MODELLING THE EFFECT OF MASS MEDIA ON INFLUENZA TRANSMISSION AND VACCINE UPTAKE

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Abstract

Influenza causes annual epidemics and occasional pandemics that have claimed millions of lives throughout history. Media reports affect social behaviour during epidemics and pandemics. Changes in social behaviour, in turn, effect key epidemic measurements such as peak magnitude, time to peak, and the beginning and end of an epidemic. The extent of this effect has not been realized. Mathematical models can be employed to study the effects of mass media. In this work, previous mathematical models concerning epidemics and mass media are studied. A novel inclusion of mass media is developed through the addition of a mass media compartment in a Susceptible-Exposed-Infected-Recovered (SEIR) model to look at the effect of mass media on an epidemic. Multiple levels of social distancing are considered in the framework of an ODE model. Vaccination is included in various models for susceptible individuals. Systems of stochastic differential equation models for each of the different scenarios have been derived. An Agent-Based Monte Carlo (ABMC) simulation is used to determine the variability in these key epidemic measurements,

so as to provide some insight in to the effects of mass media on epidemic data. Data can help to provide an epidemic outcome that is seen at the population level. Data is used in order to inform parameter values and the novel inclusion of media. A look to future work is also included.

To my family.

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would not have been possible.

'The future belongs to those who believe in the beauty of their dreams.'

Eleanor Roosevelt

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Abbreviations

GPHIN - Global Public Health Information Network

PHAC - Public Health Agency of Canada

H1N1 - Pandemic influenza strain of 2009

R_0 - Basic reproductive ratio

ODE - Ordinary differential equation

SDE - Stochastic differential equation

ABMC - Agent-Based Monte Carlo simulation

PDE - Partial differential equations

FTBS - Forward-time Backward-space

SIR - Susceptible-Infectious-Recovered

SIS - Susceptible-Infectious-Susceptible

SEIR - Susceptible-Exposed-Infectious-Recovered

SEIRV - Susceptible-Exposed-Infectious-Recovered-Vaccinated

S_1 - First socially distanced compartment of individuals

S_2 - Second socially distanced compartment of individuals

E - Exposed compartment

I - Infectious compartment

R - Recovered compartment

M - Media compartment

V - Vaccinated compartment

I_c - Critical number of infectious individuals

Severity of pandemic/epidemic - intensity of transmission

Mass media - Organized entities that generate and disseminate information [61]

1 Introduction

Epidemiological modelling has been developing since at least the time of Aristotle with the idea that invisible living creatures were agents of disease [11, 66]. This idea developed into a theory in the sixteenth century when Leeuwenhoek (1632-1723) demonstrated the existence of microorganisms under a microscope. The first formal germ theory of disease expression was proposed by Jacob Henle (1809-1885) in 1840, and was further developed by Robert Koch (1843-1910), Joseph Lister (1827-1912) and Louis Pasteur (1827-1875) [66]. The first disease model came from Bernoulli on the effect of vaccination against smallpox in increasing life expectancy [11]. This model contained the idea of differential mortality to estimate the death rate attributable to different diseases. Kermack and McKendrick were monumental in the advancement of disease modelling as they explained why epidemics do not infect everyone: mass action [11]. In this thesis we employ disease modelling to study the effects of mass media on epidemics.

1.1 Influenza

Influenza causes annual epidemics and occasional pandemics that have claimed millions of lives throughout history [10]. A pandemic is an infectious epidemic which occurs worldwide and usually affects large numbers of people [45]. In the past 100 years, the H1N1 influenza A virus has caused four pandemics: 1918, 1957, 1977 and 2009, [11, 42, 43]. According to the Public Health Agency of Canada, inter-

pandemic influenza (or seasonal influenza) affects approximately 20,000 Canadians, with approximately 2,000 to 8,000 deaths, annually [47]. Typically, a pandemic occurs in multiple waves [19].

During a pandemic it is important to reduce serious illness and death. A vaccine may not be available to the public for many months after the arrival of a pandemic influenza strain [27]. Thus, it is important to practice self-preservation techniques that include good hand hygiene practices and social distancing [27, 47]. The intent of social distancing is to minimize the transmission of influenza by reducing the amount of contact between susceptible individuals and infectious individuals. Social distancing measures include school closures, travel restrictions and restrictions on mass gatherings. These are some of the only early intervention strategies that are guaranteed to be available during the beginning stages of a novel pandemic. This type of strategy is part of the pandemic response plans for organizations such the World Health Organization (WHO) and the United States' Center for Disease Control (CDC) [51].

1.1.1 Influenza Pathogenesis

Influenza is an infectious disease caused by negative-sense RNA viruses of the Orthomyxoviridae family [66]. Three of the five genera of the Orthomyxoviridae family are comprised of influenza: influenza A virus, influenza B virus and influenza

C virus. The most common is the influenza A virus [23]. The viral particles of the three subtypes of influenza all have a similar structure. The virus has a viral envelope containing glycoproteins which are wrapped around an RNA core. Influenza A, specifically, contains 11 genes encoding for 11 proteins on 8 pieces of segmented negative-sense RNA. The glycoproteins on the outside of the viral particles, hemagglutinin (HA) and neuraminidase (NA) — regulate the virus binding to and entering the target cells, and the release of progeny virus from the infected cells, respectively. Binding typically occurs on the surface of epithelial cells in the nose throat and lungs of mammals [8]. Individuals are infectious (virus shedding occurs) for approximately 5-7 days after contracting the infection, with the highest period of infectiousness occurring at 2-3 days after becoming infected. Virus shedding starts approximately 1 day prior to the manifestation of influenza symptoms [9]. There are three main transmission pathways of influenza: direct transmission, when an infectious individual directly spreads mucous into the eyes, nose or mouth of another person; airborne transmission, inhalation of virus droplets from the sneeze or cough of an infectious individual; contact with contaminated surfaces such as touching a contaminated surface and then touching your eyes, nose or mouth [9].

1.1.2 Influenza Control

Controlling the spread of influenza can reduce the effect it has on the population. Influenza control can be achieved through various strategies including vaccination, the use of drug therapy, and through practicing social distancing, [47]. Vaccination is used preemptively for influenza whereas drug therapy and social media can be practiced before infection and while infected, to reduce the probability of infection and transmission to others.

1.1.2.1 Vaccination

Once a vaccine is available it is considered a first line of defense against a pandemic [27, 47]. Vaccination has been a topic of debate since vaccines were first introduced for smallpox [14]. Despite the debated issues, the fear of becoming ill was greater which led to a high vaccine uptake rate resulting in the eradication of smallpox and low incidences of measles, polio and other childhood diseases [14]. Due to this eradication, the perceived risk of these illnesses has decreased which has allowed the debate to turn towards vaccine safety versus risks from diseases that are not prevalent in society [14]. This has challenged the willingness of society to vaccinate against these diseases possibly leading to lower vaccine coverage than required to control transmission [14]. The influence of outside information is becoming the

critical factor as to whether or not a vaccination campaign will succeed. Modern societies are facing 'rational' exemption from vaccination with the population comparing low perceived risk of infection, due to high vaccine coverage, with the risks and side effects from vaccination. This 'rational' exemption does not consider resurgence of an illness due to decline in vaccine coverage and only considers the most publicly available information of the current and recent past spreading of the disease versus the 'risks' associate with the vaccine [13].

A vaccine for a pandemic is unlikely to be implemented in the first wave. The strain is unknown at first and takes time to develop. Moreover the vaccine is developed for the pandemic strain of the first wave. It is possible that the strain can change over time, resulting in a less effective vaccine [19].

1.1.2.2 Drug Therapy

Antivirals are available during the first wave of the epidemic and play a critical role in reducing morbidity and mortality from the virus. The antivirals may help to prevent illness and reduce the ability of the virus to replicate but do not provide immunity. Treatment with antivirals may reduce the severity and transmission rate of an infection [15, 19, 60, 62, 64]. Treatment with antivirals can occur post exposure (post-exposure prophylaxis), prior to exposure (pre-exposure prophylaxis) and in the early stages of the illness, [19]. The early stages treatment has been shown to

be effective if administered within 48 hours of the onset of clinical symptoms [18]. Resistance to drug therapy can develop with drug treatment so this may not be the best strategy for treating influenza [19].

1.1.2.3 Social Distancing

The reduction of contact between susceptible and infectious individuals through restrictions on social behaviour is defined as social distancing [51]. Restrictions to provoke social distancing include school or workplace closures, prohibitions on mass gatherings and travel restrictions. Individuals are also able to practice self-imposed social distancing by changing their own behaviour [20, 51]. These are non-pharmaceutical measures of disease prevention that are available as a prevention tool from the onset of an epidemic. The social distancing measures may also be known as public health measures. Currently, these intervention measures are part of the pandemic preparedness plans of the World Health Organization (WHO) and the U.S. Centers for Disease Control and Prevention (CDC) [51]. The Public Health Agency of Canada (PHAC) also considers social distancing measures as an illness prevention tactic. Social distancing measures are part of the influenza preparedness checklist [47].

1.1.3 Summary

Measures prescribed to help control an invading pandemic are not fail proof. A major hurdle to overcome in fighting influenza is resistance to the medications indicated to treat individuals who are ill. It is possible that the vaccine is not as effective as it was predicted to be or that not enough individuals received an influenza vaccination resulting in little to no effect on the spread of influenza. The rate at which we practise social distancing may also diminish throughout the duration of a pandemic when the effect that mass media has starts to wane. This desensitization to mass media reports can lead to a reappearance of the pandemic illness or it can increase the length of time the pandemic lasts.

1.1.4 Public Health Policy

The role of the pandemic preparedness and response plan from the Public Health Agency of Canada (PHAC) is to minimize serious illness and death and to minimize social disruptions among Canadians [47]. There are many strategies that can be used to achieve the goals of a preparedness plan that depend on pandemic epidemiology [47].

1.1.4.1 Key Measurements for Public Health

In order to meet national and global expectations there are certain observations that need to be made during a pandemic which include measures of the severity of the novel influenza strain. Measures of severity include the time when the infection has reached and surpassed the peak number of infections, what the peak number of infections is, the total number of individuals who have fallen ill with the infection and the time when the epidemic has finished [47]. These measurements help public health to gauge treatment response by predicting the number of hospital beds that may be needed, the amount of drugs needed to treat the ill, the amount of vaccination that should be purchased and the duration of the increase in service that may be necessary for the epidemic. In this thesis we will obtain these measurements from the models and simulations that we present.

1.2 H1N1 Timeline in Canada and the World

The Canadian government collects data of the number of confirmed cases of influenza every flu season through FluWatch. FluWatch is a national surveillance system under the Public Health Agency of Canada which monitors the spread of influenza throughout the year. Data was collected for the 2009, pandemic season. Using this data we can visualize the two waves that occurred: Figure (1.1), [49].

In Mexico, the first cases of a severe upper respiratory infection were reported on March 18, 2009. The 6 weeks following this date saw the rapid spread of this virus worldwide. April 12, 2009, was the beginning of the first wave of the influenza pandemic in Canada. On April 20, 2009, two cases were confirmed in California and on April 23, 2009, confirmation of the virus from Mexico being an H1N1 influenza was given by the National Microbiology Laboratory. The first reported Canadian case of H1N1 by the Public Health Agency of Canada is reported on April 26, 2009, [48]. By the end of the month the World Health Organization raised the global pandemic alert level to 5. The peak of the first wave of the influenza pandemic occurred during the first 3 weeks in June. On June 11, 2009, the World Health Organization declares a global pandemic, raising the alert level to 6. August 2009, saw the Public Health Agency of Canada order 50.4 million doses of the vaccine that was in production and the end of the first wave and beginning of the 2nd wave of the H1N1 influenza pandemic. During the first wave of the pandemic, there were 18,000 confirmed deaths due to pH1N1 worldwide [48].

The second wave of the influenza pandemic is considered to have started on August 30, 2009, and it continued to January 27, 2010. During the 2nd wave of the pandemic a vaccine was available and used. The vaccine was approved and available to the public on October 21, 2009. The peak of the second wave of the pandemic occurred in early November and by December, indicators on Flu Watch

suggested a continued decline in the number of H1N1 cases. January 2010, saw a de-escalation of the H1N1 pandemic response until the end of the pandemic in Canada was announced on January 27, 2010, [48].

1.3 Mass Media

Mass media are organized, structured, entities that generate and disseminate news and entertainment through print, radio, television and the internet. Mass media are not communication interactions between friends and family members [61]. Mass media can play an important role in disease transmission. Information about preventative measures is currently spread through mass media campaigns. Attention to health news has been increasing in importance during the past twenty-five years, focusing on such topics as smoking, HIV/AIDS and obesity. The media plays a role in spreading preventative knowledge of an emerging disease which can reduce the opportunity for contact transmission amongst an aware, susceptible population [5, 44, 58]. In the most recent pandemic, H1N1 influenza A, the World Health Organization, WHO, had an established communication plan considering misinformation was rife during the SARS outbreak in 2003. Media coverage of a novel disease follows one of two routes [58]:

1. Media report directly to the public on self-observed facts;

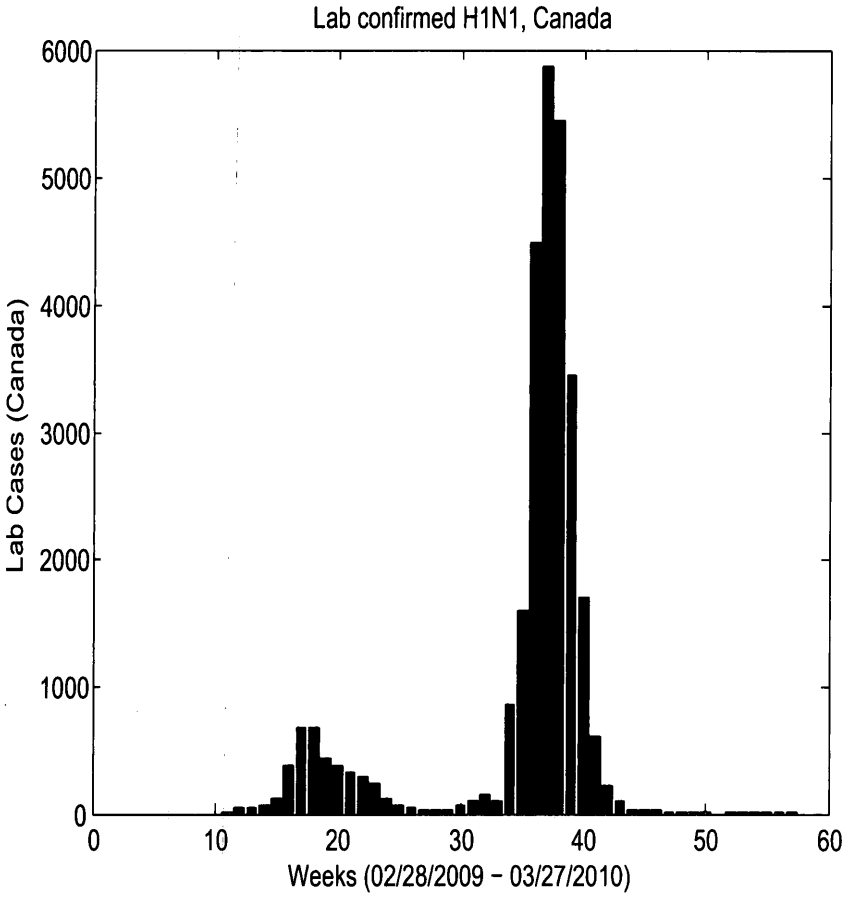

Figure 1.1: Lab confirmed cases of pandemic H1N1 in 2009. There are clearly two waves of the epidemic with the second larger than the first [49].

2. Public health authorities use mass media to communicate facts about an outbreak.

Since little information is available to the public during an outbreak, the media extend small amounts of information to the public [38, 61].

We have recently seen the effects of mass media in two infectious disease outbreaks. The first novel infectious disease of the twenty-first century was SARS. It had distinct features such as rapid spatial spread and vast media coverage [38, 59]. The media coverage of the most recent H1N1 pandemic was vast, with an increased sense of urgency as this strain of influenza was the same strain that caused approximately 50 million deaths worldwide in 1918, [11]. A Google search of 'H1N1' retrieves over 50 million results in 0.14 seconds. With the influence that mass media have on the public, they can play an important role in defining health issues, serve as a major source of information and incite avoidance behaviours early, lessening the impact of a possible pandemic [38, 59, 61].

A number of media observers have speculated that pervasive media coverage of social problems may lead to desensitization: 'the piteous site of an oil-soaked seagull or a dead soldier pales after it has been viewed even a dozen times'[33]. Pandemics are social problems [33].

In television advertising, media decay is part of the measure of effectiveness of an advertising campaign. A company considers that the most impact an advertisement

may have occurs in the first week but the effect will continue over the following weeks. The decay rate of impact is measured as a half-life, the amount of time it takes for the effectiveness of an advertising campaign to decrease by half [31]. This is called 'adstock decay'. This concept also manages to be upheld with multiple media sources [31].

The definition of desensitization is the diminished emotional responsiveness to a negative or an aversive stimulus after repeated exposure to it [17]. When subjected to media reports numerous times, the viewer is less susceptible to its effect. This waning effect was studied in [17], considering one's reaction to exposure to violent media events over time. We propose that this same phenomenon occurs with health events. Initially, when an epidemic is reported precautions may be taken to avoid illness. As time passes, however an individual will become less sensitive to the stories about the epidemic and become lax in the precautions.

There are many human reactions to the perceived presence of disease ranging from avoidance of social settings, vaccination and other more creative precautions. This type of behaviour can potentially reduce the size of an epidemic. The effects of mass media on human behaviour in a pandemic are not well understood; mathematical modelling has the ability to provide insight into possible behaviour patterns in pandemic situations.

1.3.1 Mass Media Data

The Government of Canada collects data about emerging public health threats through the Global Public Health Intelligence Network (GPHIN); information about what data is collected and from where can be found in [50]. The Global Public Health Intelligence Network (GPHIN) is a real-time early warning system based on the internet that collects information and creates reports on issues of public health significance. The media reports collected by GPHIN are informed by people falling ill around the world as well as the steps that the countries, governments and the World Health Organization are taking in order to minimize the impact of a pandemic. This network monitors global media sources in 6 languages and filters news reports for relevancy and further analysis. The languages that are monitored are Arabic, Chinese, English, French, Russian and Spanish. Public health events that are currently tracked include disease outbreaks and infectious diseases. The Global Public Health Intelligence Network (GPHIN) is managed by Health Canada's Centre for Emergency Preparedness.

Figure (1.2), is a plot of the GPHIN data from three different groups, the solid line is all media reports in the six languages, the dashed line is the English and French media reports only and the dotted line is the English only media reports. The data was collected from March 1, 2009 until January 27, 2010, when the

H1N1 epidemic was declared over in Canada [49]. The media data from GPHIN decays, after the large spike, by about half after approximately two weeks, a half-life proposed in [31] for different forms of media.

1.4 Mathematical Epidemiology

1.4.1 Deterministic Models of Ordinary Differential Equations

Disease modelling uses models of ordinary differential equations called compartmental models [10]. These models were initially described by W.O. Kermack and A.G. McKendrick in 1927, leading to what we now call the Kermack-McKendrick models [32] with mean lifetimes that are exponentially distributed. This type of model assumes that the population size in each of the compartments is large enough that they are well-mixed [10].

1.4.1.1 SIR Model

A susceptible-infectious-recovered (SIR) model represents a model that confers immunity to individuals once they have been ill. This type of model splits the total population into three groups: $S(t)$ denotes the individuals who are susceptible to disease at time t, $I(t)$ denotes the number of infected individuals and $R(t)$ denotes the number of recovered individuals [10]. The basic SIR model with demographic

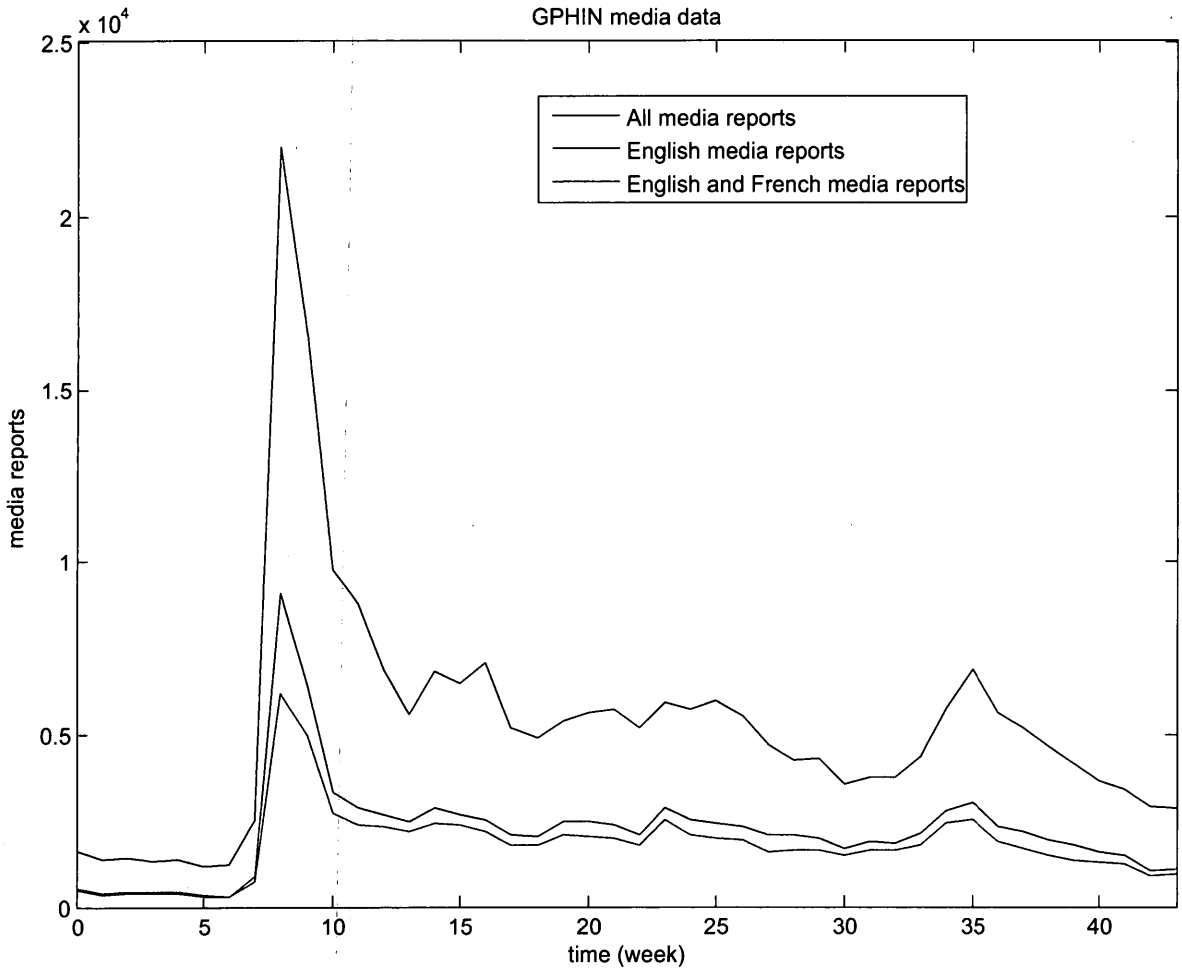

Figure 1.2: Media data collected by GPHIN from all media worldwide. The time scale is weeks with 0 corresponding to March 1, 2009. The black line is all media data collected, the red line is French and English media data and the blue line is English language media data.

parameters has the following structure

$$\begin{aligned} \dot{S} &= \lambda - \beta SI - dS \\ \dot{I} &= \beta SI - \gamma I - dI \\ \dot{R} &= \gamma I - dR, \end{aligned} \tag{1.1}$$

where λ is the birth rate of the population, d is the death rate, β is the transmission rate and γ is the recovery rate. The total population is $N = S + E + I + R$.

1.4.1.2 SEIR Model

Infectious diseases often have an exposed period, which is the period of time after transmission but before the newly infected individuals can transmit the illness [10]. This is represented by the addition of an equation to Model (1.1),

$$\begin{aligned} \dot{S} &= \lambda - \beta SI - dS \\ \dot{E} &= \beta SI - \sigma E - dE \\ \dot{I} &= \sigma E - \gamma I - dI \\ \dot{R} &= \gamma I - dR. \end{aligned} \tag{1.2}$$

The parameter σ is the mean exposed period.

1.4.1.3 SEIRV Model

The inclusion of intervention measures is also seen in compartmental models. Vaccination is included in Model (1.2) through the addition of a vaccination equation

that vaccinates individuals from the susceptible class, Model (1.3).

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - \nu V - dS \\
\dot{E} &= \beta SI - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR \\
\dot{V} &= \nu V - dV.
\end{aligned} \tag{1.3}$$

1.4.2 Mathematical tools for Disease Modelling

1.4.2.1 Uninfected equilibrium

The uninfected equilibrium is the equilibrium point at which no infection remains in the system, $I(t) = 0$. To solve for the uninfected equilibrium for Model (1.2) we set each of the equations of a model equal to 0 and solve for the variables. The uninfected equilibrium for Model (1.2) is

$$E_0 = (S, E, I, R) = (\frac{\lambda}{d}, 0, 0, 0) \tag{1.4}$$

1.4.2.2 Basic Reproductive Ratio, R_0

The basic reproductive ratio is a concept originally from demographic study [24]. In demographics, it is a measure of the number of offspring a typical member of a species produces. Mathematical thought has brought the use of R_0 to epidemic

theory, and it is now commonly used in the study of infectious diseases [24]. In epidemiology, however, the definition of R_0 is not the same as that for demography. In this case the definition of the basic reproductive ratio is the number of individuals infected by a single infected individual, in an entirely susceptible population, during the entire infectious period. It is used to determine the severity of transmissibility of an epidemic or a pandemic in the population; a larger value of R_0 translates to the possibility of more infected people, R_0 infected individuals. There is a threshold level to determine whether an infectious disease will persist or disappear. If $R_0 < 1$, the infection will disappear as each individual will, on average, produce less than one new infectious individual and if $R_0 > 1$, the infectious individual will produce more than one new infection and the disease will invade and persist in a susceptible population for a period of time. This threshold level is of critical importance to public health measures and vaccinating procedures. The smaller that preventative measures can make R_0, the better it is for the susceptible population [24].

There are various ways to determine the value of R_0 [24]. The method that we use to calculate R_0 is the next generation matrix method. In this method, R_0 is the spectral radius of the next generation operator. For this method we must determine the infectious and non-infectious compartments of the model in order to determine the next generation operator (FV^{-1}), where (F) is a matrix of actions from the system that produce new infections and (V) is the matrix of actions that moves

infections around the system. We employ the next generation matrix method here for Model (1.2).

To find the basic reproductive ratio, R_0, the equations that are needed from Model (1.2)are:

$$\begin{aligned} \dot{E} &= \beta SI - \sigma E - dE \\ \dot{I} &= \sigma E - \gamma I - dI. \end{aligned} \tag{1.5}$$

Calculating partial derivatives, the matrix that contains the creation of new infections, F is

$$F = \begin{pmatrix} 0 & \beta S \\ 0 & 0 \end{pmatrix}. \tag{1.6}$$

The disease free equilibrium of Model (1.2), is E_0 from Expression (1.4), where λ is the birth rate and d is the death rate. Evaluation of matrix (F) at the disease free equilibrium obtains

$$F = \begin{pmatrix} 0 & \beta\frac{\lambda}{d} \\ 0 & 0 \end{pmatrix}. \tag{1.7}$$

The second part of the next generation operator is the matrix (V). For this, see Matrix (1.8).

$$F = \begin{pmatrix} \sigma + d & 0 \\ -\sigma & \gamma + d \end{pmatrix}. \tag{1.8}$$

After finding the matrix inverse of (V) and multiply it with (F), we get the next

generation operator:

$$FV^{-1} = \begin{pmatrix} \frac{\beta\frac{\lambda}{d}\sigma}{(\sigma+d)(\gamma+d)} & \frac{\beta\frac{\lambda}{d}}{(\gamma+d)} \\ 0 & 0 \end{pmatrix}. \tag{1.9}$$

The spectral radius of the operator (FV^{-1}) operator, R_0, is

$$R_0 = \frac{\beta\lambda\sigma}{d(\sigma+d)(\gamma+d)}. \tag{1.10}$$

1.4.2.3 Stability Analysis

In disease modelling, typically, an epidemic occurs when $R_0 > 1$ and an epidemic will not occur if $R_0 < 1$. This threshold for R_0 corresponds to positive and negative eigenvalues of the corresponding Jacobian matrix, respectively. When the eigenvalues are positive, an epidemic will occur and when eigenvalues are negative there will be no epidemic. This analysis will be completed for Model (1.2), but similar results can be obtained for the other models herein.

The Jacobian matrix for Model (1.2) is

$$J = \begin{bmatrix} -\beta Y - d & 0 & -\beta S & 0 \\ \beta Y & -\sigma - d & \beta S & 0 \\ 0 & \sigma & -\gamma - d & 0 \\ 0 & 0 & \gamma & -d \end{bmatrix}. \tag{1.11}$$

After evaluating Matrix (1.11) at the uninfected equilibrium, we get Matrix (1.12),

$$J_0 = \begin{bmatrix} -d & 0 & -\frac{\beta\lambda}{d} & 0 \\ 0 & -\sigma - d & \frac{\beta\lambda}{d} & 0 \\ 0 & \sigma & -\gamma - d & 0 \\ 0 & 0 & \gamma & -d \end{bmatrix}. \tag{1.12}$$

Since we evaluate Matrix (1.11) at the uninfected equilibrium, all terms go to zero except those that are multiplied with (S), alone. From here we obtain the eigenvalues of the Matrix (1.12):

$$\lambda_{1,2} = -d,\ \lambda_{3,4} = \frac{-\sigma d + 2d^2 + d\gamma \pm \sqrt{\sigma^2 d^2 - 2\sigma d^2\gamma + d^2\gamma^2 + 4d\sigma\beta\lambda}}{2d}. \tag{1.13}$$

For some $\lambda_i > 0$ we can show that $R_0 > 1$, which will give an unstable equilibrium implying an epidemic will occur. For $\lambda_3 > 0$ we have

$$\frac{-\sigma d + 2d^2 + d\gamma + \sqrt{\sigma^2 d^2 - 2\sigma d^2\gamma + d^2\gamma^2 + 4d\sigma\beta\lambda}}{2d} > 0, \tag{1.14}$$

which can be rearranged to get

$$\begin{aligned} \frac{\sigma\beta\lambda}{\sigma\gamma d + \sigma d^2 + \gamma d^2 + d^3} &> 1 \\ \frac{\sigma\beta\lambda}{d(\sigma + d)(\gamma + d)} &> 1 \\ R_0 &> 1. \end{aligned} \tag{1.15}$$

It is easy to see from (1.15) that if some of the eigenvalues from Matrix (1.12) are greater than zero, $\lambda_i > 0$, then we get an epidemic, $R_0 > 1$.

1.4.3 Stochastic Models

A deterministic model can describe the general behaviour of an epidemic but it cannot capture the variability within an epidemic. A stochastic simulation lends itself well to demonstrating variation within an epidemic [25]. Also, the well-mixed assumption that is necessary for the ordinary differential equation models may not be true at the onset of an epidemic, these dynamics can be well captured with a stochastic model [10]. One such stochastic model is an Agent-Based Monte Carlo (ABMC) simulation, another is a model of stochastic differential equations.

1.4.3.1 Agent-Based Monte Carlo Simulation

An Agent-Based Monte Carlo (ABMC) simulation moves forward not in regular time intervals but following event times: the next time that an individual changes state within the system. At each event time, the underlying exponential distributions dictated by the use of ordinary differential equation models associated with each individual are examined to determine the outcome of the event and which individual moves within the system to which state, [25]. This type of ABMC has been used in various studies, [25, 26]. In Figure (1.3), we can see the progression of an individual through the epidemic.

Let us now look at Figure (1.3). Agents in each of the susceptible, exposed,

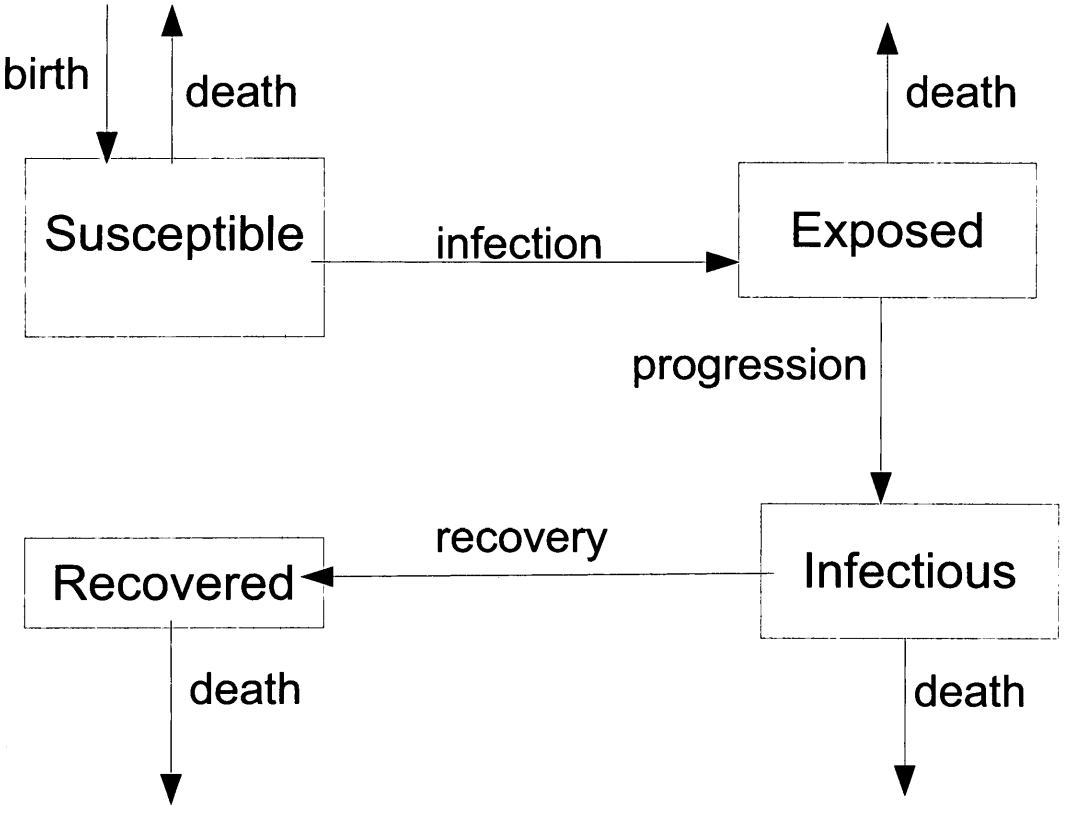

Figure 1.3: Schematic of the Agent Based Monte Carlo simulations. We start with a population with each individual assigned a set of event times. All of the event times are compared and the event that occurs is the event that has the smallest time. Once the event has occurred, event times are reassigned and reevaluated to check for the smallest time so another event can occur.

infectious and recovered compartments are assigned event times for each event that allows an individual from that compartment to change state. All of the times in each compartment are compared against one another. The compartment with the lowest time is the one from where the agent will participate in an event. Let us assume that this is the case for an agent in the infectious class. Now the event times within the infectious compartment are compared. There are event times corresponding to recovery, infecting a susceptible agent and death. After the event times are ordered from smallest to largest in the infectious compartment, the event with the lowest time is the event which occurs. Once the event has occurred, the process is repeated until a stop condition is reached. In this thesis the lifetimes of the ABMC are exponentially distributed in order to compare the ABMC to the models of ordinary differential equations herein. It is possible to change the average lifetime distribution of the parameters in the ABMC model as necessary.

