Three Models for Measles Control

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Abstract

We study the dynamics of three models consisting of infant vaccination and booster shots to control the spread of measles. Varying the rates of infant vaccination and administration of booster shots, we alter the characteristics of the basic reproductive number to study the effectiveness of both infant vaccination and booster shot control methods. In two models, we find that total effort on both birth vaccination and boosters lowers cases of recovered and infected individuals to possibly eradicate measles.

1 Introduction

Measles is one of the oldest known viruses to infect humans. It was first recorded in Arab Medical literature over 1000 years ago, but has been estimated to have had its effects for some 5000 years (Goldston, Gruemberg & Lewis, 1986, 4). Measles struck several civilizations: the Eastern Mediterranean civilizations, the Roman Empire, and the Central and South American civilizations. Measles, along with other viral infections introduced to the New World by the Europeans, killed over three and a half million natives and aided in the conquest of the New World. Even after a vaccine became licensed in 1963 to combat the virus, it was estimated in 1983 that among developing nations, approximately 900,000 lives were lost from measles infection each year (Anderson & May, 1991, 319). The effects of measles are still present after worldwide efforts to eradicate the virus.

Measles is from the species of the Morbillivirus genus of the Paramyxoviridae. The infection can be described in six stages. "The incubation period is the time interval from the moment of infection to the appearance of the first symptoms. This normally lasts from seven to eight
days. The onset is marked by a fever, respiratory catarrh and a harsh cough. The prodromal stage, which lasts about three to four days, is marked by an increase in catarrhal symptoms, a redness in the tonsils, and the appearance of Koplik’s spots (a diagnostic of measles). The rash stage is identified by an increase in temperature and an appearance of a measles rash. The rash usually starts behind the ears and along the hairline, quickly involves the face and spreads down to the body. The rash lasts around six days. The final stage, recovery, is marked by a return to regular temperature and the rash gradually fades away. The measles patient is then regarded as non-infectious” (Cliff, Haggett & Smallman-Raynor, 1993, 22-23). After one is infected by measles, the individual acquires permanent immunity. Several complications have been linked to the measles infection, such as subacute sclerosing panencephalitis, multiple sclerosis and mental disorders.

Although the number of measles cases has decreased drastically in the last half century, the virus persists. On January 1997, an outbreak was reported in British Columbia. Over 107 cases are linked to that outbreak, with a majority of the infected individuals between 19 and 29 years of age (Measles Update, vol. 5, April 1997). The question addressed here is, why does the measles virus continue to afflict so many people in this present time. Infection continues in developing countries as well as in first world countries. Eradication campaigns through immunization are much too difficult to administer in developing countries and therefore measles persists in developing countries. In the United States, there were efforts made in 1980 to “eliminate indigenous measles from the United States by October 1, 1982 ” (Cliff, Haggett & Smallman-Raynor, 1993, 217). “Although no date for global eradication is yet in prospect, the European Region of WHO (World Health Organization) has adopted a target of no indigenous measles by the year 2000” (Cliff, Haggett & Smallman-Raynor, 1993, 426). It is estimated by Anderson and May that in order to eradicate the measles virus, an immunization effort would have to immunize 90% to 95% of the world’s population (Anderson & May, 1991, p.88). Other studies conclude that in order for the population to have herd immunity (the degree of specific population immunity) from measles, 81.5% to 94% of the population would have to be vaccinated in the United States alone (Brauer & Castillo-Chavez, 1994, 12). The only virus for which herd immunity has had global success is smallpox.

In this study, we discuss various methods to continue reducing the spread of the measles virus. Through a model with age-structure, we present various results by varying certain parameters. In particular, we focus on the rate of immunization at birth (which roughly occurs the first 15 months of age) and the rate at which booster shots are administered to the general population.

In Section 2 we will introduce a general model with age-structure which involves susceptible, vaccinated, infected and recovered classes, and discuss the parameters involved. The two
parameters which we focus our study on are \( p \), the proportion of vaccinated individuals at birth (in this case 15 months of age) and on \( w(a) \), the rate at which booster shots are administered which is a function of age. We solve for steady-state age distributions and calculate the basic reproductive number in Section 3. In Section 4 we perform the local stability analysis of the steady-state age distributions.

