

A Class Structured Mathematical Model For Polio Virus In Nigeria

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July 24, 2014

Abstract

In this paper, a bilinear mathematical model of ordinary differential equations is introduced to investigate the transmission dynamics of polio virus in Nigeria. The effect of vaccines versus environmental clean-up efforts on disease transmission is discussed. In the modeling process, Monte Carlo simulations were employed to approximate the values of three unknown parameters. Transmission is assumed to occur only through the fecal-oral route. Model analysis is conducted through computer simulation, and the simulations are repeated using different probability distributions for the length of the infectious period (γ), and the likelihood of total eradication is derived in each case. The results suggest that lower environmental virus concentrations can lead to eradication when the length of the infectious period is distributed exponentially. However, in some cases increased environmental clean up efforts are more effective than continuing to increase the number of vaccinations. The model is used to collect data for the values of its three unknown parameters.

1 Introduction

Since the commencement of the Global Polio Eradication Initiative in 1988, worldwide polio cases have fallen from 350,000 to just 406 in 2013 (WHO 2014). Today, efforts for complete eradication are of critical importance in Nigeria if the disease is to be eliminated worldwide. The polio virus has hitherto remained endemic in Nigeria, but if eradication is to be achieved, policy-makers must choose the appropriate strategy for limiting transmission. More specifically, policy-makers need to know whether continued vaccinations will ultimately succeed in eliminating outbreaks, or if supplemental

environmental clean-up efforts will be necessary to reach this goal.

In order to answer this question, we will use a variation of the standard S-I-R model for infectious diseases. It is important to note that this model is only applicable on a small scale. Therefore, it can be used for predicting transmission dynamics within a specific village rather than within the entire country. The reason for this is that the infectious class at any given time holds approximately 50 individuals, or $2.7e - 5\%$ of the total population (GPEI 2014). Therefore, we cannot make the assumption of homogenous mixing of the infectious class. However, the model output can be used to determine the appropriate strategy for eliminating outbreaks on a village-by-village basis. This will be useful in maximizing the efficiency of medical and economic resource allocation.

Three major adjustments are made to the standard S-I-R model. The first is the inclusion of vaccinations, which adds a V class of vaccinated individuals to the population. Thus the model we use is an S-I-V-R model accounting for susceptible, infectious, vaccinated, and recovered individuals within the population. The second adjustment is the creation of an age structure within each class, which compartmentalizes the S, I, V, R classes into separate classes for children and adults. This is necessary because the virus is fatal less than 5% of the time when contracted by children under the age of 4, but as the age at infection increases, the fatality rate can be as high as 30% (CDC). Lastly, the third adjustment is the classification of low risk and high risk groups. Two states in northern Nigeria, Borno and Yobe, accounted for 50% of the nation's total polio cases reported in 2012 (Data Profiles 2012). Therefore, the northern villages remain at a substantially higher risk of infection than those villages further south.

There are three model parameters with unknown values: the length of the infectious period (γ), high risk contact rate (β_1), and low risk contact rate (β_2). However, there are estimations for minimum and maximum values for gamma. In order to approximate actual values for all three parameters, the model was simulated with gamma set as a random variable under several different distributions, including exponential and normal. The initial conditions for these simulations were derived from population statistics estimated from the most recent 2006 census (Demographics). The contact rate parameters were adjusted using the Monte Carlo method until the simulations began to output reasonable figures that are consistent with the available data.

After these parameter values were obtained, we were able to interpret the results. The findings suggest that eradication is possible with increased vaccinations under certain distributions for gamma. In other cases, the dis-

ease was not eradicated unless some environmental clean up efforts helped to decrease the viral load in local water sources. Of the distributions that were sampled, the exponential distribution yielded the most realistic results, as disease dynamics changed significantly throughout repeats of the Monte Carlo method. In contrast, when the gamma distribution was sampled, the results suggest that disease dynamics are not sensitive to the value of gamma within the estimated range. However, this does not seem likely, which would suggest that gamma does not follow a gamma distribution.

