 5
Malaria	>100

The Basic Reproductive/Reproduction Ratio or R_0 is defined as:

the average number of secondary infections produced when one infected individual is introduced into a population where everyone else is susceptible.

In short, the quantity R_0 governs whether an infection can spread and be sustained within a population. If R_0 is greater than one, then the number of infections in a susceptible population will increase and the infection will be sustained, whereas if R_0 is less than one, the infection should fail to take hold and will die out very quickly.

The concept of the Reproductive/Reproduction Ratio can also be extended to later time periods. R_t is the average number of secondary infections produced from one infected individual at time t during the epidemic. After the infection has started and some lives in the population are no longer susceptible (having been infected or acquired immunity after recovering from the disease), R_t for $t > 0$, should always be less than R_0 . Once R_t drops below one, an epidemic will start to decline and should gradually die out.

Estimated values of R_0 for various different infectious diseases are shown in Table 4 (see Hethcote [2000] and Keeling [2003]).

Table 3
Extensions to Basic Epidemic Models

Extension	Characteristics
Changes in the total population Nonhomogeneous mixing of subpopulations	Allowances are made for changes to the total population (e.g., births, deaths, and migration). Susceptible (S) and Infective (I) populations may be split into smaller subpopulations with different rates of interaction within and between subpopulations (e.g., HIV/AIDS models incorporate subpopulations with higher exposure to the disease, such as intravenous drug users and prostitutes).
Subpopulations with varying susceptibility to infection	S and I populations may be split into smaller subpopulations with the probability of becoming infected differing in each subpopulation (e.g., age-segmented models for measles where immune system difference mean that different ages become infected at different rates).
Seasonal/time variations in mixing of subpopulations	Models that allow for seasonal differences in the rate of interaction over time (e.g., the rate of interaction for epidemics affecting children will be higher during school terms than during vacations).
Seasonal/time variations in susceptibility to infection Carrier-borne or vector-borne diseases	Models that allow for seasonal differences in the rate of infection over time (e.g., people are more susceptible to cold and influenza infections during colder months). Rather than being passed directly between Infective (I) and Susceptible (S) lives, the disease is carried by other hosts (such as rats or mosquitoes). Often, such models will require multiple systems controlling the pattern of transmission from human to host animal (if any), transmission within the host animal (if any), and transmission from the host animal back to humans.
Stochastic models	The transmission dynamics are governed by random processes rather than deterministic relationships. Stochastic models provide for a greater understanding of the range of possible outcomes for any given epidemic, rather than focusing on a single mean or expected outcome.

3. PROPOSED MODEL FOR SARS

Various papers on SARS modelling have been published following the SARS outbreak in the first half of 2003 (see Dye and Gay [2003], Lipsitch et al. [2003], and Riley et al. [2003]). However, in order to provide further insight into the key factors governing the spread of epidemics such as SARS, we have developed our own comprehensive model to describe the whole process from the initial very few infective cases to the full outbreak of SARS and eventual extinguishment.

Amongst all the regions affected by SARS, Hong Kong appears to provide the best prospect for analysis with the combination of a relatively large volume of data together with relatively accurate reporting of SARS cases. Therefore we have used Hong Kong data to calibrate our model (published by the Clinical Trials Centre [2003] and by the World Health Organization [2003] with additional information provided in Donnelly et al. [2003]).

3.1 Proposed Model Structure

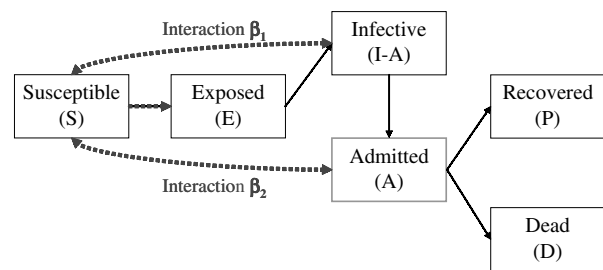
We used the Classic Susceptible, Exposed, Infective, and Removed (SEIR) model as the base for our model. Experience has shown that SARS is an acute epidemic that breaks out and spreads over a relatively short period of time, factors such as births and deaths in the susceptible population have been ignored.

Nevertheless, the classic SEIR model would appear to be insufficient to describe the SARS outbreak. In particular, when considering the actual path of the SARS epidemic, the classic SEIR model exhibits the following shortcomings:

1. It assumes a *single* rate (β) of secondary infection for lives moving from the susceptible state (S) to the exposed state (E).
2. It assumes *constant* rates of transition for movements from the exposed state (E) to the infective state (I), and from the infective state (I) to the removed state (R).
3. It assumes the susceptible population (N) is the total population.

The structure of our model and the relationships between the various states are summarized in Figure 3. To address the shortcomings outlined above, our model has been modified from the basic SEIR model as follows:

Figure 3
Proposed Model for SARS



$$S \rightarrow E$$

$$\frac{\beta_1(I-A)S + \beta_2AS}{N}$$

$$E \rightarrow (I-A)$$

as described in Donnelly et al. (2003), the number of days required for transition from state E to $(I-A)$ follows a Gamma Distribution with (mean, variance) of (6.37, 16.69)

$$(I-A) \rightarrow A$$

as described in Donnelly et al. (2003), the number of days required for transition from state $(I-A)$ to A follows a Gamma Distribution with (mean, variance) of (4.85, 12.19)

$$A \rightarrow P \text{ and } D \text{ (Recovered and Dead)}$$

as described earlier, the number of days required for transition from state A to P or D follows a Gamma Distribution with (mean, variance) of (23.47, 101.49), with proportion q_x moving to D and proportion $(1 - q_x)$ moving to P .

