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MODELING THE EFFECT OF INFORMATION CAMPAIGNS ON THE HIV EPIDEMIC IN UGANDA

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Abstract. The increasing prevalence of HIV/AIDS in Africa over the past twenty-five years continues to erode the continent's health care and overall welfare. There have been various responses to the pandemic, led by Uganda, which has had the greatest success in combating the disease. Part of Uganda's success has been attributed to a formalized information, education, and communication (IEC) strategy, lowering estimated HIV/AIDS infection rates from 18.5% in 1995 to 4.1% in 2003. We formulate a model to investigate the effects of information and education campaigns on the HIV epidemic in Uganda. These campaigns affect people's behavior and can divide the susceptibles class into subclasses with different infectivity rates. Our model is a system of ordinary differential equations and we use data about the epidemics and the number of organizations involved in the campaigns to estimate the model parameters. We compare our model with three types of susceptibles to a standard SIR model.

1. **Introduction.** The majority of all people living with HIV/AIDS in the world are found in sub-Saharan Africa. Although the region has only 10% of the world's population, it accounts for nearly two-thirds (63%) of people living with HIV/AIDS. Of the estimated 2.1 million AIDS related deaths in 2006, 72% occurred in Africa [43]. Currently, the number of people who are newly infected with HIV is still roughly equivalent to the number of people who are dying from AIDS. At the end of 2005, 24.7 million people living in sub-Saharan Africa had HIV/AIDS out of the total estimate of 39.5 million worldwide (Fig. 1).

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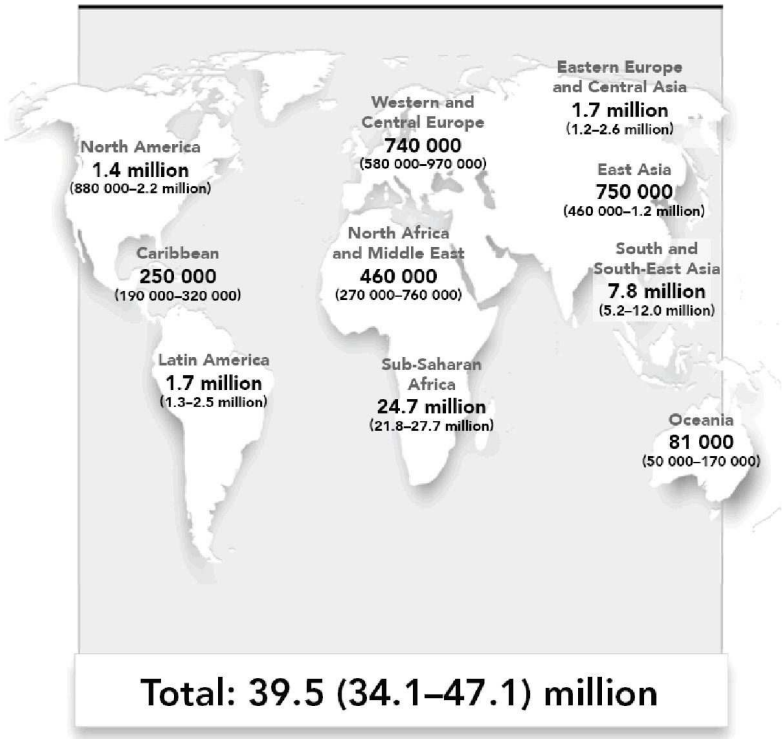

FIGURE 1. Adults and Children Estimated to be Living with HIV/AIDS (Source: UNAIDS/WHO, 2006)

The decrease in Uganda HIV rates, in contrast to other countries in the region, is an interesting phenomenon worth investigating. There are many theories about why there has been such a decrease in HIV rates. Some experts think that the abstinence, be faithful and condoms (ABC) campaigns started by the Ugandan government in 1992 had changed people's behaviors and attitudes, and thus reversed a troubling pattern of increase in HIV/AIDS [see, for example, [13, 32]]. Other government and nongovernmental agencies began campaigns to distribute information and educational materials about the disease; some organizations emphasized the AB behavior and others the C behavior. Some argue that the Ugandan government initially emphasized only AB strategies, and the effects of C did not appear until later [37]. Once the emphasis shifted to the C-type campaigns, there was a dramatic drop in new HIV infections. Others argue that the ABC strategy did not work, and what has actually happened is that a large number of deaths from HIV have dropped the prevalence rate of the disease, causing the overall rate to drop, falsely suggesting any real change in behavior [46].