1.4.3.2 Stochastic Differential Equation Models

A stochastic differential equation model is a continuous analogue of a Continuous Time Markov Chain (CTMC) model: time is continuous but the states are discrete. We construct a stochastic differential equation model from the continuous time Markov chain (CTMC) model for an SEIR model, model (1.2), by considering time as continuous on $t\epsilon[0,\infty)$, the collection of discrete random variables $S(t)$, $E(t)$,

$I(t)$, and $R(t)$ for susceptible, exposed, infectious and recovered classes or states, respectively. The transition probability from state to state is called the infinitesimal transition probability, the change in time is sufficiently small that only one event occurs in the time period, and depends only on the state of the system at the previous time: Markov property. The probability of a transition can be described by (1.16),

$$Prob\{\Delta S(t) = i, \Delta E(t) = j, \Delta I(t) = k, \Delta R(t) = l | S(t), E(t), I(t), R(t)\}, \quad (1.16)$$

where $\Delta S(t) = S(t + \Delta t) - S(t)$. Each of i, j, k, l take on values of only $+1$, -1 or 0, because only one change occurs in sufficiently small Δt, thus $o(\Delta t)$ is included in the definition of the infinitesimal transition probabilities. The probabilities for each change in state are straightforward to describe from an SEIR model and can

be seen in (1.17).

$$
\begin{aligned}
&Prob\{\Delta S(t)=i,\Delta E(t)=j,\Delta I(t)=k,\Delta R(t)=l|S(t),E(t),I(t),R(t)\} \\
&= \begin{cases}
\lambda\Delta t+o(\Delta t),\ (i,j,k,l)=(1,0,0,0) \\
\beta S(t)I(t)+o(\Delta t),\ (i,j,k,l)=(-1,1,0,0) \\
dS(t)+o(\Delta t),\ (i,j,k,l)=(-1,0,0,0) \\
\sigma E(t)+o(\Delta t),\ (i,j,k,l)=(0,-1,1,0) \\
dE(t)+o(\Delta t),\ (i,j,k,l)=(0,-1,0,0) \\
\gamma I(t)+o(\Delta t),\ (i,j,k,l)=(0,0,-1,1) \\
dI(t)+o(\Delta t),\ (i,j,k,l)=(0,0,-1,0) \\
dR(t)+o(\Delta t),\ (i,j,k,l)=(0,0,0,-1) \\
(1-(\lambda+\beta S(t)I(t)+dS(t)+\sigma E(t)+ \\
dE(t)+\gamma I(t)+dI(t)+dR(t)))\Delta t+o(\Delta t),\ (i,j,k,l)=(0,0,0,0) \\
o(\Delta t),\ \text{otherwise.}
\end{cases}
\end{aligned}
\tag{1.17}
$$

From the equations for the infinitesimal transition probabilities, Expressions (1.17), we can derive the forward Kolmogorov differential equations. From the forward Kolmogorov differential equation we derive a probability generating function (pgf), a moment generating function (mgf) and finally the differential equations for the mean and higher order moments.

The derivation for the forward Kolmogorov differential equation from Equation (1.17), representing the rate of change of the transitions from state to state

follows. The first step is to derive a discrete equation from Expression (1.17) for sufficiently small Δt for $p_{i,j,k,l}(t+\Delta t)$, the transition probability,

$$\begin{aligned} p_{i,j,k,l}(t+\Delta t) = {} & \lambda p_{i-1,j,k,l}(t)\Delta t + \beta(i+1)kp_{i+1,j-1,k,l}(t)\Delta t + \\ & d(i+1)p_{i+1,j,k,l}\Delta t + \sigma(j+1)p_{i,j+1,k-1,l}(t)\Delta t \\ & + d(j+1)p_{i,j+1,k,l}(t)\Delta t + \gamma(k+1)p_{i,j,k+1,l-1}(t)\Delta t \\ & + d(k+1)p_{i,j,k+1,l}(t)\Delta t + d(l+1)p_{i,j,k,l+1}(t)\Delta t \\ & + (1-(\lambda+\beta ik+di+\sigma j+dj+\gamma k+dk+dl))p_{i,j,k,l}(t)\Delta t, \end{aligned} \tag{1.18}$$

where $p_{i,j,k,l}(t)$ represents the probability of a susceptible, exposed, infectious and recovered population of size i, j, k, and l, respectively. Next, $p_{i,j,k,l}(t)$ is subtracted

from both sides, the expressions are divided by Δt, resulting in Expression (1.19),

$$\begin{aligned}
\frac{p_{i,j,k,l}(t+\Delta t)-p_{i,j,k,l}(t)}{\Delta t} = \sum [& \lambda p_{i-1,j,k,l}(t) S^i E^j I^k R^l \\
& + \beta(i+1)k p_{i+1,j-1,k,l}(t) S^i E^j I^k R^l \\
& + d(i+1) p_{i+1,j,k,l} S^i E^j I^k R^l \\
& + \sigma(j+1) p_{i,j+1,k-1,l}(t) S^i E^j I^k R^l \\
& + d(j+1) p_{i,j+1,k,l}(t) S^i E^j I^k R^l \\
& + \gamma(k+1) p_{i,j,k+1,l-1}(t) S^i E^j I^k R^l \\
& + d(k+1) p_{i,j,k+1,l}(t) S^i E^j I^k R^l \\
& + d(l+1) p_{i,j,k,l+1}(t) S^i E^j I^k R^l \\
& - \lambda p_{i,j,k,l}(t) S^i E^j I^k R^l - \beta i k p_{i,j,k,l}(t) S^i E^j I^k R^l \\
& - di p_{i,j,k,l}(t) S^i E^j I^k R^l - \sigma j p_{i,j,k,l}(t) S^i E^j I^k R^l \\
& - dj p_{i,j,k,l}(t) S^i E^j I^k R^l - \gamma k p_{i,j,k,l}(t) S^i E^j I^k R^l \\
& - dk p_{i,j,k,l}(t) S^i E^j I^k R^l - dl p_{i,j,k,l}(t) S^i E^j I^k R^l] \\
& + \frac{o(\Delta t}{\Delta t}.
\end{aligned} \tag{1.19}$$

Next take sum over (i,j,k,l) and take $\lim \Delta t \to 0$ and arrive at the forward Kol-

mogorov differential equation, Equation (1.20),

$$\begin{aligned}\frac{dp_{i,j,k,l}(t)}{dt} &= \lambda p_{i-1,j,k,l}(t)S^iE^jI^kR^l + \beta(i+1)kp_{i+1,j-1,k,l}(t)S^iE^jI^kR^l \\ &+ d(i+1)p_{i+1,j,k,l}S^iE^jI^kR^l \\ &+ \sigma(j+1)p_{i,j+1,k-1,l}(t)S^iE^jI^kR^l + d(j+1)p_{i,j+1,k,l}(t)S^iE^jI^kR^l \\ &+ \gamma(k+1)p_{i,j,k+1,l-1}(t)S^iE^jI^kR^l + d(k+1)p_{i,j,k+1,l}(t)S^iE^jI^kR^l \\ &+ d(l+1)p_{i,j,k,l+1}(t)S^iE^jI^kR^l \\ &- \lambda p_{i,j,k,l}(t)S^iE^jI^kR^l - \beta ikp_{i,j,k,l}(t)S^iE^jI^kR^l \\ &- dip_{i,j,k,l}(t)S^iE^jI^kR^l - \sigma jp_{i,j,k,l}(t)S^iE^jI^kR^l \\ &- djp_{i,j,k,l}(t)S^iE^jI^kR^l - \gamma kp_{i,j,k,l}(t)S^iE^jI^kR^l \\ &- dkp_{i,j,k,l}(t)S^iE^jI^kR^l - dlp_{i,j,k,l}(t)S^iE^jI^kR^l.\end{aligned} \tag{1.20}$$

Next replace the deterministic variables i, j, k, l with the state variables of S, E, I, R to arrive at the probability generating function (pgf), Function (1.21)

$$\begin{aligned}\frac{dP}{dt} &= \lambda(S-1)P(S,E,I,R,t) + \beta I(E-S)\frac{\partial^2 P}{\partial S\partial I} + \sigma(I-E)\frac{\partial P}{\partial E} \\ &+ d(1-S)\frac{\partial P}{\partial S} + d(1-E)\frac{\partial P}{\partial E} + \gamma(R-I)\frac{\partial P}{\partial I} \\ &+ d(1-I)\frac{\partial P}{\partial I} + d(1-R)\frac{\partial P}{\partial R}.\end{aligned} \tag{1.21}$$

Here convert the pgf to the mgf, since the method of derivation of the moment differential equations is simpler from the moment generating function, defined as $M(\theta,\phi,\psi,\zeta,t) = P(S,E,I,R,t)e^{S\theta+E\phi+I\psi+R\zeta}$. From the probability generating

function (pgf), take the total derivative of $M(\theta, \phi, \psi, \zeta, t)$ to get Function (1.22).

$$\begin{aligned}\frac{\partial M}{\partial t} &= \lambda(e^{\theta}-1)M + \beta(e^{\phi-\theta}-1)\frac{\partial^2 M}{\partial\theta\partial\psi} + d(e^{-\theta}-1)\frac{\partial M}{\partial\theta} \\ &+ \sigma(e^{\psi-\phi}-1)\frac{\partial M}{\partial\phi} + d(e^{-\phi}-1)\frac{\partial M}{\partial\phi} + \gamma(e^{\zeta-\psi}-1)\frac{\partial M}{\partial\psi} \\ &+ d(e^{-\psi}-1)\frac{\partial M}{\partial\psi} + d(e^{-\zeta}-1)\frac{\partial M}{\partial\zeta}.\end{aligned} \tag{1.22}$$

To derive the system of stochastic differential equations for S, E, I, and R, take the derivative of Equation (1.22) with respect to the variable that corresponds to the moment required for the differential equation; for example, if $E[S]$ is required, take the partial derivative of Equation (1.22) w.r.t θ and evaluate the results at $(\theta, \phi, \psi, \zeta) = (0, 0, 0, 0)$,

$$\frac{dE[S]}{dt} = \lambda - \beta E[SI] - dE[S], \tag{1.23}$$

depending on the higher order moment $E[SI] = E[S]E[I] + cov(S, I)$. If $cov(S, I) = 0$, the ordinary differential equation is returned and the state variables of the system are independent. Obviously this cannot be solved explicitly, as it is not closed, differential equations for the higher order moments must be considered, in an attempt to solve the equation. Take the derivative of Equation (1.22) w.r.t S and I, and end up with

$$\frac{d(E[SI])}{dt} = \lambda E[I] - \beta E[IIS] - 2dE[SI] + \sigma E[SE] - \gamma E[SI]. \tag{1.24}$$

We can see that this equation contains the third order moment. The system of

stochastic differential equations is

$$\begin{aligned}
\frac{dE[S]}{dt} &= \lambda - \beta E[SI] - dE[S] \\
\frac{dE[E]}{dt} &= \beta E[SI] - \sigma E[E] - dE[E] \\
\frac{dE[I]}{dt} &= \sigma E[E] - \gamma E[I] - dE[I] \\
\frac{dE[R]}{dt} &= \gamma E[I] - dE[R] \\
\frac{dE[S^2]}{dt} &= \lambda + 2\lambda E[S] + \beta E[SI] - 2\beta E[S^2 I] + dE[S] - 2dE[S^2] \\
\frac{dE[E^2]}{dt} &= \beta E[SI] + 2\beta E[SEI] + \sigma E[E] - 2\sigma E[E^2] + dE[E] - 2dE[E^2] \\
\frac{dE[I^2]}{dt} &= \sigma E[E] + 2\sigma E[EI] + \gamma E[I] - 2\gamma E[I^2] + dE[I] - 2dE[I^2] \\
\frac{dE[SI]}{dt} &= \lambda E[I] - \beta E[I^2 S] - 2dE[SI] + \sigma E[SE] - \gamma E[SI] \\
\frac{dE[EI]}{dt} &= \beta E[I^2 S] - \sigma E[E] - \sigma E[EI] + \sigma E[E^2] - 2dE[EI] - \gamma E[EI] \\
\frac{dE[SE]}{dt} &= \lambda E[E] - \beta E[SI] - \beta E[SEI] + \beta E[S^2 I] - 2dE[SE] - \sigma E[SE].
\end{aligned} \tag{1.25}$$

To choose which higher order moment equations to explicitly define, look at which 2^{nd} order moments appear in the equations of the first order moments. Next look at which equations of 2nd order moments appear in the already existing 2nd order moment equations. Since there are no 2nd order moments involving $E[R]$, there is no 2nd order moment equation for $E[R]$.

It must be noted that this System (1.25), is not a closed system and cannot be solved explicitly. Almost every equation depends on higher order moments; some first order equations depend on second order moments and some second order equa-

tions depend on third order moments. In order to solve this system, the system needs to be closed, which is done by the third order moments by moment closure techniques. In moment closure methods, the higher order moments are approximated by lower order moments by making assumptions about the distributions of the higher order moments. This method for deriving the system of stochastic differential equation System (1.25) can be found in [4].

1.4.3.3 Moment closure

Moment closure approximation has become more common in recent years. Closure methods are based on a grand time evolution equation, Equation (1.22), and apply to transient and equilibrium dynamics [34]. Alternative approaches — linearization and quasi-equilibrium probabilities — are limited in their applications as they can only be applied in regions close to the fixed points or equilibria [34]. In many cases, only first and second order moments are of interest so higher order moments can be approximated by the lower order moments [55]. When population levels are low, it is advisable to assume the underlying distribution of the higher order moments is log-normal, as this guarantees a positive population [39]. The moment closure approximations used for the third order moments in Model (1.25) come from the method of [55].

From [55], the log-normal moment closure equations for third order moments of

System (1.25) are

$$E[S^2I] = \frac{E[S^2]E[SI]^2}{E[S]E[I]^2}$$
$$E[I^2S] = \frac{E[I^2]E[IS]^2}{E[I]E[S]^2} \tag{1.26}$$
$$E[SEI] = \frac{E[SE]E[SI]E[EI]}{E[S]E[E]E[I]}.$$

Equation (1.26), uses lower order moment equations to approximate the higher order moments. When these Equations (1.26) are substituted into Equation (1.25), the system is now closed and can be solved.

From the equations for the approximations of the third order moments, Equation (1.26), we notice that the denominators of these equations are the first order moments. It must be ensured that the initial conditions for the first order moments that are in the denominators of the moment approximations for the system of stochastic differential equations are non-zero. This is a limitation of this moment closure technique. The set of initial conditions for the systems of ODEs and SDEs and the ABMC method must be, at least, $(S, E, I, R) = (10000, 1, 100, 0)$.

The initial conditions to solve the higher order moment equations of System (1.25), are moment closure approximations themselves. The form of the initial conditions are as follows: $(E[S], E[E], E[I], E[R], E[S^2], E[E^2], E[I^2], E[SI], E[EI], E[SE]) = (E[S], E[E], E[I], E[R], (E[S])^2, (E[E])^2, (E[I])^2, E[S]E[I], E[E]E[I], E[S]E[E])$. The values of the first order moments are multiplied together to get the initial condition values for the second order moments. Before any interaction has occurred amongst

individuals in the system, each of the classes are independent hence the form of the initial conditions.

1.5 Scope of Thesis

In this thesis we will study mass media and its affect on an influenza epidemic. In this chapter we have outlined deterministic models that have been used to model epidemics. In Chapter 2, we look at the functions that have previously modelled media will be compared in an SEIR model. Sensitivity analysis is completed for each model. Agent-based Monte Carlo simulations will be completed in order to observe variability that is present in an epidemic but not measurable in deterministic models. Although previous studies have incorporated mass media, it is incorporated as a specific function and waning of mass media is not considered. In Chapter 3, a new model to include social distancing, mass media and media waning will be proposed and stochastic simulations will be carried out to measure epidemic variability. Stability and sensitivity analysis will be completed for this new model. In Chapter 4, we will look at data pertaining to mass media and H1N1. This data will be incorporated into the model proposed in Chapter 3. Finally, Chapter 5 introduces a system of partial differential equations and ordinary differential equations to model a continuum of social behaviour throughout each compartment.

2 Previous Media Models

2.1 Previous media models

Mathematical modelling has been used to study the effect of media on epidemics by employing functions in the transmission terms of mathematical models: [5, 6, 37, 38, 44, 54, 58, 59, 65]. As globalization is a reality and technology and media play an increasing role in daily life, it is of interest to incorporate media into classical disease models. In this chapter we review some past models including the effects of media.

2.1.1 Media/psychological impact on multiple outbreaks of emerging infectious diseases

The first model that we look at is presented in [38]. This model is proposed as a first look at emphasizing media/psychological impact of emerging infectious diseases in a compartmental model. This study focuses on the SARS outbreak in Toronto.

For this model, the population is split into 5 compartments: susceptible, exposed, infectious, hospitalized and removed. The authors concentrate on the solution of the case where effective contacts are reduced as infection level increases. The model assumes that the outbreak is short thus the susceptible population remains unchanged and natural birth and death are ignored. As presented in [38],

see Model (2.1),

$$\begin{aligned} \dot{E} &= \beta_0 IS - \alpha E \\ \dot{I} &= \alpha E - dI - hI \\ \dot{H} &= hI - d_h H - rH, \end{aligned} \tag{2.1}$$

with parameters in Table 2.1. Standard methods to analyze System (2.1) were used

Parameter	Definition	Parameter	Definition
E(t)	Number exposed, not yet infected.	I(t)	Number of infectious individuals.
H(t)	Hospitalized class receiving treatment. No risk to S.	β_0	Infectiousness and contact transmission rates
S	Susceptible population; considered a parameter	α	Transfer rate per unit time (day) for exposed to become infectious
d	Death due to disease without treatment	h	Rate entering treatment
d_h	Disease induced death rate of hospitalized individuals	r	Recovery rate of hospitalized cases

Table 2.1: Parameters for system (2.1).

to identify the disease free equilibrium and the basic reproduction number. By the

next generation method, the basic reproduction number is

$$R_0 = \frac{\beta_0 S}{d+h}. \tag{2.2}$$

When $R_0 < 1$ the disease will die out since the disease free equilibrium is asymptotically stable. The disease free equilibrium is unstable when $R_0 > 1$, which means the disease will grow exponentially [38].

According to [38], System (2.1), is not representative of the reported impact of the news coverage on avoidance behaviours at the individual and societal level and does not incorporate the self-control property of the illness due to the change of avoidance patterns at different stages of infection.

In order to include the psychological impact of the mass media, [38] assumed that this phenomenon could be described by an exponentially decreasing factor, since a detailed functional description does not exist and would be extremely difficult to achieve. The exponentially decreasing factor is

$$\beta_0 = \beta e^{-a_1 E - a_2 I - a_3 H}, \tag{2.3}$$

where a_1, a_2, a_3 are non-negative parameters that measure the psychological impact of the reported cases of exposed, infected and hospitalized, respectively. The revised

system including the self-control property is

$$\begin{aligned} \dot{E} &= \beta e^{-a_1 E - a_2 I - a_3 H} IS - \alpha E \\ \dot{I} &= \alpha E - dI - hI \\ \dot{H} &= hI - d_h H - rH. \end{aligned} \tag{2.4}$$

System (2.4), from [38], is studied, therein, for its nonlinear dynamics and oscillatory behaviours in the biologically feasible region of $\mathcal{D} = \left\{(E, I, H) \in \mathbf{R}_+^3\right\}$. The disease free equilibrium of Model (2.4) is $E_0 = (0, 0, 0)$. The basic reproductive ratio for Model (2.4) is $R_0 = \frac{\beta S}{d+h}$ for any fixed values of a_1, a_2, a_3, the three parameters that measure the media/psychological impact of exposed, infected and hospitalized cases on the disease dynamics. There is an endemic equilibrium when $R_0 > 1$. The endemic equilibrium is Expression (2.5).

$$\begin{aligned} E^* &= C(d_h + t)(d + h) \\ I^* &= D(d_h + t)\alpha \\ H^* &= Ch\alpha, \end{aligned} \tag{2.5}$$

where

$$C = \frac{\ln R_0}{a_1(d+h)(r+d_h) + a_2\alpha(r+d_h) + a_3\alpha h}. \tag{2.6}$$

The endemic equilibrium is studied for stability and possible bifurcations. It is found that the stability of the model depends on the parameters a_i, $i = 1, 2, 3$ and a Hopf bifurcation is found for $a_1 = a_2 = 0$, $a_3 > 0$.

This is one of the first articles talking about media/psychological impact of reported cases of a disease on the population. This model was extended in [5], where they look at a classical SEI model and the ideas in [38] in order to study a new incidence functional reflecting the impact of media coverage and disease spread and control. The incidence functional is considered to play a key role in ensuring that a realistic description of transmission dynamics is achieved [5].

2.1.2 The impact of media on the control of infectious diseases

The model developed in [5], is

$$\begin{aligned} \dot{S} &= bS\left(1-\frac{S}{K}\right) - \mu e^{-mI} SI \\ \dot{E} &= \mu e^{-mI} SI - (c+d)\, E \\ \dot{I} &= cE - \gamma I, \end{aligned} \tag{2.7}$$

where b is the intrinsic growth rate of the human population, K is the carrying capacity of people in a given region, c is the rate per unit time that exposed individuals become infected, d is the natural death rate for the susceptible population and γ is the removal rate from the infected compartment. The removal rate incorporates recovery of hospitalized infectious individuals and natural death. With the complicated growth and transmission dynamics of Model (2.7), its aim is to show that if the real situation of the disease is not reported correctly, multiple outbreaks may occur [5].

The media incidence rate is again a decaying exponential of the form

$$\beta(I) = \mu e^{-mI}, \tag{2.8}$$

which measures virus spread. In Model (2.7), it is assumed that the spreading of the virus is not only related to the disease spreading ability but also to how alert the susceptible population is to the disease. This aspect is measured in the parameter $m > 0$, although it is assumed that this parameter is sufficiently small as it is not a determining factor in disease spread. With this for the incidence rate, as the media becomes more aware of the disease or the number of infected individuals increases, the transmission rate will decrease. If $m \approx 0$, we have the mass action transmission dynamic of classical SEI models. It is also assumed that the population of a region grows according to Logistic growth, compared with Systems (2.1), (2.4), where it is assumed that the epidemic is short enough that the population remains approximately constant.

The basic reproductive ratio, R_0 for Model (2.7) is $R_0 = \frac{\mu c K}{\gamma(c+d)}$. If the right hand side of System (2.7) is set to 0, then we can see that origin is an equilibrium point. With further analysis it is noted in the paper that there is a hyperbolic saddle point. It can also be shown that the disease free equilibrium is locally and globally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$. The details of this can be found in [5].

In addition to the disease free equilibrium, there are also endemic equilibria of

which the number depends on the value of m, the media parameter. It is possible for System (2.7) to have up to three endemic equilibria when media and psychological impacts are incorporated. The endemic equilibria appear as three different cases; the first case is $m = 0$. In this case there is a unique endemic equilibrium which is locally asymptotically stable. The case where $m > 0$, a Hopf bifurcation occurs. This implies a periodic solution appears and the disease will be endemic in the population, with multiple peaks. Although this is a periodic solution and multiple disease peaks occur, the length of time the secondary disease peak is much shorter with the incorporation of media and psychological impacts. The detailed results of this model can be seen in [5].

2.1.3 An SIS infection model incorporating media coverage

Another model incorporating a general nonlinear incidence function to reflect intrinsic characteristics of media and education on the spread of an infectious disease [6]. The model is Model (2.9),

$$\begin{aligned} \dot{S} &= A - dS - (\mu_1 - \mu_2 f(I)) \frac{SI}{(S+I) + \gamma I} \\ \dot{I} &= (\mu_1 - \mu_2 f(I)) \frac{SI}{(S+I)} - (d + \nu + \gamma) I. \end{aligned} \tag{2.9}$$

where A is the recruitment rate of the population, d is the natural death rate, γ is the recovery rate, ν is the disease induced death rate, and the parameter for disease transmission is a function that incorporates media: $c(I) = c_1 - c_2 f(I)$, where c_1 is

the contact rate of individuals and c_2 is the maximum reduced contact rate due to the presence of infected individuals.

The basic reproduction ratio is $R_0 = \frac{\mu_1}{d+\nu+\gamma}$. Without an explicit expression for $f(I)$, the authors of [6] find two biologically meaningful equilibria, one disease free equilibrium and one endemic equilibrium with the disease free equilibrium being $E_0 = \left(\frac{A}{d}, 0\right)$, which is locally and globally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$. The existence of the unique positive endemic equilibrium can be shown for any choice of $f(I)$ that satisfies the conditions of Expression (2.10) and $R_0 > 1$.

$$f(0) = 0,\ f'(I) < 0,\ \lim_{I\to\infty} f(I) = 1. \tag{2.10}$$

The endemic equilibrium is locally and globally asymptotically stable when it exists [6]. The details can be found explicitly in [6].

2.1.4 Global analysis of an epidemic model with nonmonotone incidence rate

Another model incorporating mass media is proposed in [65].

$$\begin{aligned} \dot{S} &= b - dS - \frac{kSI}{1+\alpha I^2} + \gamma R \\ \dot{I} &= \frac{kSI}{1+\alpha I^2} - (d+\mu)\, I \\ \dot{R} &= \mu I - (d+\gamma)\, R. \end{aligned} \tag{2.11}$$

The parameters are b, the recruitment rate of the population, d is the natural death rate, k is the proportionality constant, μ is the natural recovery rate and γ is the rate at which one loses immunity. Model (2.11) is an SIR model with media inclusion as an incidence rate functional, Equation (2.12),

$$g(I)S = \frac{kIS}{1+\alpha I^2}, \tag{2.12}$$

where kI measures the force of the infection and $\frac{1}{1+\alpha I^2}$, with α as the inhibitory parameter that describes the psychological effect on the susceptible individuals through behaviour modification when the population of infected individuals becomes very high, [65].

The basic reproductive ratio for Model (2.11) is $R_0 = \frac{kb}{d(d+\mu)}$. The stability of the disease free equilibrium, $E_0 = (\frac{b}{d}, 0, 0)$, and the endemic equilibrium

$$\begin{aligned} S^* &= \frac{1}{d}\lceil b - (d+\mu - \frac{\gamma\mu}{d+\gamma}I^*\rceil, \\ I^* &= \frac{-k(d+\mu-\frac{\gamma\mu}{d+\gamma}) + \hat{\Delta}}{2\alpha d(d+\mu}, \\ R^* &= \frac{\mu}{d+\gamma}I^*, \end{aligned} \tag{2.13}$$

where

$$\Delta = k^2 d + \mu - \frac{\gamma\mu}{d+\gamma}^2 - 4\alpha d^2(d+\mu)^2\lceil 1 - R_0\rceil, \tag{2.14}$$

is studied. The analytical results of [65] conclude that if $R_0 \leq 1$ the disease free equilibrium is a global attractor in the first quadrant also attracting all of the orbits in the first quadrant. For $R_0 > 1$ there is a disease free equilibrium and an endemic

equilibrium. The positive endemic equilibrium is unique when it exists. It is a global attractor for in the interior of the first quadrant and there is no endemic equilibrium for $R_0 < 1$ [65].

The authors of [65] note that there are many other functionals that can be used when considering different conditions that effect transmission of a disease. Details for other functionals can be found in [65].

2.1.5 Effect of media-induced social distancing on disease transmission in a two patch setting

As an extension to both Models (2.9) and (2.11) from [6] and [65], respectively, a two patch model assuming spatial heterogeneity is proposed since diseases spread geographically over time. All of the previous models outlined assume spatial homogeneity: the disease is confined to one population or group [58].

The following model, Model (2.15) is a two patch SIS model with each patch connected by population movement.

$$\begin{aligned}
\dot{S}_1 &= \Lambda_1 - \beta_1(I_1)\frac{S_1 I_1}{S_1 + I_1} + \gamma_1 I_1 - d_1 S_1 - m_{12} S_1 + m_{21} S_2 \\
\dot{I}_1 &= \beta_1(I_1)\frac{S_1 I_1}{S_1 + I_1} - (\gamma_1 + d_1) I_1 - m_{12} I_1 + m_{21} I_2 \\
\dot{S}_2 &= \Lambda_2 - \beta_2(I_2)\frac{S_2 I_2}{S_2 + I_2} + \gamma_2 I_2 - d_2 S_2 + m_{12} S_1 - m_{21} S_2 \\
\dot{I}_2 &= \beta_2(I_2)\frac{S_2 I_2}{S_2 + I_2} - (\gamma_2 + d_2) I_2 + m_{12} I_1 - m_{21} I_2,
\end{aligned} \tag{2.15}$$

with initial conditions

$$S_1(0) + S_2(0) > 0, \qquad I_1(0), I_2(0) \geq 0,$$
$$I_1(0) + I_2(0) > 0. \tag{2.16}$$

The parameters for Model (2.15) are Λ_i, the population recruitment rate, m_{ij}, the travel rate from patch i to patch j, γ_1, the recovery rate and d_i, the natural death rate. An assumption for Model (2.15) is that S and I have the same travel rate from patch i to patch j: the disease does not impede travel abilities [58].

Media and social distancing are being considered here and the transmission incidence functional here has a similar form to the modified, nonconstant incidence Functional (2.12):

$$\beta_i(I_i) = a_i - b_i f_i(I_i). \tag{2.17}$$

The parameters include a_i, the maximal effective contact rate between S and I in patch i, analogous to the c_1 in Function (2.12), b_i is the maximal reduced effective contact rate due to mass media alert in the presence of I, analogous to c_2. The assumptions and conditions are similar to Expression (2.10) with $a_i < b_i$, in place of $c_1 < c_2$, $0 < f_i'(I_i) \leq 1$ instead of $f'(I) < 0$ and a new condition $f''_i(I_i) < 0$.

Model (2.15), a patch model, is also analyzed for R_0, the disease free equilibrium and the endemic equilibrium. Here $E_0 = (I_{10}, I_{20}, S_{10}, S_{20}) = (0, 0, N_1^*, N_2^*)$, with N_i^* being the initial population of each patch. The basic reproduction number is calculated again through the next generation method and it is determined that

media does not play a role in the R_0 of this system. Finally it is shown that the disease free equilibrium is locally and globally asymptotically stable with no endemic equilibrium for $R_0 < 1$ and unstable for $R_0 > 1$. Also, it is proven that there is one unique locally and globally asymptotically stable endemic equilibrium for $R_0 > 1$ and an unstable disease free equilibrium [58].

For the patch Model (2.15), simulations are carried out assuming a non-fatal disease with two very different populations in order to better demonstrate the effect of the media. It is possible to use any media functional mentioned above however, the simple media coverage function chosen in [58] is,

$$f(I_i) = \frac{I_i}{1 + I_1}. \tag{2.18}$$

Although the basic reproduction number does not include media, it is concluded that if R_0 is small, media can be used to greatly reduce the effect of the disease on the population; this becomes more difficult as R_0 becomes larger. It is determined by the simulations that media cannot be used to make an epidemic go extinct if $R_0 > 1$, but it can greatly reduce the number of individuals infected and decrease the severity of the infection. Furthermore, if R_0 is brought below 1 by means other than media coverage, the media coverage can help speed up the extinction of the epidemic [58].

2.1.6 Influenza and media: The impact of media coverage on the transmission dynamics of human influenza

The models in [59] set out to monitor the dynamics of single-strain influenza without cross-immunity. The susceptible population in the model is increased by recruitment of individuals — birth, immigration and loss of immunity. It decreases through vaccination campaigns, infection, natural death and emigration. The vaccine used in the model is assumed to not be 100% effective. Media coverage, similar to the previous models, is introduced via a saturated incidence function. The model being considered is Model (2.19),

$$\begin{aligned}
\dot{S} &= \Lambda + \omega V - (theta + \mu)\, S - \left(\beta_1 - \beta_2 \frac{I}{m_I + I}\right) SI + \sigma R \\
\dot{I} &= \left(\beta_1 - \beta_2 \frac{I}{m_I + I}\right) SI + \left(\beta_1 - \beta_3 \frac{I}{m_I + I}\right) (1-\gamma)\, VI \\
&\quad - (\alpha + \mu + \lambda)\, I \\
\dot{V} &= \theta S - (\mu + \omega)\, V - \left(\beta_1 - \beta_3 \frac{I}{m_I + I}\right) (1-\gamma)\, VI \\
\dot{R} &= \lambda I - (\mu + \sigma)\, R,
\end{aligned} \tag{2.19}$$

where $S(t)$ is the susceptible population, $I(t)$ is the infective population, $V(t)$ is the vaccinated population and $R(t)$ is the recovered population. The parameters for this model are in Table (2.2).

Media in Model (2.19) is included through a half-saturation constant $m_I > 0$ in a continuous bounded function which takes into account disease saturation and

Parameter	Definition	Parameter	Definition
Λ	susceptible population recruitment rate	θ	vaccination rate
μ	natural death rate	β_1	infection rate
ω	vaccine immunity wane rate	γ	vaccine efficacy
α	death due to disease	λ	recovery rate
β_2	contact transmission rate of infected individuals	β_3	contact transmission rate of vaccinated individuals

Table 2.2: Parameters for system (2.19).

psychological effects, Equation (2.20):

$$g(I) = \frac{I}{m_I + I}. \tag{2.20}$$

With this Michaelis-Menten functional response, Equation (2.20), media coverage rises as the number of infectives rise. Media reaches a plateau when information saturation has been reached.

The analysis of Model (2.19) is completed by finding and studying the stability of the equilibria and solving for R_0. For $R_0 < 1$, there is a disease free equilibrium which is locally asymptotically stable and unstable for $R_0 > 1$. This is solved

through finding the Jacobian of Model (2.19) and evaluating it at the disease free equilibrium, Expression (2.21),

$$\begin{aligned} E_0 &= \left(\bar{S}, \bar{I}, \bar{V}, \bar{R}\right) \\ &= \left(\frac{\Lambda\left(\mu+\omega\right)}{\mu\left(\theta+\mu+\omega\right)}, 0, \frac{\Lambda\theta}{\mu\left(\theta+\mu+\omega\right)}, 0\right). \end{aligned} \tag{2.21}$$

Expression (2.21) is globally asymptotically stable, the details can be found in [59].

In addition to System (2.19), the authors of [59] include two intervention methods and create and analyze an optimal control model. The aim of the optimal control section is to determine the benefit of vaccination and media coverage. The final section of this article includes pulse vaccination.

The conclusion of the compartmental model in [59] with media coverage is that more people are encouraged to become vaccinated. From looking at the optimal control and pulse vaccination cases, the authors of [59] conclude that the media may also trigger a vaccination panic. People may become overconfident in the vaccine and see it as a cure-all which is dangerous in the face of a new epidemic with a vaccine that does not protect 100% of individuals, [59].