1. We know that as soon as a diagnosis of SARS is made, the person concerned would be hospitalized, thereby stopping the spread of infection in the general population. We also know that in the early part of the epidemic, substantial numbers of people were infected within hospitals. However, the pattern of infection within hospitals differed from the pattern of infection outside hospitals.

We have therefore split the Infective (I) state into two subgroups, with each subgroup subject to a different rate of secondary infection (β):

- “ $(I-A)$ ” represents cases not yet admitted to hospital, with β_1 representing the rate of secondary infections affecting these lives;
- “ A ” represents those cases admitted to hospital, with β_2 representing the rate of secondary infection affecting these lives. Lives are assumed to move from state $(I-A)$ to A in accordance with the gamma distribution fitted in Donnelly et al. (2003) with mean and variance of 4.85 and 12.19 respectively.

Compared to the SEIR or SIR model (see section 2.1), the total rate of secondary infection is therefore modified from:

$$\frac{\beta IS}{N} \text{ to } \frac{\beta_1(I-A)S + \beta_2AS}{N}.$$

According to reports from the Hospital Authority of Hong Kong (see World Health Organization [2003]), full control of hospital-based transmission of SARS only started to take place after 28 March 2003. We have therefore assumed that the rate of infection within hospitals (β_2) reduced to zero after 28 March.

2. A constant rate of exposed state (E) to the infective state (I), and from the infective state (I) to the removed state (R) implies that the duration spent in each states E and I follow an exponential distribution. However, the empirical data (see Donnelly et al. [2003]) appear to indicate that the duration from exposure to first symptoms, from first symptoms to hospital admission, and from hospital admission to death or discharge, all follow a gamma distribution.

We have therefore used the gamma distributions fitted in Donnelly et al. (2003) to derive transition probabilities for movements from the exposed state (E) to the infective but not yet admitted to hospital state ($I-A$), and from ($I-A$) to the admitted state (A). For the transition from the admitted state (A) to the recovered (P) and dead (D) states, we derived our own gamma distribution for the pattern of deaths and recoveries. Unlike the two separate gamma distributions fitted in Donnelly et al. (2003), we fitted a single gamma distribution (mean = 23.47, variance = 101.49, $R^2 = 0.77$) for deaths and recoveries such that:

$$\text{Deaths} = [\text{SARS mortality rate}] \times [\text{total deaths and recoveries}]$$

$$\text{Recoveries} = (1 - [\text{SARS mortality rate}]) \times [\text{total deaths and recoveries}]$$

3. With increased awareness of an infectious disease such as SARS, quarantine and restrictions imposed by either international or domestic health organizations, the susceptible population ($S_0 = N$) for an epidemic would be reduced significantly to a much smaller subset of the entire population. As it is impossible to identify the actual susceptible population, in our model, we have included N as one of the parameters that we need to estimate together with β_1 and β_2 .

3.2 Other Assumptions

In addition to the above, the following assumptions were made:

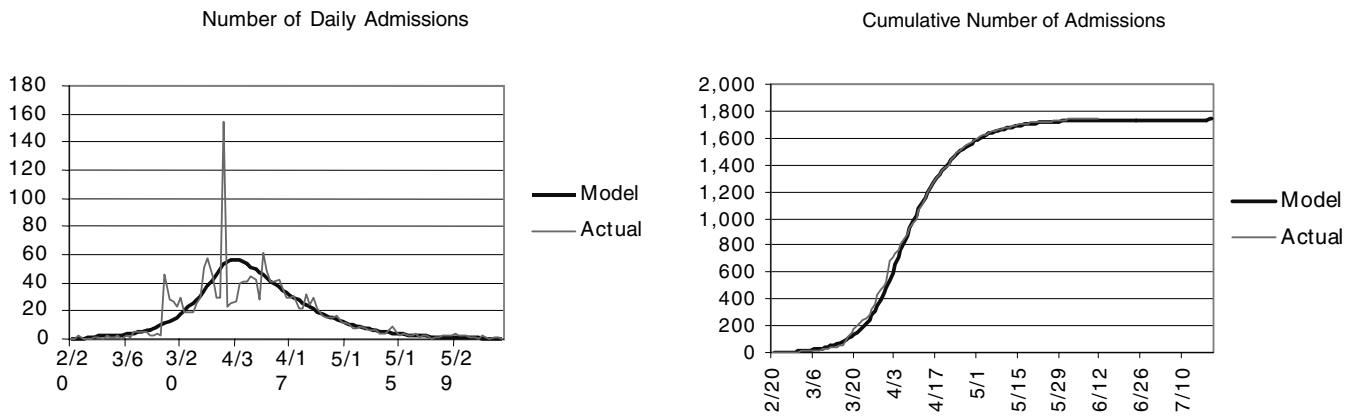
- The start date for our model is 20 February 2003 (i.e., $t = 0$ at 20 February 2003). This is approximately 2 weeks before the first reported SARS cases in Hong Kong (bearing in mind that for most SARS cases, the period from infection to onset was less than 14 days).
- The end date for our model is 20 July 2003 (i.e., $t = 150$), by which time the SARS epidemic in Hong Kong was largely finished.
- The number of index cases at 20 February 2003 is 24 (i.e., $E_0 = 24$). This figure was derived by using the reported SARS cases in hospital (A) in early March, together with the transition probabilities from the exposed state (E) to the infective but not yet admitted to hospital state ($I-A$), and from the ($I-A$) state to the admitted state (A) and working backwards. In other words, according to our model, there had to be 24 cases in the exposed (E) state at 20 February 2003 to create a SARS outbreak with the pattern observed in early March 2003.
- The hospital infection rate drops to zero from 28 March onwards (i.e., $\beta_2 = 0$ for $t \geq 36$) as noted in Section 3.1.
- The case fatality rate for SARS in Hong Kong is 17.5% (i.e., $q_x = 0.175$), which is consistent with the final mortality rate taken from Clinical Trials Centre (2003).