In areas where policy makers are unsure if current disease control methods are on track to eradication, our model can be used to simulate the results of those methods over a 5 year period. If the simulation does not show the infectious class falling to zero, adjusting the model until eradication is achieved can allow policy makers to make informed decisions about how they choose to combat this disease at local levels. Essentially, the model is a tool that can attempt to quantify the risks, costs, and benefits of existing and proposed disease management strategies.

2 Modeling Approach

2.1 High Risk

Consider a village of individuals in northern Nigeria. Because of its location, the village falls within the high risk portion of this model. Children are fully susceptible until they receive their first dose of Oral Polio Vaccine (OPV), and 4% of children never receive any doses. 59% will receive enough doses to be considered fully immune. 37% will receive between 1 and 3 doses, leaving them partially susceptible to infection. Vaccinated and infectious individuals are able to shed the virus through their stool, which increases the viral concentration in the habitat (H , equation 11 below). In this group, 30% of adults will die if they are infected with the polio virus. This percentage is greater than for low risk individuals because high risk villages have inferior access to treatment possibilities.

$$s'_H = \mu_H(V_H + V_1 + R_H + S_H) - d_H s_H - \beta_1 s_H H_1 - .04\sigma_1 s_H - .37s_H - .59s_H - \frac{.097s_H}{4} \quad (1)$$

$$S'_H = (.04(\sigma_1 s_H)) - (d_H S_H) - (\beta_1 S_H H_1) \quad (2)$$

$$v'_H = .37s_H - (\beta_1 v_H H_1) - (\sigma_1 v_H) - d_H v_H \quad (3)$$

$$V'_H = (\sigma_1 v_H) - (\beta_1 V_H H_1) - d_H V_H \quad (4)$$

$$v_1 = .59s_H - d_H v_1 - \sigma_1 v_1 \quad (5)$$

$$V_1 = \sigma_1 v_1 - d_H * V_1 \quad (6)$$

$$i'_h = (\beta_1 v_H H_1) + \beta_1 s_H H_1 - \gamma_1 i_H \quad (7)$$

$$I_{H1} = .7(\beta_1 V_H H_1) - \gamma_1 I_{H1} \quad (8)$$

$$I_{H2} = .3(\beta_1 V_H H_1) + (\beta_1 S_H H_1) - \delta_1 I_{H2} \quad (9)$$

$$r'_H = \gamma_1 i_H - d_H r_H \quad (10)$$

$$R'_H = \sigma_1 r_H + \gamma_1 I_{H1} - d_H R_H \quad (11)$$

$$H'_1 = \alpha_1(i_H + I_{H1} + I_{H2}) + .25\alpha_2(v_H + V_H + v_1 + V_1) - \alpha_3 H_1 \quad (12)$$

Parameter	Definition	Units
d_H	High risk death rate	$\frac{\text{natural deaths}}{\text{time}}$
δ_1	High risk disease death rate	$\frac{\text{deaths due to disease}}{\text{time}}$
σ_1	Child→ maturation rate	$\frac{1}{t}$
γ_1	Length of infectious period	$\frac{1}{t}$
μ_H	High risk birth rate	$\frac{\text{births}}{\text{time}}$
β_1	High risk contact transmission rate	$\frac{\text{contacts}}{\text{time}}$
α_1	Infectious individual virus shedding rate	$\frac{\text{virus}}{\text{person*time}}$
α_2	Vaccinated individual virus shedding rate	$\frac{\text{virus}}{\text{person*time}}$
α_3	Environmental clean up rate	$\frac{\text{virus}}{\text{time}}$

Table 1: High Risk Parameters

All time is measured in days

2.2 Low Risk

Now consider a village of individuals in southern Nigeria. Because of its location, the village falls within the low risk portion of this model. Children are fully susceptible until they receive their first dose of Oral Polio Vaccine (OPV), and 93% of children will receive enough doses to be considered fully immune. 7% will receive between 1 and 3 doses, leaving them partially susceptible to infection. Vaccinated and infectious individuals are able to shed the virus through their stool, which increases the viral concentration in the habitat (H , equation 11 below). Children who become infected are assumed to flow directly into the recovered class. 15% of adults flow into the fatal infectious class for those who die from contracting the virus. Because southern Nigeria is home to greater access to medicine and higher levels of affluence, 85% of adults eventually recover fully from the disease.

