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EXAMINATION OF A SIMPLE MODEL OF CONDOM USAGE AND INDIVIDUAL WITHDRAWAL FOR THE HIV EPIDEMIC

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Abstract. Since the discovery of HIV/AIDS there have been numerous mathematical models proposed to explain the epidemic of the disease and to evaluate possible control measures. In particular, several recent studies have looked at the potential impact of condom usage on the epidemic [1, 2, 3, 4]. We develop a simple model for HIV/AIDS, and investigate the effectiveness of condoms as a possible control strategy. We show that condoms can greatly reduce the number of outbreaks and the size of the epidemic. However, the necessary condom usage levels are much higher than the current estimates. We conclude that condoms alone will not be sufficient to halt the epidemic in most populations unless current estimates of the transmission probabilities are high. Our model has only five independent parameters, which allows for a complete analysis. We show that the assumptions of mass action and standard incidence provide similar results, which implies that the results of the simpler mass action model can be used as a good first approximation to the peak of the epidemic.

1. **Introduction.** Human immunodeficiency virus (HIV), which is now epidemic worldwide, is spread mainly through unprotected sexual intercourse and the sharing of contaminated needles. As such, the correct and consistent use of condoms is expected to be an effective means of slowing, or halting the spread of the virus. This paper presents a simple model for the spread of HIV and its control by condom use. Other control strategies for HIV include vaccination [5], microbicides [6] and antiretroviral therapy. However, although researchers are optimistic [7, 8], there is currently no vaccine available for HIV, and the long-term use of treatment methods risks development of resistant strains of the virus. The aim of this paper is to investigate the potential use of condoms as a control strategy for HIV when withdrawal from sexual activity is included for those individuals in the later stages of their HIV infection. In this section we review the background of the HIV epidemic and models for its spread. In Section 2 we introduce our model and discuss our analysis and results, focusing on the implications for disease control.

According to the Centre for Disease Control, the first known case of HIV was in San Francisco in the early 1980s [9]. Since then the number of individuals developing AIDS has increased rapidly each year, from approximately 8 million cases worldwide in 1990 to 39 million cases in 2006 [9]. The rapid increase in AIDS cases is most noticeable in developing countries such as Africa, where the prevalence rate among

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pregnant women has risen from 4.3% in 1999 to 30.2% in 2005. In Canada, there are approximately 60,000 people living with HIV whereas in 1996 there were 35,000. Among this group, females now account for approximately 23% of the cases, whereas between 1979-94 they accounted for 7% of the cases [10].

As with most models for disease transmission and control, our model is based on the simple SIR model [2]. The main parameter of the SIR model is the basic reproduction number, $\mathcal{R}_0$. If this parameter is below one, then the disease dies out, whereas if this parameter is above one, any small introduction of infected individuals in the population results in an oscillatory approach to an endemic equilibrium. Mathematically, there is a trivial equilibrium, known as the disease-free equilibrium, which is globally asymptotically stable whenever $\mathcal{R}_0 < 1$ [11]. For many diseases, HIV included, the initial peak is much larger than the final endemic prevalence of infection, and the period of oscillation is long compared with the time scale of interest for disease control. For this reason, the main focus of this paper is the peak of the initial epidemic.

The simplest theoretical result follows from assuming that condom usage directly affects the reproduction number by reducing transmission rates. Suppose condom use reduces transmission by a factor p, on average, with $0 \leq p \leq 1$. The simple SIR model then predicts that an epidemic can be controlled if $(1-p)\mathcal{R}_0 < 1$, or equivalently, $p < p_c = 1 - 1/\mathcal{R}_0$. A conservative estimate of 0.6 for p_c (see below) suggests that condoms alone would be sufficient to control the HIV epidemic provided $\mathcal{R}_0 < 1/(1-0.6) = 2.5$. Estimates of $\mathcal{R}_0$ for HIV vary widely, but this value is likely low and one concludes that condom use alone is not sufficient to control the epidemic. However, condoms in combination with other approaches may be sufficient.