2.1.7 Summary

The previous studies have incorporated mass media into some traditional deterministic models. Incorporating mass media is important as it plays an influential role in our lives [58]. The models that have shown here directly affect disease transmission.

Moreover, mass media are not allowed to evolve as the pandemic evolves. They do not consider how the behaviour of the epidemic itself affects the mass media coverage which then affects how the epidemic proceeds. The prescribed media functions assume that mass media has a specific, constant, effect on disease transmission.

Within these structured models it is difficult to include the evolution of mass media that occurs during a pandemic. The different functions may have a different effect on epidemic outcome. In Chapter 3 we will look at how the functions presented in this chapter affect an epidemic outcome in an SEIR model and in Chapter 4, we will propose a model that more easily includes the evolution of mass media.

3 Functions used to incorporate mass media

3.1 Introduction

Mathematical modelling has been used to study the effect of media on epidemics by employing disease modelling and extensions as outlined in Chapter 2. Although these studies do incorporate mass media, it is incorporated as a specific function, different in each case. Each of these functions results in a different epidemic behaviour and outcome, suggesting this may not be the optimal way to incorporate mass media. Moreover, mass media are not considered to evolve in the previous studies as the pandemic evolves. They do not consider how the behaviour of the epidemic itself affects the mass media coverage which then affects how the epidemic proceeds. The prescribed media functions assume that mass media has a specific, constant, effect on disease transmission. The models in the previous media studies also look solely at deterministic modelling. Deterministic modelling can describe the behaviour of the epidemic but information important during an epidemic includes variability around key measurements such as peak epidemic time, peak number of infections and the duration of the epidemic. A stochastic model is well suited to this task. We will employ agent-based Monte Carlo simulations in order to look at variability that can be present during an epidemic. This variability around the key measurements is often information that is important to public health.

It is important to know which parameters in a model have the most significant

effect on different outcomes. We will complete a sensitivity analysis for certain specified epidemic outcomes to see which parameters have the greatest effect on various outcomes. This may provide information about what should be focused on by the public to control the spread of an epidemic.

As globalization is a reality and technology and media play an increasing role in daily life, the information may have a large effect on the individual's willingness to cooperate with public health measures and vaccination. It is of interest to incorporate media into classical disease models. We look to study the role that mass media plays in an epidemic through mathematical modelling.

In the sections that follow we will compare a standardized version of functions representing mass media from previous works. These functions will also be compared in a standardized SEIR model. Stochastic models will be presented and compared in order to look at variation within an epidemic. A sensitivity analysis is completed in order to look at the importance of certain parameters for various epidemic outcomes.

3.2 Model

3.2.1 Media functions

There have been multiple functions used in the mathematical modelling studies to represent mass media during an epidemic. The models and the functions used to represent mass media are outlined in Chapter 2. Here we look at the proposed functions for media influences on an epidemic SEIR model and conduct a comparison.

From disease modelling literature and outlined in Chapter 2, we have identified three distinct functions employed to present the effects of media

$$f(I,\beta,p) = \beta e^{-p_1 \gamma I}, \quad [5, 37, 38, 44, 54] \tag{3.1}$$

$$f(I,\beta,p) = \beta\left(\frac{1}{1+p_2 I^2}\right), \quad [58, 65] \tag{3.2}$$

$$f(I,\beta,p) = \left(\beta - \beta\frac{I}{p_3+I}\right), \quad [6, 59]. \tag{3.3}$$

Each of Functions (3.1-3.3) incorporates a media parameter p_i, where $i = 1, 2, 3$. Functions (3.1-3.3) are decreasing functions that change with the value of p_i, see Figure (3.1). It is possible to write p_1 and p_2 in terms of p_3 in order to see how the functions relate to one another,

$$\begin{aligned} p_1 &= \frac{-\ln(\frac{p_3}{p_3+I_c})}{\gamma I_c} \\ p_2 &= \frac{1}{p_3 I_c}. \end{aligned} \tag{3.4}$$

Using Equation (3.4), we can determine p_1 and p_2 for some p_3 and I_c, which may be known from epidemiological data. In this study $\gamma = \frac{1}{4}$.

In order to see how these parameters affect the outcome of an epidemic, we vary p_3 for $p_3 = 10$, $p_3 = 100$ [59] and $p_3 = 1000$. The media functions plotted with these values are in Figure (3.1). We can see in Figure (3.1) that the functions are decreasing and intersect at $I_c = 300$ and $I_c = 1000$. These are the values for I_c that will be used so that the values of p_1 and p_2 coincide with p_3 for each of the models.

3.2.2 SEIR

In order to compare the Functions (3.1-3.3), we must choose a standardized model. For incorporation we have chosen the basic SEIR model with a constant population.

$$\begin{aligned} \dot{S} &= -f(I,\beta,p)SI \\ \dot{E} &= f(I,\beta,p)SI - \sigma E \\ \dot{I} &= \sigma E - \gamma I \\ \dot{R} &= \gamma I. \end{aligned} \tag{3.5}$$

The compartments with the initial conditions and the parameters with the values used for each of the models can be found in Table (3.1).

To find the basic reproductive ratio, R_0, for Model (3.5) we employ the next generation matrix method as in Chapter 1. In the next generation method, R_0 is defined as the spectral radius of the next generation operator, (FV^{-1}). The

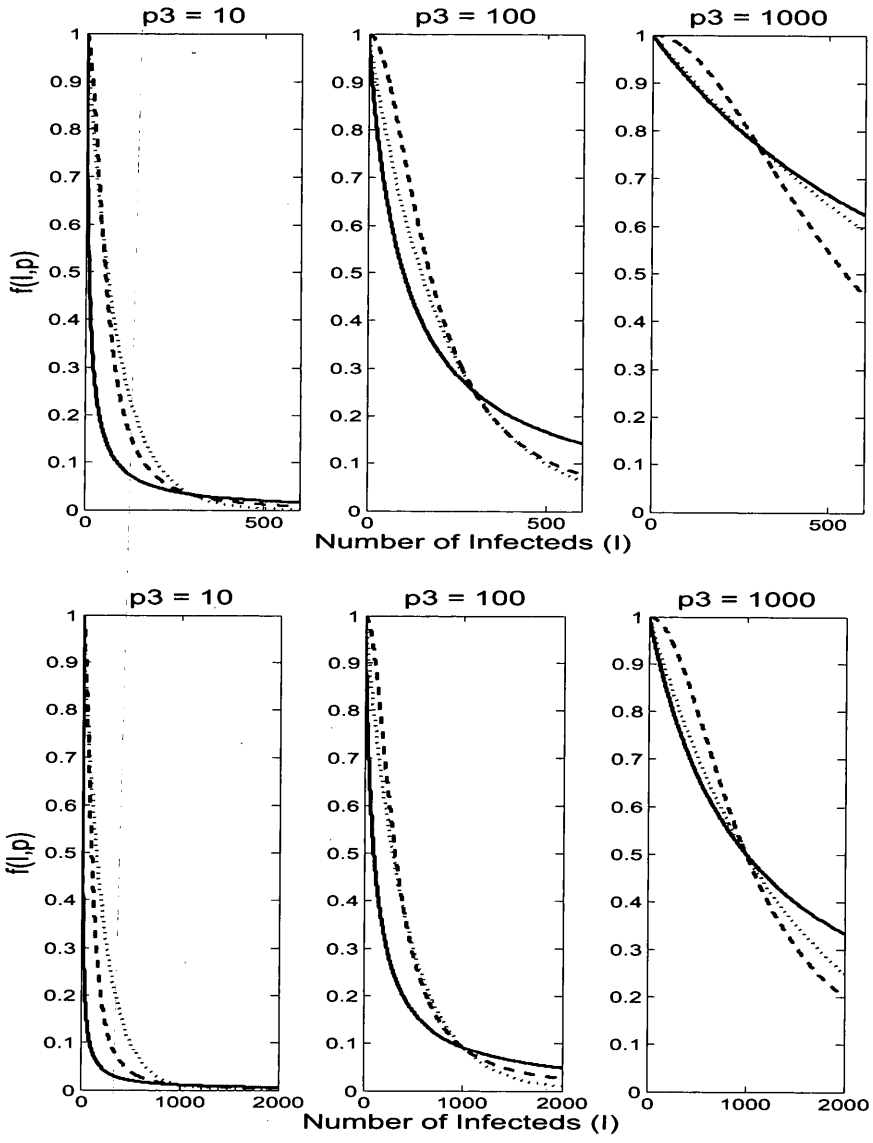

Figure 3.1: Media functions. The first row is media functions for $I_c = 300$. The second row corresponds to media functions for $I_c = 1000$. The dotted line represent Function (3.1), the dashed line represent Function (3.2) and the solid line represent Function (3.3).

Parameter	Definition	Value	Reference
S(t)	susceptible individuals	$10,000$ individuals	
E(t)	exposed individuals	0	
I(t)	infectious individuals	100 individuals	
R(t)	recovered individuals	0	
β	contact transmission rate	$6.67e{-}5$	Calculate from R_0
σ	exposed to infectious rate	$\frac{1}{2}\ \frac{1}{\text{day}}$	[46]
γ	recovery rate	$\frac{1}{4}\ \frac{1}{\text{day}}$	[46]
p_i	media parameter	$p_3 = 10, 100, 1000$ reports to change behaviour	[59]
R_0	basic reproductive ratio	2.67	[46]

Table 3.1: Parameters for model (3.5) and functions (3.1-3.3).

matrices (F) and (V) are derived from the terms in Model (3.5) that relate to the creation of new infections and the movement of infectious individuals around the system. This corresponds to

$$\begin{aligned} \dot{E} &= f(I,\beta,p)SI - \sigma E \\ \dot{I} &= \sigma E - \gamma I. \end{aligned} \tag{3.6}$$

Calculating the partial derivatives with respect to E and I of the terms that refer to the creation of new illnesses for (F) and the movement of infectious individuals (V), we obtain

$$F = \begin{pmatrix} 0 & \frac{\partial f}{\partial I} \\ 0 & 0 \end{pmatrix}, \tag{3.7}$$

and

$$V = \begin{pmatrix} \sigma & 0 \\ -\sigma & \gamma \end{pmatrix}, \tag{3.8}$$

where $FV^{-1} = \frac{\frac{\partial f}{\partial I}}{\gamma}$. This calculation for each of the Functions (3.1-3.3) is completed by fixing p_3 and setting $I = 0$, resulting in $R_0 = \frac{\beta}{\gamma}$ for all Function (3.1-3.3). Explicit calculations for Functions (3.1-3.3) can be found in Appendix A.

The above mathematical models are useful in describing the behaviour of an epidemic, but they are unable to provide estimates in variation of important public health measures. A stochastic simulation lends itself well to demonstrating variation within an epidemic [25]. Here, we employ an Agent-Based Monte Carlo (ABMC) simulation.

3.2.3 Agent-based Monte Carlo

An Agent-Based Monte Carlo (ABMC) simulation moves forward not in regular time intervals but following event times: the next time that an individual changes state within the system. The events that occur for the simulations for Model (3.5) are outlined in Figure (3.2). Table (3.2) lists the means of the corresponding parameters, assuming an exponential distribution, from Table (3.1). An exponential distribution is assumed so that we can compare our results to ODEs.

Mean	Definition	Value
$\frac{1}{\beta}$	mean contact transmission	14992.5
$\frac{1}{\sigma}$	mean days from exposed to infectious	2 days
$\frac{1}{\gamma}$	mean days to recovery	4 days
p_i	media parameter	10, 100, 1000

Table 3.2: Parameters for Model (3.5) and Functions (3.1-3.3). These are means of exponential lifetime distributions.

Corresponding to movements outlined in Figure (3.2), there are event times for recovery and infecting a susceptible agent. The procedure outlined in Chapter 1 for the ABMC is completed for model (3.5) for each of Functions (3.1-3.3) for $I_c = 300$ and $I_c = 1000$ in order to look at variation within an influenza epidemic. The

stop condition that has been chosen for an epidemic to be finished throughout this work is $E(t)+I(t) < 2$, after the epidemic has peaked. This has been chosen since, generally, there is less than one individual in each of these classes for this condition.

3.3 Results

The results for the ABMC simulations of the ODE systems and the ODE solutions can be seen in Figure (3.3). In Figure (3.3) we can see that the media functions for each value of p_i produce different results. We also notice that media, the value of p_3, does have an effect on the behaviour of the epidemic. If p_3 is very effective, Figure (3.3) first row where $p_3 = 10$, the epidemic peak is very small compared with the ODE model for all three media functions however the duration of the epidemic is very long. For $p_3 = 10$, the second row of Figure (3.3), the epidemic curves are more similar than in the first row and the epidemic peaks are higher. In the third row of Figure (3.3), the epidemic curves with media become similar to the epidemic without media, the largest curve. We can better see the differences of the functions in Figure (3.4), where the epidemic without media is not shown.

In Figure (3.4) we are better able to see the variation within an epidemic that the agent-based Monte Carlo simulations provide that we cannot obtain from a deterministic system alone. From the Monte Carlo systems we are able to determine measurement ranges for peak time, peak magnitude, end time and total infectious.

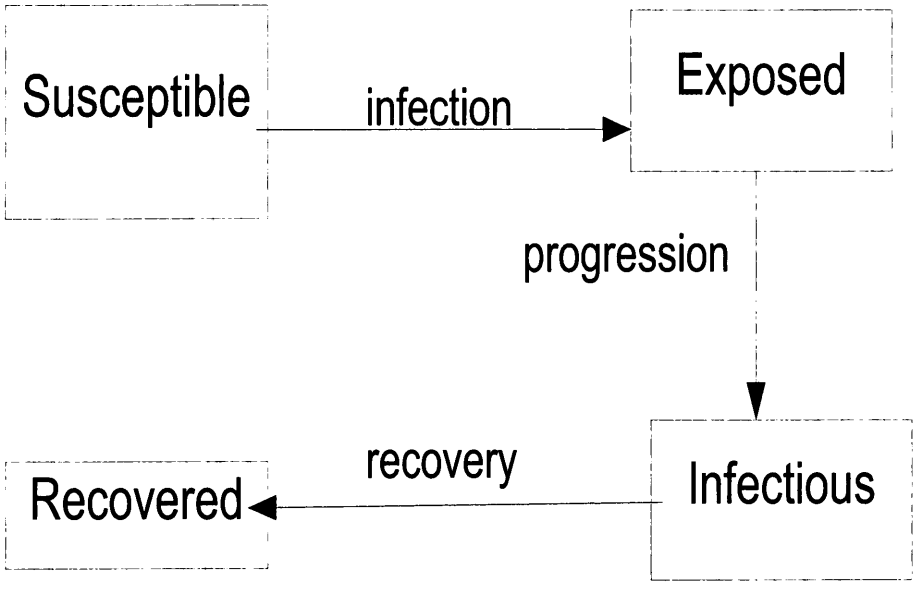

Figure 3.2: Schematic of the Agent Based Monte Carlo simulations corresponding to Table (3.2).

The results for $p_3 = 1000$ are displayed in Table (3.3). We can obtain similar measurements for the other two values of p_3. The equations for mean and standard error are in Appendix A.

As previously stated, when $p_3 = 1000$ the epidemic curves are the most similar to the epidemic without media influence. We can see from the results in Table (3.3) that even in writing p_1 and p_2 in terms of p_3 and choosing a value for I_c where the media functions intersect, the epidemic outcomes are not the same. To further demonstrate the differences in modelling mass media with a fixed media function, we have studied System (3.5) with $I_c = 1000$, and $p_3 = 10$, $p_3 = 100$ and $p_3 = 1000$, Figure (3.5). The results of the measurements that are obtained from the ABMC for $p_3 = 1000$ for Figure (3.5) are in Table (3.4). Again, similar results can be obtained for both $p_3 = 10$ and $p_3 = 100$.

3.3.1 Sensitivity Analsyis

Sensitivity analysis is now performed on Equations (3.1-3.3) to identify key parameters that affect the model outcomes corresponding to each of the functions. To conduct the sensitivity analysis of the parameters, we use Latin Hypercube Sampling (LHS) and partial rank correlation coefficient (PRCC) [7].

The Latin Hypercube Sampling (LHS) is a type of stratified Monte Carlo sampling, which is an extension of the Latin Square sampling. For this method, each

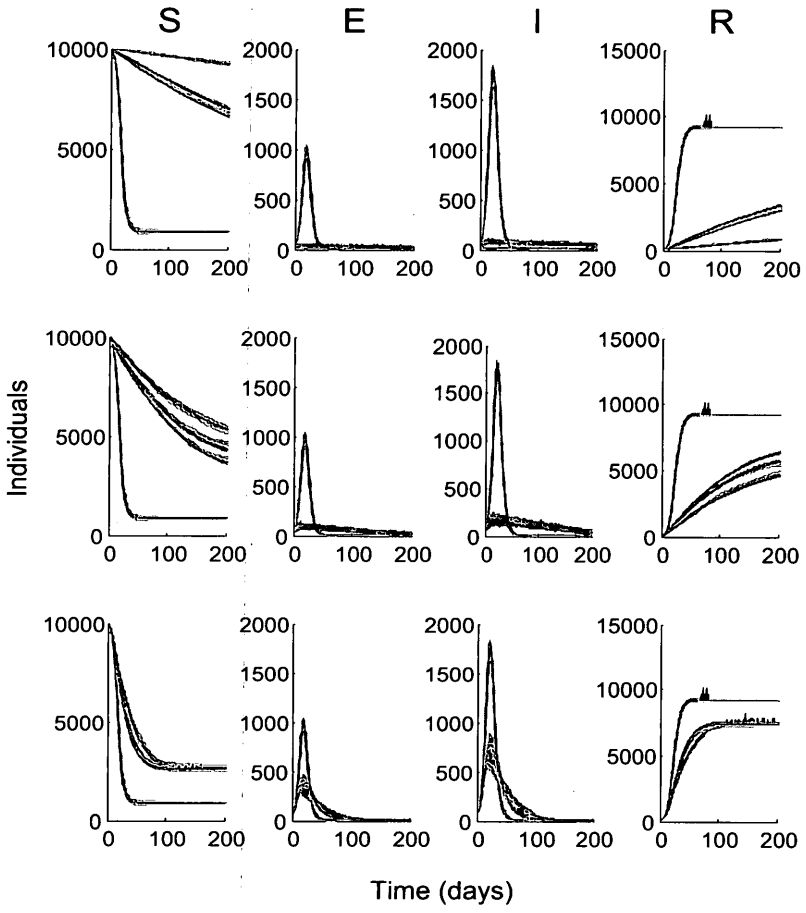

Figure 3.3: ODE and ABMC results with and without media functions for $I_c = 300$. The first row corresponds to $p_3 = 10$, the second row is $p_3 = 100$ and the third row is $p_3 = 1000$. There are four epidemic curves in each figure, one corresponding to Model (3.5) without media and the other three correspond to the model with each of the various media functions. The cyan curves are the ABMC simulations, the black are the means of the ABMC and the red curves are the ODE results. Parameter values are in Table (3.3).

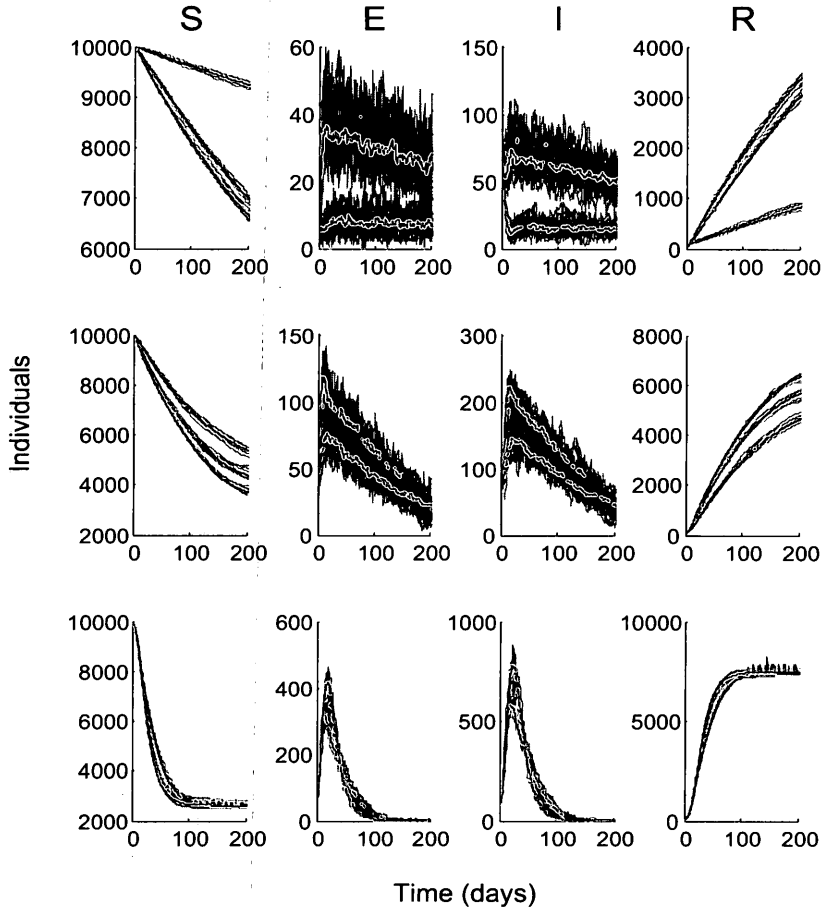

Figure 3.4: ODE and ABMC results for media functions for $I_c = 300$. There are 20 simulations here and $I_c = 300$. The first row corresponds to $p_3 = 10$, the second row is $p_3 = 100$ and the third row is $p_3 = 1000$. There are three epidemic curves in each figure. The largest epidemic curve corresponds to Function (3.1), the next largest is Function (3.2). The most shallow curve in each case corresponds to Function (3.3). The cyan curves are the ABMC simulations, the black are the means of the ABMC and the red curves are the ODE results. Parameter values are in Table (3.3).

Model	Peak Magnitude (E+I)	Peak time (days)	Epidemic end (days)	Total (I)
(a)	2676.1	21.155	76.6065	9254.8
	2623.9 ± 204.09	21.005 ± 19.9	77.1863 ± 15.59	9542.4 ± 255.37
(b)	1061.5	21.7841	162.3647	7537.5
	1095.6 ± 46.35	21.001 ± 19.99	168.077 ± 31.24	7578.13 ± 460.95
(c)	858.176	18.544	176.8229	7557.9
	902.15 ± 78.85	17.7 ± 15.99	177.07 ± 23.6	7622.2 ± 328.8
(d)	1144.7	23.5169	154.3623	7586.0
	1175.9 ± 175.09	22.01 ± 20.99	157.07 ± 28.005	7643.9 ± 229.2

Table 3.3: Key epidemic measurements for Model (3.5) and ABMC simulations. There are 200 simulations used to calculate the means; $p_3 = 1000$, $I_c = 300$. The ABMC results are $\pm$ standard error. (a) SEIR model, no media; (b) Model with Function (3.1); (c) Model with Function (3.2); (d) Model with Function (3.3). For each of (a)-(d), the top row displays the ODE results and the bottom row displays the ABMC results.

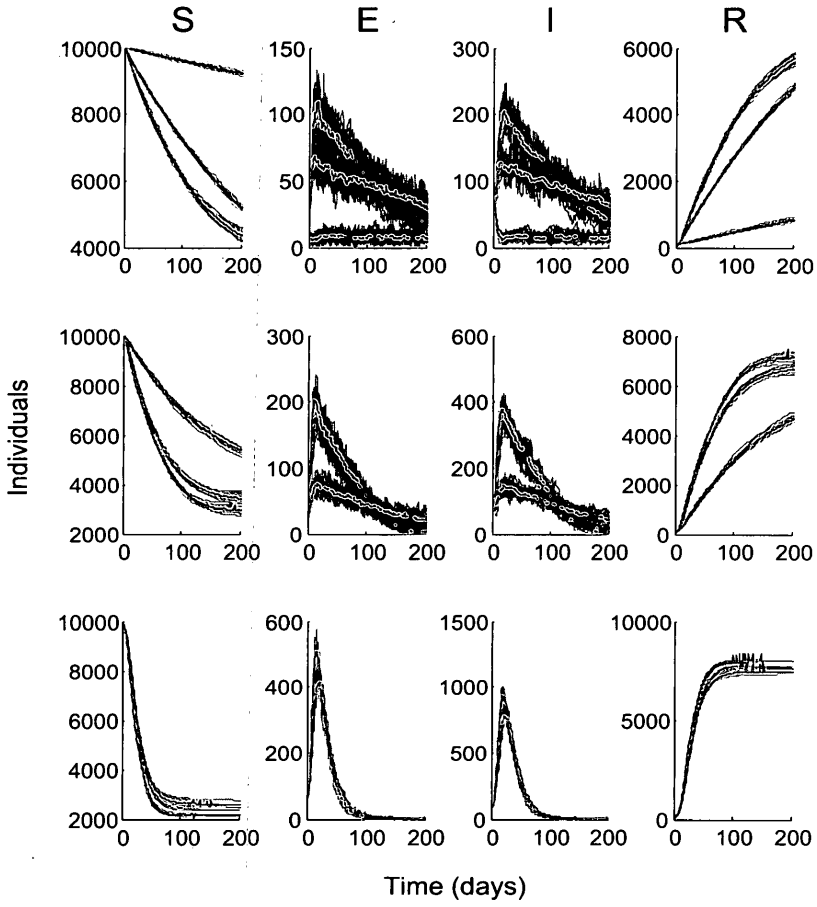

Figure 3.5: ODE and ABMC results for Model (3.5) with media. These are the results for $I_c = 1000$ and 20 simulations. The first row corresponds to $p_3 = 10$, the second row is $p_3 = 100$ and the third row is $p_3 = 1000$. There are three epidemic curves in each figure. The largest epidemic curve corresponds to Function (3.1), the next largest is Function (3.2). The most shallow curve in each case corresponds to Function (3.3). The cyan curves are the ABMC simulations, the black are the means of the ABMC and the red curves are the ODE results. Parameter values are in Table (3.4).

Model	Peak Magnitude (E+I)	Peak time (days)	Epidemic end (days)	Total (I)
(a)	2676.1	21.155	76.6065	9254.8
	2623.9 ± 204.09	21.005 ± 19.9	77.1863 ± 15.59	9542.4 ± 255.37
(b)	1219.6	22.2121	143.8281	7733.3
	1251.9 ± 130.9	21.0014 ± 19.99	142.104 ± 17.25	7836.7 ± 309.71
(c)	1334.9	19.2175	237.9880	8026.6
	1394.6 ± 113.4	19.0013 ± 17.9	131.1089 ± 23.51	8183.2 ± 331.85
(d)	1144.7	23.5169	154.3623	7586.0
	1175.9 ± 175.09	22.0014 ± 20.99	157.069 ± 28.01	7643.9 ± 229.2

Table 3.4: Key epidemic measurements for Model (3.5) and ABMC simulations. There are 200 simulations used to calculate the means; $p_3 = 1000$, $I_c = 1000$. The ABMC results are $\pm$ standard error. (a) SEIR model, no media; (b) Model with Function (3.1); (c) Model with Function (3.2); (d) Model with Function (3.3).

input parameter is treated as a random variable with probability density functions (pdfs) defined for each parameter. The marginal distributions are stratified and a the value of each parameter is chosen randomly. This process is carried out N times. Next a sensitivity analysis can be carried out by calculating the partial rank correlation coefficient (PRCC) for each outcome variable since the PRCC shows which parameters have the largest effect on the specified outcomes. PRCC also allows us to see the independent effects of each parameter even when parameters are correlated. The sign of the PRCC indicates the qualitative relationship between the input and output variables and the magnitude indicates its importance [7].

In order to complete a Latin Hypercube Sampling, partial rank correlation coefficient (LHS-PRCC) sensitivity analysis, we need to specify outcomes that are of interest. For $p_3 = 10$,100 and 1000, for $I_c = 300$ and 1000 and $\gamma = \frac{1}{4}$, we will look at the parameters that have the biggest effect on peak magnitude.

The first set of LHS-PRCC completed is for $\gamma = \frac{1}{4}$,$I_c = 300$ for $p_3 = 10$, $p_3 = 100$ and $p_3 = 1000$, Figure (3.6), with 1000 bins. Similarly, we can look at the same sensitivity analysis results for $I_c = 1000$, in Figure (3.7).

In Figures (3.6-3.7), for the outcome of peak magnitude, we can see that with keeping all of the parameters constant except R_0, σ and N, for $p_3 = 10$, $p_3 = 100$ and $p_3 = 1000$, results in R_0 and N having the most impact on the model. The parameter σ becomes more influential for both values of I_c as p_3 becomes larger.

This is true for all Functions (3.1-3.3).

We are also able to vary all parameters in the model for the sensitivity analysis. Figure (3.8) displays the sensitivity analysis for 1000 bins with all parameters varied, except p_1 and p_2, which are calculated from the results. The parameters p_3 and I_c are varied from 1 to 1000. We look at four key measurements that were outlined in the previous section, peak magnitude, peak time, end of epidemic and total number of infectious individuals.

When varying the parameters from Figure (3.8), we can see that different parameters affect the outcomes based on the function. Peak magnitude for Functions (3.1-3.2), is sensitive to both R_0 and p_3. Epidemic peak time for Function (3.1) is sensitive to R_0 and p_3, whereas peak time for Function (3.2) is sensitive mainly to p_3. Function (3.3) is not greatly influenced by any of the parameters tested. The same parameters that are influential for Function (3.1) for peak magnitude and peak time are influential for epidemic end time. For epidemic end time for Function (3.2), the sensitive parameters are σ, R_0 and γ and Function (3.3) is sensitive σ. Total infectious individuals has similar influential parameters for Functions (3.1-3.3), they are all sensitive to R_0 and σ. Total infectious individuals results from Functions (3.1-3.2) are also sensitive to p_3 and the results for Function (3.3) are sensitive to N. We can see here that depending on the function used to represent the media, different parameters are important for the outcome.

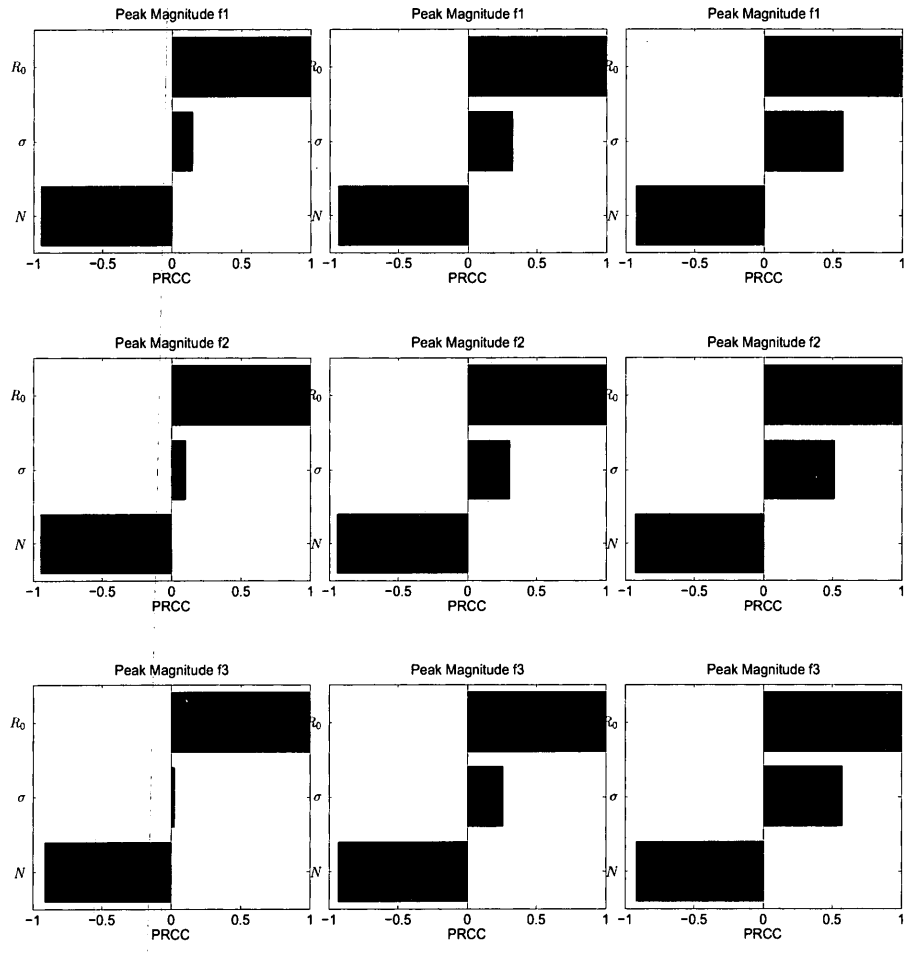

Figure 3.6: LHS-PRCC results for $I_c = 300$. This sensitivity analysis is done with 1000 bins. The rows correspond to Function (3.1), Function (3.2) and Function (3.3), respectively. The columns of this PRCC figure correspond to $p_3 = 10$, $p_3 = 100$ and $p_3 = 1000$. Here $I_c = 300$ and $\gamma = \frac{1}{4}$.

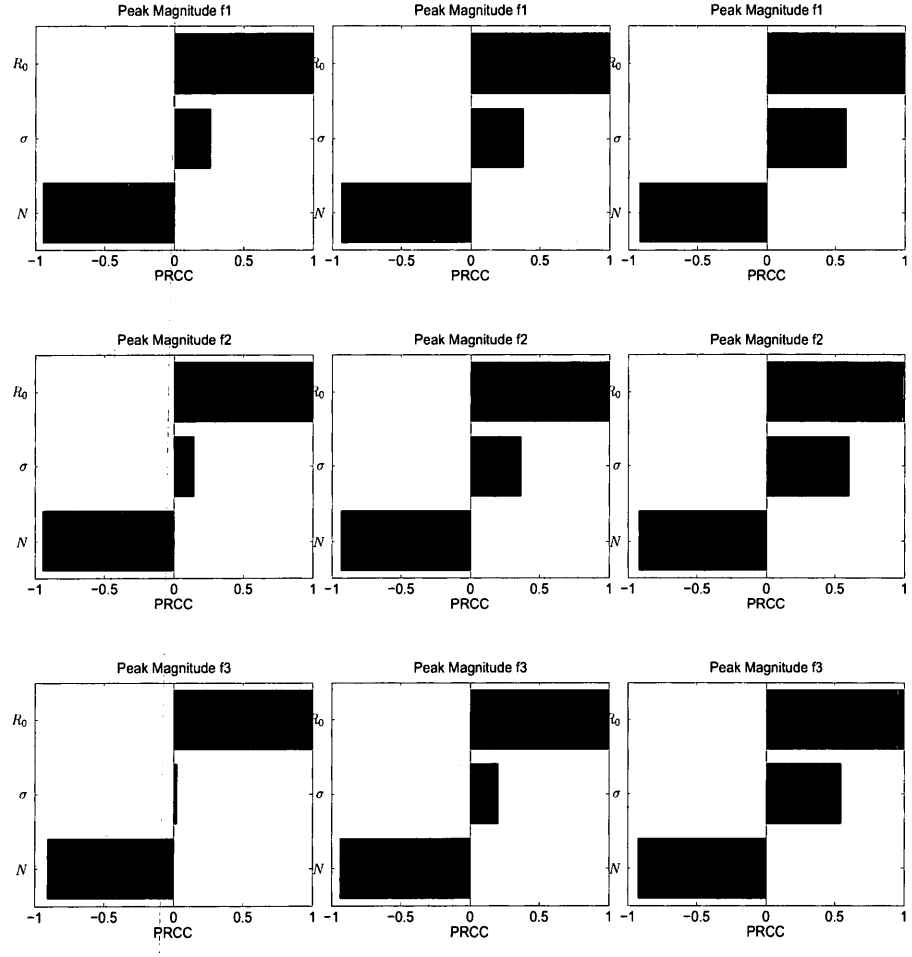

Figure 3.7: LHS-PRCC results for $I_c = 1000$. This sensitivity analysis is done with 1000 bins. The rows correspond to Function (3.1), Function (3.2) and Function (3.3), respectively. The columns of this PRCC figure correspond to $p_3 = 10$, $p_3 = 100$ and $p_3 = 1000$.