Note that, in fitting the model, the publicly reported number of SARS cases would be analogous to the number of lives in state A of our model, the number of cases diagnosed and admitted to the hospital.

3.3 Super-Spreading Events (SSEs)

From the empirical data for Hong Kong, we know that a severe localized epidemic occurred in Amoy Gardens in March 2003. Through contact tracing, it has been estimated (see Clinical Trials Centre [2003]) that 332 cases were involved in this outbreak, a large proportion of whom could be traced to a single person. Outbreaks involving the direct spread of the disease from one primary case to a very large number of secondary cases are often referred to as ‘‘Super-Spreading Events’’ (SSEs). As the inclusion of SSEs may exaggerate

Figure 4a
Model with Amoy Gardens—Reported Cases (Admitted to Hospital)



the actual infectiousness of SARS, when fitting our model to the empirical data, we used data both including Amoy Gardens cases and excluding Amoy Gardens cases to understand the impact of SSEs on the SARS outbreak.

3.4 Model Parameter Estimates and Results

Using published daily information on the number of new SARS cases as well as the number of deaths and recoveries (see Clinical Trials Centre [2003], and Daley and Gani [1999]), we derived the key parameters (see Sections 3.4.1 and 3.4.2) for our proposed model by using a least squares method. Essentially, we minimized the un-weighted sum of the squares of the differences between observed new SARS cases and new SARS

cases predicted by the model, as well as the un-weighted sum of the squares of the differences between observed new SARS deaths/recoveries and new SARS deaths/recoveries predicted by the model.

3.4.1 Model with Amoy Gardens Cases

- β_1 (rate of secondary infection for cases not yet admitted) = 0.25.
- β_2 (rate of secondary infection for cases admitted to hospital) = 0.365 before 28 March.
- β_2 (rate of secondary infection for cases admitted to hospital) = 0 afterwards.
- N (susceptible population) = 2,396.

The results are illustrated in Figures 4a, 4b and 4c.

Figure 4b
Model with Amoy Gardens—SARS Deaths

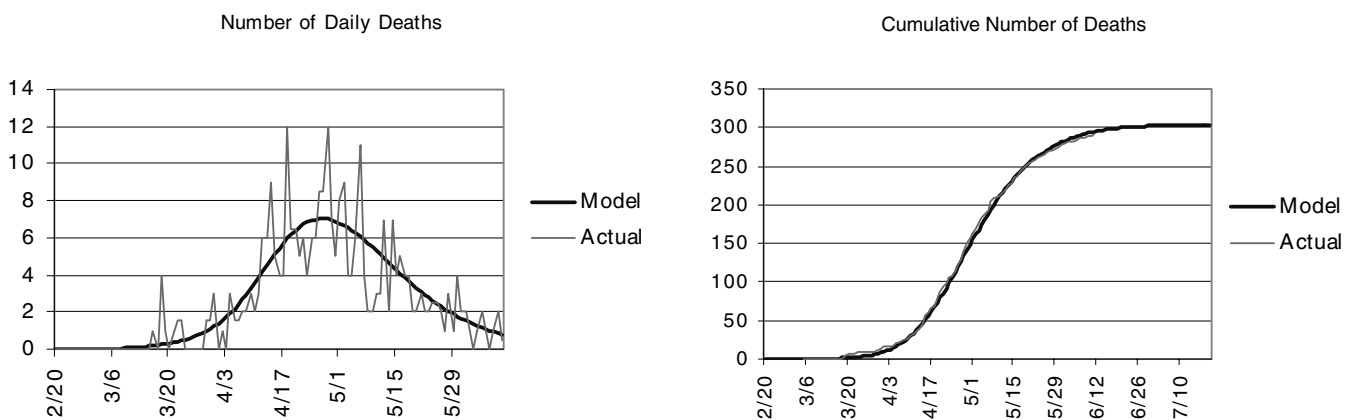
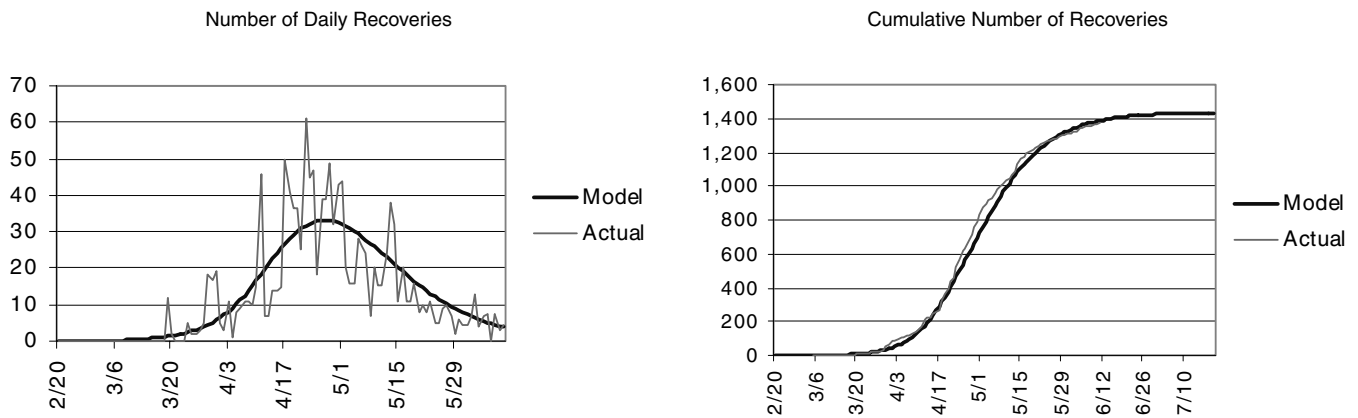