Greenhalgh et al [1] analyzed a two-group deterministic model to assess the potential effectiveness of condom usage as a control strategy for HIV/AIDS in a homosexual population. Their model exhibited a threshold for the reproduction number below which the disease-free equilibrium was locally asymptotically stable. However, they also showed the existence of a locally stable endemic equilibrium for $\mathcal{R}_0 < 1$. Hence, disease control requires restraints on the initial conditions in addition to the usual requirement that the reproduction number be below one. Greenhalgh also investigated the effects of an individual's behavioral changes based on the society they live in when determining an individual's inclination/disinclination to use condoms. They found that when accounting for behavioral changes, condoms can have a positive effect on reducing the HIV epidemic. They found such effects to include a reduction in the peak prevalence of HIV and delays in the time taken to reach this peak [1, p.252]

More recently, Moghadas et al [12] proposed a three-compartment, deterministic model to analyze the effects of condom use on the HIV epidemic. The model predicts that if the average number of sexual partners is small (fewer than seven) then condoms can be an effective control strategy for the HIV epidemic. However, if the number of sexual partners is larger, the level of condom usage required to control the epidemic is unrealistic.

Mukandavire and Garira [4] analyzed a two-sex model to investigate condom usage as a potential control strategy for HIV/AIDS. They considered only heterosexual transmission with the population divided into three compartments: susceptibles, individuals who are infected with the virus but have not developed AIDS and those who have developed AIDS. They assumed all individuals in the susceptible

class to be sexually active and all individuals who have developed AIDS and are showing symptoms withdraw from sexually activity. They first formulated a fixed time delay model without condom usage or any other control measure as a base model to compare the results from the condom usage models. The fixed time delay model was chosen to represent the long incubation period of HIV. Mukandavire and Garira considered two cases: male condoms only, and male and female condoms. Female condom usage was included because women are more vulnerable to HIV infection [4]. The results for the male condom usage with no female condom usage showed a preventability value of 90% for male condoms is needed to ensure the male condom-induced basic reproduction number is less than one. When male and female condoms are used, a preventability value of 80% for both is necessary to ensure the male and female-condom induced reproduction numbers below unity. Results also indicated there are certain values of the basic reproduction number that won't reduce the male and female-condom induced reproduction numbers below unity even with 100% preventability. They also showed that even for values where the reproduction number can be reduced below unity, the necessary preventability values are quite high and perhaps unattainable. Thus the authors concluded that condoms may not be effective in stopping the spread of the epidemic.

We extend this previous research by modeling contacts by individuals in the second infectious stage of infection differently. Rather than assume that individuals in the second stage make contacts at a different rate (the rate c_2 in [12]), we assume that the contacts are made at the same rate, but that a fraction, q, of individuals withdrawal from sexual activity. The fraction q appears in both the numerator and the denominator of the incidence term. We show that model parameters influence the peak of the epidemic through R_c, so that the effectiveness of control measures can be determined solely by analyzing R_c. This greatly simplifies the sensitivity analysis. We further show in Section 3 that although the longer term dynamics of disease prevalence are very sensitive to the choice of incidence, the peak of the epidemic is only mildly affected by a change in incidence terms. This allows one to use the simpler mass action model as a first approximation to the peak.

2. **Model and analysis.** After an initial symptomatic infection lasting 6 to 18 weeks, HIV enters a dormant stage, with low viral load, lasting 10 years, on average. This is followed by an increased viral load, decreased $CD4^+$ count, vulnerability to infection and eventually AIDS. However, it is thought that individuals in their final years before AIDS and morbidity may be highly contagious and asymptomatic [13]. We model this by the introduction of two infectious stages. The model consists of the following system of differential equations:

$$\frac{dS}{dt} = \pi - \lambda(S, I, A) - \mu S, \tag{1}$$

$$\frac{dI}{dt} = \lambda(S, I, A) - (\mu + \sigma)I, \tag{2}$$

$$\frac{dA}{dt} = \sigma I - (\mu + \gamma)A, \tag{3}$$

with

$$\lambda(S, I, A) = \frac{(1-p)cS}{N - qA}\big(\beta_1 I + (1-q)\beta_2 A\big), \tag{4}$$

together with the initial conditions

$$\begin{aligned} S(0) &= S_0, \\ I(0) &= I_0, \\ A(0) &= A_0. \end{aligned} \tag{5}$$