Section 5 takes the age structured model and converts it to a system of ordinary differential equations. In this case, we assume that the parameters are constant. We vary the proportion of vaccinated individuals at birth \( (p) \) and the rate of administrating the second dosage of vaccine \( (w) \). In Section 6 we find some criteria on the contact rate \( (c) \), the proportion of vaccinated individuals at birth \( (p) \) and the rate of administrating the second dosage of vaccine \( (w) \) where eradication of measles would be successful. Section 7 presents a second simpler model where conclusions are drawn. We conclude and discuss our results in Section 7.

2 Analysis of age-structured model with parameters dependent on age

The total population, \( n(a, t) \), is separated into four groups: \( s(a, t) \), \( v(a, t) \), \( i(a, t) \) and \( r(a, t) \), representing the susceptible, vaccinated, infected and recovered populations respectively. We define \( \int_{0}^{a_2} s(l, t)dl \), \( \int_{a_1}^{a_2} v(l, t)dl \), \( \int_{a_1}^{a_2} i(l, t)dl \), and \( \int_{a_1}^{a_2} r(l, t)dl \) as the density of individuals in their respective class from age \( a_1 \) to age \( a_2 \). The death rate per unit time, \( \mu(a) > 0 \), the booster shot (second dosage) rate per unit time, \( w(a) > 0 \), and the recovery rate per unit time, \( \gamma(a) > 0 \), are all functions of age and represent the respective rates at the age group \( a \). The vaccine wears off at a constant rate \( \alpha \). At birth \( (a = 0) \), we consider that a proportion \( p \) (\( 0 \leq p < 1 \)) of the constant recruitment of newborns, \( \rho \), are vaccinated and enter \( v(a, t) \), and the remaining proportion \( (1 - p) \) newborns become susceptible and enter \( s(a, t) \).

The model is described by the following equations.

\[
\frac{\partial s(a, t)}{\partial a} + \frac{\partial s(a, t)}{\partial t} = -\mu(a)s(a, t) + (\alpha - w(a))v(a, t) - c(a)s(a, t)\frac{\beta(t)}{N(t)}, \tag{1}
\]
\[
\frac{\partial v(a, t)}{\partial a} + \frac{\partial v(a, t)}{\partial t} = -(\mu(a) + \alpha - w(a))v(a, t), \tag{2}
\]
\[
\frac{\partial i(a, t)}{\partial a} + \frac{\partial i(a, t)}{\partial t} = -(\mu(a) + \gamma(a))i(a, t) + c(a)s(a, t)\frac{\beta(t)}{N(t)}, \tag{3}
\]
\[
\frac{\partial r(a,t)}{\partial a} + \frac{\partial r(a,t)}{\partial t} = -\mu(a)r(a,t) + \gamma(a)i(a,t),
\]

where

\[
n(a,t) = s(a,t) + v(a,t) + i(a,t) + r(a,t),
\]

\[
\alpha - w(a) > 0
\]

\[
\beta(t) = \int_0^\infty c(z)\lambda(z)i(z,t)dz,
\]

\[
\mathcal{N}(t) = \int_0^\infty c(z)n(z,t)dz,
\]

\[
s(a,0) = s_0(a),
\]

\[
s(0,t) = (1-p)p,
\]

\[
v(a,0) = 0,
\]

\[
v(0,t) = pp,
\]

\[
i(a,0) = i_0(a),
\]

\[
i(0,t) = 0,
\]

\[
r(a,0) = 0,
\]

\[
r(0,t) = 0.
\]

In this model we consider proportionate mixing. Proportionate mixing is means that the “degree of contamination of the resource is proportional to the total number of infectives present in the population” (Busenberg & Cooke, 1993, p. 16). The force of infection, denoted by \(\beta(t)\), is the total number of contacts with an infected individual multiplied by the probability of infection given a successful contact over all of the age classes. Then, \(c(a)\frac{\beta(t)}{\mathcal{N}(t)}\) is the fraction of successful contacts with infected individuals in all the age groups of the total population. This is then multiplied by the susceptible class.