Figure 4c
Model with Amoy Gardens—Recoveries from SARS



3.4.2 Model without Amoy Gardens Cases

- β_1 (rate of secondary infection for cases not yet admitted) = 0.22.
- β_2 (rate of secondary infection for cases admitted to hospital) = 0.28 before 28 March.
- β_2 (rate of secondary infection for cases admitted to hospital) = 0 afterwards.
- N (susceptible population) = 2,368.

The results are illustrated in Figures 5a, 5b, and 5c.

4. CONCLUSIONS FROM THE MODEL

4.1 Basic Reproductive Ratio (R_0)

If we exclude SSEs and ignore the high rate of in-hospital infection during the initial weeks of the

epidemic, it is relatively simple to estimate the basic reproductive ratio R_0 for the SARS epidemic in the general population. From our model excluding Amoy Gardens cases (see Section 3.4.2), the average number of secondary infections per unit time in the general population is $\beta_1 = 0.22$. Assuming that the early infections are only a very small fraction for the susceptible population (so that the susceptible population can for all practical purposes be treated as a constant), then R_0 is approximately $\beta_1 D$ where D is the average duration that a SARS case remains infective.

We know that:

D = average duration from onset of symptoms to death or recovery.

Using the gamma distributions fitted by Donnelly et al. (2003), this is:

Figure 5a
Model without Amoy Gardens—Reported Cases (Admitted to the Hospital)

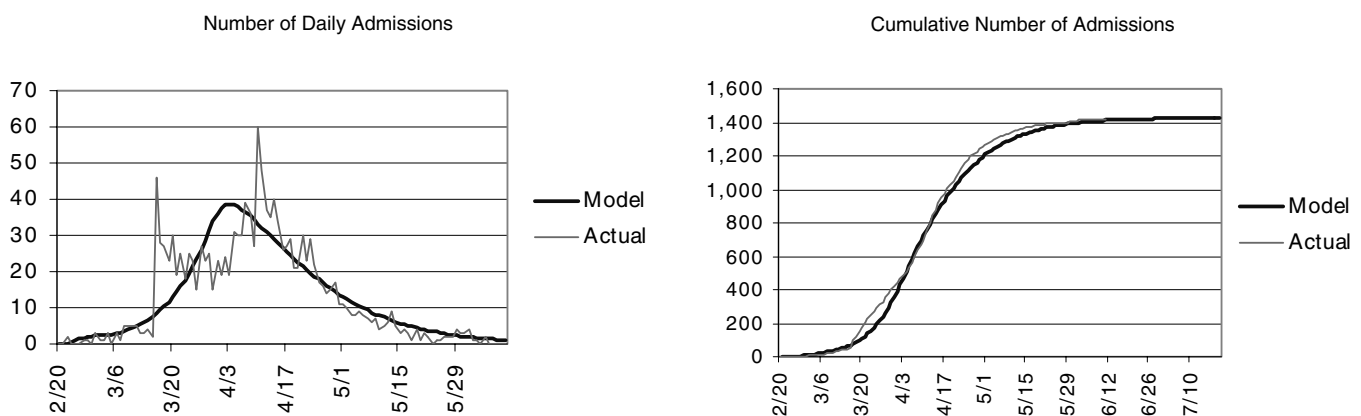
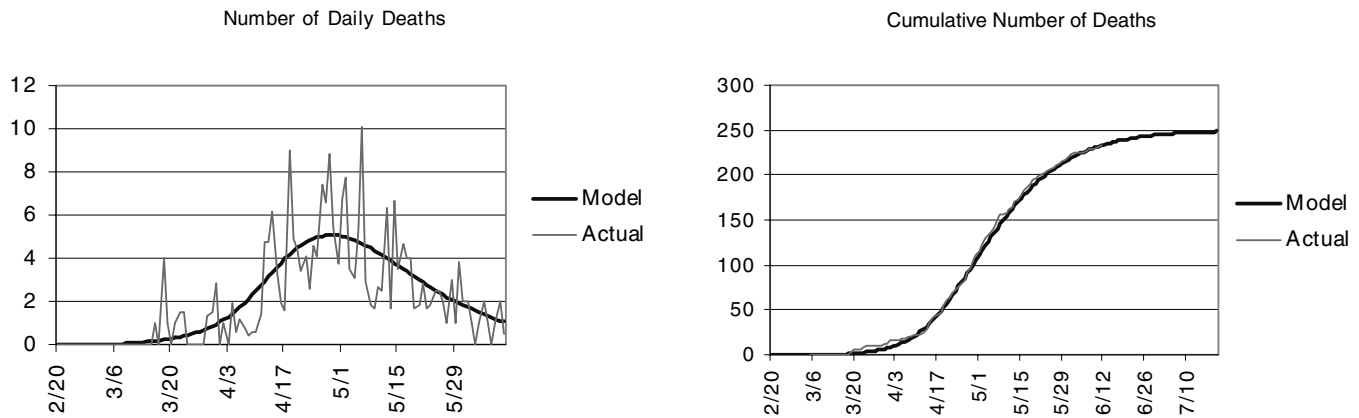