Here, $S(t)$ is the number of susceptible individuals, $I(t)$ is the number of individuals in the first infectious stage, $A(t)$ is the number of individuals in the second infectious stage and $N(t) = S(t) + I(t) + A(t)$ is the total population. Note that $S_0 = N_0 = \pi/\mu$, with $I_0 = A_0 = 0$ leads to a constant solution known as the disease-free equilibrium. We are interested in initial conditions with $S_0 < N_0 = \pi/\mu$, $0 < I_0 << N_0$ and $A_0 = 0$, which represent a small deviation from this equilibrium.

Sexually active infected individuals make contacts at a rate c, but, assuming a fraction q of the infectious individuals in the second stage withdraw from contact, only a fraction $\dfrac{S}{N - qA}$ of these contacts are with susceptible individuals [14]. In the absence of condom use, a fraction β_1 of contacts made by individuals in the first infectious compartment lead to new infections. Condom use prevents transmission in a fraction p of these potential contacts, as described in detail below. Thus, the incidence of new infections due to contacts with the $I(t)$ individuals in the first infectious compartment is $(1-p)c\beta_1 IS/(N - qA)$, which is the first component of λ. Active individuals in the second infectious compartment make contacts at the same contact rate c. However, since only a fraction, $1 - q$, of these individuals are sexually active, the incidence of infection from these contacts is given by $(1 - q)c\beta_2 AS/(N - qA)$ where a fraction β_2 of contacts with an second stage infected individual leads to new infectious. The two transmission probabilities, β_1 and β_2, reflect the fact that the probability of transmission may be higher for individuals in the second infectious stage due to a higher viral load [15]. The two terms together make up the total incidence $\lambda(S, I, A)$. Individuals progress from the first infectious compartment to the second at a rate σ, and the mortality rate for individuals in the second infectious compartment is γ. Susceptible individuals are recruited into the sexually active population at a rate π and removed at a rate μ.

Condom use is assumed to reduce transmission by a factor p, on average, with $0 \leq p \leq 1$ [12]. This parameter combines two effects: compliance and efficacy. Condom compliance measures the fraction of sexual contacts for which a condom is used consistently and correctly. Condom efficacy measures the protection individuals receive by condom usage and is mainly a product of condom breakage, leakage, or slippage [16, 17, 18, 19].

Before proceeding with the analysis, it is convenient to define several important parameter combinations.

$$\mathcal{R}_I = \frac{c\beta_1}{\mu + \sigma}, \quad \mathcal{R}_A = \frac{c\beta_2}{\mu + \gamma}, \quad \tau = \frac{\beta_2}{\beta_1}, \quad \chi = \frac{(\mu + \gamma)}{(\mu + \sigma)}, \quad \varepsilon = \frac{\mu}{(\mu + \sigma)}, \quad N_0 = \frac{\pi}{\mu}.$$

The interpretations of the parameters are as follows: $\mathcal{R}_I$ and $\mathcal{R}_A$ are the expected number of secondary infections made by an infectious individual during the first and second stages of infection respectively, in absence of prevention and withdrawal; τ is the ratio of the transmission probabilities in the first and second infectious compartments; χ is the ratio of the mean time spent in the first and second infectious compartments; ε is the ratio of the expected, or mean, duration of the first infectious period to the mean lifespan in absence of HIV/AIDS related deaths; and N_0

is the population at the disease-free equilibrium defined below. Note that these parameters are not independent, since $\tau = \chi\mathcal{R}_A/\mathcal{R}_I$.

The model (1) - (5) has a unique disease-free equilibrium given by $S(t) = N(t) = N_0 = \pi/\mu$, $I(t) = 0$ and $A(t) = 0$. The technique of van den Driessche and Watmough [11] can be applied to show that the disease-free equilibrium is locally asymptotically stable whenever the control reproduction number, $\mathcal{R}_c$, defined by

$$\mathcal{R}_c = (1-p)c\left(\frac{\beta_1}{\mu+\sigma} + \frac{\beta_2(1-q)\sigma}{(\mu+\gamma)(\mu+\sigma)}\right) \tag{6}$$

is below one, and unstable if $\mathcal{R}_c > 1$. By the following theorem, local stability implies global stability.