We call attention to some studies on the advances in HIV education and prevention and their effects on the epidemic [2, 8, 13, 21]. See the recent work by de Walque [7] about the responsiveness to information as a channel through which education has affected health outcomes in Uganda.

To understand this phenomenon, we need to look at the history of the epidemic in Uganda and the data collected from numerous sources such as the UN, WHO, Ugandan government reports and other available sources. We also need data about the numbers of organizations giving information and the percentages of the types of

behavior recommended. Using this data, we will develop a model to explain what has transpired.

We will augment the standard SIR (susceptible, infected, and removed) model with the effect of the information and education campaigns (IEC) on these populations. Crucial to this model are not only the infection and death rates for HIV in Uganda, but also the role that IECs have played during this process. In Uganda two distinct types of campaigns have been deployed to combat the infection rates. The abstinence and be faithful campaigns have focused on behavior change and are employed fairly exclusively by faith-based organizations in Uganda. The condom campaigns have gained much ground in recent years and have been used more commonly by secular organizations [1]. Data for these IECs is hard to find, and the effect of their messages is difficult to measure. Also, quantifying the actual behavior resulting from the campaigns poses a challenge.

We formulate a system of differential equations modeling the HIV epidemics in Uganda. We include the novel feature of breaking the susceptible population into three subpopulations, based on possible changes in behavior. We hypothesize the IEC affected the change in the behavior of some susceptibles. Another novel feature is the inclusion of the dynamics of the "education campaign" class, since the amount of information about HIV is changing over time. After estimating key parameters from data, we compare our model with a standard SIR model.

In Section 2, we give some historical background of the HIV epidemic in Uganda. The formulation of the model is given in Section 3. Key parameters are estimated from some epidemic data in Section 4. Section 5 contains some model simulation results and comparison with a standard SIR model. Our conclusions and possible extensions are discussed in the last section.

2. History of the HIV epidemic in Uganda.

AIDS was first discovered in the Rakai District in southeastern Uganda in 1982. Over the next four years, few campaigns or programs were geared to combat HIV, because little was known about the disease. During this time, efforts focused on care-giving for infected people at the local level. The government responded to the rapid spread of the disease and in 1986 established an AIDS control program to address ways to control the disease, focusing on ensuring safe blood products and educating people about HIV [18]. The first national survey was conducted in 1988 to determine the extent of the HIV epidemic. The results estimated the average national HIV prevalence rate to be 9%. In 1991, the prevalence rate among pregnant women aged 15 to 24 peaked at an estimated 21%. A national prevalence rate of over 10% is considered to be at the epidemic stage.

The government's approach to addressing the AIDS problem included open public discussion about the disease, led by President Museveni and other prominent figures in Ugandan society. The political openness about the problem, coupled with the level of priority assigned to the problem of AIDS in Uganda, set a new precedent in the world and is considered to be a major influence in curbing the prevalence of HIV/AIDS in Uganda [14].

Uganda's multisectoral approach has three main components [41]. The first calls for active involvement of everyone in society to participate in AIDS control activities. Everyone is responsible, both individually as well as collectively, to coordinate with other sectors of society. The second component calls for social responsibility in managing the care for infected persons as well as those affected by the disease.

The third component of the multisectoral approach calls for building relationships among organizations to promote sustainability of effort, including the coordination of communication and information between the organizations. This approach, credited with the overall success of the program, has resulted in the involvement of numerous organizations, many of which disseminate information and education to the Ugandan public. The importance of dissemination of information about HIV/AIDS has resulted in the development of a formal information, education, and communication strategy. This strategy provides goals for the dissemination of information throughout the multiple sectors working in HIV/AIDS in Uganda. The missions and services offered by these organizations vary but all provide information to the Ugandan people.