$$s'_L = \mu_L(V_L + V_2 + R_L) - d_L s_L - \frac{.097s_L}{4} - .07s_L - .93s_L \quad (13)$$

$$v'_L = .07s_L - \sigma_1 v_L - d_L v_L - \beta_2 v_L H_2 \quad (14)$$

$$V'_L = \sigma_1 v_L - d_L V_L - \beta_2 V_L H_2 \quad (15)$$

$$v'_2 = .93s_L - d_L v_2 - \sigma_1 v_2 \quad (16)$$

$$V'_2 = \sigma_1 v_2 - d_L V_2 \quad (17)$$

$$i'_L = \beta_2 v_L H_2 - \gamma_1 i_L \quad (18)$$

$$I'_{L1} = .85\beta_2 V_L H_2 - \gamma_1 I_{L1} \quad (19)$$

$$I'_{L2} = .15\beta_2 V_L H_2 - \delta_2 i_{L2} \quad (20)$$

$$r_L = \gamma_1 i_L - d_L r_L \quad (21)$$

$$R_L = \sigma_1 r_L + \gamma_1 i_{L1} - d_L R_L \quad (22)$$

$$H'_2 = \alpha_1(i_L + I_{L1} + I_{L2}) + .25\alpha_2(v_L + V_L + v_2 + V_2) - \alpha_3 H_2 \quad (23)$$

Parameter	Definition	Units
d_H	High risk death rate	$\frac{\text{natural deaths}}{\text{time}}$
δ_2	Low risk disease death rate	$\frac{\text{deaths due to disease}}{\text{time}}$
σ_1	Child \rightarrow maturation rate	$\frac{1}{t}$
γ_1	Length of infectious period	$\frac{1}{t}$
μ_L	Low risk birth rate	$\frac{\text{births}}{\text{time}}$
β_2	Low risk contact transmission rate	$\frac{\text{contacts}}{\text{time}}$
α_1	Infectious individual virus shedding rate	$\frac{\text{virus}}{\text{person*time}}$
α_2	Vaccinated individual virus shedding rate	$\frac{\text{virus}}{\text{person*time}}$
α_3	Environmental clean up rate	$\frac{\text{virus}}{\text{time}}$

Table 2: Low Risk Parameters

2.3 Data

Parameter	Definition	Value
σ_1	Child \rightarrow maturation rate	$\frac{1}{4}$
γ_1	Length of infectious period	RandomVariate[29]*
μ_H	High risk birth rate	$\frac{.0185}{365}$
μ_L	Low risk birth rate	$\frac{.0123}{365}$
d_H	High risk death rate	$\frac{.00375}{365}$
d_L	Low risk death rate	$\frac{.00125}{365}$
β_1	High risk contact transmission rate	0.000000000000000055
β_2	Low risk contact transmission rate	.00000000000000004
δ_1	High risk disease death rate	$\frac{3}{300}$
δ_2	Low risk disease death rate	$\frac{15}{3000}$
α_1	Infectious individual virus shedding rate	$\frac{1}{50}$
α_2	Vaccinated individual virus shedding rate	$\frac{1}{20}$
α_3	Environmental clean up rate	Variable**

Table 3: Parameter Values

Average range for γ_1 is between 3 and 6 weeks. The average of this is

about 4 weeks, or 29 days.

*Ran simulations with zero, “average,” and “high” clean up rates. The average value was determined using Monte Carlo simulations and identified when the results matched the current data. The “high” value was identified when the results showed faster eradication. Average rate was found to be ≈ 0.8 , and the high rate was found to be ≈ 2.8 .