Theorem: The disease-free equilibrium of (1) - (3) is globally asymptotically stable whenever $\mathcal{R}_c < 1$.

Proof. First, it is straightforward to verify that (1) - (5) with $S_0,\ I_0,\ A_0 \geq 0$ is well posed in the sense that there exists a nonnegative solution for all time. Let

$$D = \{(S, I, A) \in \mathbb{R}^3_+ \mid S,\ I,\ A \geq 0,\ S + I + A \leq N_0\}, \tag{7}$$

and let $F(I, A) = \mathcal{R}_c I + (1-p)(1-q)\mathcal{R}_A A$. Summing (1) - (3) gives

$$N' = N_0 - \mu N - \delta A.$$

It follows that $N' < 0$ if $N > N_0$, and hence D is attracting and positively invariant. Finally, since F is a Lyapunov function on D [20], it follows that $\lim_{t\to\infty} I(t) = 0$ and $\lim_{t\to\infty} A(t) = 0$. Hence, the ω - limit set of ${R_+}^3$ is contained in the set

$$\{0 \leq S \leq N_0,\ 0,\ 0\}.$$

Inspection of (1) confirms that $\lim_{t\to\infty} S(t) = N_0$, and the disease-free equilibrium is globally asymptotically stable as claimed. □

3. **The epidemic peak.** Let $x = S/N_0$ $y = I/N_0$ and $z = A/N_0$, and let $t^* = (\mu+\sigma)t$. This change of variables leads to the system

$$\dot{x} = \varepsilon(1-x) - \lambda(x, y, z), \tag{8}$$

$$\dot{y} = \lambda(x, y, z) - y, \tag{9}$$

$$\dot{z} = (1-\varepsilon)y - \chi z, \tag{10}$$

with

$$\lambda(x, y, z) = \frac{(1-p)\mathcal{R}_I(y + (1-q)(\tau/\chi)z)x}{x + y + z - qz}, \tag{11}$$

and $y(0) = y_0 = S_0/N_0$, $x(0) = 1 - y_0$, $z(0) = 0$. The dot on the righthand side of (8) - (10) represents differentiation with respect to the new time variable t^*. The scaling of $1/(\mu+\sigma)$ for time is the expected duration of the first stage of infection, before progression to AIDS.

The expression for λ given by (11) is referred to as the standard incidence. The analysis of this section compares the results using standard incidence with those of the simpler mass action model with $\lambda(x, y, z) = (1-p)\mathcal{R}_I(y + (1-q)(\tau/\chi)z)x$.

Based on estimates from several sources [1, 21], parameters for the simulations were sampled uniformly from the following ranges:

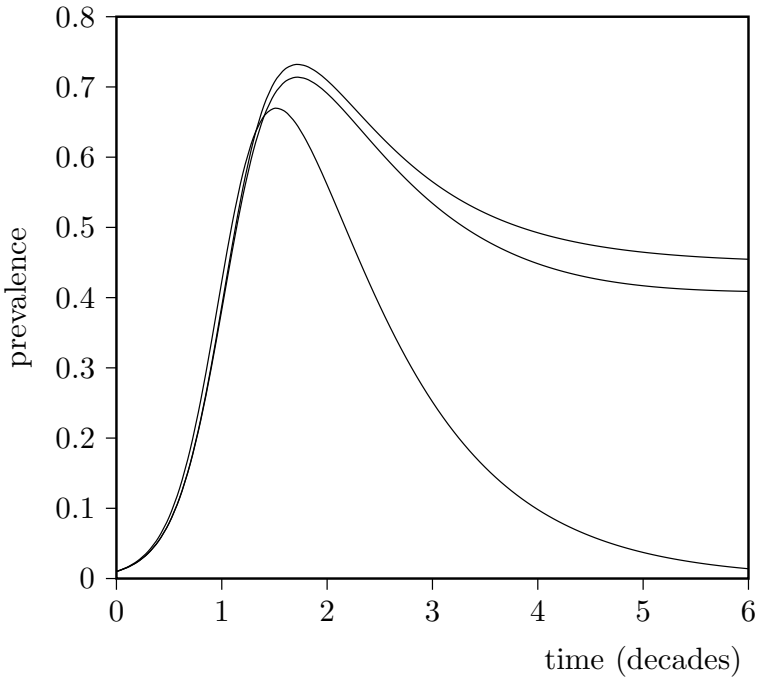

FIGURE 1. Time trace of solutions for three cases: mass action (upper curve), standard incidence (middle curve) and mass action with $\varepsilon = 0$ (lower curve). The remaining parameters are as detailed in the text.

TABLE 1. Number of outbreaks for different values of p

preventability (p)	0.0	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1.0
number of outbreaks	100	100	100	100	98	93	83	65	33	1	0

$$.05 \leq \varepsilon \leq .30$$
$$2 \leq \chi \leq 6$$
$$1 \leq \mathcal{R}_I \leq 3$$
$$1 \leq \tau \leq 10$$

During the early stages of HIV and all stages of AIDS, individuals are very infectious leading to the large range of transmission probabilities [15].

Figure 1 shows the prevalence as $y(t^*) + z(t^*)$ for three simulations: standard incidence (middle curve), mass action (upper curve) and mass action with $\varepsilon = 0$ (lower curve). The remaining parameters are as follows: $\mathcal{R}_I = 4.94$, $\tau = 1.53$, $\chi = 2.48$, $\varepsilon = 0.40$, $p = 0$, $q = 0$, $y_0 = 0.01$. These parameter values lead to $\mathcal{R}_c = 6.8$. The time scale is in units of mean infectious period, $(\mu+\sigma)^{-1}$, which can be taken as 10 years. The three curves overlap up to the peak, suggesting that the form of the incidence term is not as important for the initial epidemic. We explore the use of a simpler epidemic model in Section 4.