Figure 5b
Model without Amoy Gardens—SARS Deaths



average duration from onset to admission (4.85) + average duration from admission to death or recovery (23.47) = 28.32 days.

Hence, R_0 is approximately $0.22 * 28.32 = 6.23$.

Note that this is much higher than R_0 of 3 suggested by some other papers (see Lipsitch et al. [2003], and Riley et al. [2003]). We believe that this is caused by differences in the assumed starting point of the various models. In particular, the model in Riley et al. (2003) that was also based on the Hong Kong data was developed using an initial SSE of 195 cases traceable to the Prince of Wales Hospital, assumed to occur on 3 March 2003. Our model on the other hand was started with 24 cases assumed to occur on 20 February 2003 ($E_0 = 24$, see Section 3.2). As Riley et al. (2003) treats the 195 initial cases as an SSE,

which would be ignored in deriving the estimate for R_0 , it is not surprising that our model, which builds up from a much smaller number of initial cases, results in a higher estimate of R_0 .

4.2 Controlling the Epidemic

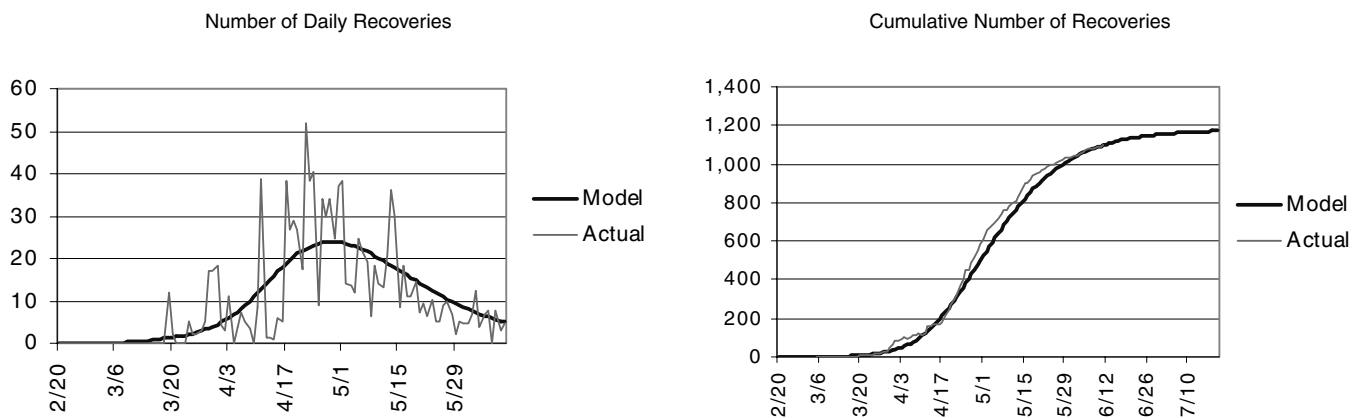
The number of secondary infections in our model is driven by

$$\beta = \frac{\beta_1(I-A) + \beta_2A}{I}$$

In order to reduce the spread of the infection (as determined by β), there are clearly a few different means:

- (a) reducing β_1 , the rate of secondary infection for cases not yet admitted to hospital;

Figure 5c
Model without Amoy Gardens—Recoveries from SARS



- (b) reducing β_2 , the rate of secondary infection for cases admitted to hospital;
- (c) increasing the hospitalization rate of infective cases, A/I (assuming that $\beta_2 < \beta_1$).

We have analyzed the effectiveness of the above controlling mechanisms below to demonstrate that restricting the rate of in-hospital infection β_2 to 0 (in other words, effective isolation of infective cases) is probably the most effective means to control an acute epidemic like SARS.