3 Results With Exponential Distribution for Gamma

Normal vaccine levels:

Low risk: 93% > 4 doses, 7% $1 < doses < 3$

High risk: 59% > 4 doses, 37% $1 < doses < 3$, 4%: 0 doses

3.1 Sample Simulations With Varying Outcomes

1. Case 1: Eventual eradication of low risk infectious

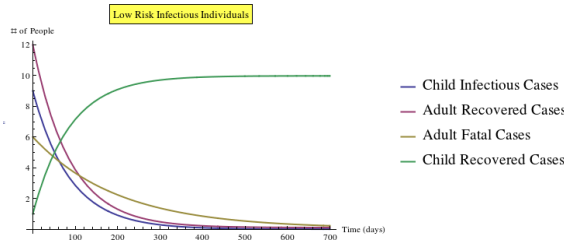


Figure 1

2. Case 1 : Eventual Eradication of high risk infectious

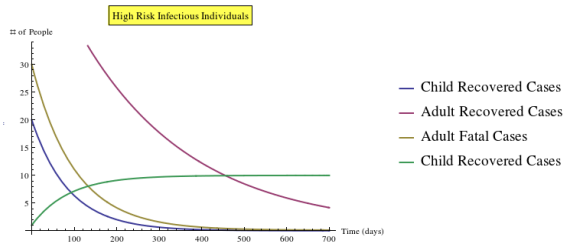


Figure 2

3. Case 2: Low risk infectious class persists

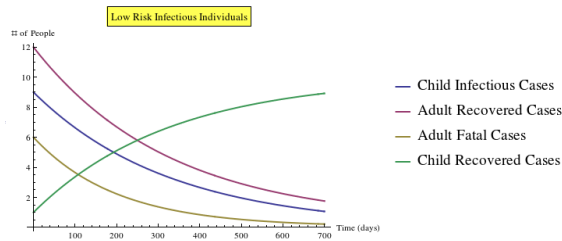


Figure 3

4. Case 2: Low risk infectious class persists

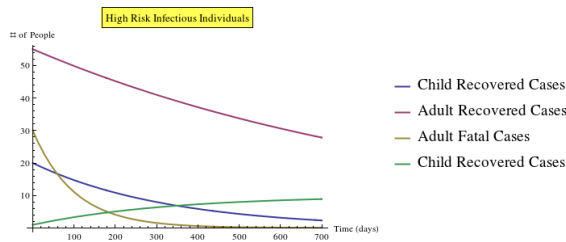


Figure 4

5. Case 3: Aggressive eradication of low risk infectious

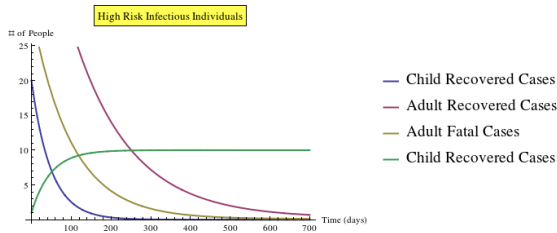


Figure 5

6. Case 3: Aggressive eradication of high risk infectious

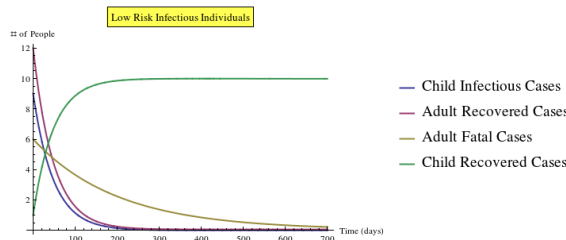


Figure 6

3.2 Discussion

The variation in each case is explained by gamma's definition as a random variable following an exponential distribution. Depending on the value chosen for gamma, the disease dynamics can vary considerably. Essentially, the value of gamma controls whether or not the disease is eradicated or persists within a village.

3.3 Outbreak Cases with Zero Initial Infectious Individuals

7. Case 4: Initial infectious class set to zero. Environmental efforts set to zero. Vaccines set to normal level.

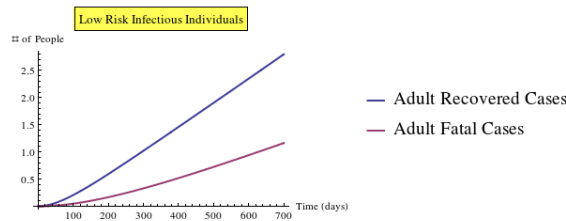


Figure 7: Infectious class returns.