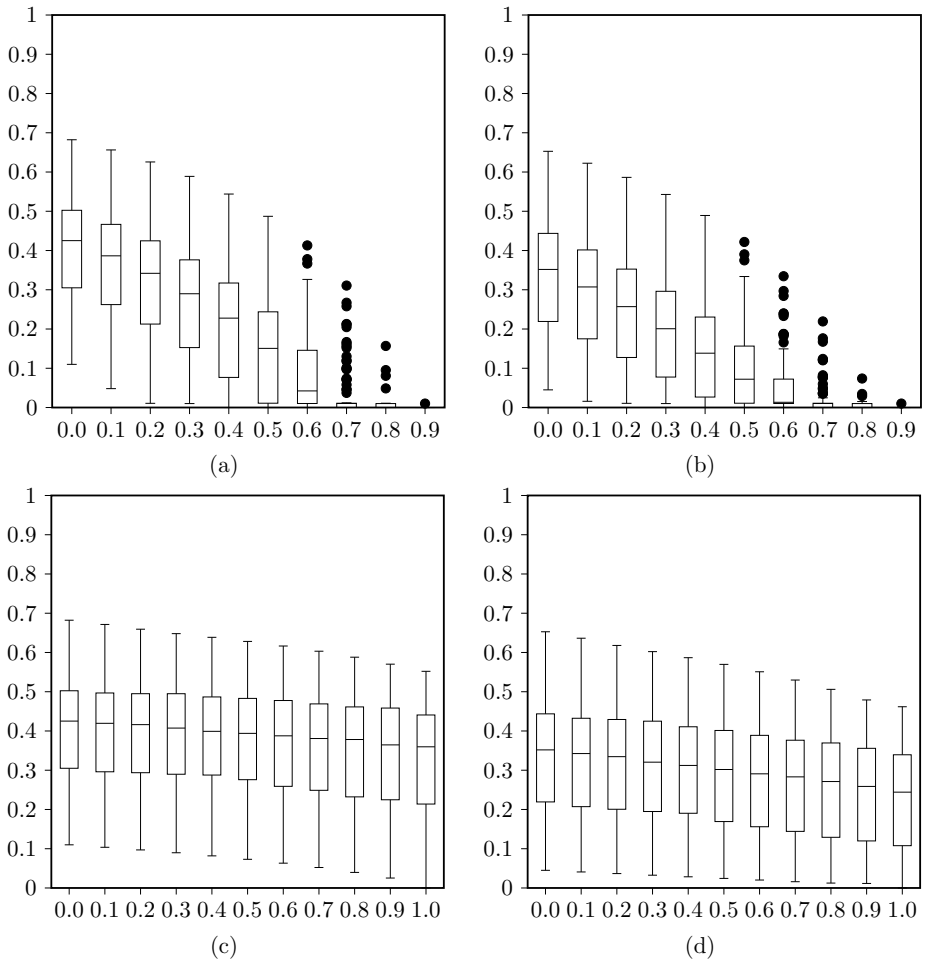

FIGURE 2. Boxplots of epidemic peaks for 100 simulations. (a) and (c) are for standard incidence, and (b) and (d) are for the model with mass action incidence. The lower two plots are peak versus the withdrawl fraction q, and the upper two plots are peaks versus the preventability p.

Figure 2 shows boxplots of the peak caseload of the epidemic vs. p and q for a random sample of 100 points in parameter space. For the 100 parameter sets, $\mathcal{R}_0$ ranged from 1.45 to 12.1 with a median of 4.05. In addition to reducing the peak of the epidemic, condom use may also prevent an epidemic. The number of outbreaks for the different values of p are given in Table 1. Since the peak values are close for the full range of parameter space for both incidence terms, this suggests that one can use the simpler mass action model as a first approximation for the peak of the epidemic. As can be seen in Figure 2, neither the number of outbreaks or the size of the peak is sensitive to the value of q. However, changing p significantly reduces the size of the peak and the number outbreaks.

4. **The epidemic model.** There are two motivating reasons to study the epidemic system given by (8) - (10) with $\varepsilon = 0$. First, the demographic timescale, as measured by ε, is thought to be long, and so ε is a small parameter that can be neglected in a first approximation. Second, and related, simulations of the model suggest that the initial peak of solutions to (8) - (10) is close to the peak of the epidemic model. Current studies suggest that HIV is still an epidemic, so that the study of this peak remains important [22, 23]. This model has no endemic equilibria, and the disease-free equilibrium is replaced with the line of equilibria $y = z = 0$.

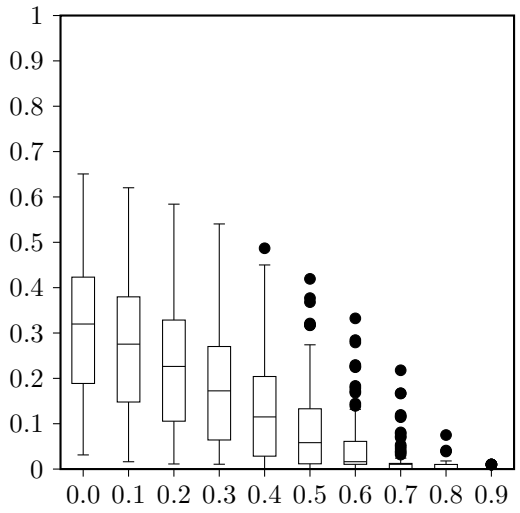

FIGURE 3. Peaks of the epidemic model for various levels of condom preventability, p.

Figure 3 shows boxplots of peaks of the epidemic for the same 100 parameter sets used previously, but with $\varepsilon = 0$ and mass action incidence. The peak values obtained are similar to those obtained for $\varepsilon > 0$ in the previous section. Figure 4 is a plot of the predictions of model (8)-(10) with the standard incidence form of λ versus the predictions of the epidemic model with mass action incidence. Each point on the plot represents the result from one of the 100 parameter sets simulated. The dashed line represents equality of the two predictions, so in almost every case the standard incidence model predicts a higher peak. However, the differences are small and consistent, which suggests that the epidemic model is a good first approximation.