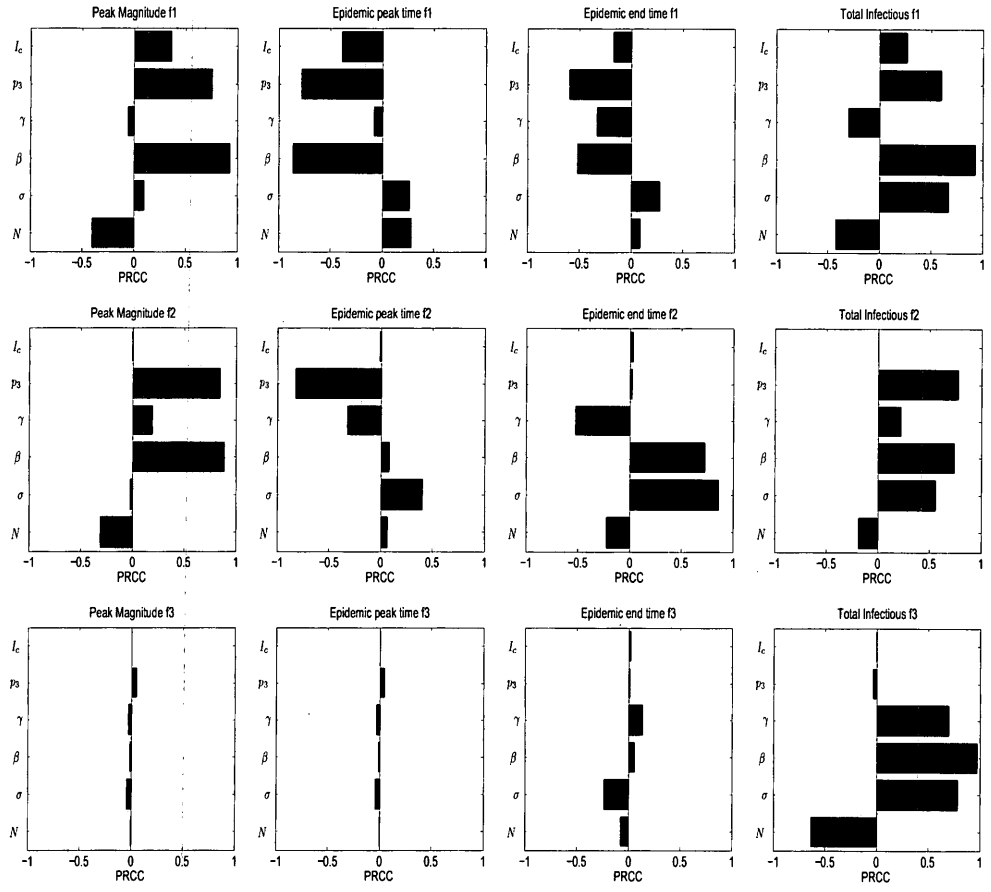

Figure 3.8: LHS-PRCC many parameters varied. This sensitivity analysis is done with 1000 bins. The rows correspond to Function (3.1), Function (3.2) and Function (3.3), respectively. The columns of this PRCC figure to the analysis for the outcomes of importance to public health.

3.4 Discussion

In this chapter we looked at how mass media has been incorporated into disease models. In surveying the literature we found that three main functions have been used, Functions (3.1-3.3). We incorporated these functions into an SEIR model for comparison. ABMC simulations were completed in order to see variation that occurs in an epidemic that is not observable from an ODE model. Sensitivity analysis was performed to investigate which parameters have the largest influence on desired outcomes from each of the functions used.

First we looked at Functions (3.1-3.3) for the transmission term of the epidemic in Model (3.5). We found that the resulting epidemics from Functions (3.1-3.3) could be different for similar values for the media parameters — p_1, p_2, and p_3 — and the same value of infectious individuals at a specific time point I_c, Figures (3.3-3.5).

For each of the resulting epidemics from Functions (3.1-3.3) the media did influence a decrease in severity of the epidemic compared with an epidemic without media influence, Figure (3.3). The key epidemic measurements, Tables (3.3) and (3.4), for each of the media influenced epidemics, Figures (3.3-3.5) were widely varied.

Deterministic models like System (3.5) are able to capture the general trend of an epidemic however they are not able to capture variability in key measurements,

which is important in public health policy and epidemic preparations. Stochastic simulations like the ABMC simulations used here lend themselves well to this. Using an ABMC simulation we were able to determine variability in the key epidemic measurements peak time, peak magnitude, epidemic end time and total number of infectious individuals for each of Functions (3.1-3.3), with different values of p_3 and I_c. As shown in Figures (3.3-3.5) and Tables (3.3) and (3.4), the mean of the epidemic measurements from the ABMC simulations agree with the results from System (3.5) but the magnitude of the standard error can be quite large.

To further our study of media on key epidemic measurements, we conducted a sensitivity analysis by LHS-PRCC on System (3.5) for each of Functions (3.1-3.3). Our initial look consisted of testing the model when the effect of media, p_3, and critical level of infecteds, I_c, were held constant. We found that all epidemic measurements were significantly affected by R_0 and population size, N. When the media parameter p_3 and I_c were allowed to vary, we could not identify parameters that were important in all of the epidemic outcomes. Thus, depending on the media function chosen, different parameters will be important making it difficult to inform public health policy. Therefore, including a decreasing function, such as Functions (3.1-3.3), in the transmission term of an epidemic model may not be the best way to include mass media. Furthermore, determining media parameters $p_1 - p_3$ and I_c from the population may be difficult, and mass media may affect

other terms than disease transmission.

In Chapter 4, we develop a new model where mass media is incorporated into a deterministic model as a separate compartment.

4 Novel mass media inclusion model

4.1 Introduction

Controlling the spread of influenza to reduce the effect of infection on the population is an important practice of public health. Influenza control can be achieved through various strategies including vaccination, the use of drug therapy, and through social distancing practices — removing oneself as much as possible from the population [47, 51]. Vaccination is used preemptively for seasonal influenza and as a preventive measure in pandemic influenza to help control the spread. A vaccine may not always be available. Drug therapy can be used preemptively and during infection, but may also not be available. Social distancing can be practiced at all times to reduce the probability of contracting the infection, and also if infected, to reduce the probability of spreading the disease to others [47].

To better inform individuals regarding preventative measures and risk of infection information regarding vaccination, drug therapies and preventative measures is relayed to the public through mass media campaigns [47]. For example, SARS and H1N1 had vast media coverage [53, 63]. Currently information about the new coronavirus and H7N9 have been the subject of many media reports [1, 52]. Media coverage can influence vaccine uptake [2, 16], drug therapy [22] and social behaviours [33, 54]. It has been reported that the influence of outside information is becoming the critical factor as to whether or not a vaccination campaign will

succeed [12].

A number of media observers have speculated that pervasive media coverage of social problems may lead to desensitization to the media [17, 33, 56]: a diminished emotional responsiveness to a negative or an aversive stimulus after repeated exposure. This same phenomenon can occur with health events i.e., initially, when one hears about an epidemic, one may take precautions against becoming ill. As time passes, however, an individual will become less sensitive to the reports about the epidemic and become lax in the precautions [21, 33, 40, 58]. It is imperative that we better understand how individuals respond to media and uptake precautions, so that in future epidemics/pandemics media messages can be optimized to reduce the effect of diseases on a population.

As outlined in Chapter 2, mathematical models can be employed to study the effects of media in an epidemic [5, 6, 37, 38, 44, 54, 58, 59, 65]. In past studies media has been included as a function which directly affects disease transmission. The structure of these models makes it difficult to incorporate effects such as waning effect of behaviour changes to media reports, or any change in mass media explicitly with respect to time. A further drawback of the previous models is that variability in key epidemic measures to public health can vary drastically depending on what function is chosen to represent mass media in the transmission term, Chapter 3. We propose a new model of disease dynamics that incorporates a media compartment

that allows media to change with respect to time, and media waning. The proposed model is translated to a stochastic model.

In this chapter we consider preventative measures of multiple levels of susceptible individuals S, S_1 and S_2 and vaccination V. In Section 4.2 we derive the proposed models. In Section 4.3 we look at the derivation of a system of stochastic differential equations and a method for moment closure. Section 4.3 also outlines an ABMC method used to capture variability within an epidemic allowing range prediction in key epidemic measurements important to public health such as peak time, peak magnitude, end time and total infectious individuals. Section 4.4 discusses analytical and numerical results, and sensitivity analysis of the model in Section 4.2.

4.2 Models

Traditionally, a deterministic three or four compartmental model has been used to study epidemics. This section will extend a traditional SEIR compartmental model to include vaccination, social distancing and mass media.

4.2.1 SEIR

The new model of disease dynamics that we propose is an extension of a standard SEIR model, Model (4.1), with new equations added for for media (M), S_1, S_2 and

V. As a reminder, the standard SEIR model is, Model (4.1)

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - dS \\
\dot{E} &= \beta SI - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR.
\end{aligned} \tag{4.1}$$

The parameter values for Model (4.1) are in Table (4.1).

4.2.2 SEIR with media and social distancing

We extend the SEIR model, Model (4.1) to include a mass media compartment and a second susceptible class practicing social distancing, Model (4.2),

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - \alpha SM - dS \\
\dot{S}_1 &= -\beta_1 S_1 I + \alpha SM - dS_1 \\
\dot{E} &= \beta SI + \beta_1 S_1 I - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR \\
\dot{M} &= \rho \sigma E.
\end{aligned} \tag{4.2}$$

The media equation (M) influences movement to the socially distanced compartment (S_1) by considering that only a portion of the media reports will affect an individual, (αM). This novel media inclusion allows the media to grow as the

Parameter	Definition	Value	Reference
S(t)	susceptible individuals		
$S_1(t)$	susceptible, socially distanced		
$S_2(t)$	susceptible, further socially distanced		
E(t)	exposed individuals		
I(t)	infectious individuals		
R(t)	recovered individuals		
V(t)	vaccinated individuals		
M(t)	media reports		
λ	birth rate	0.355	[46]
d	natural death rate	$3.52e-005$	[46]
β_i	contact transmission rates	$3.712e-5$	calculated
σ	exposed to infectious	$\frac{1}{2}$	[46]
γ	recovery rate	$\frac{1}{4}$	[46]
α_i	enter socially distanced class $S_i(t)$	0.04, 0.004	
ν_i	vaccination rate from $S_i(t)$	0.002, $1e-5$, $4e-5$	[35]
q_i	relax social distancing rate	0.06, $1e-3$	
ρ	media rate	0.01	
ρ_1	media waning rate	0.015	

Table 4.1: Parameters for the models in Chapter 4. Some parameter values are chosen so that the final vaccination population proportion is between 25% and 40%, [35, 47, 51].

epidemic progresses, can change depending on assumptions and data availability.

In Model (4.2) we must note that although the S_1 population has partially removed itself from the general population, they are still part of the susceptible class, thus are still able to move through the system although the rate of exposure is lower. The parameter values for this model can be found in Table (4.1).

4.2.3 SEIR with vaccination and media

Model (4.2) represents a model of, for example, the first wave of a pandemic, when a vaccine has not yet been produced, or when vaccination is not available. Here, the only method of prevention is social distancing. In subsequent epidemic waves, once a vaccine has been successfully created, a vaccinated class of individuals needs to be included. Once a vaccine has been successfully created, an SEIR model with an equation for vaccination describes the behaviour of the epidemic, Model (4.3). It is assumed that once an individual is vaccinated, they remain in the vaccinated class; i.e., no waning immunity from the vaccinated class and the vaccine is perfectly effective. The parameter for the vaccination rate is (νM), as vaccination is affected

by media.

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - \nu SM - dS \\
\dot{V} &= \nu SM - dV \\
\dot{E} &= \beta SI - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR \\
\dot{M} &= \rho \sigma E.
\end{aligned} \tag{4.3}$$

4.2.4 SS_1EIRM with vaccination

Models (4.2) and (4.3) allow only one effect of media for either social distancing or vaccination. Here, in Model (4.4), we include a media effect with both methods of prevention. Note that, it is likely that individuals will practice multiple prevention measures during an epidemic. Model (4.4) is one that includes a choice to either

practice social distancing or vaccinate during the epidemic.

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - \alpha SM - \nu SM - dS \\
\dot{S}_1 &= -\beta_1 S_1 I + \alpha SM - \nu_1 S_1 M - dS_1 \\
\dot{V} &= \nu SM + \nu_1 S_1 M - dV \\
\dot{E} &= \beta SI + \beta_1 S_1 I - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR \\
\dot{M} &= \rho\sigma E.
\end{aligned} \tag{4.4}$$

4.2.5 Multiple levels of social distancing

It is possible that individuals will practice social distancing with varying degrees. We now introduce an S_2 class and assume that S_1 slightly limits regular activities and S_2 limits regular activities as much as possible and may even exclude themselves from the population. It can also be considered that individuals in S_2 have great fears about becoming vaccinated until a time when threat of illness becomes great

and they may then choose to vaccinate. This behaviour is explained by Model (4.5).

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - \alpha SM - \nu SM - dS \\
\dot{S}_1 &= -\beta_1 S_1 I + \alpha SM - \alpha_1 S_1 M - \nu_1 S_1 M + q_2 S_2 - dS_1 \\
\dot{S}_2 &= \alpha_1 S_1 M - q_2 S_2 - \nu_2 S_2 M - dS_2 \\
\dot{V} &= \nu SM + \nu_1 S_1 M + \nu_2 S_2 M - dV \\
\dot{E} &= \beta SI + \beta_1 S_1 I - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR \\
\dot{M} &= \rho\sigma E.
\end{aligned} \tag{4.5}$$

The parameters for this model are explained in Table (4.1).

4.2.6 Desensitization to media

As previously mentioned, desensitization to media can occur [33, 31]. Thus, if the public becomes bored with reporting of the epidemic, disease outcome will be affected. It is also thought that the public does not fully believe messages from the media [17, 61]. A term that incorporates this boredom factor can be seen as a constant media 'death' term, (ρ_1) included in the media equation. This model also allows individuals to relax social distancing practices from both socially distanced classes (q_1) and (q_2). The models for the boredom with the media are

Models (4.6,4.7) with parameters in Table (4.1),

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - \alpha SM - \nu SM - dS \\
\dot{S}_1 &= -\beta_1 S_1 I + \alpha SM - \alpha_1 S_1 M - \nu_1 S_1 M - dS_1 \\
\dot{V} &= \nu SM + \nu_1 S_1 M - dV \\
\dot{E} &= \beta SI + \beta_1 S_1 I - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR \\
\dot{M} &= \rho\sigma E - \rho_1 M,
\end{aligned} \tag{4.6}$$

and

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - \alpha SM - \nu SM + q_1 S_1 - dS \\
\dot{S}_1 &= -\beta_1 S_1 I + \alpha SM - \alpha_1 S_1 M - \nu_1 S_1 M + q_2 S_2 - dS_1 - q_1 S_1 \\
\dot{S}_2 &= \alpha_1 S_1 M - q_2 S_2 - \nu_2 S_2 M - dS_2 \\
\dot{V} &= \nu SM + \nu_1 S_1 M + \nu_2 S2M - dV \\
\dot{E} &= \beta SI + \beta_1 S_1 I - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR \\
\dot{M} &= \rho\sigma E - \rho_1 M.
\end{aligned} \tag{4.7}$$

4.3 Stochastic differential equations

A stochastic differential equation model is derived for each of the proposed models herein. As an example, we look at the stochastic differential equation model for a standard SEIR model, derived as in [4].

Following the derivation for the stochastic differential equation model outlined in Chapter 1.4.3.2, we obtain a system of stochastic differential equations for Model (4.7, Model (4.8). The systems of stochastic differential equations for the other models presented in this chapter can be seen in Appendix B.

$$
\begin{aligned}
\frac{dE[S]}{dt} &= \lambda - \beta E[SI] - \alpha E[SM] - \nu E[SM] - dE[S] + q_1 E[S_1] \\
\frac{dE[S_1]}{dt} &= -\beta_1 E[S_1 I] - \nu_1 E[S_1 M] - \alpha_1 E[S_1 M] - \alpha E[SM] \\
&\quad - dE[S_1] + q_2 E[S_2] - q_1 E[S_1] \\
\frac{dE[S_2]}{dt} &= \alpha_1 E[S_1 M] - \nu_2 E[S_2 M] - q_2 E[S_2] - dE[S_2] \\
\frac{dE[V]}{dt} &= \nu E[SM] + \nu_1 E[S_1 M] + \nu_2 E[S_2 M] - dE[V] \\
\frac{dE[E]}{dt} &= \beta E[SI] + \beta_1 E[S_1 I] - \sigma E[E] - dE[E] \\
\frac{dE[I]}{dt} &= \sigma E[E] - \gamma E[I] - dE[I] \\
\frac{dE[R]}{dt} &= \gamma E[I] - dE[R] \\
\frac{dE[M]}{dt} &= \rho\sigma E[E] - \rho_1 E[M]
\end{aligned}
$$

$$\frac{dE[S^2]}{dt} = 2\lambda E[S] - 2\beta E[SIS] - 2\alpha E[SMS] - 2\nu E[SMS] - 2dE[SS]$$
$$+ \lambda + \beta E[SI] + \alpha E[SM] + \nu E[SM] + dE[S] + 2q_1 E[SS_1]$$
$$+ q_1 E[S_1]$$
$$\frac{dE[S_1^2]}{dt} = \alpha E[SM] + 2\alpha E[SMS_1] + \beta_1 E[S_1 I] - 2\beta_1 E[S_1 I S_1] + \alpha_1 E[S_1 M]$$
$$- 2\alpha_1 E[S_1 M S_1] + \nu_1 E[S_1 M] - 2\nu_1 E[S_1 M S_1] + 2q_2 E[S_1 S_2]$$
$$+ q_2 E[S_2] - 2q_1 E[S_1^2] + dE[S_1] - 2dE[S_1 S_1]$$
$$\frac{dE[S_2^2]}{dt} = dE[S_2] + \alpha_1 E[S_1 M] + qE[S_2] - 2dE[S_2^2] - 2qE[S_2^2]$$
$$+ 2\alpha_1 E[S_1 M S_2] + \nu_2 E[S_2 M] - 2\nu_2 E[S_2 M S_2]$$
$$\frac{dE[E^2]}{dt} = 2\beta E[SEI] + 2\beta_1 E[EIS_1] - 2\sigma E[EE] - 2dE[EE] + \sigma E[E]$$
$$+ dE[E] + \beta_1 E[S_1 I] + \beta E[SI]$$
$$\frac{dE[I^2]}{dt} = \sigma E[E] + 2\sigma E[EI] + \gamma E[I] - 2\gamma E[I^2] + dE[I] - 2dE[I^2]$$
$$\frac{dE[SI]}{dt} = \lambda E[I] - \beta E[I^2 S] - \alpha E[SIM] - \nu E[SIM] - 2dE[SI] + \sigma E[SE]$$
$$- \gamma E[SI] + q_1 E[S_1 I]$$
$$\frac{dE[S_1 I]}{dt} = q_2 E[S_2 I] + \alpha E[SIM] - \beta_1 E[I^2 S_1] - \alpha_1 E[IMS_1] - \nu_1 E[IMS_1]$$
$$- 2dE[S_1 I] + \sigma E[S_1 E] - \gamma E[S_1 I] - q_1 E[S_1 I]$$
$$\frac{dE[EI]}{dt} = \beta E[I^2 S] + \beta_1 E[I^2 S_1] - \sigma E[E] - \sigma E[EI] + \sigma E[E^2] - 2dE[EI]$$
$$- \gamma E[EI] \tag{4.8}$$
$$\frac{dE[SE]}{dt} = \lambda E[E] - \beta E[SI] - \beta E[SEI] + \beta E[SIS] - \alpha E[MES]$$

$$- \nu E[MES] - 2dE[SE] + \beta_1 E[SIS_1] - \sigma E[SE] + q_1 E[S_1 E]$$

$$\frac{dE[S_1E]}{dt} = \beta E[SIS_1] + \alpha E[MES] - \beta_1 E[S_1 I] - \beta_1 E[EIS_1] + \beta_1 E[S_1 IS_1]$$

$$- 2dE[S_1E] - \sigma E[S_1E] - \nu_1 E[MES_1] + q_2 E[S_2E]$$

$$- \alpha_1 E[MES_1] - q_1 E[S_1E]$$

$$\frac{dE[SM]}{dt} = \lambda E[M] - \beta E[SIM] - \alpha E[SM^2] - \nu E[SM^2] - dE[SM]$$

$$+ \rho\sigma E[SE] - \rho_1 E[SM] + q_1 E[S_1 M]$$

$$\frac{dE[S_1M]}{dt} = \alpha E[SM^2] - \beta_1 E[IMS_1] - \nu_1 E[S_1 MM] - dE[S_1M]$$

$$+ \rho\sigma E[S_1E] - \alpha_1 E[S_1M^2] + qE[S_2M] - \rho_1 E[S_1M] - q_1 E[S_1M]$$

$$\frac{dE[IM]}{dt} = \sigma E[ME] - \gamma E[IM] - dE[IM] + \rho\sigma E[EI] - \rho_1 E[IM]$$

$$\frac{dE[SS_1]}{dt} = \lambda E[S_1] - \beta E[SIS_1] - \alpha E[SM] - \alpha E[SMS_1] + \alpha E[SMS]$$

$$- \alpha_1 E[SMS_1] - \nu E[SMS_1] - \nu_1 E[SMS_1] - 2dE[SS_1]$$

$$- \beta_1 E[SIS_1] + q_2 E[S_2S] - q_1 E[SS_1] - q_1 E[S_1] + q_1 E[S_1^2]$$

$$\frac{dE[ME]}{dt} = \beta E[SIM] + \beta_1 E[IMS_1] - \sigma E[ME] - dE[ME]$$

$$+ \rho\sigma E[E^2] - \rho_1 E[MY]$$

$$\frac{dE[M^2]}{dt} = \rho\sigma E[E] + 2\rho\sigma E[ME] + \rho_1 E[M] - 2\rho_1 E[M^2]$$

$$\frac{dE[S_2M]}{dt} = \alpha_1 E[S_1M^2] - qE[S_2M] - \nu_2 E[S_2M^2] - dE[S_2M]$$

$$+ \rho\sigma E[S_2E] - \rho_1 E[S_2M]$$

$$\frac{dE[S_2E]}{dt} = \alpha_1 E[MES_1] - q_2 E[S_2E] - 2dE[S_2E] - \sigma E[S_2E] + \beta E[SIS_2]$$

$$
\begin{aligned}
&+ \beta_1 E[S_1 I S_2] - \nu_2 E[S_2 M^2] \\
\frac{dE[S_2 I]}{dt} &= \alpha_1 E[IMS_1] - q_2 E[S_2 I] - 2dE[S_2 I] + \sigma E[S_2 E] - \gamma E[S_2 I] \\
&- \nu_2 E[IMS_2] \\
\frac{dE[S_2 S]}{dt} &= -q_2 E[S_2 S] + \lambda E[S_2] - \beta E[SIS_2] - \alpha E[SMS_2] - \nu E[SMS_2] \\
&- 2dE[S_2 S] + \alpha_1 E[SMS_1] - \nu_2 E[SMS_2] + q_1 E[S_1 S] \\
\frac{dE[S_1 S_2]}{dt} &= -q_2 E[S_1 S_2] + \alpha_1 E[S_1 MS_1] - q_2 E[S_2] + q_2 E[S_2 S_2] - \beta_1 E[S_1 I S_2] \\
&- \alpha_1 E[S_1 M] - 2dE[S_1 S_2] - \alpha_1 E[S_1 MS_2] - \nu_1 E[S_1 MS_2] \\
&+ \alpha E[SMS_2] - \nu_2 E[S_1 MS_2] - q_1 E[S_1 S_2].
\end{aligned}
$$

4.3.1 Agent-based Monte Carlo simulations

In addition to the system of stochastic differential equations that has been derived for each of the above models, an agent-based Monte Carlo simulation was carried out in order to look at the variability that is present during an epidemic and obtain key measurements that are important to public health: peak magnitude, peak time, epidemic end time and total number of infectious individuals.

Recall that as an Agent-Based Monte Carlo (ABMC) simulation moves forward not in regular time intervals but following event times: the next time that an individual changes state within the system. To compare to a system of ordinary differential equations (ODEs) we must assume exponential distributions for all pa-

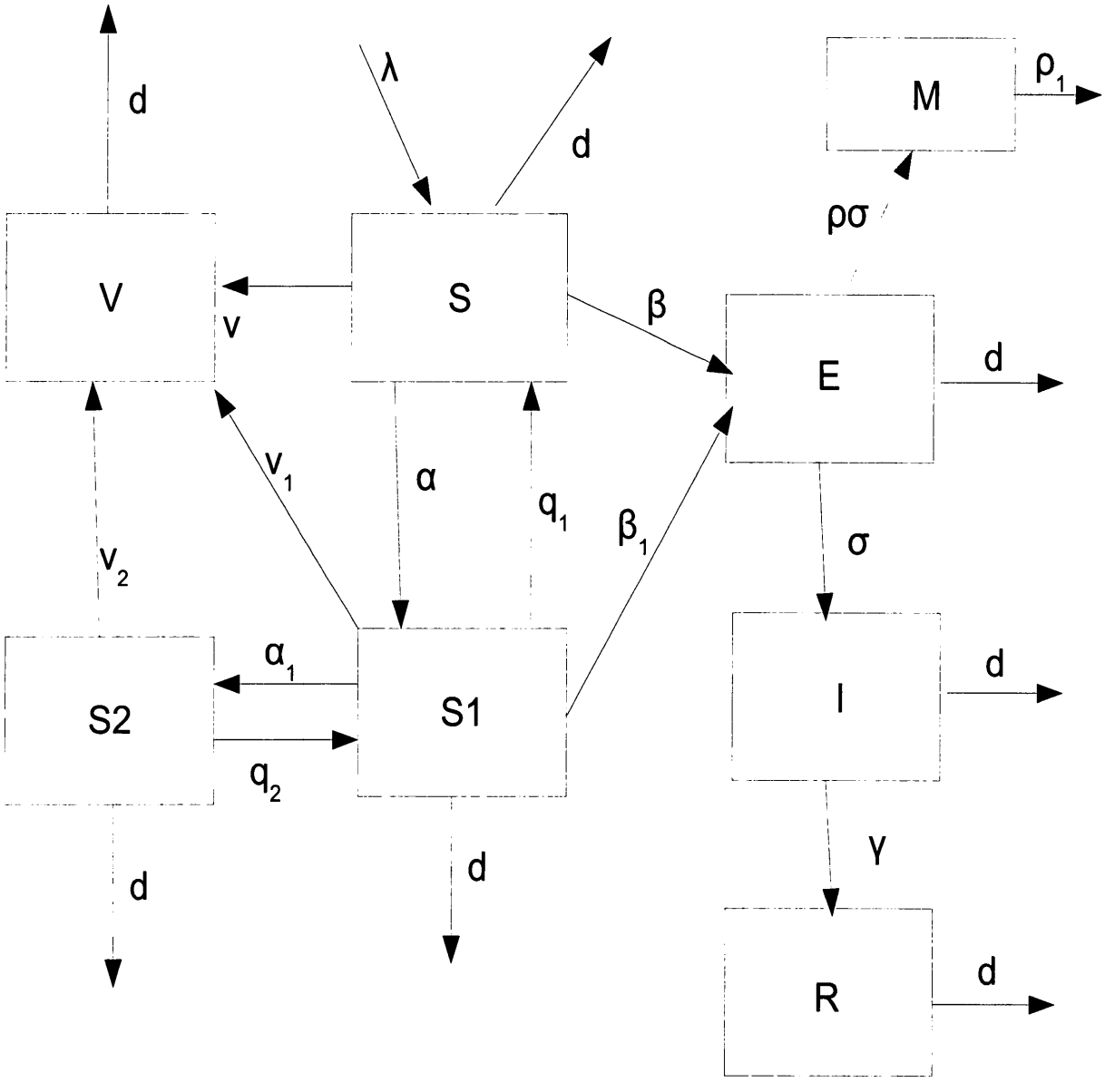

Figure 4.1: Event diagram of Model (4.7) for the ABMC. Parameters are the inverse of the values in Table (4.1).

rameters. Here we assume the lifetime distributions with means of the inverse of the parameter values from Table (4.1).

At each event time, the underlying exponential distributions dictated by the use of ordinary differential equation models associated with each individual are examined to determine the outcome of the event and which individual moves within the system to which state [25]. In Figure (4.1), we can see the progression of an individual through the epidemic.

Agents in each of the susceptible, exposed, infectious and recovered compartments are assigned event times corresponding to Table (4.1), for each event that allows an individual from that compartment to change state. Let us assume that this is the case for an agent in the infectious class. Now the event times within the infectious compartment are compared. There are event times corresponding to recovery and infecting a susceptible agent. After the event times are ordered from smallest to largest in the infectious compartment, the event with the lowest time is the event which occurs. Once the event has occurred, the process is repeated until a stop condition is reached. The next event that will occur corresponds to the mean of all times in the space.

4.4 Results

4.4.1 Basic Reproductive Ratio

The basic reproductive ratio, R_0, of an epidemic model states the local stability of the model. If $R_0 < 1$, the system is locally stable and the disease will die out, if $R_0 > 1$, the system is locally unstable and the illness will persist. To find the basic reproductive ratio for Model (4.7), we use the next generation method as discussed in Chapter 1.4.2.

The equations that are needed for R_0 calculation here are:

$$\begin{aligned} \dot{E} &= \beta SI + \beta_1 S_1 I - \sigma E - dE \\ \dot{I} &= \sigma E - \gamma I - dI. \end{aligned} \tag{4.9}$$

After taking partial derivatives, the matrix that contains the creation of new infections, F is:

$$F = \begin{pmatrix} 0 & \beta S + \beta_1 S_1 \\ 0 & 0 \end{pmatrix}. \tag{4.10}$$

The disease free equilibrium of Model (4.7), is $E_0 = (\frac{\lambda}{d}, 0, 0, 0, 0, 0, 0, 0)$, where λ is the birth rate and d is the death rate. After evaluating matrix F at the disease free equilibrium, it becomes

$$F = \begin{pmatrix} 0 & \beta\frac{\lambda}{d} \\ 0 & 0 \end{pmatrix}. \tag{4.11}$$

The second part of the next generation operator is the matrix V. In this case, the matrix is:

$$V = \begin{pmatrix} \sigma + d & 0 \\ -\sigma & \gamma + d \end{pmatrix}. \tag{4.12}$$

After inverting matrix V and multiplying, we get the next generation operator:

$$FV^{-1} = \begin{pmatrix} \frac{\beta\frac{\lambda}{d}\sigma}{(\sigma+d)(\gamma+d)} & \frac{\beta\frac{\lambda}{d}}{(\gamma+d)} \\ 0 & 0 \end{pmatrix}. \tag{4.13}$$

The spectral radius of the operator, which is R_0 is

$$R_0 = \frac{\beta S_0 \sigma}{(\sigma + d)(\gamma + d)}. \tag{4.14}$$

The basic reproductive ratio, R_0, for each of the models presented in this chapter can be calculated in the same way as for Model (4.7). Here $S_0 = \frac{\lambda}{d}$ for the ODE and SDE models. The calculations for the other models result in the same R_0 as Model (4.7).

4.4.2 Stability Analysis

In disease modelling, an epidemic occurs when $R_0 > 1$ and an epidemic will not occur if $R_0 < 1$. This threshold for R_0 corresponds to positive and negative eigenvalues of the corresponding Jacobian matrix. When the eigenvalues are positive, an epidemic will occur and when eigenvalues are negative there will be no epidemic.

This analysis will be completed for Model (4.7), but similar results can be obtained for the other models herein.

The Jacobian matrix for Model (4.7) is

$$\begin{bmatrix} -\beta Y - \alpha M - \nu M - d & q_1 & 0 & 0 & -\beta S & 0 & -\alpha S - \nu S & 0 \\ -\beta_1 I + \alpha M & -\alpha_1 M - \nu_1 M - q_1 - d & q_2 & 0 & -\beta_1 S & 0 & \alpha S - \alpha_1 S_1 - \nu_1 S_1 & 0 \\ 0 & \alpha_1 M & -\nu_2 M - d - q_2 & 0 & 0 & 0 & \alpha_1 S_1 - \nu_2 S_2 & 0 \\ \beta I & \beta I & 0 & -\sigma - d & \beta S + \beta S_1 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma & -\gamma - d & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & -d & 0 & 0 \\ 0 & 0 & 0 & \rho\sigma & 0 & 0 & -\rho_1 & 0 \\ \nu M & \nu_1 M & \nu_2 M & 0 & 0 & 0 & \nu S + \nu_1 S_1 + \nu_2 S_2 & -d \end{bmatrix}. \tag{4.15}$$

After evaluating Matrix (4.15) at the uninfected equilibrium, we get Matrix (4.16),

$$\begin{bmatrix} -d & q_1 & 0 & 0 & -\frac{\beta\lambda}{d} & 0 & -\frac{\alpha\lambda}{d} - \frac{\nu\lambda}{d} & 0 \\ 0 & -q_1 - d & q_2 & 0 & -\frac{\beta_1\lambda}{d} & 0 & \frac{\alpha\lambda}{d} & 0 \\ 0 & 0 & -d - q_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\sigma - d & \frac{\beta\lambda}{d} & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma & -\gamma - d & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & -d & 0 & 0 \\ 0 & 0 & 0 & \rho\sigma & 0 & 0 & -\rho_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\nu\lambda}{d} & -d \end{bmatrix}. \tag{4.16}$$

Since we evaluate Matrix (4.15) at the uninfected equilibrium, all terms go to zero

except those that are multiplied with (S). Because everything goes to zero, the stability analysis for the other, simpler models in this chapter will obtain the same results as the analysis for Model (4.7). From here we obtain the eigenvalues of the system:

$$\begin{aligned} \lambda_{1,2,3} &= -d,\ \lambda_4 = -(q_1 - d),\ \lambda_5 = -(q_2 - d),\ \lambda_6 = -\rho_1, \\ \lambda_{7,8} &= \frac{-\sigma d - 2d^2 - d\gamma \pm \sqrt{\sigma^2 d^2 - 2\sigma d^2\gamma + d^2\gamma^2 + 4d\sigma\beta\lambda}}{2d}, \end{aligned} \tag{4.17}$$

where $q_1, q_2 >> d$. For some $\lambda_i > 0$ we can show that $R_0 > 1$, which will give an unstable equilibrium implying an epidemic will occur. For $\lambda_7 > 0$ we have

$$\frac{-\sigma d - 2d^2 - d\gamma \pm \sqrt{\sigma^2 d^2 - 2\sigma d^2\gamma + d^2\gamma^2 + 4d\sigma\beta\lambda}}{2d} > 0, \tag{4.18}$$

which can be rearranged to get

$$\begin{aligned} \frac{\sigma\beta\lambda}{\sigma\gamma d + \sigma d^2 + \gamma d^2 + d^3} &> 1 \\ \frac{\sigma\beta\lambda}{d(\sigma + d)(\gamma + d)} &> 1 \\ R_0 &> 1. \end{aligned} \tag{4.19}$$

From Expression (4.19) we can see that if some of the eigenvalues from Matrix (4.16) are greater than zero, $\lambda_i > 0$, then we get an epidemic, $R_0 > 1$.