- (a) As described earlier, β_1 represents the natural rate of secondary infection in the general population assuming that no control measures have been introduced. As this depends on the nature of the infectious disease, it is difficult to reduce β_1 .
- (b) In contrast, β_2 , the rate of secondary infection within hospitals, can be controlled much more easily. According to our model, if the rate of infection within hospitals is reduced to zero, R_0 is reduced significantly as the infection can only be transmitted during the short period of time from onset of the disease to hospital admission.

$$D = \text{average duration from onset to admission} = 4.85$$

Therefore

$$R_0 = \beta D = 0.22 \times 4.85 = 1.07$$

This compares to R_0 of 6.23 (see Section 4.1) where there is no control. Figure 6 shows var-

ious scenarios based on different values of β_2 after 28 March.

- (c) To increase A/I relative to $(I-A)/I$ by shortening the time lag from SARS onset to admission into the hospital is not effective by itself. Because our model suggests that, up to 28 March, the rate of secondary infection for cases admitted to the hospital (β_2) was higher than the rate of secondary infection for cases not yet admitted (β_1), increasing the rate of admission to the hospital would do little to slow the epidemic. As shown in Figure 7, even if the average duration from onset to admission is reduced from 4.85 days to 1 day, it would only reduce the peak number of SARS by less than 100. Obviously however, shortening the time lag from SARS onset to admission into the hospital would have a larger impact if admitted cases are successfully isolated (i.e., $\beta_2 = 0$).

The most significant implication from the above discussion is that a zero rate of infection in the hospital will naturally bring the disease under control as the proportion of infective cases admitted to the hospital (A/I) increases. In other words, keeping infection contained within the hospital is absolutely critical to bringing the disease under control. In our model, with the in-hospital secondary infection rate $\beta_2 = 0$, once the proportion of cases in the hospital (A/I) is more than 6%, the reproductive ratio at time t , R_t reduces to less than one. Once this occurs, the ep-

Figure 6
Effect of Varying the Rate of In-Hospital Infection (β_2)

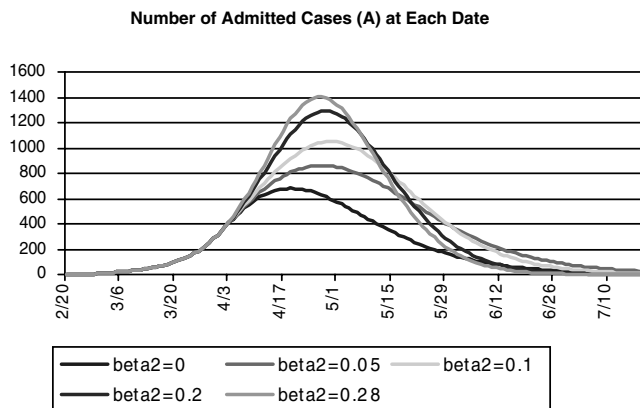
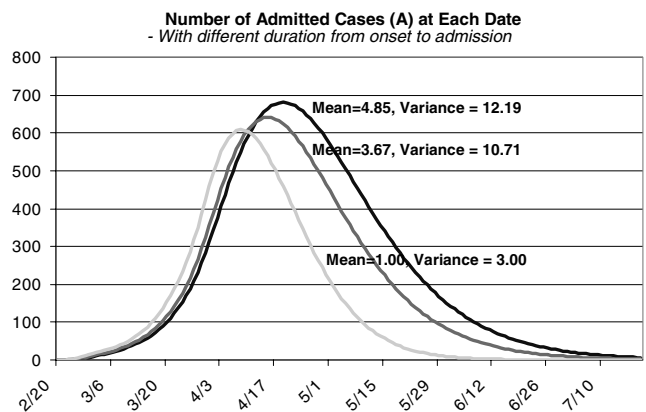


Figure 7
Effect of the Duration between Onset of SARS and Admission to the Hospital



idemic is unable to sustain itself and will gradually die out.

The path of β over the life of the epidemic in Hong Kong is shown in Figure 8.

4.3 Controlling the Susceptible Population

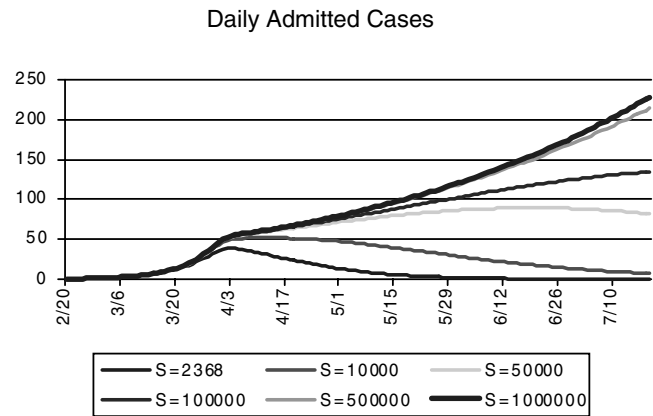
Other than reducing β_2 , another effective way to control the epidemic is to reduce the susceptible population or N . Figure 9 demonstrates the number of new exposed SARS cases for various different values of N .

In summary, without the significant reduction in the susceptible population brought about by isolation of SARS cases, increased awareness of the epidemic within the population and other control means, the SARS epidemic could have been much more serious and the epidemic would have lasted much longer than actually was the case. From another perspective, this illustrates the important role of public awareness and control measures in containing the SARS epidemic.