3 Steady-state solutions

We begin by solving for the steady state age distributions. Taking the time derivative at zero and solving for functions of age results in the following system:

\[
\frac{ds^*(a)}{da} = -\mu(a)s^*(a) + (\alpha - w(a))v^*(a) - c(a)s^*(a)\frac{\beta^*}{\mathcal{N}^2},
\]

\[
\frac{dv^*(a)}{da} = -\mu(a)s^*(a) + (\alpha - w(a))v^*(a),
\]

4
\[
\frac{di^*(a)}{da} = -(\mu(a) + \gamma(a))i^*(a) + c(a)s^*(a)\frac{\beta^*}{N^*},
\]
\[
\frac{dr^*(a)}{da} = -\mu(a)r^*(a) + \gamma(a)i^*(a),
\]

where
\[
n^*(a) = s^*(a) + v^*(a) + i^*(a) + r^*(a),
\]
\[
\beta^* = \int_0^\infty c(z)\lambda(z)i^*(z)dz,
\]
\[
N^* = \int_0^\infty c(z)n^*(z)dz,
\]
\[
s^*(0) = (1 - p)p,
\]
\[
v^*(0) = pp,\]
\[
i^*(0) = r^*(0) = 0.
\]

The steady-state solutions, which are all positive, are:
\[
n^*(a) = pe^{-M(a)},
\]
\[
s^*(a) = e^{-M(a) - \frac{\beta^*}{N^*}C(a)}\left((1 - p)p + \int_0^a (\alpha - w(a))v^*(x)e^{M(x)} + G(x)dx\right),
\]
\[
v^*(a) = pp e^{-M(a)} + aa - W(a),
\]
\[
i^*(a) = \frac{\beta^*}{N^*}e^{-M(a) - G(a)}\int_0^a c(x)s^*(x)e^{M(x)} + G(x)dx,
\]
\[
r^*(a) = e^{-M(a)}\int_0^a i^*(x)\gamma(x)e^{M(x)}dx,
\]

where
\[
M(a) = \int_0^a \mu(x)dx, C(a) = \int_0^a c(x)dx, G(a) = \int_0^a \gamma(x)dx, W(a) = \int_0^a w(x)dx.
\]

Substituting (14) into (10) we get the following equation:
\[
\beta^* = \int_0^\infty c(x)\lambda(x)e^{-M(x)}e^{-G(x)}\int_0^x c(z)s^*(z)e^{M(z)} + G(z)dzdx.
\]

One solution of this equation is $\beta^* = 0$. In this case, the force of infection is equal to zero, resulting in the disease free state:
\[
n^*(a) = pe^{-M(a)},
\]
\[
\begin{align*}
s^*(a) &= e^{-M(a)} \left( \rho - ppe^{-a-W(a)} \right), \\
v^*(a) &= ppe^{-M(a)+a-W(a)}, \\
i^*(a) &= 0, \\
r^*(a) &= 0,
\end{align*}
\]

Dividing through by \( \beta^* \neq 0 \) results in the following equation:

\[
1 = \int_0^\infty \frac{c(x)\lambda(x)}{N^*} e^{-M(x)-G(x)} \int_0^x c(z)s^*(z)e^{M(z)+G(z)}dzdx.
\]

Define

\[
H(\beta^*) = \int_0^\infty \frac{c(x)\lambda(x)}{N^*} e^{-M(x)-G(x)} \int_0^x c(z)s^*(z)e^{M(z)+G(z)}dzdx,
\]

where \( s^*(z) \) is the function that depends on time. In order to obtain a real and positive value for \( \beta^* \), \( H(\beta^*) \) must be a monotone decreasing function, thus we must have \( H'(\beta^*) < 0 \). Taking the derivative with respect to \( \beta^* \):

\[
H'(\beta^*) = \int_0^\infty \frac{c(x)\lambda(x)}{N^*} e^{-M(x)-G(x)} \int_0^x c(z)\frac{\partial s^*(z)}{\partial \beta^*}e^{M(z)+G(z)}dzdx.
\]