4.4.3 Comparison of ODE to SDE

The results for the first order moments and some of the second order moments can be seen in Figures (4.2) and (4.3). A similar figure can be produced for all of the

second order moments for each system herein. We can see in Figure (4.2) that the first order moment equation results, the mean behaviour of the ODEs, are very similar to the ODE results. Many of the second order moment equations go to zero. In Figure (4.3), we can see the second order moments that are not zero at the end of the epidemic. Since the ODE and SDE results are similar in Figure (4.2), the nonzero second order moments have limited influence on the behaviour of the epidemic.

4.4.4 Variability within an epidemic

The results for the models in Section 4.2 along with the ABMC can be seen in the following figures. The ABMC results display variability in an epidemic that is not seen from the ODE results alone. Figure (4.4) displays the results of Model (4.1) with the corresponding ABMC results. Figure (4.5) displays the results of Models (4.2, 4.3, 4.4, 4.5) and Figure (4.6) displays the results of Models (4.6, 4.7) with the ABMC results. The SDE results are also presented in these figures but as discussed in Section 4.4.3, there is little difference between the ODE and the SDE first order moment results. As we increase the preventative measures during the pandemic, the peak time, peak magnitude and length of the epidemic decrease. Social distancing and vaccine decrease the total number of infected individuals and when both social distancing and vaccination are included as prevention measures

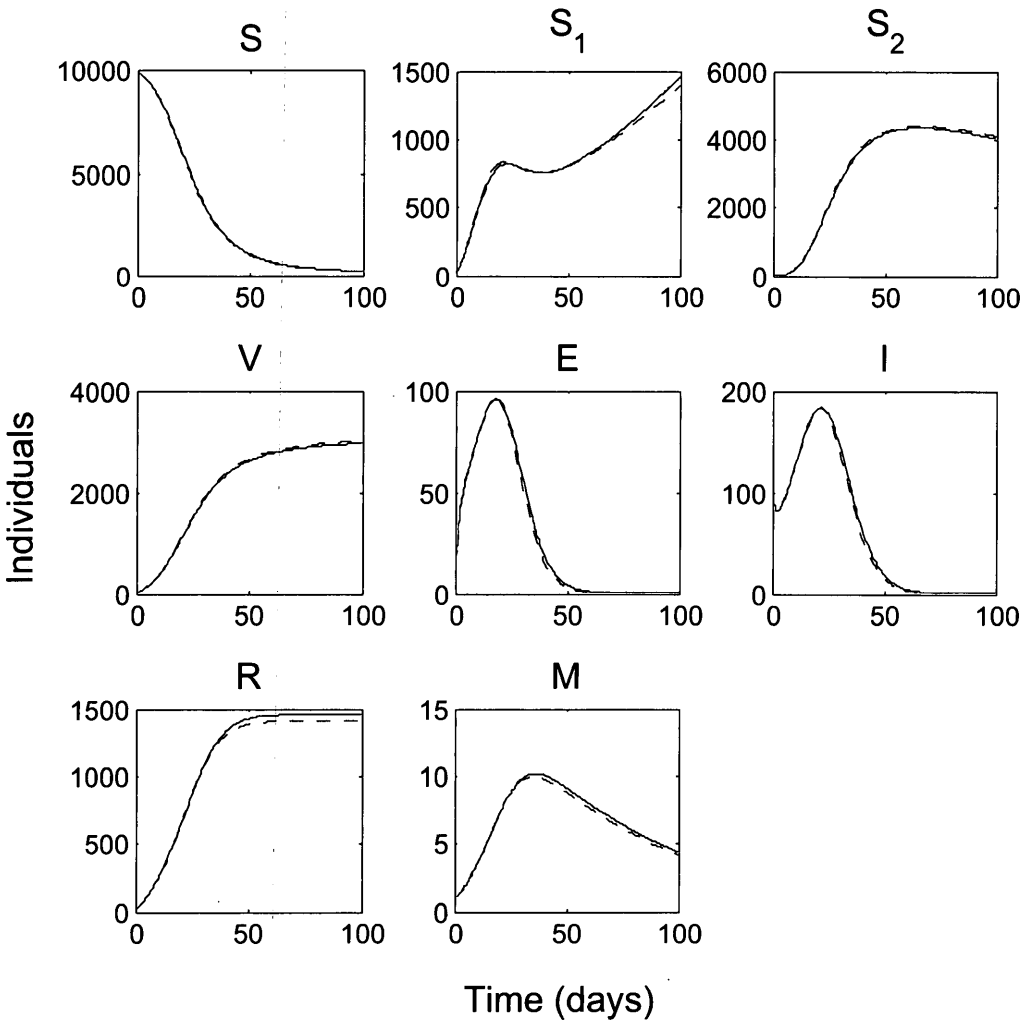

Figure 4.2: First order moments of Model (4.8) and Model (4.7). Both models result in the same epidemic.

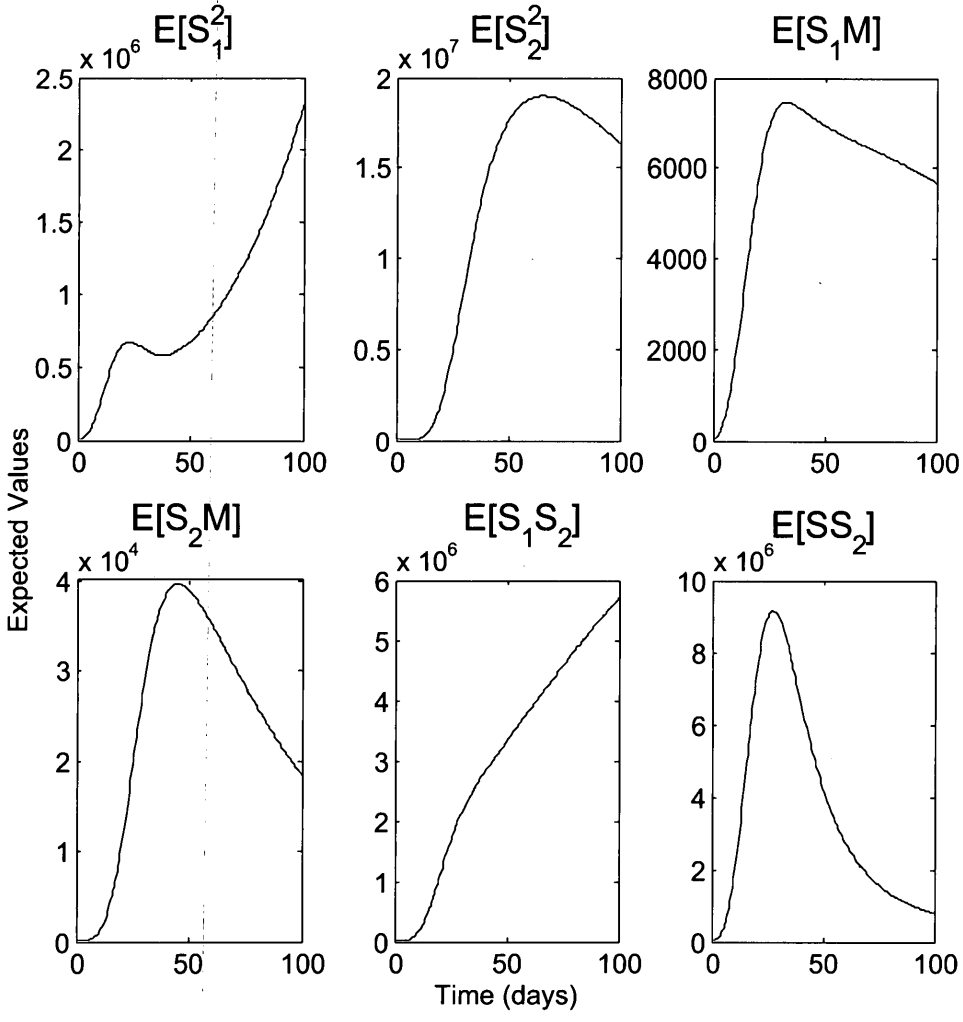

Figure 4.3: The non-zero second order moments from Model (4.8).

the total number of infectious individuals decreases further. When media waning is included the total number of vaccinated individuals decreases. The numerical results are in Table (4.2).

The results in Table (4.2) demonstrate that with the inclusion of mass media and more intervention measures, the size of the key epidemic measurements decrease implying that the effect of the epidemic on the population is less with more intervention measures and a mass media campaign. However, when a boredom factor is included in mass media, the effect of the epidemic on the population is slightly larger and the number of individuals who receive a vaccine decreases.

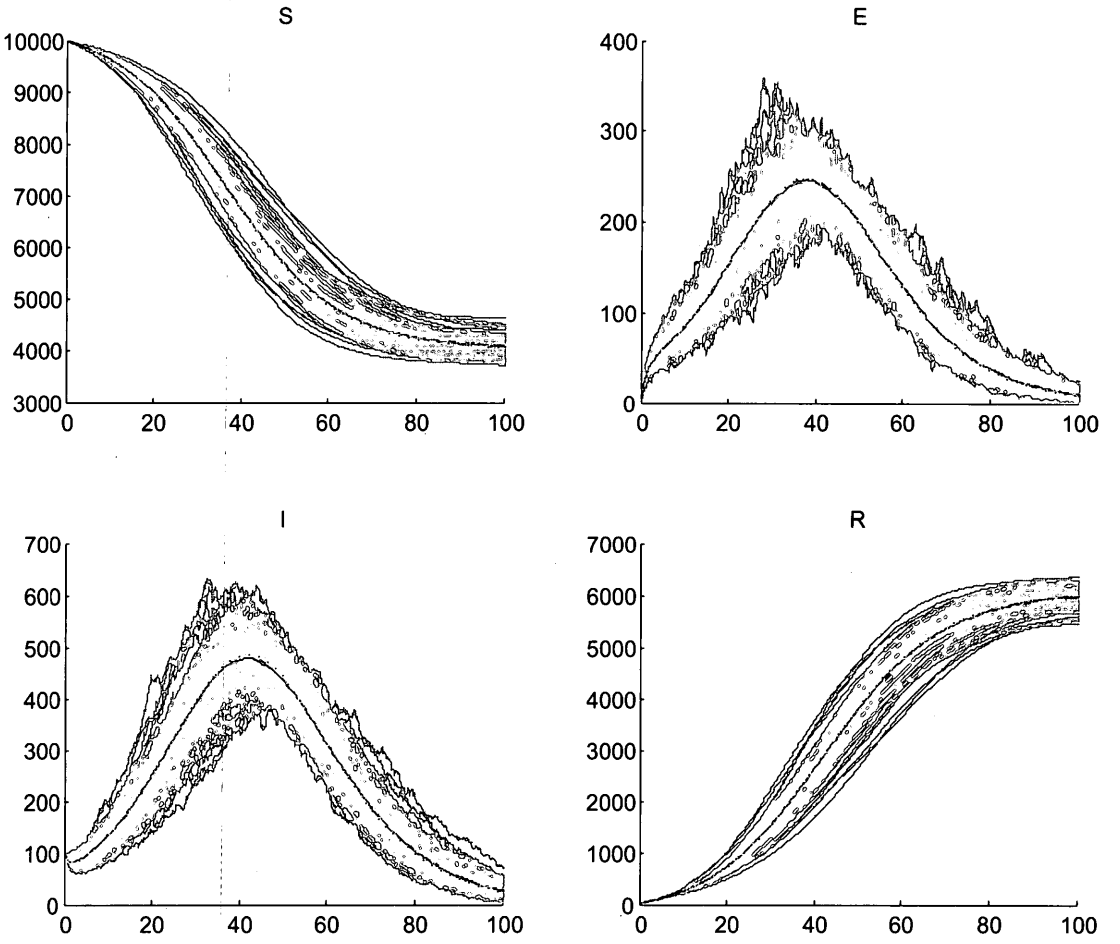

Figure 4.4: ODE and ABMC results for Model (4.1). Parameters can be found in Table (4.1).

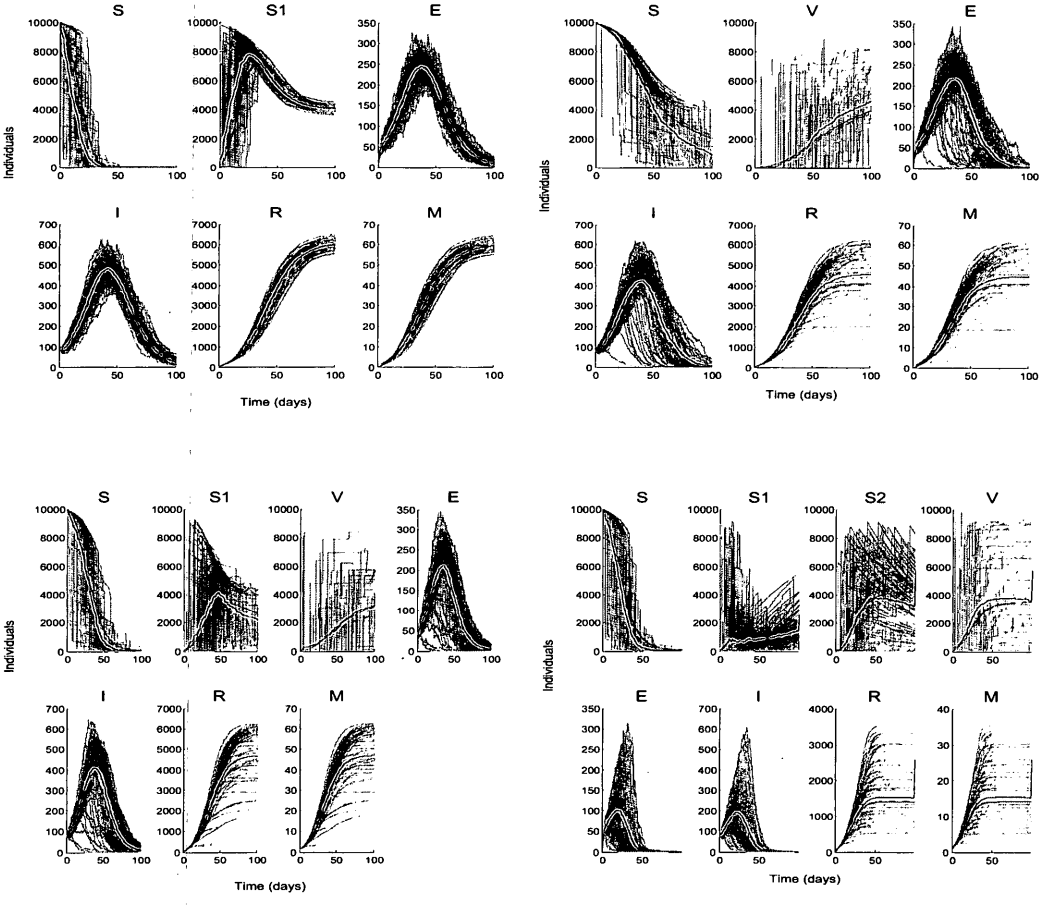

Figure 4.5: ODE and ABMC results for Models (4.2), (4.3), (4.4), (4.5). The red solid line is the ODE, the red dashed line is the SDE, the black line is the mean of the ABMC and the cyan lines are the ABMC results. Parameters can be found in Table (4.1).

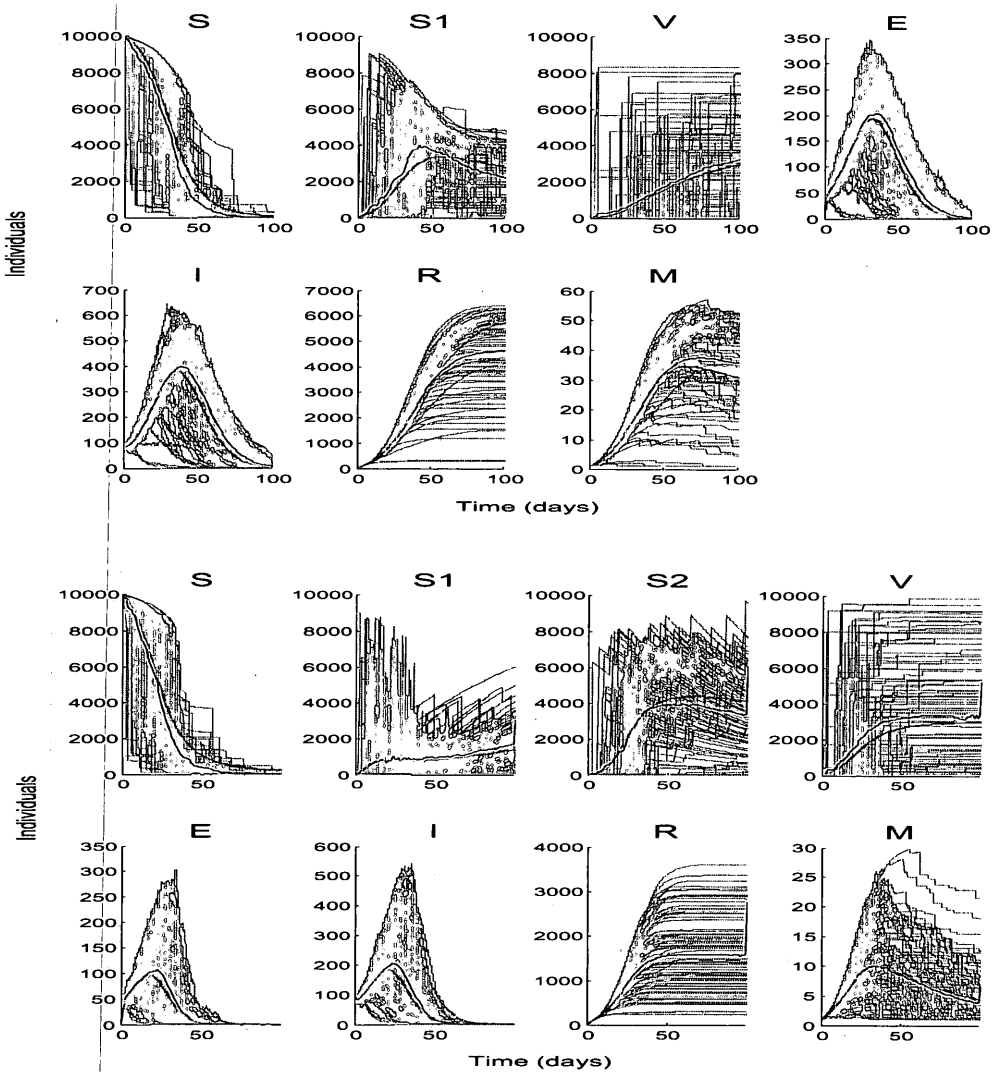

Figure 4.6: ODE and ABMC results for Models (4.6) and (4.7). The red solid line is the ODE, the red dashed line is the SDE, the black line is the mean of the ABMC and the cyan lines are the ABMC results. Parameters can be found in Table (4.1).

4.4.5 Sensitivity Analysis

In order to determine which parameters play the greatest role in various model outcomes of interest for Model (4.7), sensitivity analyses, Latin Hypercube Sampling (LHS) and Partial Rank Correlation Coefficient (PRCC), are performed. These methods are said to be the most efficient methods for performing sensitivity analysis with variation in multiple parameters simultaneously [7].

The outcomes that are of interest for Model (4.7) include the peak time of the epidemic, the end time of the epidemic, the peak magnitude of infectious and exposed individuals and the total number of infectious individuals. These outcomes are important measurements for public health as they provide estimates of the amount of care that may be required during the pandemic. Sensitivity analysis for each of the models in this chapter can be completed with similar outcomes.

The sensitivity analysis shows that each outcome has different influential parameters, however, R_0 is significant for each of the outcomes of interest. Three of the outcomes, peak magnitude, epidemic end time and total number of infectious individuals are sensitive to changes in one of the media parameters, ρ for peak magnitude and total infectious and ρ_1 for epidemic end time.

Model	Peak Time (days)	Peak Magnitude (I)	Epidemic end (day)	Total (I)	Total (V)
(a)	40.4959	730.6339	149.0844	6066.5	N/A
	40.10 ± 38.1	719.57 ± 234.43	135.98±83.03	6061.2 ± 81.77	N/A
(b)	40.82	704.03	151.81	5967	N/A
	41.6 ± 39.5	722.6 ± 17.34	187.5 ± 64.90	5838.23 ± 471.67	N/A
(c)	35.53	573.47	105.34	4126.1	4079.94
	38.78 ± 36.68	638.8 ± 148.2	113.43±16.04	4568.7 ± 781.29	4407.7±449.2
(d)	35.50	566.38	112.67	4183.6	3983.4
	36.8 ± 34.7	663.34 ± 204.32	113.2 ± 8.77	4313.2 ± 321.6	3874.5±220.5
(e)	35.52	570.97	115.06	4261.7	3370.5
	38.795±36.68	599.62 ± 211.38	126.63±30.42	4644.93 ± 694.16	3520.1±495.1
(f)	19.71	275.03	66.73	1402.48	3524.66
	22.236 ± 20.6	293.3 ± 57.9	61.82 ± 8.13	1536.7 ± 441.43	3811.6±435.8
(g)	20.11	275.98	67.72	1404.18	3008.3
	23.13 ± 21.01	311.25 ± 55.78	64.99 ± 7.58	1586.3 ± 409.87	3418.9±615.5

Table 4.2: Key epidemic measurements for ODEs and ABMC simulations. 100 ABMC simulations. The first row — ODE results,the second row — the ABMC mean ± standard error. (a) Results for an SEIR model; (b)Model (4.2) — SS_1EIRM, ODE and ABMC; (c)Model (4.3); (d) Model (4.4) — SS_1VEIRM, vaccinate S and S_1, ODE and ABMC; (e) Model (4.6) — waning media; (f)Model (4.5) — SS_1S_2VEIRM, vaccinate S, S_1 and S_2, ODE and ABMC; (g)Model (4.7): SS_1S_2VEIRM, vaccinate S, S_1 and S_2, waning media, ODE and ABMC.

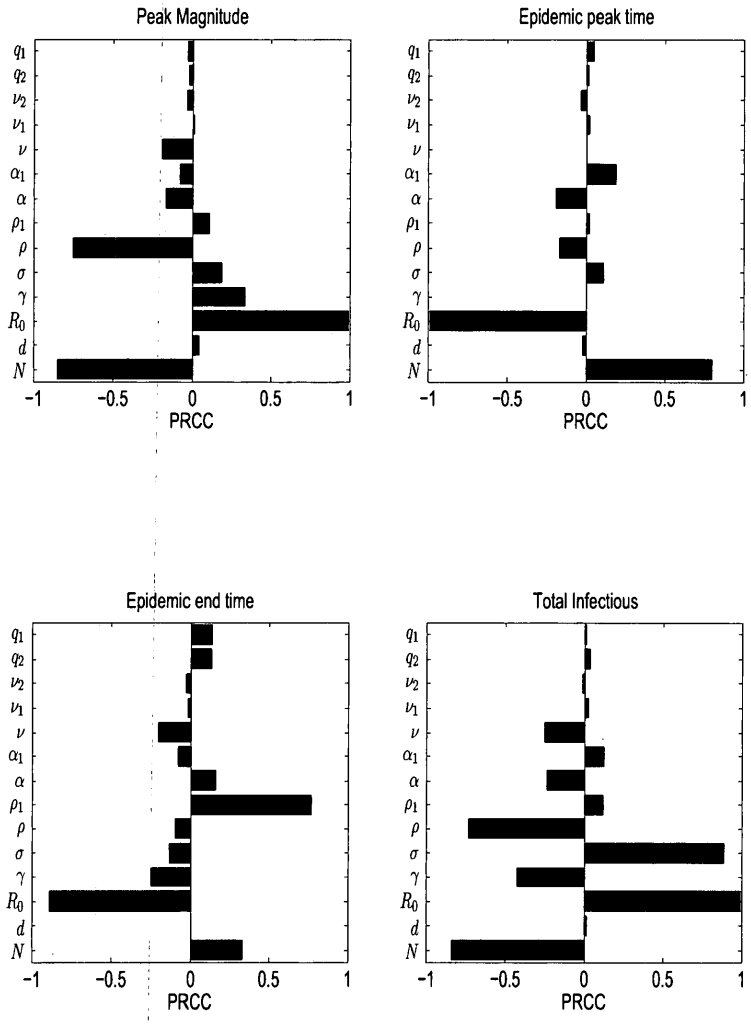

Figure 4.7: LHS-PRCC for Model (4.7). This figure shows which of the parameters have the greatest effect on Model (4.7) for the outcomes specified. (a) Peak magnitude; (b) Peak time (c) End time; (d) Total number of individuals who were infectious.

4.5 Discussion

Previous works that included mass media included mass media as a specific function as discussed in Chapter 3. Some of the drawbacks of the inclusion of a decreasing function to represent media include the different epidemic outcomes resulting from different choices in the media function, the key media parameters in the functions may be hard to obtain from the population and including the evolution of mass media, growth and waning, throughout the epidemic is not possible. In this chapter we developed a model with a novel method of media inclusion in a system of ODEs by including a new compartment for media, Model (4.7). Using a compartment to include mass media allows for the evolution of media during the epidemic. The new model can be employed to study the effects of mass media on social distancing and vaccination uptake. We studied changes in key epidemic measurements with the inclusion of multiple intervention strategies and how media waning effects the epidemic measurements.

First we developed a set of ODE models that include a media compartment which describe different scenarios possible during an epidemic. We looked at multiple levels of social distancing and vaccination and how mass media affects these scenarios. We found that in all cases without media waning, the media did decrease the severity of the epidemic on population. Moreover, the more intervention

measures included resulted in a less severe epidemic. When mass media waning was included the number of individuals who received vaccination decreased and the other key epidemic measurements increased slightly, Table (4.2).

The ODE models developed are able to describe the general behaviour of the epidemic, however, the variability in the key measurements is important to public health. To obtain variability we looked at stochastic models, a system of stochastic differential equations and ABMC simulations for each model.

The results of the SDE for Model (4.7), System (4.8) and Figures (4.2) and (4.3), show that the SDE system is not very different from its ODE model. The second order moments captured in Figure (4.3) are those which are not zero by the end of the epidemic however they are not very significant in the SDE results.

We obtained variability in key epidemic measurements, Table (4.2), from performing ABMC simulations for each model, Figures (4.4-4.6). From the variability in the key measurements, we are able to better inform public health of the rate of preparation needed during an epidemic. As shown in Figures (4.4-4.6), and Table (4.2), the mean of the epidemic measurements determined by the ABMC agree with the results of the corresponding ODE systems with the standard error.

From the stability analysis we know that when the eigenvalues of a Jacobian of a system are greater than zero, $\lambda_i > 0$, the system is unstable. In mathematical epidemiology, an unstable system corresponds to a basic reproductive ratio, $R_0 > 1$,

which means an epidemic will occur. We were able to confirm that when some of the eigenvalues are greater than zero, specifically $\lambda_7 > 0$, the basic reproductive ratio, $R_0 > 1$, and an epidemic occurs.

To further our investigation into our new model, Model (4.7), we performed a sensitivity analysis to identify key parameters that affect the key epidemic measurements. From the sensitivity analysis for Model (4.7), Figure (4.7), we see that R_0 is the most important parameter for each outcome, N is also significant in three of the four outcomes, peak magnitude, peak time and total number of infectious. Peak magnitude and total infectious are both sensitive to a media parameter, the proportion of stories that the media report (ρ). Epidemic end time is sensitive to media boredom (ρ_1). Mass media can play a role in the severity and length of a pandemic. Attention should be paid to how the media effect wanes so that this can be minimized. In addition to the parameters already mentioned peak magnitude, peak time and total infectious are sensitive to (N). The total number of infectious individuals is also sensitive to (σ) and (γ), the rate individuals become infectious and the recovery rate, respectively.

Mass media has an effect on an epidemic. This was demonstrated both in Chapter 3 and here in Chapter 4. The addition of preventative measures helps to control a pandemic. When media wanes, the proportion of individuals vaccinated decreases. The sensitivity analysis demonstrated that Model (4.7) is sensitive to

changes in media parameters. Mass media can play an important role in decreasing the severity of a pandemic and should be used with care.

As an extension to this project, it has been suggested to research a threshold criteria for media as media waning for a particular news story does not commence at the beginning of the news coverage but after repeated viewing of a specific event. This type of threshold criteria has been suggested in violent and shocking news events and video games: the event has less effect on an individual after it has been viewed a number of times [33].

In the following chapter we look to incorporate mass media data from the H1N1 epidemic in Canada. We will count the number of media stories in Canada per day and incorporate that into a compartmental model to see how media reports affect the outcome of an epidemic. This type of data has been collected and studied in [56], and by the Global Public Health Information Network (GPHIN) administered by the Public Health Agency of Canada (PHAC).

5 Data to inform the media components

5.1 Introduction

In March of 2009 a novel influenza virus appeared and spread throughout North America and the world. This novel influenza virus, pH1N1, is the first influenza pandemic of the 21st century [41]. In facing a pandemic, the immediate risk is not known and requires public health agencies, scientists and journalists to make quick decisions about informing the public about the events [38]. Investigating how news coverage develops during and affects an epidemic, provides insight into the role that mass media can play during a pandemic [56]. As novelty wanes about an issue, boredom sets in creating challenges in information dissemination to the public [33, 56]; however, understanding the nature of media during an epidemic and the role that it plays in the epidemic's progression can help to create effective communication plans for the future [56].

The Global Public Health Intelligence Network (GPHIN) is a real-time early warning system based on the internet that collects information and creates reports on issues of public health significance. This network monitors global media sources in 6 languages and filters news reports for relevancy and further analysis. The languages that are monitored are Arabic, Chinese, English, French, Russian and Spanish. Public health events that are currently tracked include disease outbreaks and infectious diseases. The Global Public Health Intelligence Network (GPHIN)

is managed by Health Canada's Centre for Emergency Preparedness.

In this section, we look at a model proposed in Chapter 4 with mass media GPHIN data in place of the equation for mass media. We also incorporate a discussion of media waning (ρ_1) informed by data presented in [56]. We compare our model output to the Canadian pandemic H1N1 data, Chapter 1.2.

5.2 Model

The model that was proposed in Chapter 4 and will be used here to study the effects of media is

$$\begin{aligned}
\dot{S} &= \lambda - \beta SI - \alpha SM - \nu SM - dS + q_1 S_1 \\
\dot{S}_1 &= -\beta_1 S_1 I + \alpha SM - \alpha_1 S_1 M - \nu_1 S_1 M + qS_2 - q_1 S_1 - dS_1 \\
\dot{S}_2 &= \alpha_1 S_1 M - qS_2 - \nu_2 S_2 M - dS_2 \\
\dot{V} &= \nu SM + \nu_1 S_1 M + \nu_2 S2M - dV \\
\dot{E} &= \beta SI + \beta_1 S_1 I - \sigma E - dE \\
\dot{I} &= \sigma E - \gamma I - dI \\
\dot{R} &= \gamma I - dR \\
\dot{M} &= \rho\sigma E - \rho_1 M.
\end{aligned} \tag{5.1}$$

Data about epidemics along with mathematical models can provide insight in to the role of the media in creating and disseminating information [56]. This study can

provide insight in to the way a pandemic progresses and and aid in future pandemic preparedness plans. To this end, we use parameter estimates from data collected and analysed in [41], for some of the parameters values in Model (5.1) and data collected about mass media in [56] and from the Global Public Health Intelligence Network (GPHIN), to better inform the model about mass media behaviour during a pandemic and to help better understand how mass media can affect the behaviour of a pandemic.

5.3 Parameter information

Each novel infectious disease where a mathematical model is created needs to see the estimation of parameters related to the transmissibility and severity of that illness. According to [41], epidemiological parameters for pandemic H1N1 of 2009 are congruent with values similar to regular seasonal influenza. Parameter estimates for the basic reproductive ratio R_0 for the first and second waves of the pandemic were made in [41] through data collected concerning cases of influenza in Ontario and Manitoba. The parameters in [41] will be used in the models presented in this section.

In [56], information about mass media reports was collected from newspaper homepages across 12 websites between April 29, 2009, the day that the World Health Organization upgraded this illness to level 5 on the pandemic scale, and May

28, 2009. The data is shown in Figure (5.1). This data collection was specifically designed to capture variation by region and county level differences in pandemic planning and previous influenza or influenza like illnesses (ILI). Data capturing occurred in 'real-time' using a single daily screenshot of the home page from each of the newspapers designated to be monitored. In this case the newspapers that were monitored were primarily in English with 2 exceptions, on newspaper in French and one in Spanish.

The amount of news for the media data of [56] was measured as a proportion of total news called, news hole. A 'news hole' is the portion of non-advertising space of a given news outlet. A news report was considered relevant to the H1N1 news hole if on the landing page of the newspaper, the news report contained at least one of 'flu', 'influenza', 'swine', 'H1N1' and 'pandemic'. An example about how a news hole is defined and how each media measurement was calculated is taken from [56]. For the *Sydney Morning Herald*, the total news hole space was 185.89, an average of the total number of available links/reports on 3 randomly selected days on the home page for this newspaper. This average available news hole of 185.89 for the *Sydney Morning Herald* is then used as a denominator to determine proportion of the news hole that is used for influenza. On May 3, 2009, the number of links that pertained to H1N1 for this newspaper was 12: for that day, the proportion of news hole for H1N1 is 6.5%. Further details about how this data was collected can be

found in [56].

In this section, the data that was collected in [56] is used to estimate the parameter that refers to media boredom for Model (5.1), (ρ_1). To estimate this parameter an exponential function was fit to the data and the decay rate of the function will be used as the media boredom parameter in Model (5.1), Figure (5.1) displays the data collected in [56] in blue and the fitted curve is is red.

The curve that is fit to the data in Figure (5.1) is an equation of the form $f(t) = ae^{bt}$, where $a = 0.09911$ with a 95% confidence interval of $(0.08611, 0.1121)$ and $b = -0.0469$ with a 95% confidence interval of $(-0.05788, -0.03592)$, where $b = \rho_1$. The R^2 value is 0.7499. A better fit can be obtained by a more complicated polynomial however the general trend of the data appears to be exponential and in order to use the value of b in our system of ordinary differential equations, Model (5.1), an exponential fit works best. The value obtained from the data in [56] for $b = \rho_1$, along with the parameter values estimated in [41], result in an epidemic in Figure (5.3). The results in this figure are considered for only the second wave of the epidemic as vaccine is available for individuals from the beginning of the model. It is evident that mass media increases as the epidemic increases but the media decays more rapidly as the epidemic is starting to decline.