System (8) through (10) with $\varepsilon = 0$ becomes

$$\dot{x} = -(1-p)\mathcal{R}_I c(x,y,z)\left(y + \tau(1-q)z\right)x \tag{12}$$

$$\dot{y} = (1-p)\mathcal{R}_I c(x,y,z)\left(y + \tau(1-q)z\right)x - y \tag{13}$$

$$\dot{z} = y - \chi z \tag{14}$$

where,

$$c(x,y,z) = \frac{1}{x + y + (1-q)z}. \tag{15}$$

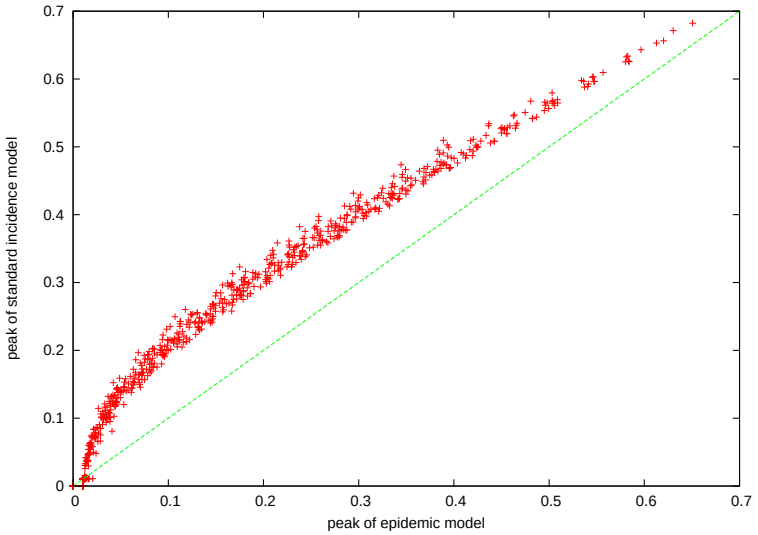

FIGURE 4. Plot of peaks predicted from model (8) through (10) with standard incidence versus the prediction of the epidemic model with mass action incidence.

This simpler system, which we refer to as the epidemic model, no longer has a unique disease-free equilibrium and is similar to the epidemic model studied by Kermack and McKendrick [24]. Kermack and McKendrick showed the final size of a simple epidemic model with no disease induced death depends only on $\mathcal{R}_0$. The final size relation assumes μ is much less then σ or that the life expectancy in absence of HIV is much larger then the lifespan with HIV. It is expected that the parameter ε is on the order of 0.1, and thus the study of the simpler system with $\varepsilon = 0$ is merited. It should be noted that neglecting μ increases $\mathcal{R}_0$, but does allow us to consider a worst-case scenario.

The final size of an epidemic is defined as the fraction of the initial population infected over the course of the epidemic which, with our notation, is $x_0 - x_\infty$. In this section, we develop the final size relation for (12)- (14) and compare the results to the ones obtained by solving (12) through (14) numerically. Let $y_\infty = \lim_{t\to\infty} y(t)$ and $\hat{y} = \int_0^\infty y(t)dt$, provided these limits exist. Summing (12) through (13) and integrating leads to the relation

$$(x_0 - x_\infty) + (y_0 - y_\infty) = \hat{y}. \tag{16}$$

From (12), $x(t)$ is decreasing and bounded below, hence $x_\infty = \lim_{t\to\infty} x(t)$ exists. Further, $\int_0^T y(t)\,dt$ is increasing, and, by (16), bounded above. Hence, $\hat{y}$ exists. Similarly, from (14),

$$(z_0 - z_\infty) = \hat{y} - \chi\hat{z}, \tag{17}$$

so that $\hat{z}$ and z_∞ must also exist. It follows that $z_\infty = y_\infty = 0$. Dividing (12) through by x and integrating yields

$$\log(x_\infty/x_0) = -(1-p)\mathcal{R}_I \int_0^\infty c(x(t), y(t), z(t)) \left(y(t) + \tau(1-q)z(t)\right)\, dt. \tag{18}$$