We need \( \frac{ds^*(a)}{d\beta^*} < 0 \).

\[
\frac{ds^*(a)}{d\beta^*} = -\frac{C(a)}{N^*}e^{-M(a)-\frac{s^*}{T_C(x)}} \left( (1-p)\rho + \int_0^a (\alpha - w(x))v^*(x)e^{M(x)+G(x)}dx \right) +
\]

\[
e^{-M(a)-\frac{s^*}{T_C(x)}} \left( \int_0^a (\alpha - w(x))v^*(x)\frac{C(x)}{N^*}e^{M(x)+G(x)}dx \right);
\]

Let \( \Pi(x) = (\alpha - w(x))v^*(x)e^{M(x)+G(x)} \). Then we can rewrite:

\[
\frac{ds^*(a)}{d\beta^*} = \frac{e^{-M(a)-\frac{s^*}{T_C(x)}}}{N^*} \left( -C(a) \left( (1-p)\rho + \int_0^a \Pi(x)dx \right) + \int_0^a \Pi(x)C(x)dx \right).
\]

Using integration by parts, we see that:

\[
\int_0^a \Pi(x)C(x)dx = C(a) \int_0^a \Pi(x)dx - \int_0^a C'(x) \int_0^a \Pi(z)dzdx.
\]

Then:

\[
\frac{ds^*(a)}{d\beta^*} = \frac{e^{-M(a)-\frac{s^*}{T_C(x)}}}{N^*} \left( -C(a)(1-p)\rho - \int_0^a C'(x) \int_0^a \Pi(z)dzdx \right) < 0,
\]
since \( C'(x) = c(x) > 0 \). Therefore \( H(\beta^*) \) is a monotone decreasing function. We define \( R_0 \),
the basic reproductive number, as \( H(0) \). Hence, when \( R_0 > 1 \), a positive \( \beta^* \) exists such that
equation (22) holds. The graph of Figure 1 illustrates what is occurring with \( H(\beta^*) \).

\[
R_0 = \int_0^\infty \frac{c(x)\lambda(x)}{N^*} e^{-(M(z)+G(z))} \int_0^x c(z)s^*(z) e^{M(z)+G(z)} dz dx,
\]

(24)

where

\[
s^*(a) = e^{-M(a)} \left( \rho - ppe^{\alpha W(a)} \right).
\]

If we consider all of the parameters as constants, we have:

\[
R_0(p) = \frac{c\lambda}{\mu + \gamma} \left( 1 - \frac{p\mu}{\mu + \alpha - w} \right)
\]

(25)

Taking \( p = 0 \), i.e. no vaccination at birth, the basic reproductive number is \( R_0(0) = \frac{c\lambda}{\mu + \gamma} \).

This corresponds to a pre-vaccination model where \( c\lambda \) was the force of infection and \( \frac{1}{\mu + \gamma} \) the
average time an individual spends in the infected stage. Thus, by introducing the parameter
\( p \), you decrease \( R_0(0) \). It is clear that \( R_0(p) < R_0(0) \). To solve for the critical value of \( p \) which
makes \( R_0(p) < 1 \) and \( w = 0 \), we need

\[
p > \frac{\mu + \alpha}{\mu} \left( 1 - \frac{1}{R_0(0)} \right).
\]

4 Stability of the steady-state age distributions

To perform the local stability analysis of the steady-state age distributions, we take the fol­
lowing perturbations of the steady-states:

\[
s(a, t) = s^*(a) + \xi(a, t)
\]

(26)

\[
v(a, t) = v^*(a) + \zeta(a, t)
\]

(27)

\[
i(a, t) = i^*(a) + \eta(a, t)
\]

(28)

\[
\beta(t) = \beta + \theta(t)
\]