5. STOCHASTIC EXTENSIONS TO THE MODEL

As with the classic SEIR model, the proposed model described above implicitly assumes that transitions between different states will occur in a deterministic manner. However, in order to develop a better understanding of the epidemic, we extended the model described above to incorporate stochastic transitions between the various

Figure 9
Effect of Varying the Susceptible Population (N)



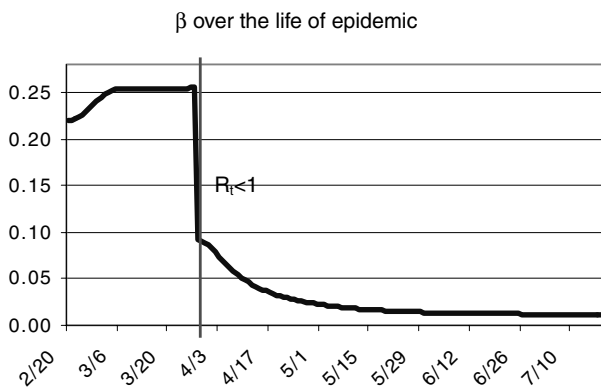
different states. Such an extension has enabled us to explore issues such as:

1. The range of likely outcomes for the SARS epidemic in Hong Kong (in terms of both the number of SARS cases and the number of deaths). That is, from the same starting point, how much better or how much worse could the outcome of the epidemic have been?
2. The potential impact of future SARS outbreaks. That is, given one index case of SARS, what is the range of likely outcomes?

In order to change from a deterministic to a stochastic model, the following changes were made:

- The number of new cases exposed to SARS in each period (moving from state S to state E) was changed into a Poisson random variable.
- The number of cases becoming infective in each period (moving from E to $[I-A]$) and being admitted to the hospital in each period (moving from $[I-A]$ to A) were changed into a binomial random variable.
- The number of deaths and recoveries in each period (moving from A to P and D) were also changed into a binomial random variable, with the relative proportions of deaths and recoveries (as determined by q_x) also set to be stochastic.
- In order to take account of the possibility of Super Spreading Events (SSEs), a parameter was added such that on any given day, there

Figure 8
Overall Rate of Secondary Infection (β)



would be a 0.5% chance that the number of new SARS cases would be 30 times the normal level. In order that the average number of new SARS cases remain the same for the models with and without SSEs, on normal days (i.e., without an SSE), the expected number of new SARS cases was multiplied by 170/199.

The SSE parameters were chosen so that, using the parameters fitted to the data excluding Amoy Gardens cases, an outcome equal to or worse than the final outcome including Amoy Gardens would arise with a probability of approximately 5%.

One feature of the stochastic results is that the mean number of cases and deaths produced by the stochastic model is sometimes substantially less than the mean number of cases and deaths produced by the deterministic model (see Tables 5 and 6). This is a common observation when comparing results from stochastic and deterministic models arising because under the stochastic model, it is possible for the epidemic to die out in the first few days without any further spread. The deterministic model does not allow for this possibility.

5.1 Range of Likely Final Outcomes for the Hong Kong SARS Epidemic

With 10,000 simulations, the results of our stochastic modelling were as shown in Table 5 and Figures 10a and 10b. The range of possible outcomes is highly dependent on the assumptions concerning the operation of Super Spreading Events (SSEs). Without any allowance for SSEs, the distribution of possible final outcomes in our SARS model is fairly compact.

One other point to note from Figures 10a and 10b is that the distribution of final SARS cases and final SARS deaths with SSEs is not especially smooth. This may indicate that the number of simulations is not quite sufficient to fully capture the effect of SSEs.

5.2 The Potential Impact of Future SARS Outbreaks

Finally, we used our model to gauge the potential impact of future SARS epidemics. In order to do this, we ran the stochastic model with a single index case and monitored the final results. We

ran the model parameterized using the data excluding Amoy Gardens both without SSEs and with SSEs.

Of particular interest to us were the following questions:

- Given one index case, what is the probability that there is no further spread of the epidemic?

Looking at Table 6, with respect to the total number of cases, we examined the probability that the total number of cases remained at one (i.e., the index case) and with respect to the total number of deaths, we considered the probability that there would be no deaths originating from the single index case.

- Given what we now know about controlling the SARS epidemic, what would be the impact of applying our current knowledge to future outbreaks?

In order to do this, we ran the model both with in-hospital infections (as assumed in our base model) and without in-hospital infections (effectively setting β_2 to zero from the beginning of the epidemic).

From the results in Table 6, it is also clear that our assumptions concerning in-hospital transmission during the early stages of the epidemic have a major impact on the final outcome. The final numbers for SARS cases and deaths produced by the model without in-hospital transmission are substantially lower than those produced by the model with in-hospital transmission.

In addition, it is notable that the inclusion of SSEs has a significant impact on the tail of the distribution of final outcomes. Compared to the results without SSEs, the 99th percentile results for final SARS cases and deaths using our SSE assumptions are 15–25% higher.