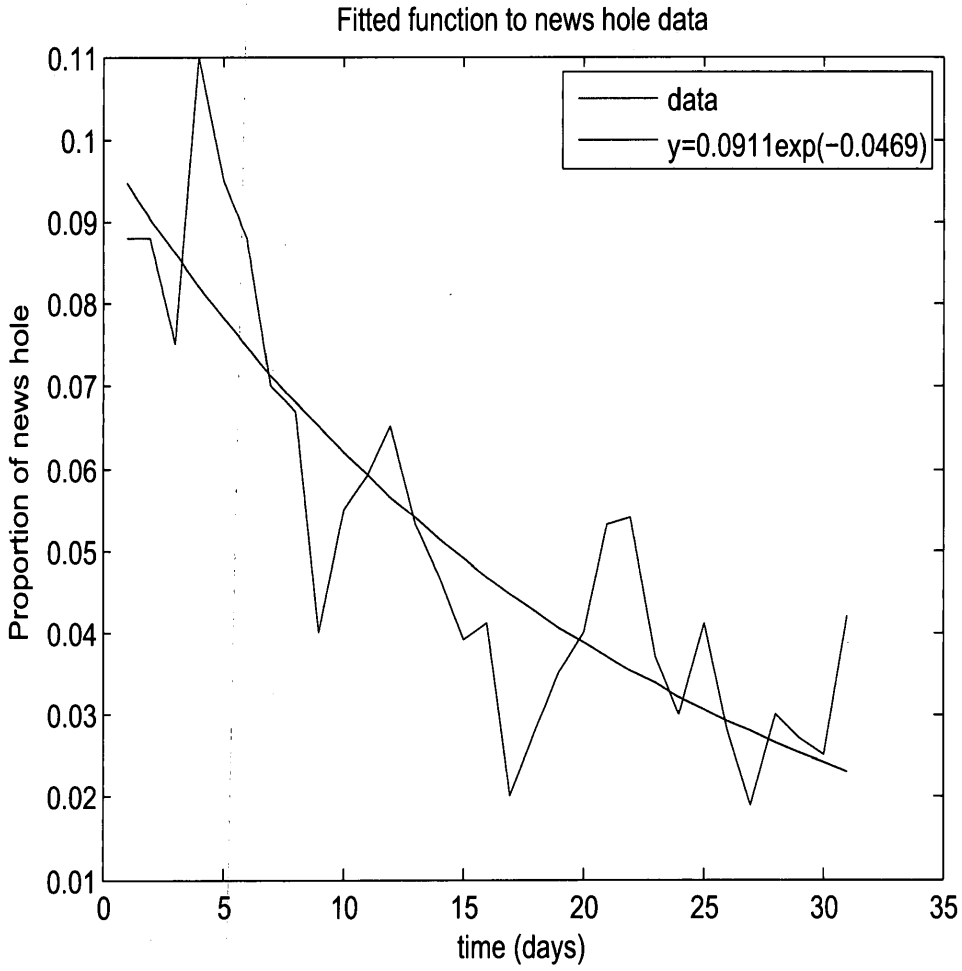

Figure 5.1: Media waning data from [56], blue, fit by an exponential curve, red. The curve has equation $f(t) = 0.09911e^{-0.0469t}$. The x-axis is time, in days.

5.4 Media report data

In this section we will use the GPHIN data introduced in Chapter 1.3.1 to inform Model (5.1) about the behaviour of mass media during the two waves of the H1N1 pandemic in Canada. The data for media will replace the equation for media $\dot{M}$ in the model.

Figure (5.2), is a plot of three different categories of data that will be used with Model (5.1). The figure is a graph of English media reports, dashed line, French and English media reports combined, dotted line, and all of the media reports, solid line, collected from March 1, 2009 to December 27, 2009. The media data from GPHIN decays, after the spike, by about half after approximately two weeks, a half-life proposed in [31] for different forms of media.

Also in Chapter 1.2 is the data for the PHAC laboratory confirmed cases of the H1N1 pandemic in Canada was introduced. We will compare the results of Model (5.1) with GPHIN data included with the number of infections in Canada.

5.5 Results

The parameter value that was obtained from the data from [56] for the media decay rate is used in Model (5.1) for ρ_1. The epidemic results are in Figure (5.3). Note that media decay, also a measure of advertisement impact, has a half-life between

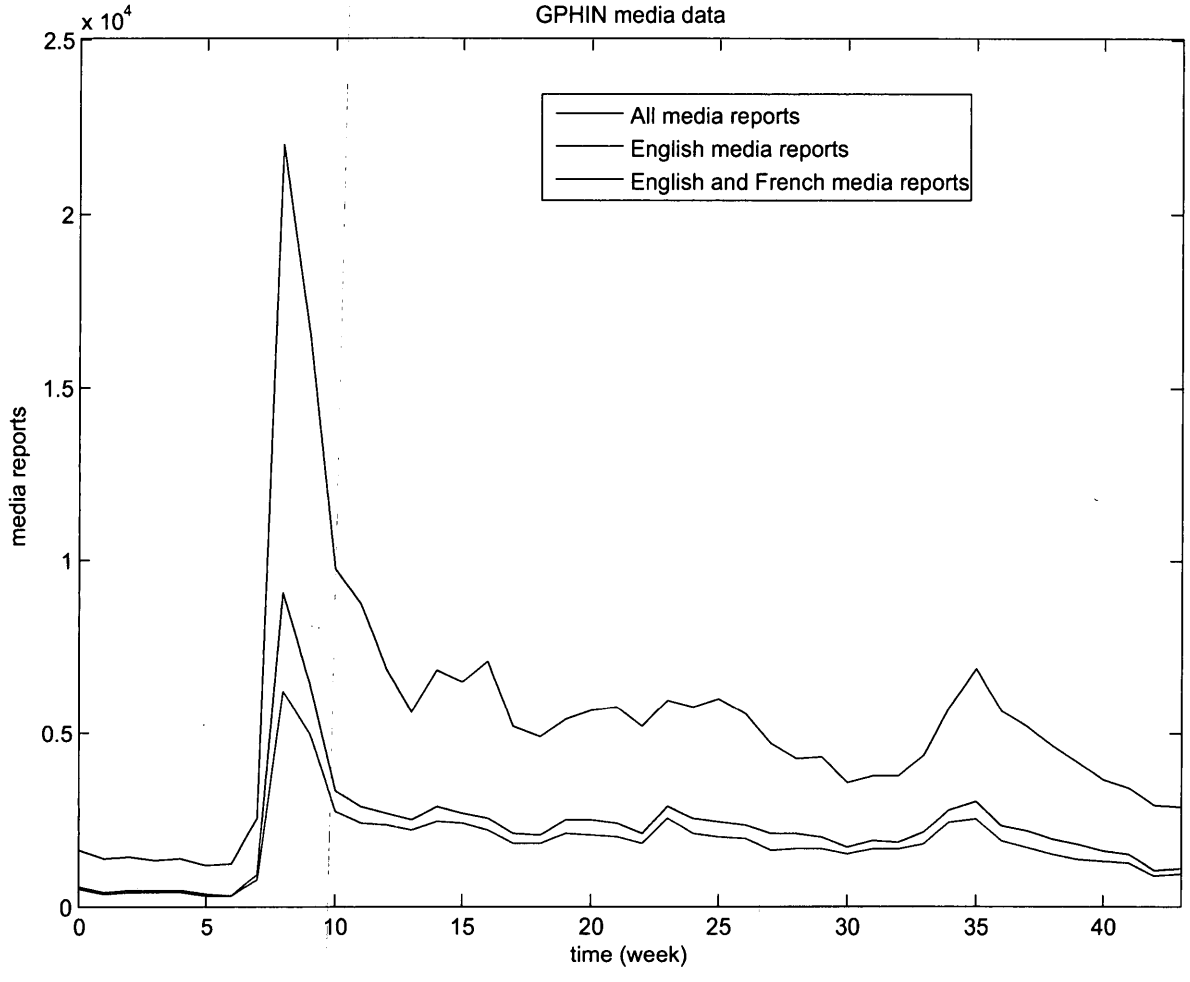

Figure 5.2: Media data collected by GPHIN from all media worldwide [50]. The time scale is weeks with 0 corresponding to March 1, 2009. The solid line is all media data collected, the dotted line is French and English media data and the dashed line is English language media data.

two and six weeks, [31]. In Figure (5.3), we can see that the fitted curve of the media level decays by about half within two weeks.

Including the data from Figure (5.2) for the media equation in System (5.1) demonstrates how mass media affects epidemic outcomes. We will look at two epidemic scenarios, an epidemic with only social distancing and an epidemic with social distancing and vaccination. The vaccination rate for each of the following models is between 25% to 40% [51] of the population by the end of the data collection or when the epidemic is finished, $I = 0$. For both scenarios, when the GPHIN data is included for the media equation, we obtain a two wave epidemic like we see in the PHAC data, Figure (1.1), in Chapter 1.2.

The GPHIN media collection stopped 43 weeks on December 27, 2010, after it began. The number of H1N1 cases in Canada was not yet zero. For this investigation, we considered five different scenarios surrounding what could happen with variations on the way the media continued after the declared end date of the pandemic. The five scenarios we studied were the data alone, media constant until the epidemic ended, a linear decline of media similar to the decline already observed, an abrupt stop of media reports at the end of the collection period and media constant until the declared end of the epidemic and then cut off. We also considered each scenario when vaccination was not available at all during the pandemic and when a vaccination was available during the second wave of the pandemic. Figure (5.4)

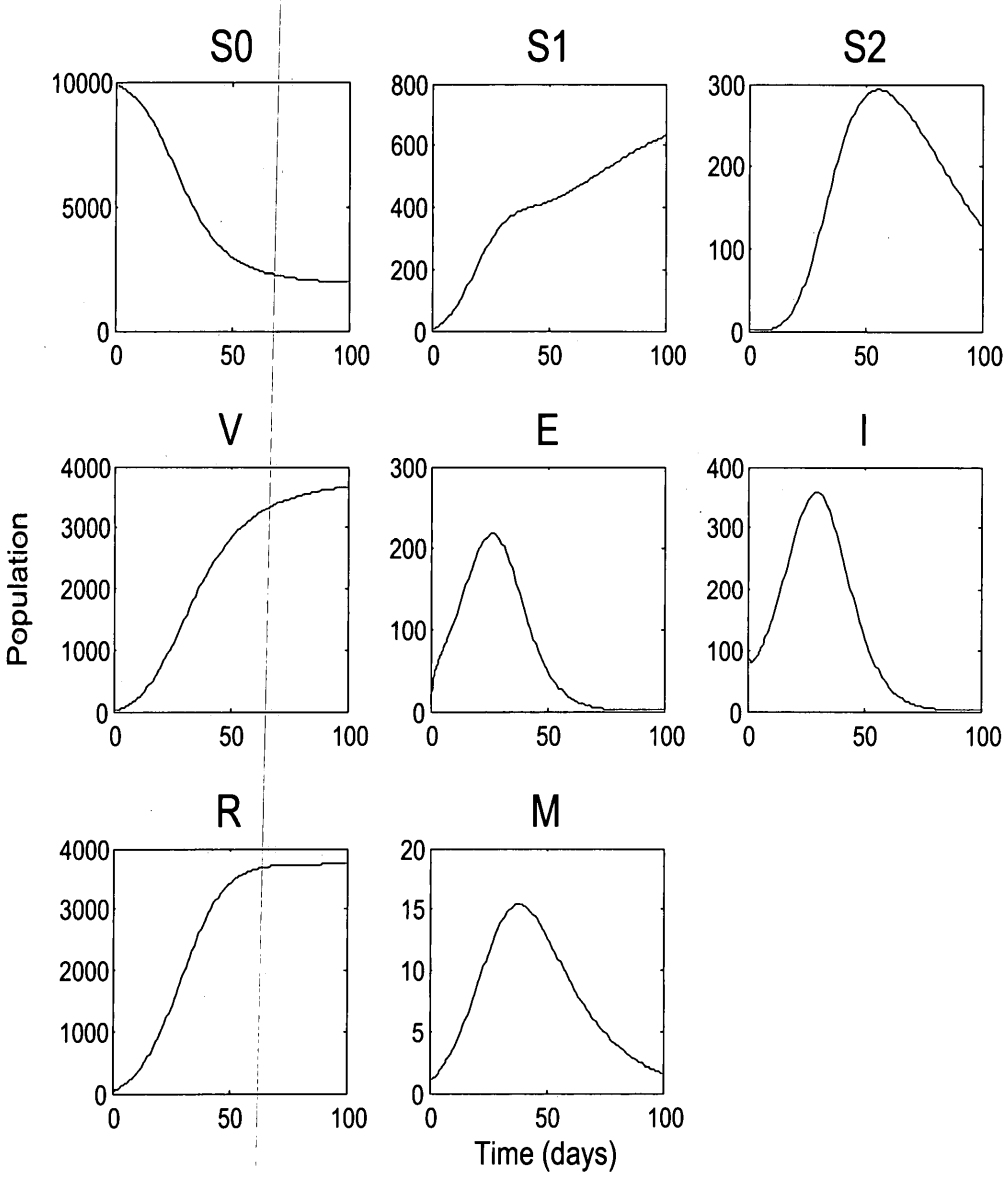

Figure 5.3: Model (5.1) with fitted media waning parameter from [56]. Second wave of an epidemic produced from the fitted value for (ρ_1) from [56] and parameter values in [41]. The x-axis is time, in days.

and Figure (5.5) display the results of the five scenarios without vaccination and with vaccination, respectively.

In addition to English, French is another official language in Canada. If the location of this population of individuals is in a bilingual area of Canada, it is possible that the population is affected by both English and French media reports, Figure (5.6). When the population is influenced by media of two languages fewer individuals fall ill during an epidemic than with fewer media reports with the same proportion of individuals who are vaccinated, Figure (5.6).

In Figure (5.7) all of the individuals are affected by all of the media reports that are collected by GPHIN. The influence of a very large amount of media in a location relative to the population, Figure (5.7), seems to stop the epidemic at the beginning of the first wave. As the number of media reports increases an individual may need to be less sensitive to media to appreciate how mass media can influence behaviour during an epidemic.

5.6 Discussion

In this chapter we looked at the incorporation of media data to inform the media equation of Model (5.1) in two different ways. First we estimated a media waning rate from the data in [56] and second we used media reports collected by GPHIN instead of the $\dot{M}$ equation for the media. With the GPHIN data we looked at

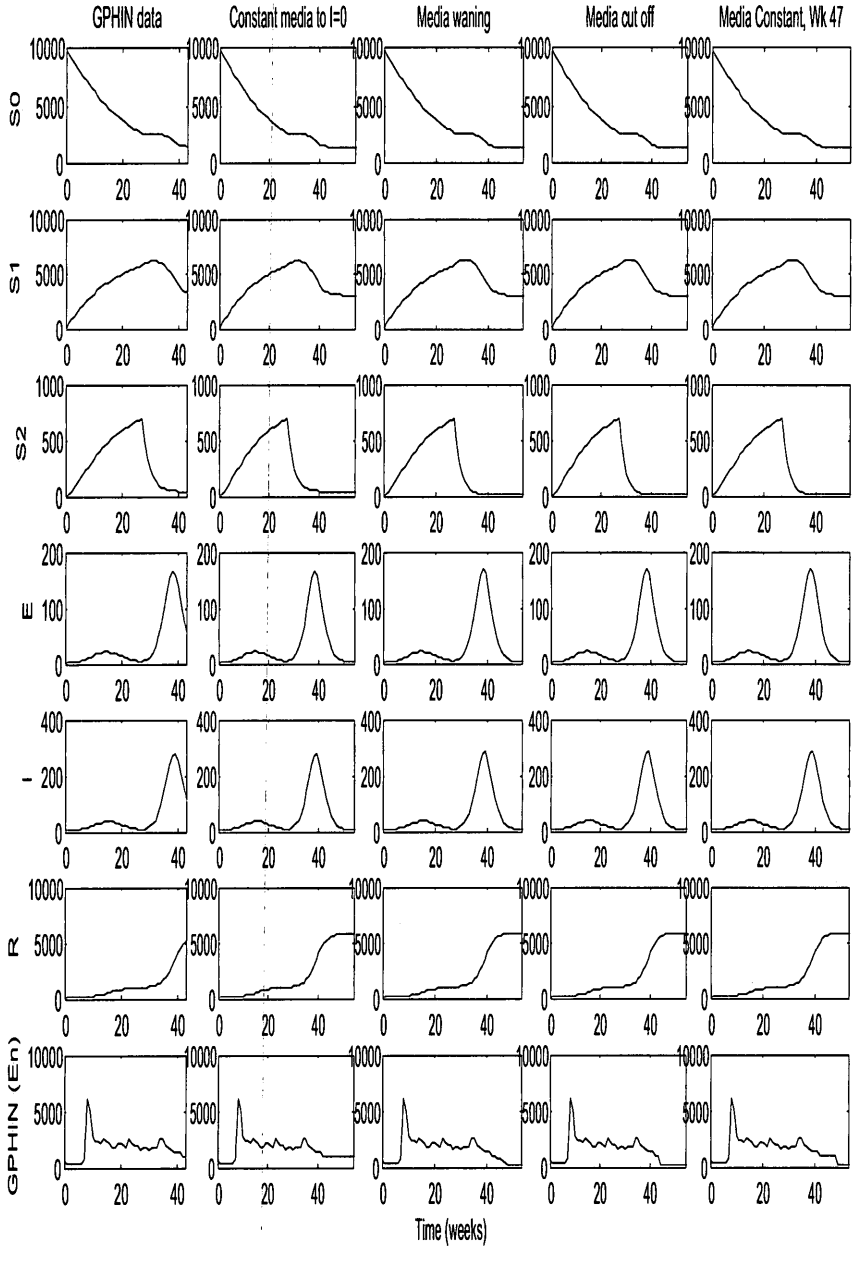

Figure 5.4: Model (5.1) with GPHIN data, Figure (5.2). These are the results without a vaccination compartment for different media end behaviour. There is a two wave epidemic like in the PHAC data, Figure (1.1).

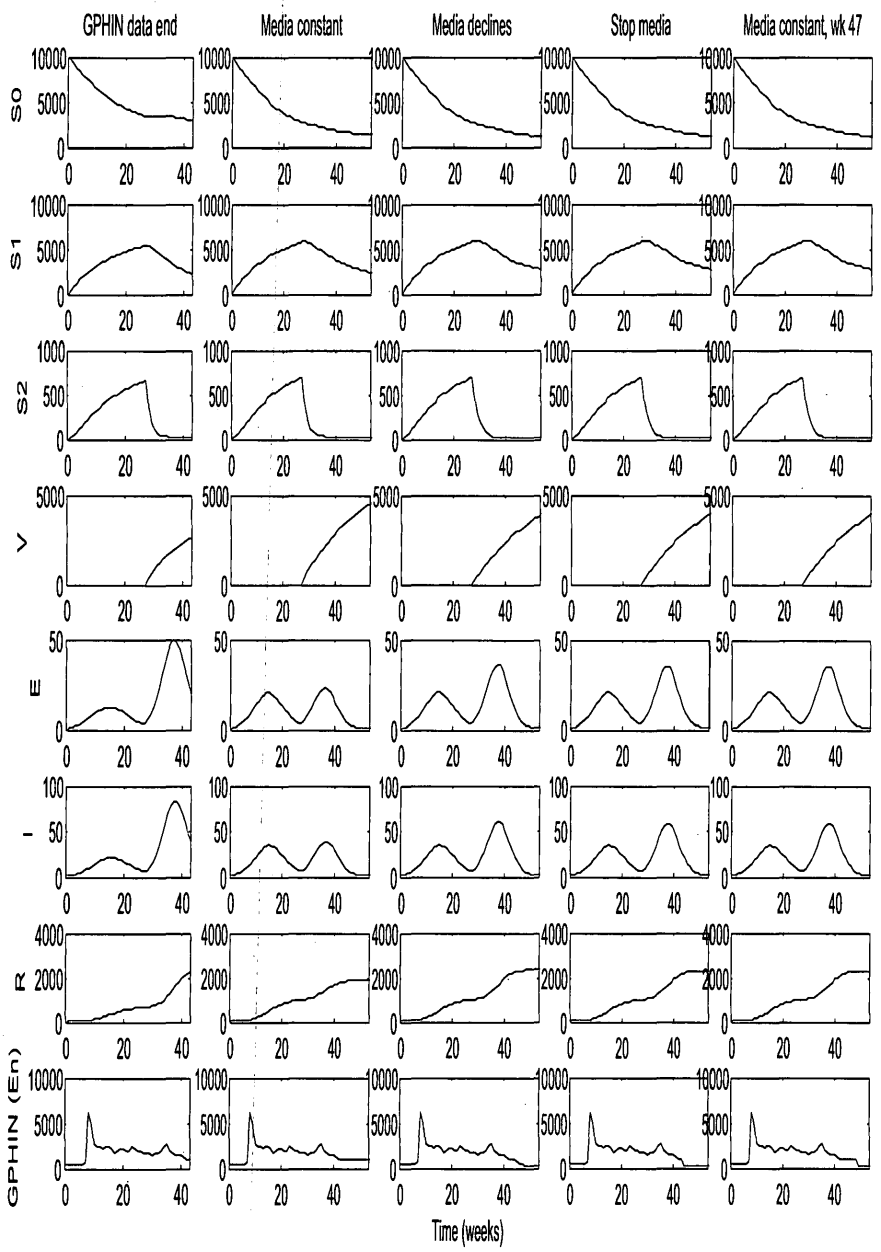

Figure 5.5: Model (5.1) with GPHIN data, Figure (5.2). These are the results with a vaccination compartment for different media end behaviour. There is a two wave epidemic like in the PHAC data, Figure (1.1).

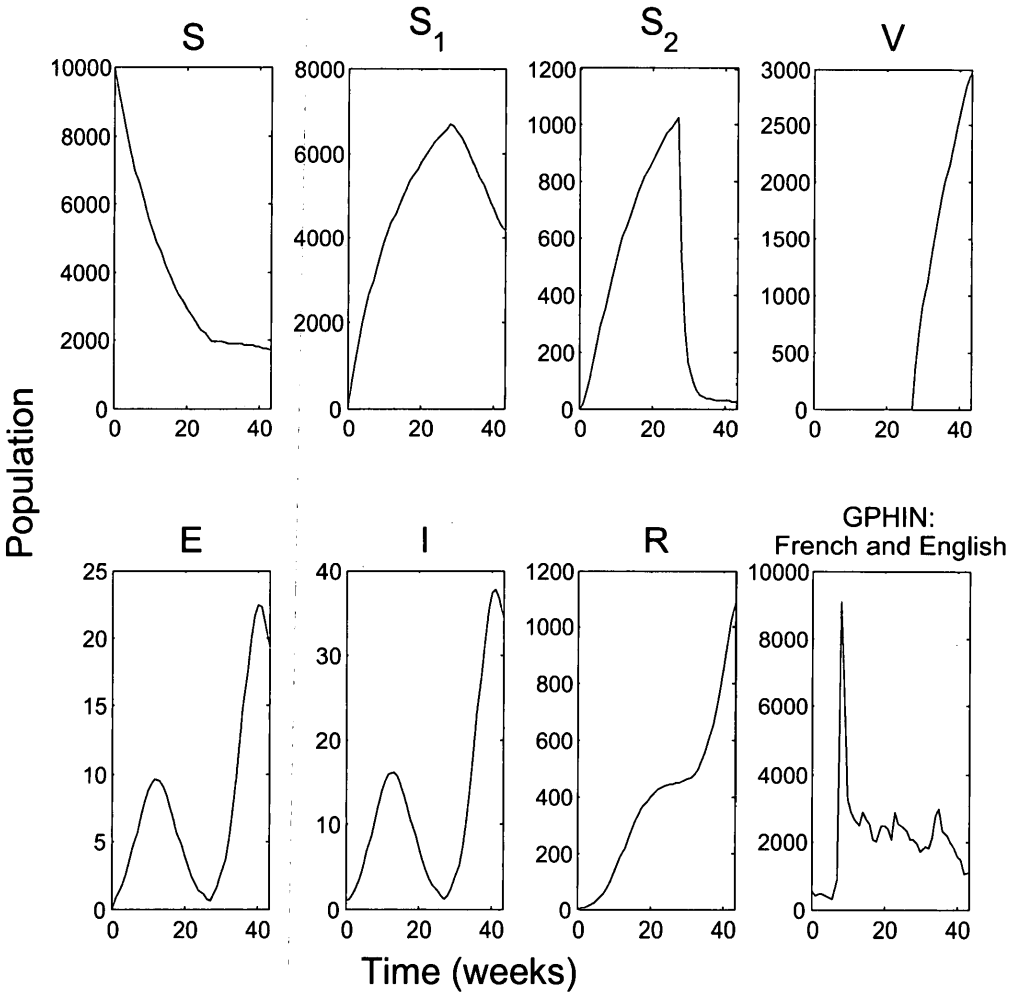

Figure 5.6: Model (5.1) using French and English GPHIN data. Both waves of the pandemic are influenced by both French and English media.

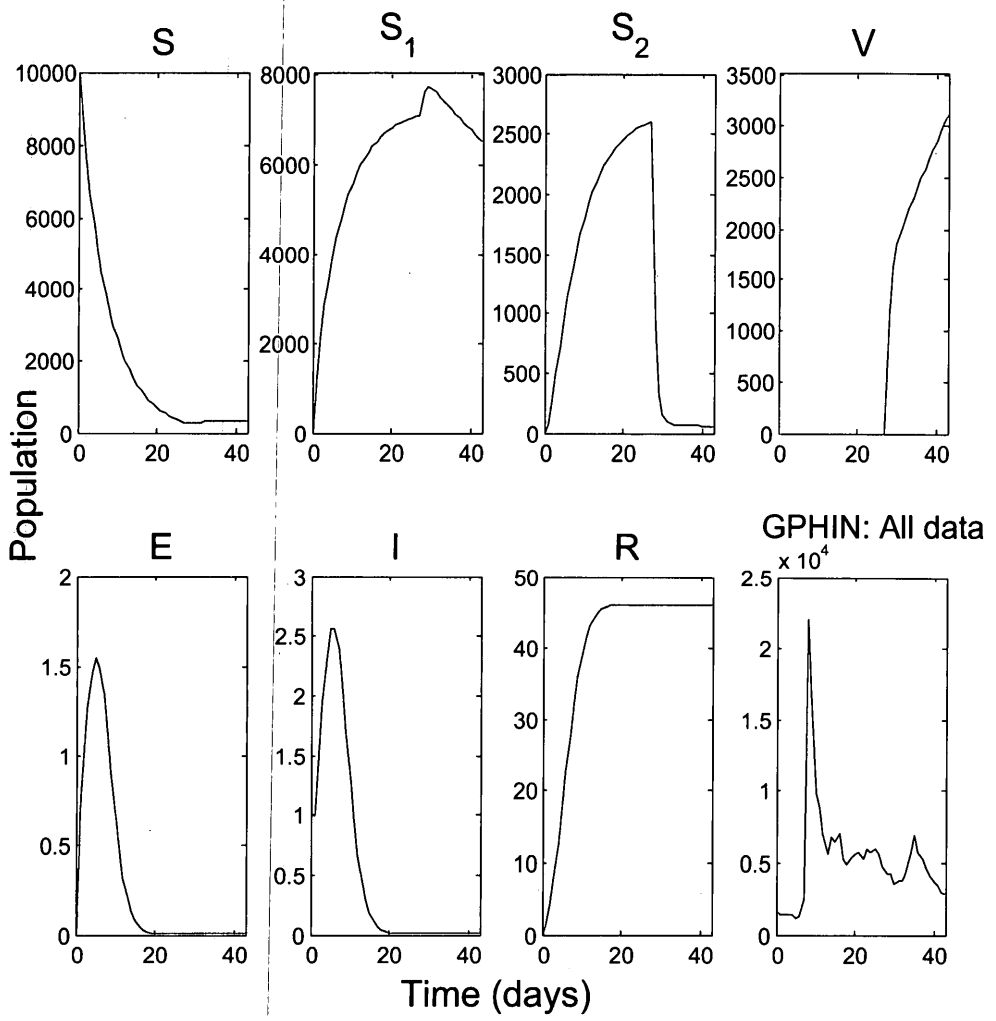

Figure 5.7: All GPHIN data. The epidemic is influenced by all of the media reports that GPHIN collects.

scenarios with and without available vaccination and we looked at whether differences in media behaviour at the end of the epidemic can change the outcome of the epidemic. We also considered scenarios if all individuals in the population were able to be influenced by both French and English data and all of the data collected by GPHIN.

The model from Chapter 4 seems to accurately portray the behaviour of media during an epidemic. We were able to estimate a value for the media waning rate, ρ_1, from the data collected in [56] and use this estimate to demonstrate the effect that waning media has on an epidemic. The data from [56] has a decreasing slope. We fit the data with a decreasing exponential function. Using a decreasing function to represent media, like Functions (3.1-3.3) does capture some of the behaviour of media. In this case vaccination was an option as the epidemic is modelled only for the second wave. The media information in [56] was collected only for one month after the World Health Organization raised its pandemic alert level to 5. It may be possible that the data does not continue on this same path of decline over the entire pandemic.

Mass media informed by only people falling ill may have a different effect on an epidemic than mass media reports that are informed by people becoming ill and by outside sources. A possible limitation to this would be that the parameter estimate of ρ_1 comes from only one month's worth of media data from a very limited number

of sources. This could present a bias towards the severity of H1N1 in the countries from where the data was collected. The data collected by Global Public Health Information Network (GPHIN) takes into account media in 6 languages from all around the world, including translated reports [50].

Figures (5.4) and (5.5), demonstrate that the number of individuals who were ill when vaccination was not included in Model (5.1), using GPHIN English language data for the media equation, is much higher than when vaccination is available for the population. Both models generate a two wave pandemic as in Figure (1.1), however we see in Figure (5.5) that with vaccination, the peak of the second wave is much less than without vaccine, Figure (5.4).

In comparing the different end strategies for the media, we see that in most cases the epidemics without vaccination, although a greater burden on the population, are slightly shorter. This is especially true for the scenario that media is kept constant until the declared end of the epidemic and then stop reporting about the pandemic. In this scenario, the epidemic without vaccination lasts 52 weeks whereas the epidemic with vaccination is 55 weeks. A possible explanation for the similarities between each of the media scenarios is that the media behaviour was changed almost near the end of the epidemic. If the behaviour was changed earlier in the epidemic it is possible that the epidemic outcomes may be different.

For Model (5.1), using the media data from GPHIN, including influence from

multiple languages, fewer individuals become ill and more individuals practice social distancing than for the epidemics using only English language data. When all of the media data is used, this epidemic with these parameters can be stopped in the first wave.

Considering different behaviours of the media after the final data collection point allows variation in the duration of the epidemic by a few weeks while the end behaviour of the epidemic is mostly unchanged. This result appears consistent in situations with and without vaccination. The similar epidemic end patterns displayed with the different media scenarios could be due to the fact that the change in behaviour of the media occurs very late in the epidemic and has little influence on the actions of the population. The differences in epidemic end time and the final populations for Model (5.1) suggests that an agent-based Monte Carlo simulation would be useful in further examining the variability of the effect of different media scenarios during an epidemic. Another possible extension would be to investigate stochastic and deterministic models designed to look at the results of earlier changes in media behaviour on an epidemic.

6 Continuous social distancing

6.1 Introduction

In developing an ODE model with different levels of social distancing, it became interesting to consider a continuum of social distancing classes. First we looked at a PDE model for an SEIR model that included mass media without an effect on individuals and without waning,

$$\begin{aligned}
\frac{\partial S(t,x)}{\partial t} + \frac{\partial S(t,x)}{\partial x} &= \lambda(x) - \beta(x)S(t,x)I(t,x) - dS(t,x) \\
\frac{\partial E(t,x)}{\partial t} + \frac{\partial E(t,x)}{\partial x} &= \beta(x)S(t,x)I(t,x) - \sigma E(t,x) - dE(t,x) \\
\frac{\partial I(t,x)}{\partial t} + \frac{\partial I(t,x)}{\partial x} &= \sigma E(t,x) - \gamma I(t,x) - dI(t,x) \\
\frac{dR}{dt} &= \gamma \int_0^\infty I(t,x)dx - dR(t) \\
\frac{dM}{dt} &= \rho\sigma \int_0^\infty E(t,x)dx,
\end{aligned} \tag{6.1}$$

where x is the distance of an individual from his/her normal behaviour during a pandemic, $\lambda(x) = \lambda N(x,t)$ and $\beta(x)$ is the transmission rate. The time scale for media and influenza transmission are both considered as the same giving a wave speed of of 1.

System (6.1), is a mixed system of 3 PDEs and 2 ODEs. The initial boundary

conditions for Model (6.1) are

$$
\begin{aligned}
S(0,t) &= \begin{cases} 10000, \ t = 0 \\ 0, \ \text{otherwise} \end{cases} \\
S(x,0) &= \begin{cases} 10000, \ x = 0 \\ 0, \ \text{otherwise} \end{cases} \\
E(0,t) &= 0 \\
E(x,0) &= 0 \\
I(0,t) &= \begin{cases} 1, \ t = 0 \\ 0, \ \text{otherwise} \end{cases} \\
I(x,0) &= \begin{cases} 1, \ x = 0 \\ 0, \ \text{otherwise} \end{cases} \\
R(0,t) &= 0 \\
M(0,t) &= 0.
\end{aligned}
\tag{6.2}
$$

The next PDE model that we considered includes the effect of mass media on susceptible individuals without the media boredom rate, similar to Model (4.2), except instead of one level of social distancing, there is an continuum of social distancing classes. The proposed PDE model with movement in social distancing

is

$$\begin{aligned}
\frac{\partial S(t,x)}{\partial t} + \frac{\partial S(t,x)}{\partial x} &= \lambda(x) - \beta(x)S(t,x)\int_0^\infty I(t,x)dx - \alpha(x)S(x,t)M(t) \\
&\quad + \int_{a<x} \alpha(a-x)S(a,t)M(t)da \\
\frac{\partial E(t,x)}{\partial t} + \frac{\partial E(t,x)}{\partial x} &= \beta(x)S(t,x)\int_0^\infty I(t,x)dx - \sigma E(t,x) - dE(t,x) \\
\frac{\partial I(t,x)}{\partial t} + \frac{\partial I(t,x)}{\partial x} &= \sigma E(t,x) - \gamma I(t,x) - dI(t,x) \\
\frac{dR}{dt} &= \gamma \int_0^\infty I(t,x)dx - dR(t) \\
\frac{dM}{dt} &= \rho\sigma \int_0^\infty E(t,x)dx,
\end{aligned} \tag{6.3}$$

where x represents the distance of an individual from regular behaviour during a pandemic situation as influenced by mass media. The initial boundary conditions are the same as for Model (6.1), Expression (6.2).