8. normal vaccines derived from PolioInfo.org
9. Case 4: Initial infectious class set to zero. Environmental efforts set to zero. Vaccines set to normal level.

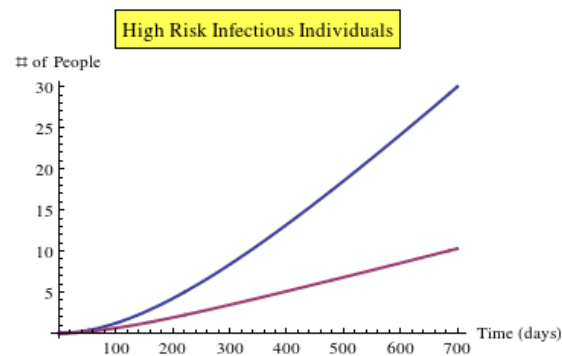


Figure 8: Infectious class returns.

10. Case 5: Initial infectious class set to zero. Environmental efforts set to zero, vaccines increased

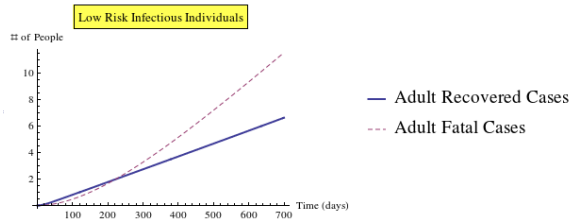


Figure 9: As expected, infectious class size is smaller than in Case 4. low risk: 98% > 4 doses, 2% $1 < doses < 3$ high risk: 66% > 4 doses, 30% $1 < doses < 3$, 4%: 0 doses

11. Case 5: Initial infectious class set to zero. Environmental efforts set to zero, vaccines increased

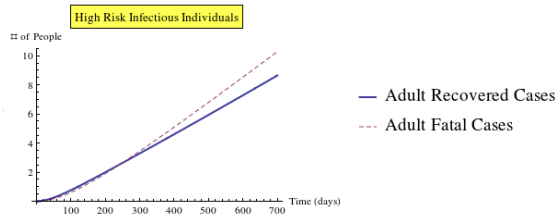


Figure 10: As expected, infectious class size is smaller than in Case 4.

3.4 Discussion

With no environmental efforts and increased vaccinations, the size of the high risk infectious class can be suppressed by about 33% compared to the case with “normal” vaccinations. However, the increased vaccinations by themselves are not sufficient to eradicate the disease within two years. Increased environmental efforts cause a 94% suppression of the size of the high risk group.

12. Case 6: Initial infectious class set to zero. Vaccines restored to normal levels. Environmental efforts increased 1%

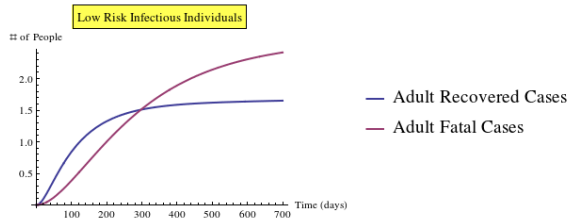


Figure 11: Infectious class size approaches 2 individuals

13. Case 6: Initial infectious class set to zero. Vaccines restored to normal levels. Environmental efforts increased 1%

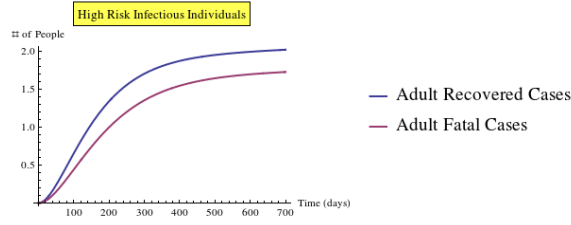


Figure 12: Infectious class size approaches 2 individuals

3.5 Eradication Cases with Initial Infectious Individuals Existing

Standard initial conditions:

$$i_L[0] = 9, I_{L1}[0] = 12, I_{L2}[0] = 6i_H[0] = 20, I_{H1}[0] = 55, I_{H2}[0] = 30$$

14. Case 7: Initial infectious class set to standard values. Vaccines restored to normal levels. Environmental efforts set to zero.