(29)

Linearizing the system of partial differential equations results in the first order approximation
for \( \xi, \zeta, \eta \) and \( \theta \):

\[
\frac{\partial \xi(a, t)}{\partial a} + \frac{\partial \xi(a, t)}{\partial t} = -\left( \mu(a) + \frac{c(a)}{\kappa} \beta \right) \xi(a, t) + \left( \alpha - w(a) \right) \zeta(a, t) - \frac{c(a)}{N^*} s^*(a) \theta(t)
\]

(30)
\[
\frac{\partial \zeta(a, t)}{\partial a} + \frac{\partial \zeta(a, t)}{\partial t} = -(\mu(a) + \alpha - w(a)) \zeta(a, t)
\]
(31)
\[
\frac{\partial \eta(a, t)}{\partial a} + \frac{\partial \eta(a, t)}{\partial t} = -(\mu(a) + \gamma(a))\eta(a, t) + \frac{c(a)}{N^*} \beta \xi(a, t) + \frac{c(a)}{N^*} s^*(a) \theta(t)
\]
(32)
\[
\theta(t) = \int_0^\infty c(z) \lambda(z) \eta(z, t) dz
\]
(33)
\[
\xi(0, t) = \zeta(0, t) = \eta(0, t) = 0,
\]
\[
\xi(a, 0) = \xi_0(a), \; \zeta(a, 0) = 0, \; \eta(a, 0) = \eta_0(a)
\]

Since the equations on the right hand side are linear, we assume separable perturbations of the form:

\[
\xi(a, t) = \xi^*(a)e^{pt},
\]
(34)
\[
\zeta(a, t) = \zeta^*(a)e^{pt},
\]
(35)
\[
\eta(a, t) = \eta^*(a)e^{pt},
\]
(36)
\[
\theta(t) = \theta^*e^{pt}, \text{ where } \theta^* \text{ is a constant.}
\]
(37)

The solution for equations (30-32) are:

\[
\xi^*(a) = -\theta^* e^{-M(a)-G(a)} \int_0^a \frac{c(z)s(z)}{N^*} e^{-p(a-z)+M(z)+\frac{c(z)a}{N^*}} dz,
\]
(38)
\[
\zeta^*(a) = 0,
\]
(39)
\[
\eta^*(a) = e^{-M(a)-G(a)} \int_0^a \left( \xi^*(z) \frac{c(z)\beta}{N^*} + \theta^* \frac{s(z)c(z)}{N^*} \right) e^{-p(a-z)+M(z)+G(z)} dz,
\]
(40)
\[
\theta^* = \int_0^\infty c(z) \lambda(z) \eta^*(z) dz.
\]
(41)

Substituting \(\eta^*(a)\) into \(\theta^*\) results in the Lotka-type characteristic equation for \(p \; (\theta \neq 0)\):

\[
1 = \int_0^\infty c(z) \lambda(z) e^{-M(z)-G(z)} \int_0^z e^{-p(z-x)} \times \\
\left( \frac{s(x)c(x)}{N^*} e^{M(x)+G(x)} - \frac{c(x)\beta}{N^*} e^{G(x)-\frac{c(x)a}{N^*}} \int_0^x \frac{s(y)c(y)}{N^*} e^{-p(x-y)+M(y)+\frac{c(y)a}{N^*}} dy \right) dx dz.
\]
(42)

We see that for any negative value of \(p\) satisfying equation (42), equations (34-37) approach zero as time approaches infinity. For the trivial steady-state age distribution, that is when \(\beta = 0\), we have the following equation:

\[
1 = \int_0^\infty c(z) \lambda(z) e^{-M(z)-G(z)} \int_0^z \frac{s(x)c(x)}{N^*} e^{M(x)+G(z)-p(a-z)} dx dz.
\]
(43)
Solving all roots of $p$ for equation (42) is much more complicated and is omitted. For equation (43), we see that the right side of the equality, call $F(p)$, is a decreasing function. Using $R_0$, we see that at $p = 0$, $F(0)$ is equal to $R_0$. Thus when $R_0 < 1$, we have that $F(0) < 1$ and therefore $p < 0$. Now look at the case where $p$ is a complex number, say $u + iv$. Then we have:

$$F(p) = \int_0^\infty c(z)\lambda(z)e^{-M(z)G(z)} \times \int_0^z \frac{s(x)c(x)}{N_*}e^{M(x)G(x)-u(a-x)}(\cos(v(a-x)) - i\sin(v(a-x)))dxdz.$$  