The organizations involved in the dissemination of HIV/AIDS information include governmental, nongovernmental, and faith-based organizations, community-based organizations, private companies, multi- or bilateral organizations, and other international organizations. Information that is disseminated focuses on a variety of topics including abstinence, faithfulness, and condoms. In 2003, Albright, Kawooya, and Hoff [1] identified the types of information and how it is being disseminated by these organizations.The government centered its IEC efforts on ABC beginning in 1992. From 1991 to 1993, the prevalence rate for young pregnant women began to decrease [18]. In 1992, Ugandan governmental agencies borrowed $50 million from the World Bank to fight HIV. They also created the Sexually Transmitted Infections Project with money raised in 1992. By 1995, the Ugandan government announced that the HIV prevalence rate had declined. Uganda participated in drug trials to reduce mother-to-child transmissions in 1997 [18]. Prevalence rates for young pregnant women had dropped to 9.7% in 1998. Additionally, the Drug Access Initiative was launched to reduce HIV drug prices and to create the infrastructure to distribute these drugs to the public. A voluntary door-to-door testing program was started by the Ministry of Health in 1999. In 2001 the World Bank approved $47.5 million over the next five years for HIV prevention and treatment programs. By this time, UNAIDS determined that the national HIV prevalence rate was 5% [18]. Also in 2001, a countrywide survey of agencies involved in HIV/AIDS activities was conducted to identify agencies, examine their interventions, and determine their level of operation [20].

The dissemination of information has been unprecedented in its effects on behavior change in Uganda, particularly over the past fifteen years, leading to a dramatic decrease in the number of nonregular partners and reduction in sexual networks [26]. The number of nonregular sexual partners was 60% lower in Uganda in 1995 than in Kenya in 1998 and Zambia and Malawi in 1996 [39].

3. **SIRE model.** A great deal of work has addressed HIV epidemic models. Here we mention some of the models related to education [7] or to Africa [4, 22, 31, 34, 36, 44, 47] and suggest that the reader also see some of the references in [9, 17, 25, 28, 30, 40]. The work of Velasco-Hernandez [45] models the behavior change that could occur in infecteds who know their infected status in a homosexual population; the contact rates, incubation time, and probability of disease transmission are affected by behavior change. In the papers by Behncke [3] and Castilho [5], the influence of the education campaign enters in the infectivity coefficient and the coefficient of death due to the disease, but the education campaign does not have its own dynamics. Lungu et. al. [27] investigated the rate at which

the adult population should be exposed to education on prevention, the effectiveness of this education, and the treatment rates to bring the disease reproduction number below one. Their model has two susceptible classes, susceptibles and educated susceptibles, and the level of education is reflected in the rate that susceptibles become educated susceptibles. But the education level is not a dynamic variable there. Mogahadas et. al. [29] analyzed a model for assessing the effect of condom use in the spread of the epidemic; the condom usage affected the coefficient of the infectivity term.

A basic SIR model, [24] with susceptibles, infecteds, and removed, has the form

$$\begin{aligned} S' &= -\beta SI + b(S+I) - dS \\ I' &= \beta SI - \gamma I \\ R' &= \gamma I \end{aligned} \tag{1}$$

where R collects the total number of persons who have died from the disease. We will modify this basic model to take into account the change in behavior of some susceptibles to the education campaigns.

For our model, we will examine the adult population, because educational campaigns are geared toward changing the behaviors of adults. First, we need to analyze the granularity of our model to determine if the classes of S, I and R are still appropriate. We have at least three different types of behaviors that the susceptible population could exhibit as a result of the IEC. First, the population that has not yet changed its behavior in response to the campaign will be denoted by S, the general susceptible population. Part of the population could change its behavior and practice the abstinence and be faithful behaviors, which we will denote by S_{AB}. The other type of behavior change for the susceptible population is condom usage, and those susceptibles will be indicated by S_C. Hence, this divides the previous susceptible class into S, S_{AB} and S_C. We also need to add an E class to reflect the amount of educational information from the IEC promoting AB and C behaviors. Interaction with the E class causes a portion of the S class to move to either the S_{AB} or S_C category. We assumed the size of the E class was proportional to the number of organizations giving out information and education on HIV. The split into AB and C behavior effects was based on the proportions of organizations giving out information on the respective behaviors [1]. We estimated that the interaction of E and S will have rates of .8 to go to the S_C class and .1 to the S_{AB} class. Our work differs from [27] in that our rate of movement into the different susceptibles classes depends on the time-dependent level of educational campaigns. Thus the E class has its own dynamics.