The final PDE model that we consider for numerical solution here is very similar to Model (6.3) except we consider media waning,

$$\begin{aligned}
\frac{\partial S(t,x)}{\partial t} + \frac{\partial S(t,x)}{\partial x} &= \lambda(x) - \beta(x)S(t,x)\int_0^\infty I(t,x)dx - \alpha(x)S(x,t)M(t) \\
&\quad + \int_{a<x} \alpha(a-x)S(a,t)M(t)da \\
\frac{\partial E(t,x)}{\partial t} + \frac{\partial E(t,x)}{\partial x} &= \beta(x)S(t,x)\int_0^\infty I(t,x)dx - \sigma E(t,x) - dE(t,x) \\
\frac{\partial I(t,x)}{\partial t} + \frac{\partial I(t,x)}{\partial x} &= \sigma E(t,x) - \gamma I(t,x) - dI(t,x) \\
\frac{dR}{dt} &= \gamma \int_0^\infty I(t,x)dx - dR(t) \\
\frac{dM}{dt} &= \rho\sigma \int_0^\infty E(t,x)dx - \rho_1 M(t),
\end{aligned} \tag{6.4}$$

with the initial boundary conditions from Expression (6.2)

A full PDE model that represents the dynamics of Model (4.7) of Chapter 4.2.6 includes relaxing of social distancing practices and vaccination.

$$\begin{aligned}
\frac{\partial S(t,x)}{\partial t} + \frac{\partial S(t,x)}{\partial x} &= \lambda(x) - \beta(x)S(t,x)\int_0^\infty I(t,x)dx - \alpha(x)S(x,t)M(t) \\
&\quad + \int_{a<x} \alpha(a-x)S(a,t)M(t)da - \nu S(x,t)M(t) - q(x)S(x,t) \\
&\quad + \int_{a>x} q(a-x)S(a,t)da \\
\frac{\partial E(t,x)}{\partial t} + \frac{\partial E(t,x)}{\partial x} &= \beta(x)S(t,x)\int_0^\infty I(t,x)dx - \sigma E(t,x) - dE(t,x) \\
\frac{\partial I(t,x)}{\partial t} + \frac{\partial I(t,x)}{\partial x} &= \sigma E(t,x) - \gamma I(t,x) - dI(t,x) \\
\frac{dR}{dt} &= \gamma\int_0^\infty I(t,x)dx - dR(t) \\
\frac{dM}{dt} &= \rho\sigma\int_0^\infty E(t,x)dx - \rho_1 M(t) \\
\frac{dV}{dt} &= \nu\int_0^\infty S(t,x)dx - dV(t),
\end{aligned} \tag{6.5}$$

where again (x) represents the distance an individual is from regular daily behaviours or how much an individual has changed his/her behaviour in order to socially distance him/herself from the population as a result of media influence. Note that here we do not consider media influence and social distancing within the exposed or infectious classes although this is a likely scenario. The initial boundary

conditions for Model (6.5) are in Expression (6.6).

$$
\begin{aligned}
S(0,t) &= \begin{cases} 10000, \ t=0 \\ 0, \ \text{otherwise} \end{cases} \\
S(x,0) &= \begin{cases} 10000, \ x=0 \\ 0, \ \text{otherwise} \end{cases} \\
E(0,t) &= 0 \\
E(x,0) &= 0 \\
I(0,t) &= \begin{cases} 1, \ t=0 \\ 0, \ \text{otherwise} \end{cases} \\
I(x,0) &= \begin{cases} 1, \ x=0 \\ 0, \ \text{otherwise} \end{cases} \\
R(0,t) &= 0 \\
M(0,t) &= 0 \\
V(0,t) &= 0.
\end{aligned}
\tag{6.6}
$$

The parameter values for Models (6.1,6.3) and (6.4) can be found in Table (6.1).

In order to solve Systems (6.1, 6.3, 6.4), we propose a numeric scheme that can solve hyperbolic systems of equations: forward time backward space. For the integrals in System (6.4), we approximate with the trapezoidal method.

The numerical solutions for Systems (6.1, 6.3, 6.4) consider $\beta(x)$ and $\alpha(x)$ as

Parameter	Definition	Value	Reference
S(x,t)	susceptible individuals		
E(x,t)	exposed individuals		
I(x,t)	infectious individuals		
R(t)	recovered individuals		
M(t)	media reports		
$\lambda(x)$	birth rate	0.355	[46]
d	natural death rate	$3.52e-005$	[46]
β	contact transmission rates	$3.712e-5$	calculated
$\beta(x)$	transmission rate function	$\beta e^{-\rho x}$	
σ	transition rate from exposed to infectious	$\frac{1}{2}$	[46]
γ	recovery rate	$\frac{1}{4}$	[46]
$\alpha(x)$	rate to enter socially distanced class $S_i(t)$	0.04	
$q(x)$	relax social distancing rate	0.06	
ρ	media rate	0.01	
ρ_1	media waning rate	0.015	

Table 6.1: Parameters for Models (6.1),(6.3), (6.4).

constants β and α, initially. Modelling these parameters as constants is similar to modelling space, alone. Since we propose that social distancing decreases transmission of influenza we will consider a structure for $\beta(x)$ that represents the decreasing transmission of influenza with social distancing. Since possible functions for these values are unknown, here we will consider only a structure for $\beta(x)$. We consider $\beta(x) = \beta e^{-\rho x}$, where the decrease in transmission is influenced by the mass media rate, ρ.

6.2 Forward-time backward-space (FTBS) scheme

We solve a system of PDEs and ODEs, numerically, by the forward time backward space method (FTBS), Method (6.8), an explicit scheme [3, 36, 57]. As with all explicit schemes, FTBS, Scheme (6.8), is only conditionally stable. To ensure stability, the Courant-Friedrichs-Lewy (CFL) condition, is both necessary and sufficient. The CFL condition requires that

$$c = \left| \alpha \frac{\Delta t}{\Delta x} \right| \leq 1. \tag{6.7}$$

A restriction on the time step is imposed by this condition of $\Delta t \leq \frac{\Delta x}{|\alpha|}$ [36].

A discretization for the FTBS scheme for a general advection equation: $\frac{\partial u}{\partial t} + \alpha \frac{\partial u}{\partial x} = g(t, x)$, is

$$\frac{u_i^{j+1} - u_i^j}{\delta t} + \alpha \frac{u_i^j - u_{i-1}^j}{\delta h} = g(t, x), \tag{6.8}$$

where i is the spatial index and j is the time index on the solution grid. In order to solve the integrals in system (6.4), we use the trapezoidal rule. The trapezoidal rule is an implicit method that is obtained by averaging two Euler methods. This method is second order accurate and it is a one-step method: it requires only the previous step to get the next [57]. The trapezoidal method for a general function $f(x)$ is

$$\int_a^b f(x)dx = \frac{1}{2}\left[f(a) + f(b)\right] + \sum_{n=2}^{N-1} f_n. \tag{6.9}$$

For the solution of Model (6.4), we assume that $\lambda(x) = \lambda$, $q(x) = q$,and $\alpha(x) = \alpha_1$ from the parameters for Model (4.7) from Chapter 4. The FTBS scheme for the System (6.4) without movement in the spatial direction is

$$\begin{aligned}
s(i,j+1) &= s(i,j) - c(s(i,j) - s(i-1,j)) - \beta s(i,j)(\sum_{i=2}^{n-1} y(i,j) + 0.5(y(a,j) \\
&\quad + y(b,j))) - \alpha s(i,j)m(j) + \alpha\left[\frac{1}{2}\left(s(0,j) + s(n,j)\right) + \sum_{i=2}^{N-1} s(i,j)\right] - ds(i,j) + \lambda \\
e(i,j+1) &= e(i,j) - c(e(i,j) - e(i-1,j)) + \beta s(i,j)(\sum_{i=2}^{n-1} y(i,j) + 0.5(y(a,j) \\
&\quad + y(b,j))) - \sigma e(i,j) - de(i,j) \\
y(i,j+1) &= y(i,j) - c(y(i+1,j) - y(i-1,j)) + \sigma e(i,j) - \gamma y(i,j) - dy(i,j) \\
r(j+1) &= r(j) - dr(j) + \gamma(\sum_{i=2}^{n-1}(y(i,j)) + 0.5(y(a,j) + y(b,j))) - dr(j) \\
m(j+1) &= m(j) + \rho\sigma(\sum_{i=2}^{n-1}(e(i,j)) + 0.5(e(a,j) + e(b,j))) - \rho 1 m(j),
\end{aligned} \tag{6.10}$$

where $c = \frac{0.1}{10.0} = 0.01 \leq 1$.

6.3 Results

Initially we look at the results of System (6.1) without media. This is to verify that the model corresponds to the ODE model and that the initial boundary conditions, Condition (6.2) are correct. The numerical results can be seen in Figure (6.1). We can see that without movement in x, no social distancing, the results are what would be expected of an ODE model.

To include the influence of mass media on individuals during the epidemic, it is necessary to include movement of individuals in the spatial direction, those who practice social distancing. First, we include movement in the spatial direction in order to see how media affects social distancing without media boredom effecting the epidemic outcome. The model that is used in this case is Model (6.3). The results of the model with a continuous social distancing class and no media boredom are in Figure (6.2).

The results for Model (6.4) are presented in Figure (6.3). These results demonstrate a continuum of individuals practicing social distancing in each of the three pdes in the model.

Including media waning increases the impact of the epidemic on the population when $\beta(x)$ is modelled as a constant, Figures (6.2) and (6.3).

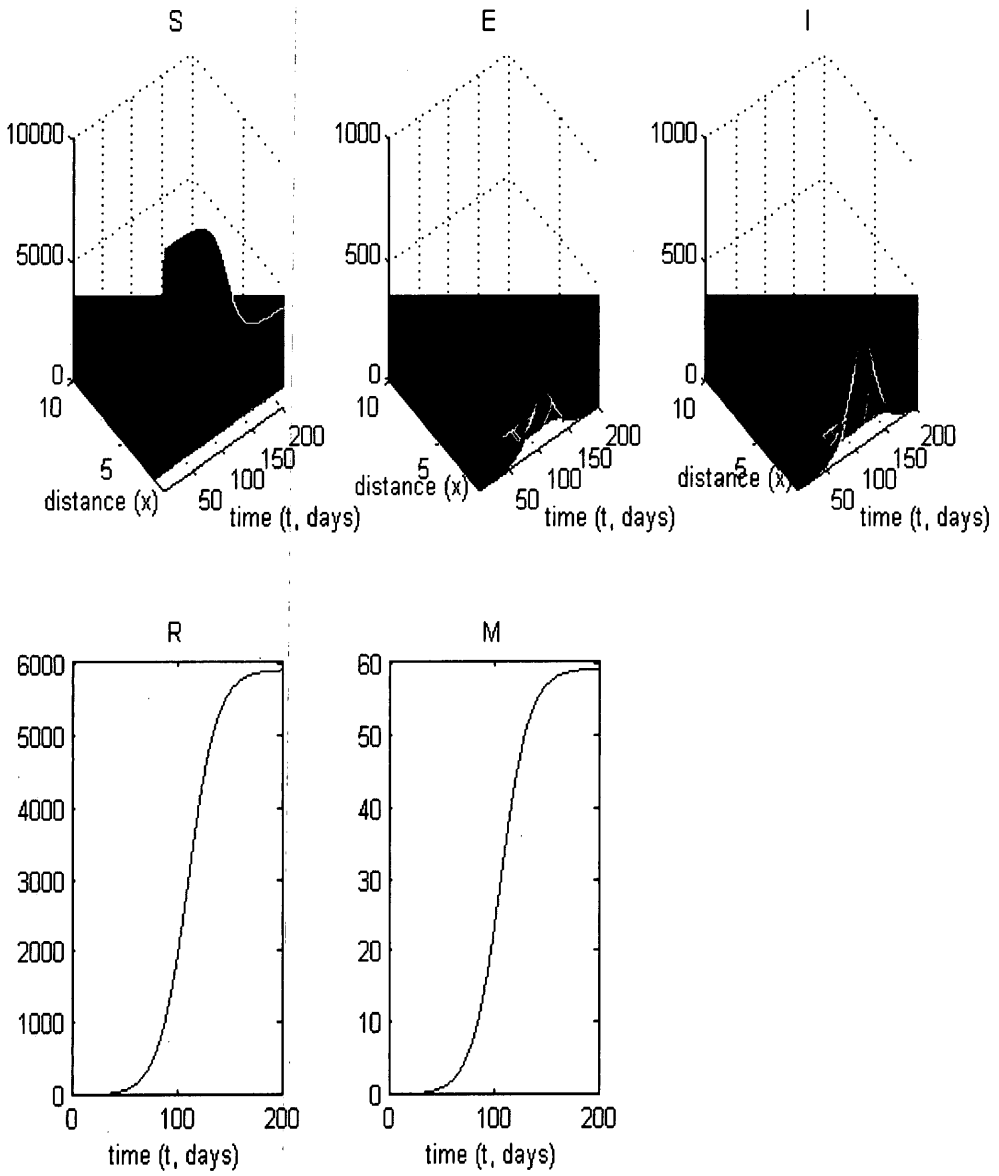

Figure 6.1: Model (6.1) — No movement in the social distancing classes and no media boredom term. The colours help to show a decrease in the population in time and space.

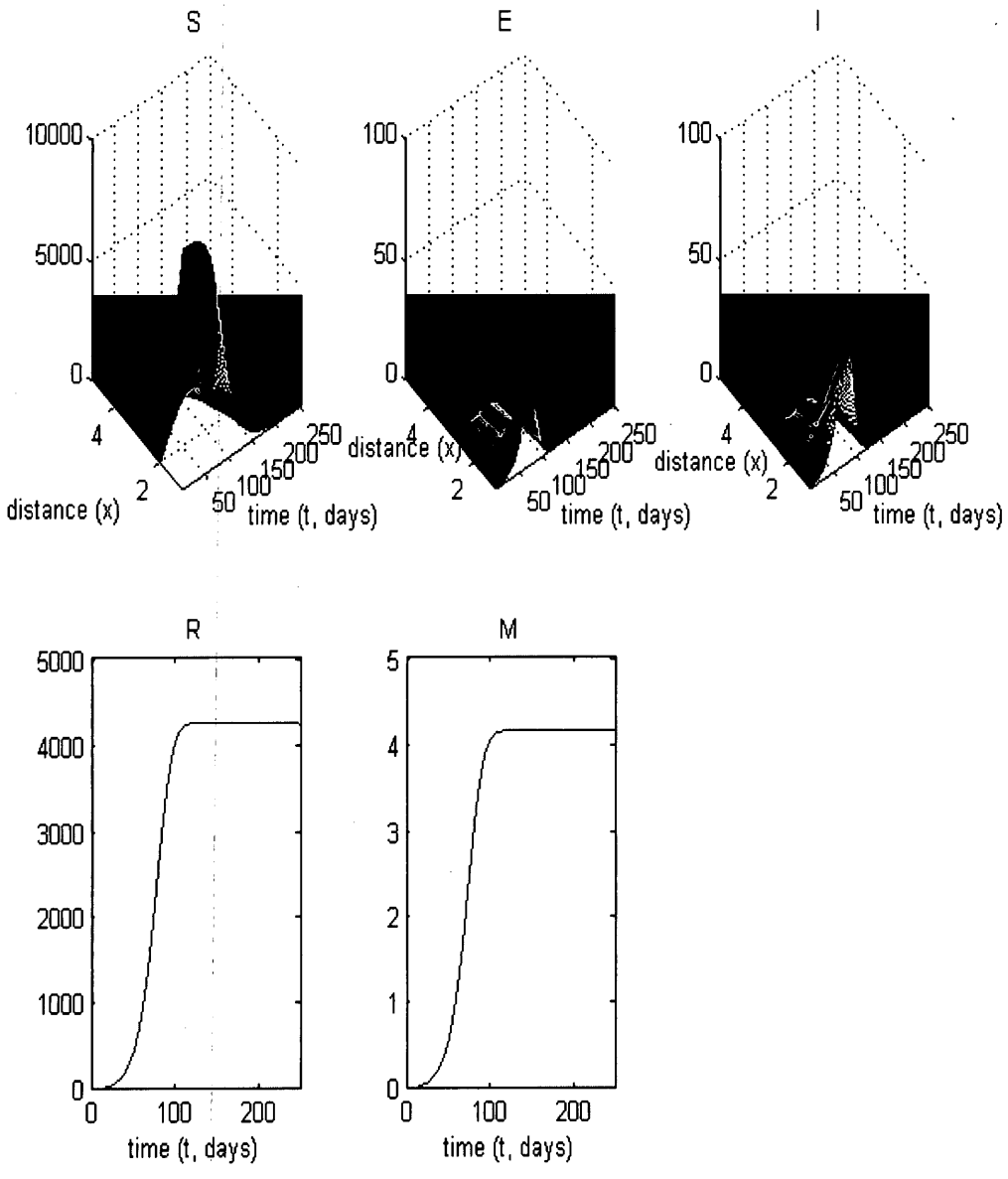

Figure 6.2: Model (6.3) — No media boredom considered. The colours help to show a decrease in the population in time and space.

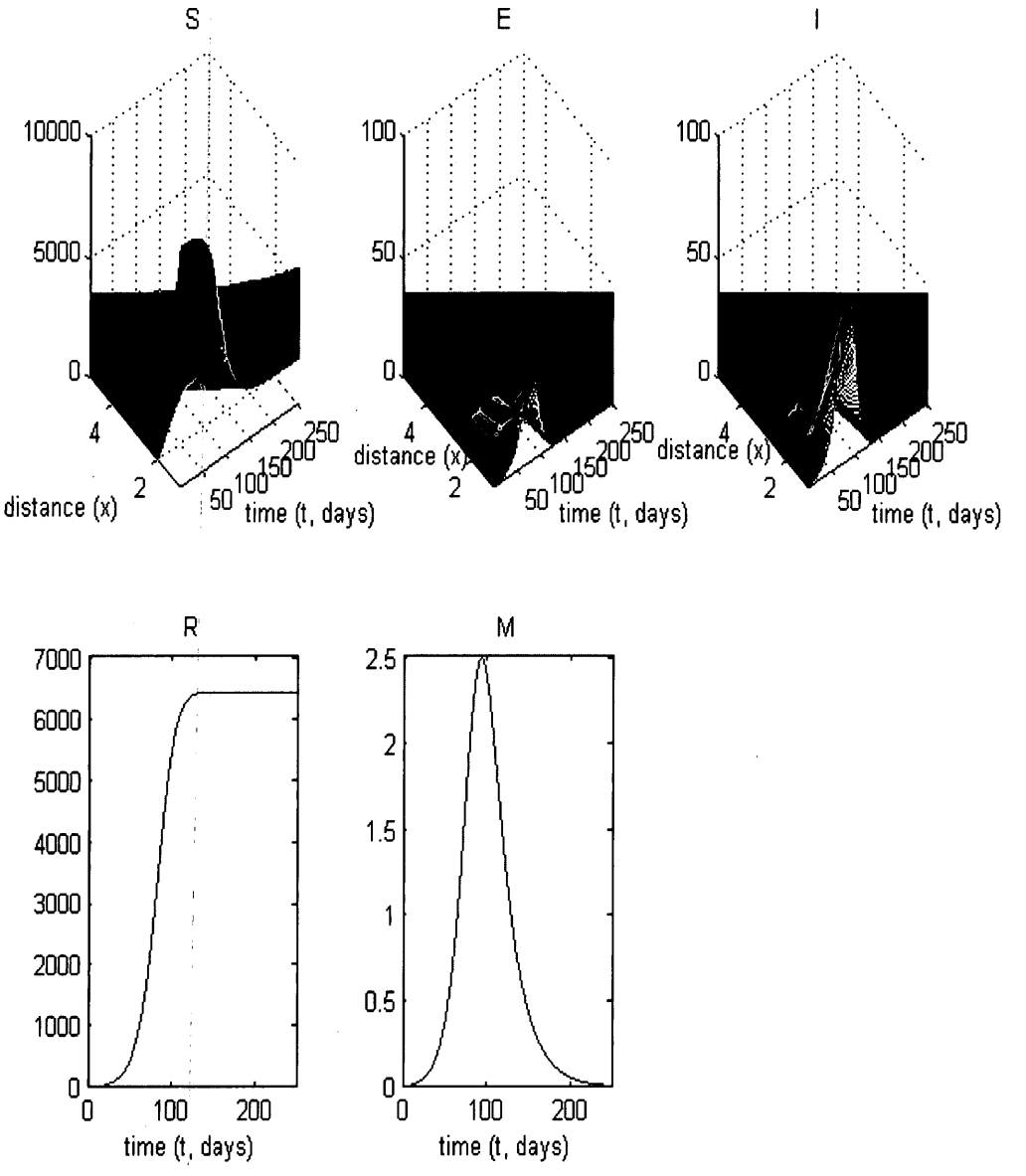

Figure 6.3: Model (6.4) — Social distancing and media boredom. The colours help to show a decrease in the population in time and space.

Figure (6.4) displays the results for Model (6.4) when a decreasing exponential function is considered for the contact transmission term. We can see that media waning increases the effect of an epidemic on the population when comparing Figures (6.2) and (6.3). It is also evident that when we include a function to represent the decreasing transmission rate that occurs with social distancing, the resulting epidemic is less potent on the population even with media waning, Figure (6.4).

6.4 Discussion

In this chapter we have proposed a PDE extension to Model (4.7) of Chapter 4 that includes continuous social distancing instead of two distinct classes of social distancing, Model (6.5). We numerically solve the PDE models presented in this chapter that include only social distancing and media as preventative measures during a pandemic. To solve the PDE models we use the FTBS algorithm and the Trapezoidal Rule to approximate the integrals.

The numerical solutions for Models (6.1), (6.3) and (6.4) are presented in Figures (6.1), (6.2) and (6.3), respectively. We can see that in comparing this model, Figure (6.1), with Figure (6.2), that including social distancing in the models through movement in the spatial direction, the effect of the epidemic on the population is reduced. The number of individuals who are in the R class at the end of the epidemic is much less than in the previous case.

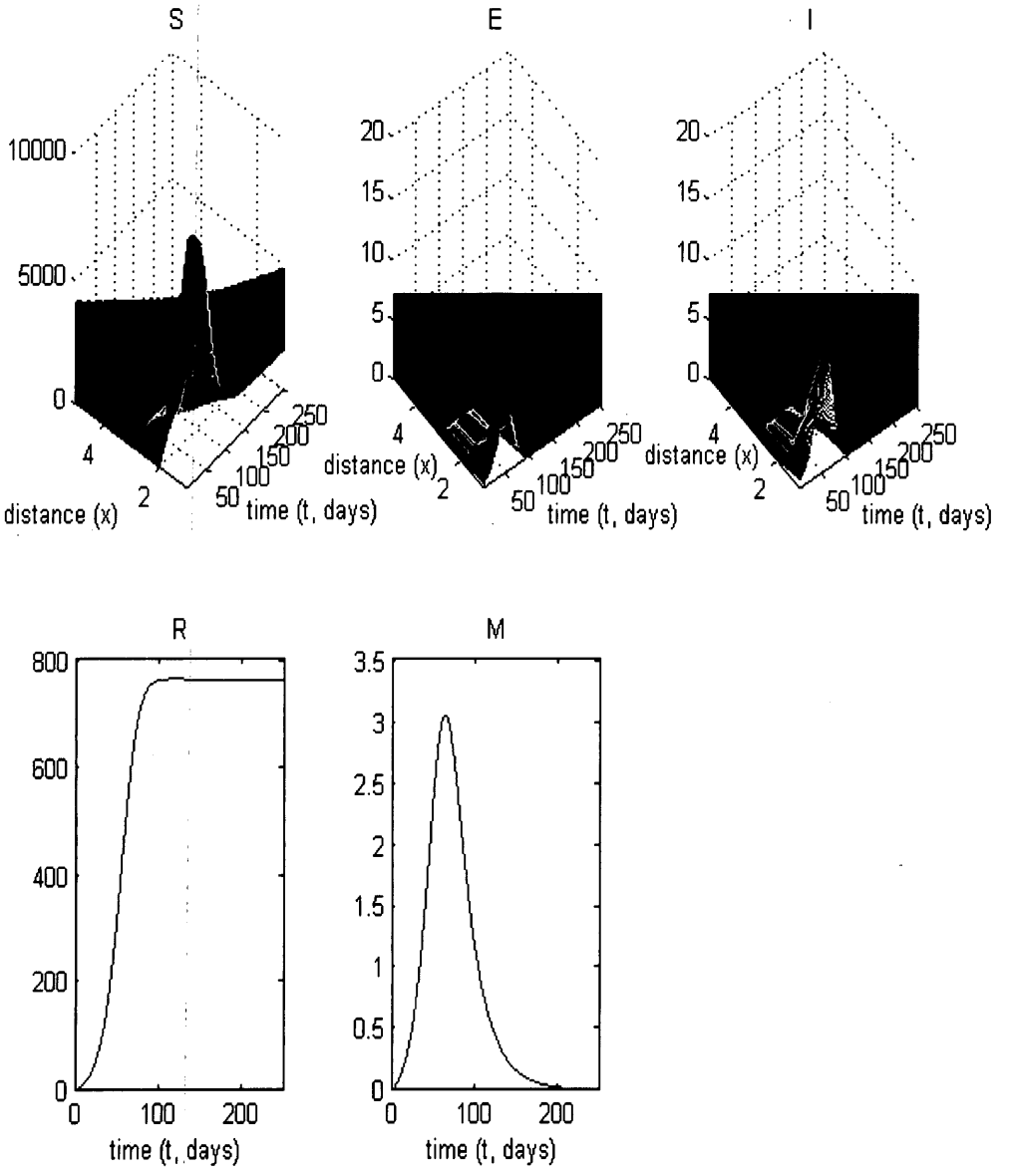

Figure 6.4: Model (6.4) — Social distancing and media boredom and a decreasing exponential function for $\beta(x)$. The colours help to show a decrease in the population in time and space.

When mass media boredom is considered and there is a reduction in the amount a story is reported, there are negative consequences for the population: the number of individuals who become ill is higher as seen by the individuals in the R class at the end of the epidemic in Figure (6.3) compared with Figure (6.2).

Practicing social distancing will decrease the transmission rate of influenza. To incorporate this, we considered a decreasing exponential function for the contact transmission rate. The function captures the fact that the more social distancing an individual practices, the lower the rate of influenza transmission. The resulting epidemic, Figure (6.4), with $\beta(x) = \beta e^{-rhox}$ has less of an effect on the population, even with the inclusion of media waning, than without this function used for a decrease in transmissibility. Since possible function structures for other parameters, like $\alpha(x)$ is unknown, we included only a function for $\beta(x)$.

From the numerical solutions to the models presented in this chapter we can determine that including social distancing as a method of combating epidemic spread that is informed by mass media reports may be beneficial to the population. We also notice that the social distance the susceptible individuals are from the 'normal' population of susceptibles is approximately three units. Model (4.7) with two socially distanced classes seems to be a fair representation of the PDE model above for population behaviour. It is important that the media and the population do not become bored with the story too early or the epidemic may be more severe

than without media boredom. It appears it may be necessary, in the future, to find a balance for mass media reporting that remains beneficial to the population for the duration of the epidemic. As an extension to the current numerical solution to the PDE, Model (6.5), we can take a further look into numerical methods to solve this system. The solution of this system appears to demonstrate isolated pulse behaviour. It may be beneficial to look at a solution in the neighbourhood of the pulse so that only local values appear in the equation. The optimal way to solve this would be by deriving a solution using coupled-mode theory and the Nyquist sampling frequency. Another possibility is to incorporate the mass media data from GPHIN as in Chapter 5 into the mass media equation in Models (6.4) and (6.5). Furthermore, social distancing and media influenced movement within each of the exposed and infectious classes may also have an effect on the epidemic. Finally, considering different function structures for $\beta(x)$ and for $\alpha(x)$ could provide further insight into how mass media affects epidemic progression. These suggested projects are left for future work.

7 Conclusion and Future work

The primary goal of this work was to study the effect of mass media on an epidemic and to be able to provide information about key measurements that are important to public health: peak epidemic time, peak epidemic magnitude, epidemic end time and total number of infected individuals after the epidemic. Previous mathematical models incorporating mass media were studied, a novel incorporation of mass media was introduced, mass media data collected by the Global Public Health Information Network was used to inform our novel media model equation and a system of PDEs was proposed in order to look at continuous population for social distancing.

In Chapter 3, three key functions were identified that have been previously used to incorporate media. In order to compare these functions, each was used in a standard SEIR model with constant population. It was demonstrated that the media parameter, p_1 and p_2, in Functions (3.1) and (3.2), respectively, could be written in terms of p_3, from function (3.3). With these three functions and I_c, which could be determined from epidemic data, we were able to see that the incorporation of mass media as a specified function does affect an epidemic. For the model with Functions (3.1-3.3), an Agent-based Monte Carlo simulation was implemented in order to look at variation in the previously specified key measurements of public health.

The epidemic curves produced by Functions (3.1-3.3) were different. The epi-

demics also changed depending on the values of p_i and I_c. The variability around the epidemic curves was displayed by the ABMC simulations. The variability around the mean of the simulations also changed depending on the function that was used to represent media and the value of I_c. A sensitivity analysis by LHS-PRCC was completed for Functions (3.1-3.3) within Model (3.5). Sensitivity analysis of this type has not previously been performed on models with the types of functions summarized in Chapter 3. Outcomes that are of interest to public health — peak epidemic time, peak number of infectious and exposed individuals, epidemic end time and total number of individuals — were investigated to see which parameters affect these outcomes. From the sensitivity analysis we were able to identify which parameters are most influential for the outcomes considered.

In studying Functions (3.1-3.3) as a method of incorporating mass media into a standard epidemic model, it became obvious that the form of the function influences the epidemic similar to the fact that the media themselves influence an epidemic. The inclusion of media as a function suggests that it continues on a similar path over time whereas, we are aware that it develops dynamically as events in the public occur.

In Chapter 4, a novel method of media incorporation was proposed. A compartment for mass media was added to Model (1.1). Compartments that represent one and two levels were also added to look at how distancing oneself as a suscep-

tible from the susceptible population may affect an influenza epidemic. The mass media equation acts to move individuals from one level of susceptibles to a socially distanced class of susceptible individuals or to a vaccinated class. Social distancing is only considered to be practiced by susceptible individuals. A natural extension to the models of Chapter 4 would be to include social distancing for the exposed and recovered classes.

While looking at incorporating how the growth of media during an epidemic affects it, it was posited that it is possible for the impact or effect of mass media to decline or wane over time. Media effect waning has been studied in cases concerning media violence and social issues, [14, 17, 33], but not in the context of an infectious illness like influenza. Chapter 3 saw the incorporation of a media waning term in a standard model with media in the equation used to incorporate media and in a term that allows for the relaxation of social distancing practices in the equation of the most socially distanced individuals to a less isolated group, model (4.7).

For the models in Chapter 4, agent-based Monte Carlo simulations were again completed to shed light on key measurements for public health through looking at variability measurements. Stochastic differential equation models were also developped for the models of Chapter 4. In developing a system of stochastic differential equations for moments it was evident that the system could not be solved without closing it as equations were written in terms of higher order moments. Traditional

methods of moment closure, [39, 34, 30, 28, 29], suggest to equate higher order moments to zero as they minimally influence the behaviour around the mean. In the case of our models, equating these moments to zero did not sufficiently account for the behaviour around the mean of the curves. It was necessary to look elsewhere. In [55], the authors developed a novel method of moment closure for higher order moments that estimated the higher order moment as a product of lower order moments. This was the method employed in Chapter 3 in order to close our systems of stochastic differential equations and solve them.

Sensitivity analysis by LHS-PRCC was used in Chapter 4 to provide insight into which parameters affect various outcomes of interest to public health. We were able to identify that for outcomes concerning peak magnitude, peak time and total number of infectious individuals different aspect of mass media are influential.

In Chapter 5, we looked at the inclusion of data to inform the media compartment of Model (4.7). The authors of [56] collected media data in twelve languages and looked at media decay. The data found therein was fit with an exponential decay function and the decay rate obtained was used in Model (4.7) as the media boredom rate. The deterministic results with this data informed parameter were similar to the results of the same model with a rate not informed by data suggesting that Model (4.7) may be an accurate way to incorporate mass media in epidemic modelling.

Canadian media data became available for use during the course of this study. The Global Public Health Information Network (GPHIN) collected media data during the H1N1 influenza pandemic of 2009, in six different languages. This data was collected from March 1, 2009, to December 27, 2009. This data was used as the media equation in the second part of the investigation of Chapter 4. With this media data, a two wave epidemic occurred with epidemic peaks occurring at the same time in the model as the time of peaks from the FluWatch data for the 2009 influenza season [49].

In Chapter 6, a system of partial differential equations is proposed in order to study a continuous population distribution for the susceptible, exposed and infectious individuals when influenced by mass media. The previous models have only two compartments of social distancing for the susceptible individuals and do not consider social distancing for the exposed and infectious individuals. The results of model (6.4) demonstrate a continuum of socially distanced individuals in the susceptible classes. From these results, it appears that after a 'distance' of three units from the unaffected level of individuals, the number of susceptibles becomes small. This suggests that the inclusion of two levels of social distancing as in model (4.7) represents the population distribution of susceptible individuals. We have not included movement in social distancing within the exposed and infectious classes.

A function for $\beta(x)$, the contact transmission rate, was also considered with the media waning model in Chapter 6. This function accounts for the waning transmission rate that occurs when individuals practice social distancing. We found that including a waning transmission rate, that decreases with the increase of social distancing, has major effects on the epidemic outcome. With a waning transmission rate, the epidemic has much less of an effect on the population.

From the results in this work, it is observed that mass media affects epidemic outcomes and key epidemic measurements. We saw in Chapter 3 that the way media has been included in epidemic models in the past was not consistent and resulted in very different outcomes depending on the way it was included. In Chapter 4, a novel method of inclusion was proposed that allowed mass media to develop with the epidemic since media is not static. Media waning was also considered as part of the influence of an epidemic. Stochastic models were considered for all the models of Chapters 3 and 4 in order to better extract pertinent information from the results. We also saw in Chapter 4 that when social distancing and vaccination are used together as a prevention strategy, it is the most effective. Media waning reduced the effectiveness of this strategy. The results of Chapter 5 confirmed that media waning does reduce the effectiveness of prevention measures. The variation in media of media reports kept constant until the very end of the epidemic resulted in a decrease in the length of the epidemic compared to the other scenarios considered.

Even though the differences in the media data implemented almost near the end of the epidemic they did make a difference in epidemic length. The results of the PDE models of Chapter 6 further demonstrate the results that were observed in the previous chapters: including mass media and social distancing decreases the severity of an epidemic but if the media wanes, the effect a pandemic has on the population can worsen.

It is necessary to keep in mind the behaviour of mass media during an epidemic. Media outlets need to be mindful not to become bored with reporting about the epidemic as it seems to be necessary to keep some level of media awareness in order for the public to continue practicing public health measures for prevention. Mass media seems to play an important role in spreading awareness to the population.

7.1 Summary of Contribution

The novel contributions of this thesis are the summary of previous methods that have been used to incorporate mass media in compartmental models, studying these methods with ABMC simulations and completing LHS-PRCC sensitivity analysis in order to identify parameters in the models that have a significant effect on various outcomes. A novel method of media inclusion in a compartmental model was developed and the waning of mass media and social distancing was considered. This novel model was studied through ODE, SDE and ABMC, and key parameters

were identified through LHS-PRCC. Mass media data collected by two sources was incorporated into the novel mass media model. The first PDE model to incorporate mass media and social distancing as a continuum was proposed.