Setting $\Re F(p) = 1$ and $\Im F(p) = 0$, we reduce the relation to:

$$F(p) = \int_0^\infty c(z)\lambda(z)e^{-M(z)G(z)} \int_0^z \frac{s(x)c(x)}{N_*}e^{M(x)G(x)-u(a-x)}\cos(v(a-x))dxdz.$$  

Comparing the inner integrand in the right hand of the equality to the equation in the right of (43), which is $F(p)$, we have:

$$e^{-u(a-x)}\cos(v(a-x)) = e^{-p(a-x)}.$$  

Since $|\cos(v(a-x))| < 1$, then $e^{-u(a-x)} > e^{-p(a-x)}$ therefore we have that $u < p$. Thus, for complex values of $p$, the real part is also negative. We conclude that when $R_0 < 1$, the disease free state is asymptotically stable.

5 Model with constant parameters and time dependency

Taking the system (1-4) and integrating over all the ages, we can convert to a system of ordinary differential equations:

$$\frac{dS(t)}{dt} = (1-p)p\mu - (\alpha - w)V(t) - \frac{c\lambda I(t)S(t)}{N(t)}, \quad (44)$$

$$\frac{dV(t)}{dt} = pp - (\mu + \alpha - w)V(t), \quad (45)$$

$$\frac{dI(t)}{dt} = -(\mu + \gamma)I(t) + \frac{c\lambda I(t)S(t)}{N(t)}, \quad (46)$$

$$\frac{dR(t)}{dt} = -\mu R(t) + \gamma I(t), \quad (47)$$

$$N(t) = S(t) + V(t) + I(t) + R(t), \quad (48)$$
This model poses some problems because of the rate of the second vaccination. The rate of the second vaccination occurs approximately 5 years after the initial, if any, vaccination was given. Since the vaccine wears off on an average of 20 years, the entire term \( \alpha - w \) would be negative.

We can modify the system by introducing two classes of vaccinated individuals, called \( V_1(t) \) and \( V_2(t) \), corresponding to those vaccinated at birth and those administered the mandatory dosage of vaccine prior to entering school. We also introduce a second set of susceptible populations. The first set, \( S_1 \), is the group of susceptibles that were not vaccinated at birth and only receive the mandatory dosage of the vaccine. The second group of susceptibles, \( S_2 \), is composed of those individuals who received the mandatory dosage which wears off at the rate \( \alpha \). In either vaccination class, the vaccine wears off at the same rate \( \alpha \) and both enter the \( S_2 \) class. They do not enter \( S_1 \) because the individuals can then receive a second mandatory dosage, which does not occur. Out of those individuals in \( S_2 \), a small proportion of those whose vaccine wore off after 20 years receives a second or maybe even third dosage at a rate \( \epsilon \). Although people rarely take the initiative of revaccinating after a 20 year immunity, it does occur.

The new model is:

\[
\begin{align*}
\frac{dS_1(t)}{dt} &= (1 - p)\rho - \mu S_1(t) - \frac{\beta(t)S_1(t)}{N(t)} - wS_1(t), \\
\frac{dS_2(t)}{dt} &= \alpha(V_2(t) + V_1(t)) - \frac{\beta(t)S_2(t)}{N(t)} - (\mu + \epsilon)S_2(t), \\
\frac{dV_1(t)}{dt} &= pp - (\mu + w + \alpha)V_1(t), \\
\frac{dV_2(t)}{dt} &= -(\mu + \alpha)V_2(t) + w(V_1(t) + S_1(t)) + \epsilon S_2(t), \\
\frac{dI(t)}{dt} &= -(\mu + \gamma)I(t) + \frac{\beta(t)(S_1(t) + S_2(t))}{N(t)}, \\
\frac{dR(t)}{dt} &= \gamma I(t) - \mu R(t).
\end{align*}
\]