Nevertheless, one comforting conclusion that could be drawn from the results shown below is that, should another outbreak of SARS occur, given our current knowledge about the disease (assuming that no transmission of the infection takes place within hospitals), there is a reasonable probability (around 40% according to our model) that the epidemic will not spread further than the index case. There is a slightly higher probability (around 50%) that no deaths would result. However, assuming that the epidemic is not contained, there is a small probability that another fairly significant outbreak could occur re-

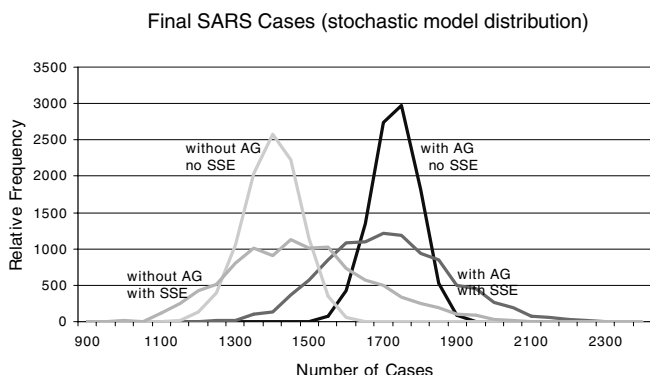
Table 5
Results of Stochastic Modelling

	With Amoy Gardens		Without Amoy Gardens	
	Final Cases	Final Deaths	Final Cases	Final Deaths
Deterministic	1,759	308	1,426	248
Stochastic without SSEs				
Mean	1,754	307	1,423	248
Standard deviation	63	19	75	19
1st percentile	1,601	262	1,237	202
5th percentile	1,648	276	1,295	216
10th percentile	1,673	282	1,326	223
25th percentile	1,712	294	1,374	235
50th percentile (median)	1,756	307	1,426	248
75th percentile	1,798	320	1,474	261
90th percentile	1,834	331	1,519	272
95th percentile	1,855	339	1,542	279
99th percentile	1,894	353	1,584	293
Stochastic with SSEs				
Mean	1,738	303	1,402	243
Standard deviation	163	34	183	36
1st percentile	1,392	231	1,039	173
5th percentile	1,490	250	1,123	187
10th percentile	1,536	262	1,170	198
25th percentile	1,623	280	1,270	219
50th percentile (median)	1,729	301	1,391	241
75th percentile	1,844	325	1,512	266
90th percentile	1,960	348	1,649	290
95th percentile	2,020	363	1,729	305
99th percentile	2,144	389	1,860	332

Table 6
Results of Stochastic Modelling for One Index Case

	With In-Hospital Transmission		Without In-Hospital Transmission	
	Total Cases	Total Deaths	Total Cases	Total Deaths
Deterministic	387	52	38	5
Stochastic without SSEs				
Probability of zero spread	0.50%	1.07%	39.49%	48.83%
Mean	375	47	39	4
Standard deviation	217	31	84	9
1st percentile	3	0	1	0
5th percentile	30	5	1	0
10th percentile	83	10	1	0
25th percentile	205	23	1	0
50th percentile (median)	370	43	2	1
75th percentile	535	67	22	4
90th percentile	670	90	153	16
95th percentile	737	105	220	24
99th percentile	864	132	405	45
Stochastic with SSEs				
Probability of zero spread	0.95%	2.58%	39.53%	50.68%
Mean	308	44	38	4
Standard Deviation	254	37	104	11
1st percentile	2	0	1	0
5th percentile	14	2	1	0
10th percentile	28	6	1	0
25th percentile	126	17	1	0
50th percentile (median)	281	34	2	0
75th percentile	496	63	11	2
90th percentile	708	98	126	14
95th percentile	829	117	242	25
99th percentile	994	156	508	54

Figure 10a
Results of Stochastic Modelling—SARS Cases



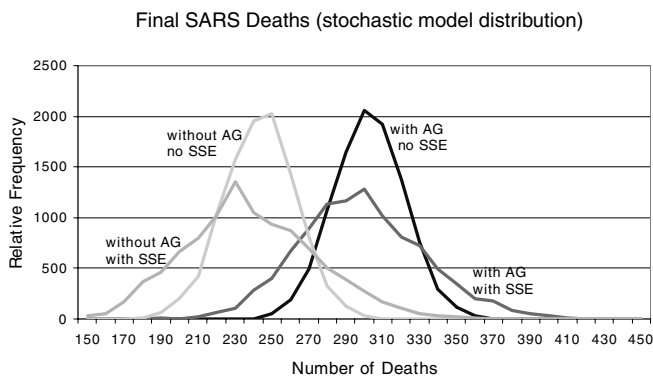
sulting in up to approximately 50 deaths for a 1-in-100 scenario.

6. CONCLUSIONS

Referring back to the three main aims of epidemic modelling espoused by Daley and Gani (1999) in Section 1:

1. We have developed a simplified model for the progress of an acute infectious disease such as SARS, enabling us to quantify the impact of various control measures. A similar model could be used for other acute infectious disease such as Avian influenza.
2. We have identified the rate of in-hospital infections clearly as one of the key drivers of the SARS epidemic. This should also provide us with lessons for tackling future acute infectious disease epidemics.

Figure 10b
Results of Stochastic Modelling—SARS Deaths



3. We have used estimates of the reproductive ratio (R_t) and stochastic modelling procedures to predict the course of future outbreaks of SARS and similar diseases, both with and without appropriate control measures. These are tools that we believe will help actuaries to assess the potential impact of any future epidemics on the insurance industry.

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