The main results of this thesis come from studying the novel model that was proposed in Chapter 4. The novel media model allowed us to see that employing various preventative measures can decrease the length and severity of a pandemic but if media boredom occurs, the severity of the epidemic can increase and the number of individuals who receive vaccination may decrease. The inclusion of media data from GPHIN for the media equation of Model (4.7) resulted in a two wave pandemic, which is what is typically seen in an epidemic or pandemic situation. The numerical solutions to the first PDE models to include mass media in an epidemic confirmed the results that the addition of interventions during a pandemic can decrease its severity but media waning can slow the gains achieved by various intervention measures.

7.2 Limitations and Future Work

There are multiple extensions to this project to consider. It is of interest to include the media data from GPHIN into an agent-based Monte Carlo simulation in place of the media compartment. It has also been suggested to research a sort-of media threshold. In the current models media waning occurs as soon as there is

a population of media reports. It is possible that this decay does not occur until a certain level of media infusion has been reached. Also, this type of inclusion of mass media, through the inclusion of an extra differential equation in a standard model, can be implemented in studies of other diseases.

The thesis looked at the effect of mass media on decreasing the transmission of influenza in the population. It is also possible that mass media can increase the mixing of susceptible individuals with infectious individuals by encouraging emergency room visits of the population when they have symptoms of the pandemic illness. The increased mixing can lead to more individuals becoming ill and could cause another pandemic wave. We did not consider this possibility here.

This thesis also does not look at the age-structure that is involved with influenza and vaccination. Seasonal influenza disproportionately attacks the very young and the elderly. It is also known that the elderly are more willing to vaccinate against influenza and that the influenza vaccine is not perfectly effective. These issues could be incorporated into the models through using age structure and with the addition of terms that represent effectiveness of the vaccine.

When an individual becomes ill, it is likely there is social distancing practiced within the home. Social distancing practices within the home are not considered here but could be through the inclusion of household structure in our models.

Pandemic influenza was considered throughout this thesis for a population of

10,000 individuals. It is possible to adapt the work of the thesis to consider seasonal influenza by varying the media parameter ρ of Model (4.7). Small populations can also be considered within this model by using ABMC simulations.

It was outside the scope of this thesis to derive systems of stochastic differential equations for model (3.5) functions (3.1-3.3). In order to do this it is necessary to be able to calculate the the variance-covariance matrices without time dependence. It is also possible to do further analysis on the functions in Chapter 3 and consider other models that may produce two waves. A comprehensive stability analysis on the functions presented may provide further insight to various situations these functions may represent.

In this thesis media was considered to be mass media — newspaper and television — reported about H1N1 influenza pandemic in 2009: there is no distinction between negative media reports and positive media reports and there is no distinction between reports about the general epidemic and reports about prevention. It seems that considering different types of media reports or reports from acquaintances via social media, Facebook or Twitter, may have different influences on an epidemic. For instance, if there are negative reports about vaccination, individuals may be less likely to vaccinate due to 'rational exemption' [13].

A partial differential equation model was proposed in Chapter 6, this is the current direction of future projects. In addition to looking at further epidemic situ-

ations, it is of interest to look further into the numerical properties of this problem. The solution of this system appears to demonstrate isolated pulse behaviour. It may be beneficial to look at a solution in the neighbourhood of the pulse so that only local values appear in the equation. The optimal way to solve this would be by deriving a solution using coupled-mode theory and the Nyquist sampling frequency. Another direction for this project is to consider different function structures for the contact transmission rate and the social distancing rate. We did consider one function for $\beta(x)$ but since the structure of these functions, $\beta(x)$ and $\alpha(x)$, are unknown it would be of interest to research the structure further and see the effect of different functions on the outcome of an epidemic.

A Appendix A: Information for Chapter 3

A.1 Basic Reproductive Ratio

In Chapter 2, the basic reproductive ratio, R_0 for model (3.5) with function (3.1)was briefly calculated. Here we look at the in depth calculation for the basic reproductive ratio for model (3.5) for each of functions (3.1-3.3).

There are two ways to calculate the basic reproductive ratio, the survival function method and the next generation matrix method. Here, the next generation method is used. A good overview of both methods can be found in [24].

To calculate R_0 by the next generation method, it is necessary to look at the equations the spread the infection the exposed and infectious classes:

$$\begin{aligned} \dot{E} &= f(I,\beta,p)SI - \sigma E \\ \dot{I} &= \sigma E - \gamma I. \end{aligned} \tag{A.1}$$

To make the next generation operator, FV^{-1}, we need to make the jacobian matrices F and V. The matrix F is the matrix with the terms for the appearance of new infections: the appearance of new infections happen with $f(I,\beta,p)SI$. The matrix F is:

$$F = \begin{pmatrix} 0 & \frac{\partial f(I,\beta,p)SI}{\partial I} \\ 0 & 0 \end{pmatrix}, \tag{A.2}$$

which is evaluated at the disease-free equilibrium. The disease free equilibrium is found by setting each of the equations in model (3.5) to zero and solving. In this case, the disease-free equilibrium is: $(S,E,I,R) = (N,0,0,0)$ where N is the total

population. If we substitute function (3.1), $f(I,\beta,p) = \beta e^{-p_1\gamma I}$ for $f(I,\beta,p)$ in matrix (A.2) and evaluate at the disease free equilibrium, we get:

$$F = \begin{pmatrix} 0 & \beta N \\ 0 & 0 \end{pmatrix}. \tag{A.3}$$

We can do the same thing for function (3.2),

$f(I,\beta,p) = \beta\left(\frac{1}{1+p_2 I^2}\right)$, which results in a matrix F of:

$$F = \begin{pmatrix} 0 & \beta N \\ 0 & 0 \end{pmatrix}. \tag{A.4}$$

Finally, repeating the derivatives and substitution for function (3.3),

$f(I,\beta,p) = \left(\beta - \beta\frac{I}{p_3+I}\right)$, the matrix F is:

$$F = \begin{pmatrix} 0 & \beta N \\ 0 & 0 \end{pmatrix}. \tag{A.5}$$

The second part of the next generation operator is matrix V. The matrix V is the matrix that accounts for the transfer of infectious individuals. The terms of equations (A.1) that account for this are σE and γI. The matrix V is:

$$V = \begin{pmatrix} -\frac{\partial \sigma E}{\partial E} & 0 \\ -\frac{\partial \sigma I}{\partial I} & -\frac{\partial \gamma I}{\partial I} \end{pmatrix}. \tag{A.6}$$

The terms that move the infectious individuals are the same for the equations (A.1)

for each of the functions. The resulting V matrix is

$$V = \begin{pmatrix} \sigma & 0 \\ -\sigma & \gamma \end{pmatrix}. \tag{A.7}$$

The next step is to take the inverse of V and determine the spectral radius, which is R_0, of the operator FV^{-1}. Since each matrix F for the individual functions turns out to be the same, the operator FV^{-1} is:

$$FV^{-1} = \begin{pmatrix} \frac{\beta N}{\gamma} & \frac{\beta N}{\gamma} \\ 0 & 0 \end{pmatrix}. \tag{A.8}$$

It is obvious that the spectral radius is $\frac{\beta N}{\gamma}$, so $R_0 = \frac{\beta N}{\gamma}$, for model (3.5) for each of the functions (3.1-3.3) in Chapter 2.

A.2 Means, Standard errors and Standard deviations

In order to verify the accuracy of the agent-based Monte Carlo simulations and compare the behaviour of the simulations with a system of ordinary differential equations we calculate the mean of the simulations and the standard error of the mean. Since the underlying distribution of the agent-based Monte Carlo simulation is assumed as exponential the means of the simulations should produce approximately the same results as the ordinary differential equation models.

To calculate the mean, the results at each time point are summed and divided

by the total number of simulations, equation (A.9),

$$\bar{x} = \frac{1}{N}\sum_{i=1}^{N} x_i, \tag{A.9}$$

where $\bar{x}$ is the mean of a simulation, x_i is the result of a simulation at the i^{th} time point and N is the total number of simulations. With the mean, there is also a calculation of the standard error.

The standard error of the mean is an estimate of the standard deviation of the sample mean. It is considered an unbiased estimator. This is calculated for the sample means of the agent-based Monte Carlo simulations. A sample calculation of the standard error is in equation (A.10),

$$SE_{\bar{x}} = \frac{s}{\sqrt{(n)}}, \tag{A.10}$$

where s is the sample standard deviation and n is the number of observations in the sample. The equation for the sample standard deviation is equation (A.11),

$$s_N = \sqrt{\frac{1}{N}\sum_{i=1}^{N}(x_i - \overline{(x)})^2}, \tag{A.11}$$

where $\bar{x}_N$ is the mean value of the observations, N is the sample size and x_i is the observation being considered at point i.

B Appendix B: Information for Chapter 4

B.1 Systems of stochastic differential equations

A system of stochastic differential equations was derived for each of the models in Chapter 3. The procedure presented in Chapter 3 is followed for each of the systems therein. The resulting systems of stochastic differential equations are presented here.

The first system of stochastic differential equations is for model (4.2):

$$\frac{dE[S]}{dt} = \lambda - \beta E[SI] - \alpha E[SM] - dE[S]$$

$$\frac{dE[S_1]}{dt} = -\beta E[S_1 I] + \alpha E[SM] - dE[S_1]$$

$$\frac{dE[E]}{dt} = \beta E[SI] + \beta_1 E[S_1 I] - \sigma E[E] - dE[E]$$

$$\frac{dE[I]}{dt} = \sigma E[E] - \gamma E[I] - dE[I]$$

$$\frac{dE[R]}{dt} = \gamma E[I] - dE[R]$$

$$\frac{dE[M]}{dt} = \rho\sigma E[SM]$$

$$\frac{dE[SS]}{dt} = -\lambda E[S] + 2\lambda E[SS] + \beta E[SI] - 2\beta E[SIS] + \alpha E[SM]$$
$$- 2\alpha E[SMS] + dE[S] - 2dE[SS]$$

$$\frac{dE[S_1 S_1]}{dt} = \alpha E[SM] + 2\alpha E[SMS_1] + \beta_1 E[S_1 I] - 2\beta_1 E[S_1 I S_1] + dE[S_1]$$
$$- 2dE[S_1 S_1]$$

$$\frac{dE[EE]}{dt} = \beta E[SI] + 2\beta E[SEI] + \beta_1 E[S_1 I] + 2\beta_1 E[EIS_1] + \sigma E[E]$$
$$- 2\sigma E[EE] + dE[E] - 2dE[EE]$$

$$\frac{dE[II]}{dt} = \sigma E[E] + 2\sigma E[EI] + \gamma E[I] - 2\gamma E[II] + dE[I] - 2dE[II] \qquad \text{(B.1)}$$

$$\frac{dE[SI]}{dt} = \lambda E[SI] - \beta E[IIS] - \alpha E[SIM] - 2dE[SI] + \sigma E[SE] - \gamma E[SI]$$

$$\frac{dE[S_1I]}{dt} = \alpha E[SIM] - \beta_1 E[IIS_1] - 2dE[S_1I] + \sigma E[S_1E] - \gamma E[S_1I]$$

$$\frac{dE[EI]}{dt} = \beta E[IIS] - \sigma E[E] - \sigma E[EI] + \sigma E[EE] - 2dE[EI] - \gamma E[EI]$$

$$\frac{dE[SE]}{dt} = \lambda E[SE] - \beta E[SI] - \beta E[SEI] + \beta E[SIS] - \alpha E[MES]$$
$$- 2dE[SE] + \beta_1 E[SIS_1] - \sigma E[SE]$$

$$\frac{dE[S_1E]}{dt} = \beta E[SIS_1] + \alpha E[MES] - \beta_1 E[S_1I] - \beta_1 E[EIS_1] + \beta_1 E[S_1IS_1]$$
$$- 2dE[S_1E] - \sigma E[S_1E]$$

$$\frac{dE[SM]}{dt} = \lambda E[SM] - \beta E[SIM] - \alpha E[SMM] - dE[SM] + \rho\sigma E[SMS]$$

$$\frac{dE[S_1M]}{dt} = \alpha E[SMM] - \beta_1 E[IMS_1] - dE[S_1M] + \rho\sigma E[SMS_1]$$

$$\frac{dE[IM]}{dt} = \sigma E[ME] - \gamma E[IM] - dE[IM] + \rho\sigma E[SIM]$$

$$\frac{dE[SS_1]}{dt} = \lambda E[SS_1] - \beta E[SIS_1] - \alpha E[SM] - \alpha E[SMS_1] + \alpha E[SMS]$$
$$- 2dE[SS_1] - \beta_1 E[SIS_1]$$

$$\frac{dE[ME]}{dt} = \beta E[SIM] + \beta_1 E[IMS_1] - \sigma E[ME] - dE[ME] + \rho\sigma E[MES]$$

$$\frac{dE[MM]}{dt} = -\rho\sigma E[SM] + 2\rho\sigma E[SMM].$$

The SDE for model (4.3) is

$$\frac{dE[S]}{dt} = \lambda - \beta E[SI] - \nu E[SM] - dE[S]$$

$$\frac{dE[V]}{dt} = \nu E[SM] - dE[V]$$

$$
\begin{aligned}
\frac{dE[E]}{dt} &= \beta E[SI] - \sigma E[E] - dE[E] \\
\frac{dE[I]}{dt} &= \sigma E[E] - \gamma E[I] - dE[I] \\
\frac{dE[R]}{dt} &= \gamma E[I] - dE[R] \\
\frac{dE[M]}{dt} &= \rho\sigma E[E] \\
\frac{dE[SS]}{dt} &= \lambda + 2\lambda E[S] + \beta E[SI] - 2\beta E[SIS] + \nu E[SM] - 2\nu E[SMS] \\
&\quad + dE[S] - 2dE[SS] \\
\frac{dE[EE]}{dt} &= \beta E[SI] + 2\beta E[SEI] + \sigma E[E] - 2\sigma E[EE] + dE[E] - 2dE[EE] \\
\frac{dE[II]}{dt} &= \sigma E[E] + 2\sigma E[EI] + \gamma E[I] - 2\gamma E[II] + dE[I] - 2dE[II] \qquad \text{(B.2)} \\
\frac{dE[VV]}{dt} &= \alpha E[SM] + 2\alpha E[SMV] + \beta_1 E[VI] - 2\beta_1 E[VIV] + dE[V] \\
&\quad - 2dE[VV] \\
\frac{dE[SI]}{dt} &= \lambda E[I] - \beta E[IIS] - \nu E[SIM] - 2dE[SI] + \sigma E[SE] - \gamma E[SI] \\
\frac{dE[EI]}{dt} &= \beta E[IIS] - \sigma E[E] - \sigma E[EI] + \sigma E[EE] - 2dE[EI] - \gamma E[EI] \\
\frac{dE[SE]}{dt} &= \lambda E[E] - \beta E[SI] - \beta E[SEI] + \beta E[SIS] - \nu E[MES] \\
&\quad - 2dE[SE] - \sigma E[SE] \\
\frac{dE[SM]}{dt} &= \lambda E[M] - \beta E[SIM] - \nu E[SMM] - dE[SM] + \rho\sigma E[SE] \\
\frac{dE[IM]}{dt} &= \sigma E[ME] - \gamma E[IM] - dE[IM] + \rho\sigma E[EI] \\
\frac{dE[ME]}{dt} &= \beta E[SIM] - \sigma E[ME] - dE[ME] + \rho\sigma E[EE] \\
\frac{dE[MM]}{dt} &= \rho\sigma E[E] + 2\rho\sigma E[ME]
\end{aligned}
$$

$$\frac{dE[VS]}{dt} = \lambda E[V] - \beta E[SIV] - \nu E[S] + \nu E[SS] - \nu E[VS] - 2dE[VS]$$
$$\frac{dE[IV]}{dt} = \nu E[SI] + \sigma E[VE] - \gamma E[IV] - 2dE[IV]$$
$$\frac{dE[VE]}{dt} = \beta E[SIV] + \nu E[SE] - \sigma E[VE] - 2dE[VE]$$
$$\frac{dE[VM]}{dt} = \alpha E[SMM] - \beta_1 E[IMV] - dE[VM] + \rho\sigma E[VE].$$

The system of SDEs for model (4.4) is

$$\frac{dE[S]}{dt} = \lambda - \beta E[SI]\alpha E[SM] - \nu E[SM] - dE[S]$$
$$\frac{dE[S_1]}{dt} = -\beta E[S_1 I] + \alpha E[SM] - \nu_1 E[S_1 M] - dE[S_1]$$
$$\frac{dE[V]}{dt} = \nu E[SM] + \nu_1 E[S_1 M] - dE[V]$$
$$\frac{dE[E]}{dt} = \beta E[SI] + \beta_1 E[S_1 I] - \sigma E[E] - dE[E]$$
$$\frac{dE[I]}{dt} = \sigma E[E] - \gamma E[I] - dE[I]$$
$$\frac{dE[R]}{dt} = \gamma E[I] - dE[R]$$
$$\frac{dE[M]}{dt} = \rho\sigma E[E]$$
$$\frac{dE[SS]}{dt} = 2\lambda E[S] - 2\beta E[SIS] - 2\alpha E[SMS] - 2\nu E[SMS] - 2dE[SS]$$
$$+ \lambda + \beta E[SI] + \alpha E[SM] + \nu E[SM] + dE[S]$$
$$\frac{dE[S_1 S_1]}{dt} = \alpha E[SM] + 2\alpha E[SMS_1] + \beta_1 E[S_1 I] - 2\beta_1 E[S_1 I S_1]$$
$$+ \nu_1 E[S_1 M] - 2\nu_1 E[S_1 M S_1] + dE[S_1] - 2dE[S_1 S_1]$$
$$\frac{dE[EE]}{dt} = 2\beta E[SEI] + 2\beta_1 E[EIS_1] - 2\sigma E[EE] - 2dE[EE] + \sigma E[E]$$
$$+ dE[E] + \beta_1 E[S_1 I] + \beta E[SI]$$

$$\frac{dE[II]}{dt} = \sigma E[E] + 2\sigma E[EI] + \gamma E[I] - 2\gamma E[II] + dE[I] - 2dE[II] \tag{B.3}$$

$$\frac{dE[SI]}{dt} = \lambda E[I] - \beta E[IIS] - \alpha E[SIM] - \nu E[SIM] - 2dE[SI]$$
$$+ \sigma E[SE] - \gamma E[SI]$$

$$\frac{dE[S_1I]}{dt} = \alpha E[SIM] - \beta_1 E[IIS_1] - \nu_1 E[IMS_1] - 2dE[S_1I]$$
$$+ \sigma E[S_1E] - \gamma E[S_1I]$$

$$\frac{dE[EI]}{dt} = \beta E[IIS] + \beta_1 E[IIS_1] - \sigma E[E] - \sigma E[EI] + \sigma E[EE]$$
$$- 2dE[EI] - \gamma E[EI]$$

$$\frac{dE[SE]}{dt} = \lambda E[E] - \beta E[SI] - \beta E[SEI] + \beta E[SIS] - \alpha E[MES]$$
$$- \nu E[MES] - 2dE[SE] + \beta_1 E[SIS_1] - \sigma E[SE]$$

$$\frac{dE[S_1E]}{dt} = \beta E[SIS_1] + \alpha E[MES] - \beta_1 E[S_1I] - \beta_1 E[EIS_1] + \beta_1 E[S_1IS_1]$$
$$- 2dE[S_1E] - \sigma E[S_1E] - \nu E[MES_1]$$

$$\frac{dE[SM]}{dt} = \lambda E[M] - \beta E[SIM] - \alpha E[SMM] - \nu E[SMM] - dE[SM] + \rho\sigma E[SE]$$

$$\frac{dE[S_1M]}{dt} = \alpha E[SMM] - \beta_1 E[IMS_1] - \nu_1 E[S_1MM] - dE[S_1M] + \rho\sigma E[S_1E]$$

$$\frac{dE[IM]}{dt} = \sigma E[ME] - \gamma E[IM] - dE[IM] + \rho\sigma E[EI]$$

$$\frac{dE[SS_1]}{dt} = \lambda E[S_1] - \beta E[SIS_1] - \alpha E[SM] - \alpha E[SMS_1] + \alpha E[SMS]$$
$$- \nu E[SMS_1] - \nu_1 E[SMS_1] - 2dE[SS_1] - \beta_1 E[SIS_1]$$

$$\frac{dE[ME]}{dt} = \beta E[SIM] + \beta_1 E[IMS_1] - \sigma E[ME] - dE[ME] + \rho\sigma E[EE]$$

$$\frac{dE[MM]}{dt} = \rho\sigma E[E] + 2\rho\sigma E[ME].$$

The SDE model derived for model (4.5) is:

$$\frac{dE[S]}{dt} = \lambda - \beta E[SI] - \alpha E[SM] - \nu E[SM] - dE[S]$$

$$\frac{dE[S_1]}{dt} = -\beta_1 E[S_1 I] - \nu_1 E[S_1 M] - \alpha_1 E[S_1 M] - \alpha E[SM]$$

$$- dE[S_1] + qE[S_2]$$

$$\frac{dE[S_2]}{dt} = \alpha_1 E[S_1 M] - \nu_2 E[S_2 M] - qE[S_2] - dE[S_2]$$

$$\frac{dE[V]}{dt} = \nu E[SM] + \nu_1 E[S_1 M] + \nu_2 E[S_2 M] - dE[V]$$

$$\frac{dE[E]}{dt} = \beta E[SI] + \beta_1 E[S_1 I] - \sigma E[E] - dE[E]$$

$$\frac{dE[I]}{dt} = \sigma E[E] - \gamma E[I] - dE[I]$$

$$\frac{dE[R]}{dt} = \gamma E[I] - dE[R]$$

$$\frac{dE[M]}{dt} = \rho\sigma E[E]$$

$$\frac{dE[SS]}{dt} = 2\lambda E[S] - 2\beta E[SIS] - 2\alpha E[SMS] - 2\nu E[SMS]$$

$$- 2dE[SS] + \lambda + \beta E[SI] + \alpha E[SM] + \nu E[SM] + dE[S]$$

$$\frac{dE[S_1 S_1]}{dt} = \alpha E[SM] + 2\alpha E[SMS_1] + \beta_1 E[S_1 I] - 2\beta_1 E[S_1 I S_1]$$

$$+ \alpha_1 E[S_1 M] - 2\alpha_1 E[S_1 M S_1] + \nu_1 E[S_1 M] - 2\nu_1 E[S_1 M S_1]$$

$$+ 2qE[S_1 S_2] + qE[S_2] + dE[S_1] - 2dE[S_1 S_1]$$

$$\frac{dE[S_2 S_2]}{dt} = dE[S_2] + \alpha_1 E[S_1 M] + qE[S_2] - 2dE[S_2 S_2] - 2qE[S_2 S_2]$$

$$+ 2\alpha_1 E[S_1 M S_2] + \nu_2 E[S_2 M] - 2\nu_2 E[S_2 M S_2]$$

$$\frac{dE[EE]}{dt} = 2\beta E[SEI] + 2\beta_1 E[EIS_1] - 2\sigma E[EE] - 2dE[EE] + \sigma E[E]$$

$$
\begin{aligned}
&+ dE[E] + \beta_1 E[S_1 I] + \beta E[SI] \\
\frac{dE[II]}{dt} &= \sigma E[E] + 2\sigma E[EI] + \gamma E[I] - 2\gamma E[II] + dE[I] - 2dE[II] \\
\frac{dE[SI]}{dt} &= \lambda E[I] - \beta E[IIS] - \alpha E[SIM] - \nu E[SIM] - 2dE[SI] \\
&+ \sigma E[SE] - \gamma E[SI] \\
\frac{dE[S_1 I]}{dt} &= qE[S_2 I] + \alpha E[SIM] - \beta_1 E[IIS_1] - \alpha_1 E[IMS_1] - \nu_1 E[IMS_1] \\
&- 2dE[S_1 I] + \sigma E[S_1 E] - \gamma E[S_1 I] \\
\frac{dE[EI]}{dt} &= \beta E[IIS] + \beta_1 E[IIS_1] - \sigma E[E] - \sigma E[EI] + \sigma E[EE] \\
&- 2dE[EI] - \gamma E[EI] \\
\frac{dE[SE]}{dt} &= \lambda E[E] - \beta E[SI] - \beta E[SEI] + \beta E[SIS] - \alpha E[MES] \\
&- \nu E[MES] - 2dE[SE] + \beta_1 E[SIS_1] - \sigma E[SE] \qquad \text{(B.4)} \\
\frac{dE[S_1 E]}{dt} &= \beta E[SIS_1] + \alpha E[MES] - \beta_1 E[S_1 I] - \beta_1 E[EIS_1] + \beta_1 E[S_1 IS_1] \\
&- 2dE[S_1 E] - \sigma E[S_1 E] - \nu_1 E[MES_1] + qE[S_2 E] - \alpha_1 E[MES_1] \\
\frac{dE[SM]}{dt} &= \lambda E[M] - \beta E[SIM] - \alpha E[SMM] - \nu E[SMM] - dE[SM] \\
&+ \rho\sigma E[SE] \\
\frac{dE[S_1 M]}{dt} &= \alpha E[SMM] - \beta_1 E[IMS_1] - \nu_1 E[S_1 MM] - dE[S_1 M] \\
&+ \rho\sigma E[S_1 E] - \alpha_1 E[S_1 MM] + qE[S_2 M] \\
\frac{dE[IM]}{dt} &= \sigma E[ME] - \gamma E[IM] - dE[IM] + \rho\sigma E[EI] \\
\frac{dE[SS_1]}{dt} &= \lambda E[S_1] - \beta E[SIS_1] - \alpha E[SM] - \alpha E[SMS_1] + \alpha E[SMS]
\end{aligned}
$$

$$
\begin{aligned}
&- \alpha_1 E[SMS_1] - \nu E[SMS_1] - \nu_1 E[SMS_1] - 2dE[SS_1] \\
&- \beta_1 E[SIS_1] + qE[S_2S] \\
\frac{dE[ME]}{dt} &= \beta E[SIM] + \beta_1 E[IMS_1] - \sigma E[ME] - dE[ME] + \rho\sigma E[EE] \\
\frac{dE[MM]}{dt} &= \rho\sigma E[E] + 2\rho\sigma E[ME] \\
\frac{dE[S_2M]}{dt} &= \alpha_1 E[S_1MM] - qE[S_2M] - \nu_2 E[S_2MM] - dE[S_2M] + \rho\sigma E[S_2E] \\
\frac{dE[S_2E]}{dt} &= \alpha_1 E[MES_1] - qE[S_2E] - 2dE[S_2E] - \sigma E[S_2E] + \beta E[SIS_2] \\
&+ \beta_1 E[S_1IS_2] - \nu_2 E[S_2MM] \\
\frac{dE[S_2I]}{dt} &= \alpha_1 E[IMS_1] - qE[S_2I] - 2dE[S_2I] + \sigma E[S_2E] - \gamma E[S_2I] \\
&- \nu_2 E[IMS_2] \\
\frac{dE[S_2S]}{dt} &= -qE[S_2S] + \lambda E[S_2] - \beta E[SIS_2] - \alpha E[SMS_2] - \nu E[SMS_2] \\
&- 2dE[S_2S] + \alpha_1 E[SMS_1] - \nu_2 E[SMS_2] \\
\frac{dE[S_1S_2]}{dt} &= -qE[S_1S_2] + \alpha_1 E[S_1MS_1] - qE[S_2] + qE[S_2S_2] - \beta_1 E[S_1IS_2] \\
&- \alpha_1 E[S_1M] - 2dE[S_1S_2] - \alpha_1 E[S_1MS_2] - \nu_1 E[S_1MS_2] \\
&+ \alpha E[SMS_2] - \nu_2 E[S_1MS_2].
\end{aligned}
$$

The system of stochastic differential equations for model (4.7) is

$$
\begin{aligned}
\frac{dE[S]}{dt} &= \lambda - \beta E[SI] - \alpha E[SM] - \nu E[SM] - dE[S] \\
\frac{dE[S_1]}{dt} &= -\beta_1 E[S_1I] - \nu_1 E[S_1M] - \alpha_1 E[S_1M] - \alpha E[SM] - dE[S_1] + qE[S_2] \\
\frac{dE[S_2]}{dt} &= \alpha_1 E[S_1M] - \nu_2 E[S_2M] - qE[S_2] - dE[S_2]
\end{aligned}
$$

$$\frac{dE[V]}{dt} = \nu E[SM] + \nu_1 E[S_1 M] + \nu_2 E[S_2 M] - dE[V]$$

$$\frac{dE[E]}{dt} = \beta E[SI] + \beta_1 E[S_1 I] - \sigma E[E] - dE[E]$$

$$\frac{dE[I]}{dt} = \sigma E[E] - \gamma E[I] - dE[I]$$

$$\frac{dE[R]}{dt} = \gamma E[I] - dE[R]$$

$$\frac{dE[M]}{dt} = \rho\sigma E[E] - \rho_1 E[M]$$

$$\frac{dE[SS]}{dt} = 2\lambda E[S] - 2\beta E[SIS] - 2\alpha E[SMS] - 2\nu E[SMS] - 2dE[SS]$$
$$+ \lambda + \beta E[SI] + \alpha E[SM] + \nu E[SM] + dE[S]$$

$$\frac{dE[S_1 S_1]}{dt} = \alpha E[SM] + 2\alpha E[SMS_1] + \beta_1 E[S_1 I] - 2\beta_1 E[S_1 I S_1] + \alpha_1 E[S_1 M]$$
$$- 2\alpha_1 E[S_1 M S_1] + \nu_1 E[S_1 M] - 2\nu_1 E[S_1 M S_1] + 2qE[S_1 S_2]$$
$$+ qE[S_2] + dE[S_1] - 2dE[S_1 S_1]$$

$$\frac{dE[S_2 S_2]}{dt} = dE[S_2] + \alpha_1 E[S_1 M] + qE[S_2] - 2dE[S_2 S_2] - 2qE[S_2 S_2]$$
$$+ 2\alpha_1 E[S_1 M S_2] + \nu_2 E[S_2 M] - 2\nu_2 E[S_2 M S_2]$$

$$\frac{dE[EE]}{dt} = 2\beta E[SEI] + 2\beta_1 E[EIS_1] - 2\sigma E[EE] - 2dE[EE] + \sigma E[E]$$
$$+ dE[E] + \beta_1 E[S_1 I] + \beta E[SI]$$

$$\frac{dE[II]}{dt} = \sigma E[E] + 2\sigma E[EI] + \gamma E[I] - 2\gamma E[II] + dE[I] - 2dE[II]$$

$$\frac{dE[SI]}{dt} = \lambda E[I] - \beta E[IIS] - \alpha E[SIM] - \nu E[SIM] - 2dE[SI] + \sigma E[SE]$$
$$- \gamma E[SI]$$

$$\frac{dE[S_1 I]}{dt} = qE[S_2 I] + \alpha E[SIM] - \beta_1 E[IIS_1] - \alpha_1 E[IMS_1] - \nu_1 E[IMS_1]$$

$$
\begin{aligned}
&- 2dE[S_1I] + \sigma E[S_1E] - \gamma E[S_1I] \\
\frac{dE[EI]}{dt} &= \beta E[IIS] + \beta_1 E[IIS_1] - \sigma E[E] - \sigma E[EI] + \sigma E[EE] - 2dE[EI] \\
&- \gamma E[EI] \qquad \text{(B.5)} \\
\frac{dE[SE]}{dt} &= \lambda E[E] - \beta E[SI] - \beta E[SEI] + \beta E[SIS] - \alpha E[MES] \\
&- \nu E[MES] - 2dE[SE] + \beta_1 E[SIS_1] - \sigma E[SE] \\
\frac{dE[S_1E]}{dt} &= \beta E[SIS_1] + \alpha E[MES] - \beta_1 E[S_1I] - \beta_1 E[EIS_1] + \beta_1 E[S_1IS_1] \\
&- 2dE[S_1E] - \sigma E[S_1E] - \nu_1 E[MES_1] + qE[S_2E] - \alpha_1 E[MES_1] \\
\frac{dE[SM]}{dt} &= \lambda E[M] - \beta E[SIM] - \alpha E[SMM] - \nu E[SMM] - dE[SM] \\
&+ \rho\sigma E[SE] - \rho_1 E[SM] \\
\frac{dE[S_1M]}{dt} &= \alpha E[SMM] - \beta_1 E[IMS_1] - \nu_1 E[S_1MM] - dE[S_1M] \\
&+ \rho\sigma E[S_1E] - \alpha_1 E[S_1MM] + qE[S_2M] - \rho_1 E[S_1M] \\
\frac{dE[IM]}{dt} &= \sigma E[ME] - \gamma E[IM] - dE[IM] + \rho\sigma E[EI] - \rho_1 E[IM] \\
\frac{dE[SS_1]}{dt} &= \lambda E[S_1] - \beta E[SIS_1] - \alpha E[SM] - \alpha E[SMS_1] + \alpha E[SMS] \\
&- \alpha_1 E[SMS_1] - \nu E[SMS_1] - \nu_1 E[SMS_1] - 2dE[SS_1] \\
&- \beta_1 E[SIS_1] + qE[S_2S] \\
\frac{dE[ME]}{dt} &= \beta E[SIM] + \beta_1 E[IMS_1] - \sigma E[ME] - dE[ME] \\
&+ \rho\sigma E[EE] - \rho_1 E[MY] \\
\frac{dE[MM]}{dt} &= \rho\sigma E[E] + 2\rho\sigma E[ME] + \rho_1 E[M] - 2\rho_1 E[MM]
\end{aligned}
$$

$$\frac{dE[S_2M]}{dt} = \alpha_1 E[S_1MM] - qE[S_2M] - \nu_2 E[S_2MM] - dE[S_2M]$$

$$+ \rho\sigma E[S_2E] - \rho_1 E[S_2M]$$

$$\frac{dE[S_2E]}{dt} = \alpha_1 E[MES_1] - qE[S_2E] - 2dE[S_2E] - \sigma E[S_2E] + \beta E[SIS_2]$$

$$+ \beta_1 E[S_1IS_2] - \nu_2 E[S_2MM]$$

$$\frac{dE[S_2I]}{dt} = \alpha_1 E[IMS_1] - qE[S_2I] - 2dE[S_2I] + \sigma E[S_2E] - \gamma E[S_2I] - \nu_2 E[IMS_2]$$

$$\frac{dE[S_2S]}{dt} = -qE[S_2S] + \lambda E[S_2] - \beta E[SIS_2] - \alpha E[SMS_2] - \nu E[SMS_2]$$

$$- 2dE[S_2S] + \alpha_1 E[SMS_1] - \nu_2 E[SMS_2]$$

$$\frac{dE[S_1S_2]}{dt} = -qE[S_1S_2] + \alpha_1 E[S_1MS_1] - qE[S_2] + qE[S_2S_2] - \beta_1 E[S_1IS_2]$$

$$- \alpha_1 E[S_1M] - 2dE[S_1S_2] - \alpha_1 E[S_1MS_2] - \nu_1 E[S_1MS_2]$$

$$+ \alpha E[SMS_2] - \nu_2 E[S_1MS_2].$$

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