We have also included an entry rate for people into the general susceptible class, and death rates where people leave the three S classes.

Interaction of the three susceptible classes with the infected class gives rise to new infecteds. Notice now instead of one infection rate β, we now have three infection rates to deal with. So β_1 is the infection rate for the general susceptible class S. The infection rates β_2 and β_3 are for the susceptible classes, S_{AB} and S_C, respectively. The infected class I moves to the removed class R as in the SIR model.

We use logistic growth for the E class with a growth coefficient that increases with the number of infectives. Such an equation is similar to those in models for advertising campaigns [35]. The growth coefficient, r, is multiplied by a ratio of the infected to the living population. The equations for the infected and removed classes are similar as in the basic SIR model.

So our new differential system is:

$$\begin{aligned} S' &= -0.1ES - 0.8ES - \beta_1 SI + b(S + S_{AB} + S_C + I) - dS \\ S'_{AB} &= 0.1ES - \beta_2 S_{AB} I - dS_{AB} \\ S'_C &= 0.8ES - \beta_3 S_C I - dS_C \\ I' &= \beta_1 SI + \beta_2 S_{AB} I + \beta_3 S_C I - \gamma I \\ R' &= \gamma I \\ E' &= \left(\frac{I}{I + S + S_{AB} + S_C}\right) rE(1 - E) \end{aligned} \tag{2}$$

with initial conditions $S(0), S_{AB}(0), S_C(0), I(0), R(0)$, and $E(0)$. The entering rate is b and the general death rate is d. Individuals enter the general susceptible class S only. Now that we have three susceptible classes, we need three infection rates $\beta_1, \beta_2, \beta_3$ for S, S_{AB}, and S_C, respectively, for the susceptibles' interactions with the infected class I. Notice that as a result of interactions of individuals in class S with the educational campaign class E, a proportion of the susceptibles leave the general susceptible class S and move to S_{AB} and S_C. Also, as a result of each susceptible class interacting with the infected class, we have individuals leaving at their respective rates and moving to the infected class. The death rate from HIV is still γ as in the basic model, where individuals leave the infected class I and move to the removed class R. In this model, we have mass action incidence terms like $\beta_1 SI$.

Recognizing the variety of viewpoints about the form of the incidence terms, [15, 23] such as $\beta_1 SI$, we consider parameter estimates and error calculations for mass action incidence terms as well as standard incidence terms. Standard incidence terms, such as $\frac{\beta_1 SI}{N}$, with $N = S + S_{AB} + S_C + I$ are frequently used for epidemics in human populations. The standard incidence terms like $\frac{\beta_1 SI}{N}$ can be interpreted with β_1 as the average number of adequate contacts (i.e., contacts sufficient for transmission) of a person per unit time, $\frac{\beta_1 I}{N}$ as the average number of contacts with infecteds per unit time of one susceptible, and thus $\frac{\beta_1 SI}{N}$ is the number of new infected cases per unit time due to the S class [15]. In our model (2), we have mass action incidence terms such as $\beta_1 SI$, implying that the contact rate increases linearly with the population size. In our estimates, we will see which type of incidence terms give a better fit.

We did not include vertical transmission terms in this model, because the number of adults entering the population with HIV is quite small. Between 1992 and 2005, the percentage of children (under age 15) living with HIV, ranged from 0.1 to 0.8 percent. Also in this period, about half of the total population were children.

We remark that some asymptotic behavior can be obtained with the model with standard incidence terms. The equation for N in that case is

$$N' = (b - d)N - (\gamma - d)I.$$

Consider the proportions, $i = I/N, s = S/N, s_{AB} = S_{AB}/N$, and $s_C = S_C/N$, and it follows that

$$i' = i[\beta_1 s + \beta_2 s_{AB} + \beta_3 s_C - (b + \gamma - d) + (\gamma - d)i].$$

With $\beta_1 > \beta_3 > \beta_2$ and $\beta_1 < b + \gamma - d$ (which holds here), one can show that $i(t)$ goes to 0 as $t \to \infty$. But our emphasis in this paper is not on long term dynamics;

we are analyzing the effect of IEC on the HIV epidemic in Uganda in a short span of years before drug treatment was introduced in a significant way.