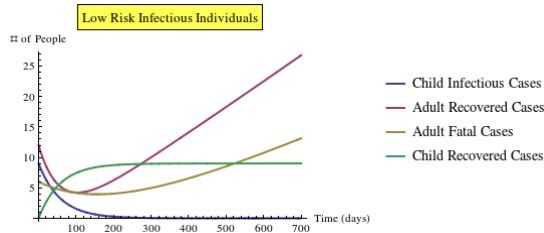


Figure 13: Infectious class increases quickly

15. Case 7: Initial infectious class set to standard values. Vaccines normal. Environmental efforts set to zero.

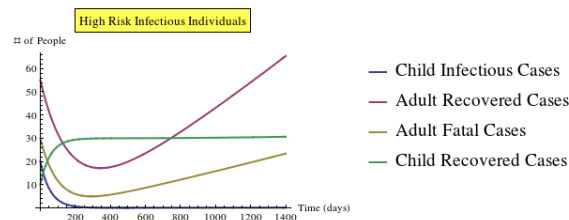


Figure 14: Infectious class size remains high

16. Case 8: Initial infectious class set to standard values. Vaccines increase. Environmental efforts set to zero.

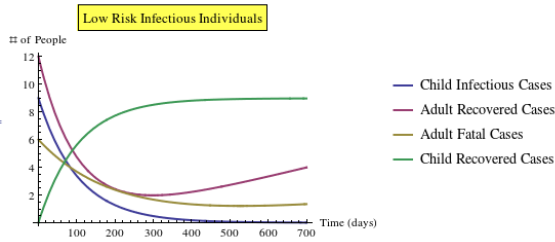


Figure 15: Infectious class size suppressed compared to case 7. Still no eradication yet.

17. Case 8: Initial infectious class set to standard values. Vaccines increase. Environmental efforts set to zero.

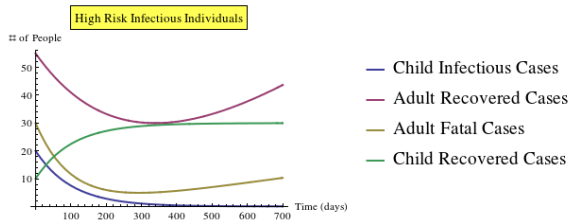


Figure 16: Infectious class suppressed compared to case 7. Still no eradication yet

18. Case 9: Initial infectious class set to standard values. Vaccines increased. Environmental efforts increased 1% ($0.01 = \alpha_3$).

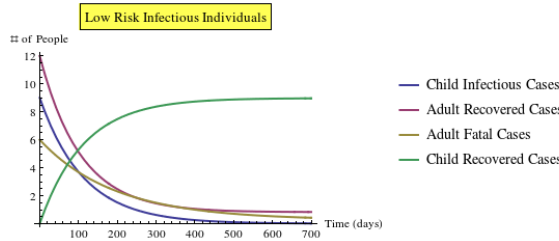


Figure 17: Infectious class sizes suppressed compared to case 7. Still no eradication yet.

19. Case 9: Initial infectious class set to standard values. Vaccines normal. Environmental efforts increased 1%.

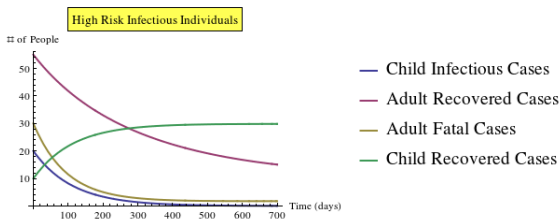


Figure 18: Infectious class suppressed compared to case 7. Still no eradication yet

20. Case 10: Initial infectious class set to standard values. Vaccines increase. Environmental efforts increased 1% ($0.01=\alpha_3$).