The compartmental diagram of Figure 2 illustrates the dynamics of the model. Individuals become infected at the rate \( \beta \), which is the number of contacts per unit of time multiplied by the probability of catching the virus given a successful contact with an infected individual. This force of infection is multiplied by the probability of coming in contact with an infected individual, \( \frac{I}{N} \). This rate is then multiplied by the total number of susceptibles in the class \( S_t \).

The steady states of this system are three: one disease free-state and two endemic equi-
librium which are conjugates. The basic reproductive model for this model is dependent on two important parameters, $p$ and $w$:

$$R_0(p, w, \epsilon) = \frac{\beta \alpha}{(\gamma + \mu)(\alpha + \epsilon + \mu)} \left(1 + \frac{\mu(1-p)(\epsilon + \mu)}{\alpha(\mu + \omega)}\right).$$

(55)

Note that $R_0(0, 0, 0) = \frac{\beta}{(\gamma + \mu)} = R_0^*$ is the case when there is no vaccination and no booster shot. For the disease-free state, we see that it is stable if and only if $R_0(p, w, \epsilon) < 1$. We wish to find $p^*$ such that $R_0(p^*, 0, 0) < 1$. We find that we need:

$$p^* > \frac{\alpha(1 - R_0^*) + \mu}{\mu R_0^*}. \tag{56}$$

Here we have that $\alpha(1 - R_0^*) + \mu < 0$, since $R_0^* > 1$. Thus, right side of the inequality in equation (56) is greater than one, implying that $p^*$ must be greater than one. Since $0 < p^* < 1$, then it would be impossible to eradicate measles through birth vaccination alone. We assume that the other two endemic points are two in which may be locally stable, with $E_a > E_b$. Further efforts will be focused on the study of the endemic equilibria of this model. We do see that if we vaccinate everyone at birth, then it would be possible to eradicate the disease and the only important parameter is the rate at which they population is revaccinated after the vaccine wears of, i.e. $\epsilon$. In this case, $R_0(0, 0, \epsilon) < 1$ if $\alpha(1 - R_0^*) - \mu < \epsilon < 1$. Figure 3 illustrates when $R_0(p, w, \epsilon)$, plotted with respect to $p$ and $w$, is less than one. We need present rates with the exception that 0.5% of the second susceptible receives a second or third dosage of the vaccine.

Figures 4-7 illustrate that for higher values of $p$, the population of infected and recovered individuals remain relatively low but persists. The time runs for 70 years.

6 A second model with constant parameters and time dependency

Here we present a less complicated version of a measles model. In this case, we assume that we have a constant population consisting of susceptible, infected, vaccinated and recovered individuals. At birth, there is a proportion $p$ babies who are vaccinated and a proportion $(1-p)$ babies who become susceptible. We have booster shots being administered at a rate $\alpha$ and a proportion of $(1-q)$, receive the boosters, while the remaining $q$ return to the susceptible class.
The model is:
\[
\begin{align*}
\frac{dS(t)}{dt} &= (1 - p)\mu N(t) - \beta S(t) \frac{I(t)}{N(t)} - \mu S(t) + (1 - q)\alpha V(t), \\
\frac{dI(t)}{dt} &= \beta S(t) \frac{I(t)}{N(t)} - (\mu + \gamma) I(t), \\
\frac{dV(t)}{dt} &= p\mu N(t) - (1 - q)\alpha V(t) - \mu V(t), \\
\frac{dR(t)}{dt} &= \gamma I(t) - \mu R(t), \\
N(t) &= S(t) + V(t) + I(t) + R(t),
\end{align*}
\]

The compartmental diagram of Figure 8 illustrates the dynamics of the model.