4. **Empirical data and parameter estimation.** We decided to start our model with 1992, the year that the information and educational campaigns started. We collected data from UN, UNAIDS, and the Uganda AIDS Commission for population, death rates, percentage of adults (ages 15 to 59) per year, growth of the adult class, and the adult prevalence rates, percent of adult population infected. We used subject matter experts, literature and surveys to determine organizational estimates of IEC, rates for interaction of E and S classes, and efficacy of AB and C type behavior.

First let us discuss the initial conditions for our set of differential equations. Our model considers only adults in the population classes. Since we are starting with 1992, we will base $S(0)$ and $I(0)$ on data collected. We will assume that no one is initially practicing the AB and C type behaviors. Note that the R class accumulates the deaths from HIV. Based on the UN population reports for 1991, there were 18.38 million individuals in Uganda and the adult population, ages 15 to 59, was 32.1%. This gives 5,899,980 individuals for the starting susceptible and infected $(S(0)+I(0))$ class for that year. During that time the adult prevalence rate was about 15%, which gives 884,997 individuals for the starting infected $(I(0))$ class. Thus there are 5,014,980 individuals in the susceptible $(S(0))$ class. For $E(0)$, we estimate that initially 240 organizations (20%) were involved in ABC campaigns. Since this estimate of the initial number of organizations is uncertain, we will vary this in some of numerical runs. So we have the following set of initial conditions:

$$\begin{aligned} S(0) &= 5.014980 \\ S_{AB}(0) &= 0 \\ S_C(0) &= 0 \\ I(0) &= 0.884997 \\ R(0) &= 0.1 \\ E(0) &= 0.20. \end{aligned} \tag{3}$$

Next we turn to the parameter estimates.

5. **Model simulation and comparison.**

5.1. **SIRE simulation and parameter estimation.** We will be running our model for 15 years (1992 through 2006). For any model run, all rates $b, d, r, \beta_1, \beta_2, \beta_3, \gamma$ are assumed to be constant.

Based on data,[10, 11, 16, 33, 38, 42] we can calculate the number of new adults as a percentage of the existing adults. Since we have the UN data for every year as the table on the next page shows, the entering adults from 1990 to 2005 were averaged to get entry rate into susceptibles. The general death rate is also an average based on given death rates for each five-year period from the UN. For our model simulation, we use susceptible adult population entering rate $b = 0.55$ and general population death rate $d = 0.0176$.

The UNAIDS Ugandan reports give the following death rates from HIV: 17.2% (1997), 13.36% (1999), 14% (2001) and 14.42% (2003). Therefore, we decided to estimate removal rate $\gamma = 0.14$.

The infection rate parameters, β_1, β_2 and β_3, as well as the growth rate r for the E class are the hardest to estimate. So we will assume that β_2 and β_3 are each directly proportional to β_1. This will help simplify the model so that we need to determine only one infection parameter. We will assume that β_2 which is the infection rate for the susceptible population practicing AB behavior, is much smaller than β_1, i.e., $\beta_2 = 0.05\beta_1$ ($\beta_2 << \beta_1$) [6, 12, 31]. Note that due to some uncertainty in β_1, in the next section, we also record the error when $\beta_2 = 0$. The infection rate for the susceptible population practicing C behavior with the infected population is given by β_3. We estimated that $\beta_3 = 0.40\beta_1$ ($\beta_2 << \beta_3 < \beta_1$) [48]. The general infection rate β_1 is the hardest to determine; therefore we will consider a range of values for β_1 to determine; the best fit. We will vary r in increments as well, to determine which value, together with β_1 gives us the model, fits the data best.

The general infection rate β_1 and growth rate r for the educational campaigns E are the hardest to determine; therefore we will run a series of values for β_1 and r to determine the best fit. Based on [27, 31], we estimated the bounds of $0.0001 \leq \beta_1 \leq 0.1$, which we will use as starting points for β_1, varying it in increments of 0.001. This will give us 100 values for the β_1 parameter. The growth rate r is assumed to be between 0.20 and 2. We will vary the value r in increments of 0.01 for a set of 180 r values. For each pair of (β_1, r) we will run the set of differential equations with a MATLAB DE solver. This will give model estimates for the values for each class for each year. These model numbers will be compared against a set of estimated data from the UN from 1999, 2000, 2001, 2003, and 2005 to compare the various simulation runs to determine the smallest model error. The following Uganda data are collected from the UNAIDS reports and databases [10, 11, 16, 33, 38, 42]. We decided to scale these numbers of organizations by a total capacity of 1200 organizations. Organizations estimates for E came from Inventory of Agencies [19, 20]. Also the field activities conducted by Albright et. al. [1] gave further data. Personal correspondence with Rosemary Kabugo at the Uganda AIDS Commission filled in other data about organizations. The data points are below:

TABLE 1. Data table

Year	**Susceptible**	**Infected**	**Deaths/Yr**	**Effort E**
1997				600/1,200
1999	6,700,000	606,000	83,492	
2000	6,597,470	573,693	80,317	700/1,200
2001	7,130,000	383,000	47,000	717/1,200
2003	7,462,000	544,000	55,000	
2005	7,636,000	548,261	45,000	778/1,200

Using the data points and the corresponding points from the model simulation, the model error for each run of r and β_1 is given by

$$\text{Model error}(r, \beta_1) = \sum_i \left[\frac{|\text{Model}(S + S_{AB} + S_C)_i - \text{Data}(S)_i|}{\text{Data}(S)_i} + \frac{|\text{Model}(I)_i - \text{Data}(I)_i|}{\text{Data}(I)_i} + \frac{|\text{Model}(D)_i - \text{Data}(D)_i|}{\text{Data}(D)_i} \right] + \sum_j \frac{|\text{Model}(E)_j - \text{Data}(E)_j|}{\text{Data}(E)_j} \tag{4}$$

where the removed data is used to calculate deaths per year, $D_i = (R_i - R_{i-1})$. In our sums above, we use years

$$i = 1999, 2000, 2001, 2003, 2005 \text{ and } j = 1997, 2000, 2001, 2005.$$

We will select the parameter pair giving the lowest model error as the best estimated pair for the model. As a result of our model runs, the best values for the pair of (β_1, r) is $\beta_1 = 0.0201$ and $r = 1.87$, with corresponding model error 2.0225. Figure 2 shows the various β_1 and r pairs and their respective model errors.

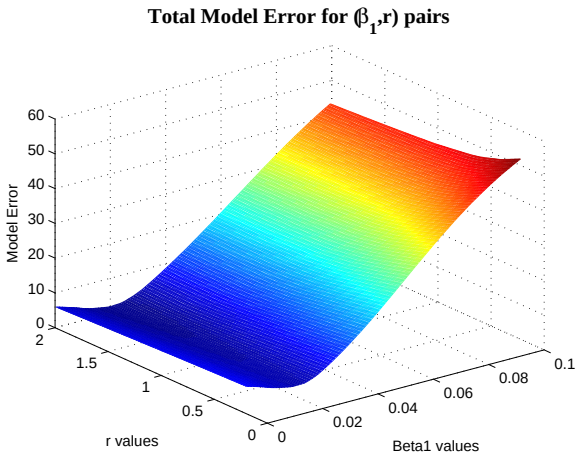

FIGURE 2.

Figure 3 shows the model S classes (total of S, S_{AB} and S_C) compared against our data points. In the following three graphs, note that each population is given in millions. Notice the model graph has a similar shape but is lower than the data.

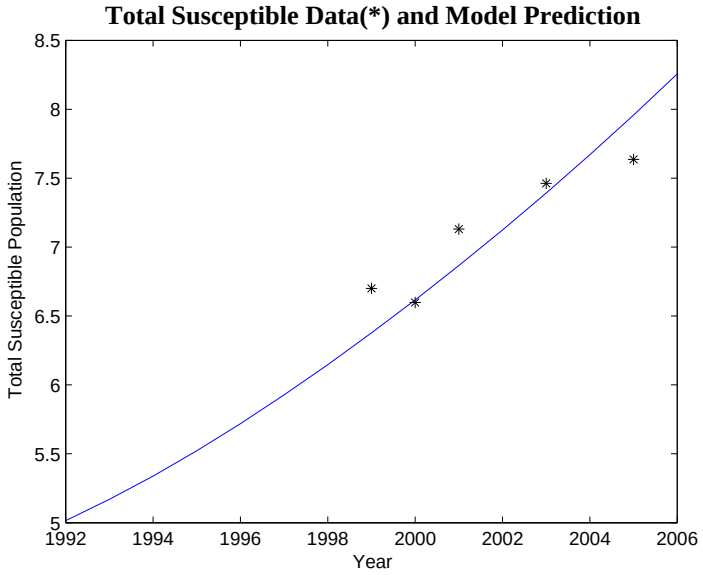

FIGURE 3.