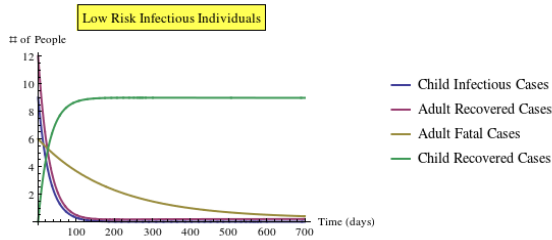


Figure 19 : Infectious class nearly eradicated

21. Case 10: Initial infectious class set to standard values. Vaccines increased. Environmental efforts increased 1%.

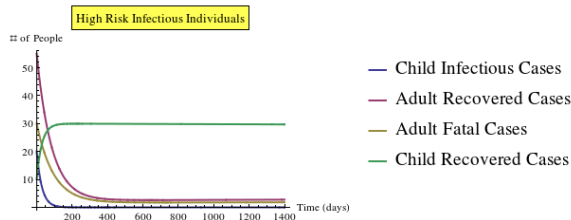


Figure 20: Infectious class nearly eradicated

3.6 Discussion

In the eradication case, a lack of environmental efforts led to explosion of the disease because of increased shedding. Thus, a combination of increased vaccinations and a 1% increase in clean up efforts was most effective

4 Conclusions

The optimal strategy for stopping transmission depends on the type of population being studied as well as the initial conditions of the infectious class. Consider the case where the infectious class is very small and the goal is to prevent an outbreak. In low risk areas, maintaining vaccines can be effective as evident in Figure 9. In high risk areas, preventing an outbreak with only vaccines is more difficult, but Figure 10 shows that increasing vaccines may still suppress the size of the infectious class in the event of an outbreak. However, this may not always be the case as some environmental efforts will

be likely be necessary in the future as the Nigerian population continues to grow.

Now consider the case where the infectious class size is already high and the goal is to eradicate the disease from that portion of the population. In this case, the impact of environmental efforts is tremendous on the high risk group as evident in comparing Figure 16 (no environmental efforts) to Figure 18 (1% increase in environmental efforts).

Therefore, whether or not vaccinations by themselves are sufficient depends on the goal of the policy as well as the location and state of the infectious class.

Also, gamma is assumed to follow an exponential distribution, which controlled the disease dynamics in this model.

4.1 Mathematica Code

```

\[Mu]L = .0123/(365);
\[Mu]H = .0185/(365);
\[Sigma] = 1/4;
dL = .00125/365;
dH = .00375/365;
\[Delta] = .15/(30);
death = .3/30;
\[Alpha]1 = 1/50;
\[Alpha]2 = 1/20;

\[Gamma]c = RandomVariate[ExponentialDistribution[29]];
\[Alpha]3 = 0.01;
\[Beta]1 = .0000000000000000055;
\[Beta]2 = .0000000000000000004;

soln1 =
NDSolve[
  {s1'[t] == (\[Mu]L*(vL[t] + v2a[t] + rL[t])) - (dL*s1[t]) - (.097*
    s1[t]/4) - (.02*s1[t]) - (.98*s1[t]),
    v1'[t] == .02*s1[t] - \[Sigma]*v1[t] - (dL*v1[t]) - \[Beta]2*
    v1[t]*h1[t],
    vL'[t] == \[Sigma]*v1[t] - dL*vL[t] - \[Beta]2*vL[t]*h1[t],
    v2c'[t] == .98*s1[t] - dL*v2c[t] - \[Sigma]*v2c[t],
    v2a'[t] == \[Sigma]*v2c[t] - dL*v2a[t],
    i1'[t] == (\[Beta]2*v1[t]*h1[t]) - \[Gamma]c*i1[t],
    iL1'[t] == .85*(\[Beta]2*vL[t]*h1[t]) - \[Gamma]c*iL1[t],
    iL2'[t] == .15*(\[Beta]2*vL[t]*h1[t]) - \[Delta]*iL2[t],
    r1'[t] == (\[Gamma]c*i1[t]) - (dL*r1[t]),