The resulting basic reproductive number is a function of both \( p \) and \( q \):
\[
R_0(p, q) = R_0 \frac{\alpha(1 - q) + (1 - p)\mu}{\alpha(1 - q) + \mu}
\]

where
\[
R_0 = \frac{\beta}{\gamma + \mu} = R_0(0, 0).
\]

Because boosters come only out of the vaccinated class, failure to vaccinate results in no effect on the basic reproductive number.
\[
R_0(0, q) = R_0.
\]

In order to eliminate the disease with pure birth vaccination, we wish to find the critical value \( p^* \) for which \( R_0(p^*, 0) < 1 \). This implies that \( p^* > 1 - \frac{\alpha(1-R_0)+\mu}{\beta} \). This is the same condition we arrived to in model 2. We know that it is impossible to eliminate the disease without booster shots. However, we also know that \( R_0(1, 1) = 0 \), so it is possible to eliminate the disease with a combination of vaccination at birth and booster shots. Figure 9 shows the basic reproductive number as it crosses the \( R_0 = 1 \) plane. Note that we must have very high proportions of vaccination and boosters in order to eradicate measles.

7 Conclusions

Through the study of these three models, we are left with more questions than answers. But even though the models give wildly different results, we can still make some conclusions. We
found that eradicating the measles disease is much more difficult than presently thought. We still ask why have we not been as successful in eradicating this disease as we have been with small pox. We feel that the fact that the infection only lasts 20 days, with a nine day infectious period, keeps the disease persistent. Also, since measles is not viewed as a life threatening disease, the public does not take the necessary precautions to prevent infection.

For the first time dependent model, we found that it was possible to eradicate the spread of the measles disease by increasing the rate of vaccination for individuals in the second susceptible class. The stability analysis and study of the two endemic points remains to be solved. We feel that these two endemic points are such that one will be larger than the other and although measles will not be eradicated, it may be reduced. The last model gave some positive results which were predicted. Although it seems to show that only a massive campaign of vaccination and booster shots can eliminate the disease. The fact that the vaccine lasts so long supports a campaign of vaccinating at birth. However, it does not support the idea of vaccinating at the beginning of school. Perhaps vaccinating individuals at birth and after their completion of high school could be more effective.

We are left with the task of continuing the analysis of the endemic state for the age-structured model. Although we could not find criteria for eradicating or reducing the reproductive number, we still believe that the abandoning of boosters and focus on birth vaccination may reduce the reproductive model.

Acknowledgments

This study was supported by the following institutions and grants: National Science Foundation (NSF Grant DMS-9600027); National Security Agency (NSA Grants MDA 904-96-1-0032 and MDA 904-97-1-0074); Presidential Faculty Fellowship Award (NSF Grant DEB 925370) and Presidential Mentoring Award (NSF Grant HRD 9724850) to Carlos Castillo-Chavez; and the Office of the Provost of Cornell University. We would especially like to thank those who supported us through this study: Dr. Carlos Castillo-Chavez, Dr. Jorge Velasco-Hernández, Dr. Carlos Hernández-Súaìrez, Stephen Wirkus, Song Baojun and any one else we forgot to mention.
References


Figure 1: Graphical representation of the function H.

Figure 2: Compartmental diagram of Section 5.
Figure 3: $R_0(p, w, \epsilon)$, plotted with respect to $p$ and $w$.

Figure 4: Population of infected and recovered individuals. $w = \frac{1}{(5)(53)}$, $p = 75\%$. $R_0 = 4.36044$. 
Figure 5: Population of infected and recovered individuals. $w = \frac{1}{3(53)}$, $p = 85\%$. $R_0 = 4.34643$.

Figure 6: Population of infected and recovered individuals. $w = 0$, $p = 90\%$. $R_0 = 4.53552$.

Figure 7: Population of infected and recovered individuals. $w = 0$, $p = 95\%$. $R_0 = 4.32542$. 
Figure 8: Compartmental diagram of Section 6.

Figure 9: Graph of $R_0(p, q)$ and the plane $R_0 = 1$. 

18