In Figure 4, the graph for the infected class I has a similar shape to the data.

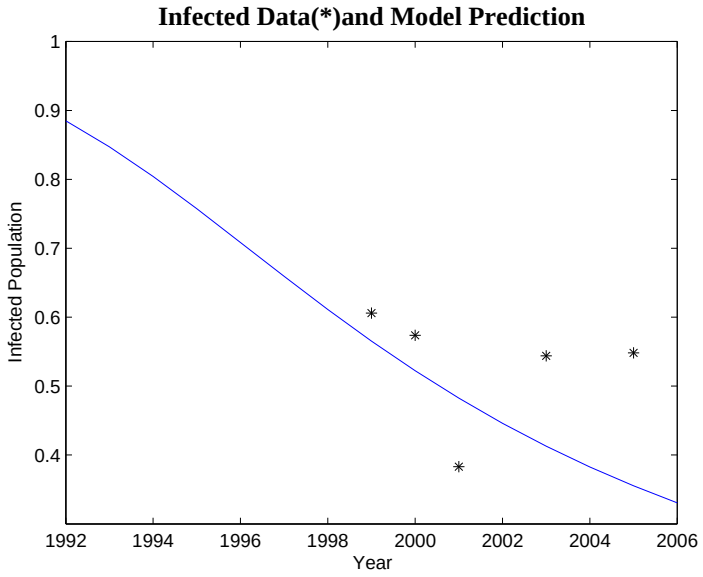

FIGURE 4.

In Figure 5, the graph for the deaths per year is in between the data points.

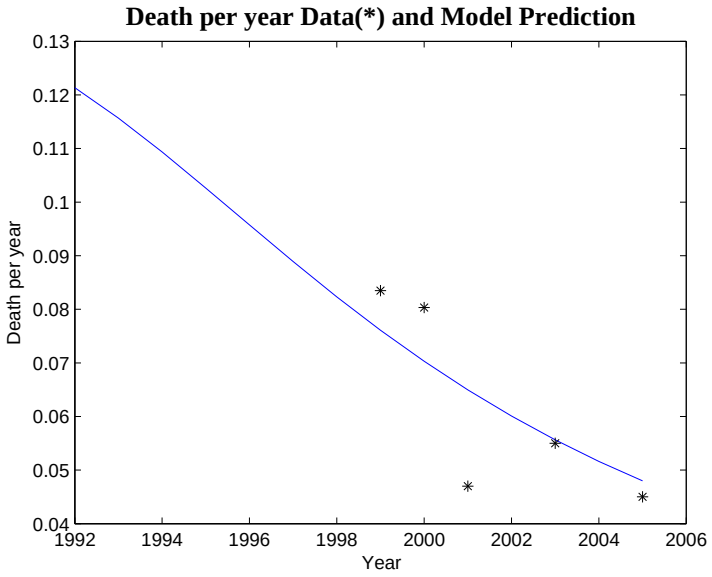

FIGURE 5.

The graph for the E class is fairly close to the E data points in Figure 6.

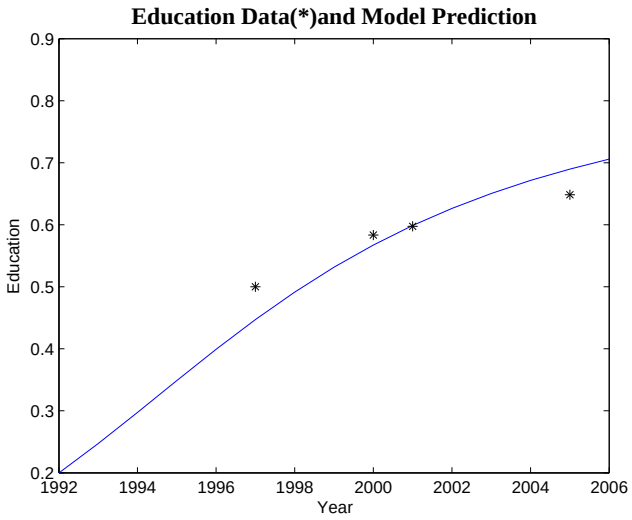

FIGURE 6.