```

```

rL'[t] == \[Sigma]*rL[t] + (\[Gamma]c*iL1[t]) - dL*rL[t],
sh'[t] == (\[Mu]H*(vH[t] + v1a[t] + rH[t] + sH[t])) - (dH*
sh[t]) - (\[Beta]1*sh[t]*h2[t]) - (.04*\[Sigma]*sh[t]) - (.3*
sh[t]) - .66*sh[t] - (.097*sh[t]/4),
sH'[t] == (.04*\[Sigma]*sh[t]) - (dH*sH[t]) - (\[Beta]1*sH[t]*
h2[t]),
vh'[t] == .3*sh[t] - (\[Beta]1*vh[t]*h2[t]) - (\[Sigma]*vh[t]) -
dH*vh[t],
vH'[t] == (\[Sigma]*vh[t]) - (\[Beta]1*vH[t]*h2[t]) - dH*vH[t],
v1c'[t] == .66*sh[t] - dH*v1c[t] - \[Sigma]*v1c[t],
v1a'[t] == \[Sigma]*v1c[t] - dH*v1a[t],
ih'[t] ==
10*(\[Beta]1*h2[t]*vh[t]) + \[Beta]1*sh[t]*
h2[t] - (\[Gamma]c*ih[t]),
iH1'[t] == .7*(\[Beta]1*vH[t]*h2[t]) - (\[Gamma]c/3)*iH1[t],
iH2'[t] == .30*(\[Beta]1*vH[t]*h2[t]) + (\[Beta]1*sH[t]*h2[t]) -
death*iH2[t],
rh'[t] == (\[Gamma]c*ih[t]) - (dH*rh[t]),
rH'[t] == \[Sigma]*rh[t] + (\[Gamma]c*iH1[t]) - dH*rH[t],
h1'[t] == \[Alpha]1*(il[t] + iL1[t] +
iL2[t]) + .25*\[Alpha]2*(v1[t] + vL[t] + v2c[t] +
v2a[t]) - \[Alpha]3*h1[t],
h2'[t] == \[Alpha]1*(ih[t] + iH1[t] +
iH2[t]) + .25*\[Alpha]2*(vH[t] + v1c[t] + v1a[t] +
vh[t]) - \[Alpha]3*h2[t],
s1[0] == 20000000, v1[0] == 1400000, vL[0] == 8000000,
v1c[0] == 4500000, v1a[0] == 25000000, v2c[0] == 18600000,
v2a[0] == 107000000, il[0] == 9, iL1[0] == 12, iL2[0] == 6,
r1[0] == 0, rL[0] == 0, sh[0] == 300000, sH[0] == 1500000,
ih[0] == 20, iH1[0] == 55, iH2[0] == 30, vh[0] == 2000000,
vH[0] == 11500000, rh[0] == 10, rH[0] == 35, h1[0] == 1,
h2[0] == 1},
{s1, v1, vL, v1c, v1a, v2c, v2a, sh, sH, il, iL1, iL2, vh, ih, iH1,
iH2, vH, r1, rL, rH, rh, h1, h2},
{t, 0, 2000}];\
\
Plot[{il[t] /. soln1, iL1[t] /. soln1, iL2[t] /. soln1 ,
r1[t] /. soln1}, {t, 0, 700},
AxesLabel -> {"Time (days)", "# of People"},
PlotLegends -> {"Child Infectious Cases", "Adult Recovered Cases",
"Adult Fatal Cases", "Child Recovered Cases"},
PlotLabel ->
Style[Framed["Low Risk Infectious Individuals"], Thick,
Background -> Lighter[Yellow]],

```

```
PlotStyle -> {Thick, Thick, Thick }]\ \
\ \
Plot[{ih[t] /. soln1, iH1[t] /. soln1, iH2[t] /. soln1 ,
  rh[t] /. soln1}, {t, 0, 1400},
AxesLabel -> {"Time (days)", "# of People"},
PlotLegends -> {"Child Infectious Cases", "Adult Recovered Cases",
  "Adult Fatal Cases", "Child Recovered Cases"},
PlotLabel ->
  Style[Framed["High Risk Infectious Individuals"], Thick,
  Background -> Lighter[Yellow]], PlotStyle -> {Thick, Thick, Thick }]
```

Acknowledgments

I'd like to thank Dr. Jay Walton for his invaluable input throughout this research project. I'd also like to thank Adrian Barquero-Sanchez for his technical support and mastery of Mathematica.

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