5.2. **Model comparisons with a standard SIR model.** In this section we compare total error from our SIRE model and total error from the base SIR model. But first in this subsection, we simulate our SIRE model for

$$E_0 = 0.20, 0.25, 0.30, 0.35, 0.40$$

and using the method in the previous section, tabulate the best β_1 and r pairs. Note that in tables 2 and 3 we used mass action incidence terms.

TABLE 2. Lowest model SIRE error

Model SIRE error (with E)	2.0225	1.9557	1.8915	1.8816	1.9370
β_1 value	0.0201	0.0201	0.0211	0.0211	0.0221
r value	1.87	1.58	1.31	1.08	0.84
E_0 value	0.2	0.25	0.3	0.35	0.4

Using the β_1 and r ranges above, we ran simulations of the base SIR model and calculated the lowest error between the data points and the corresponding points from the simulation. To compare these errors to the errors for the SIRE model, we subtracted from the SIRE model error the part of the error from the E component. The comparisons of both errors are shown below and we find that our SIRE model always has lowest error.

TABLE 3. Compare lowest SIRE error with base model SIR error

Model SIRE error (without E)	1.6803	1.6911	1.6970	1.7065	1.7126
Base Model SIR error	3.5161	3.6189	3.6892	3.7391	3.7760
E_0 value	0.2	0.25	0.3	0.35	0.4

Next, we show the corresponding results for the use of the standard incidence terms in both the SIRE and SIR models. Note that the errors with mass action incidence terms (like $\beta_1 SI$) are smaller. Thus we conclude the the model with the mass action incidence terms gives a better fit than the one with the standard incidence terms. Also we record that the triple $(\beta_1, r, E_0) = (0.0211, 1.08, 0.35)$ is the one that gives the lowest error over both of types of incidence terms.

TABLE 4. Lowest model SIRE error for standard incidence terms

Model SIRE error (with E)	2.0893	2.0042	1.9296	1.9213	1.9716
β_1 value	0.0661	0.0661	0.0661	0.0661	0.0661
r value	1.95	1.64	1.36	1.1	0.88
E_0 value	0.2	0.25	0.3	0.35	0.4

Since there is some uncertainty in the choice of β_2 as a small multiple of β_1, we ran results with a few different small multiples and did not see much difference. For example, when we ran the above results with $\beta_2 = 0$ and $E_0 = 0.2$ and mass action incidence terms, we obtained the SIRE error 2.0165 with $\beta_1 = 0.0201$ and $r = 1.87$. We also noticed that the SIRE error (without E) is 1.6756 and the SIR error is 3.5160. Similarly, with the standard incidence terms and that β_2 and E_0, we obtained the SIRE error 2.0744 with $\beta_1 = 0.0661$ and $r = 1.95$. The SIRE error (without E) is 1.7383 and the SIR error is 3.4966.

6. **Model enhancements and conclusions.** This work indicates that refining the susceptible class based on behavior change and including the dynamics of the information level are valid features. The SIRE model with mass action incidence terms gives the lowest error when compared with the SIRE model with standard incidence terms and with the base SIR model with our limited amount of data. Of course, we note the importance of finding more data and including other features to make this model more realistic. Other formats for the E equation could be considered.

Many enhancements should be considered for future work. First, one would consider further refining the S_{AB} class into S_A and S_B classes. The S classes and I classes could be refined based on age, gender and stages of the disease. The work of de Walque [7] indicates that women have been more responsive to the dissemination of information about the epidemic in Uganda and this issue would be interesting to further investigate. The gender issue may be important to consider in future modeling efforts.

Later, one could consider including differential infectivity or staged progression in HIV in an expanded model such as that of Hyman et. al. [17]. But one would need to search for data to get initial conditions and other data for the subclasses of the infecteds.

It would also be interesting to add drug treatments to the model even if the treatments are only available in limited numbers. It would be helpful to look at the different types of organizations involved and formats of information disseminated.

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