For our model, $c(x, y, z)$ is not bounded; however, in practice, this function will be bounded above and below by strictly positive constants c_M and c_m, and it follows

that

$$\log(x_0/x_\infty) \le c_M(1-p)\mathcal{R}_I(\hat{y} + \tau(1-q)\hat{z}). \tag{19}$$

From (16) and (17), with $z_0 = 0$, it follows that

$$\log(x_0/x_\infty) \le c_M(1-p)\mathcal{R}_I(1 + \tau(1-q)/\chi)(x_0 - x_\infty + y_0). \tag{20}$$

Since the righthand side is finite, $x_\infty > 0$. That is, the epidemic passes leaving a strictly positive fraction, x_∞, of the initial population untouched by the disease. Equation (20) with inequality replaced by equality gives a lower bound for x_∞. An upper bound for x_∞ can be found by replacing c_M by c_m in (20) and reversing the inequality. Note that for the standard incidence model with $c(x, y, z)$ unbounded, this approach does not yield a lower bound for x_∞. Derivation of a final size relation for a more general model can be found in Arino et al. [25].

For the mass action model, $c_m = c_M = 1$, and the final size relation becomes the equality

$$\log(x_0/x_\infty) = \mathcal{R}_c(x_0 - x_\infty + y_0). \tag{21}$$

Based on the simulation results of the previous section, we conjecture that the initial peak of the model of Section 2 will be similar to that of the simpler mass action model with $\varepsilon = 0$, and since the peak of the epidemic model is related to the final size of the epidemic, study of (21) will lead to insights into the peak of the model of Section 2.

Setting $p = q = 0$ in (6) gives the basic reproduction number

$$\mathcal{R}_0 = c\left(\frac{\beta_1}{\mu+\sigma} + \frac{\beta_2\sigma}{(\mu+\gamma)(\mu+\sigma)}\right) \tag{22}$$

The numbers $\mathcal{R}_c$ and $\mathcal{R}_0$ are usually interpreted as the average number of secondary infections caused by one infectious individual in a totally susceptible population with and without control measures in place [26]. These two numbers are important thresholds for disease spread in epidemiological models. It is evident from (6) that R_c is an increasing function of the partial reproduction numbers, $\mathcal{R}_I$ and $\mathcal{R}_A$, and the fraction, $1-\varepsilon = \dfrac{\sigma}{\mu+\sigma}$, of individuals progressing to the second infectious stage, and is decreasing with the control parameters p and q. It is clear that increasing p from 0 to 1 with $q = 0$, decreases $\mathcal{R}_c$ from $\mathcal{R}_0$ to 0. Further, increasing q from 0 to 1 decreases $\mathcal{R}_c$ from $(1-p)\mathcal{R}_0$ to $(1-p)\mathcal{R}_I$. Moreover, the elasticity [27] of $\mathcal{R}_c$ with respect to p and q can be computed as follows:

$$\frac{p}{\mathcal{R}_c}\frac{\partial\mathcal{R}_c}{\partial p} = -\frac{p}{1-p}, \tag{23}$$

$$\frac{q}{\mathcal{R}_c}\frac{\partial\mathcal{R}_c}{\partial q} = -q\left(\frac{(1-p)\mathcal{R}_A}{\mathcal{R}_c}\right). \tag{24}$$

These elasticities measure the effect a change in p or q has as a proportional change in $\mathcal{R}_c$. The elasticity to p increases nonlinearly with p, so that the proportional change of $\mathcal{R}_c$ to p is small for p near zero, and very large for p near one. It may be more informative to note that the elasticity of $\mathcal{R}_c$ to $(1-p)$ is unity. That is, percent changes in the fraction of people *not* using condoms produced equal percent changes in $\mathcal{R}_c$. In contrast, the elasticity of $\mathcal{R}_c$ to q depends on the ratio $\mathcal{R}_A$ to $\mathcal{R}_c$. Since the viral loads and duration of infection vary between individuals it is important to examine the model for a range of R_I and R_A. If R_I is much larger then R_A then its elasticity to q is small and withdrawl will have little effect. However, if,

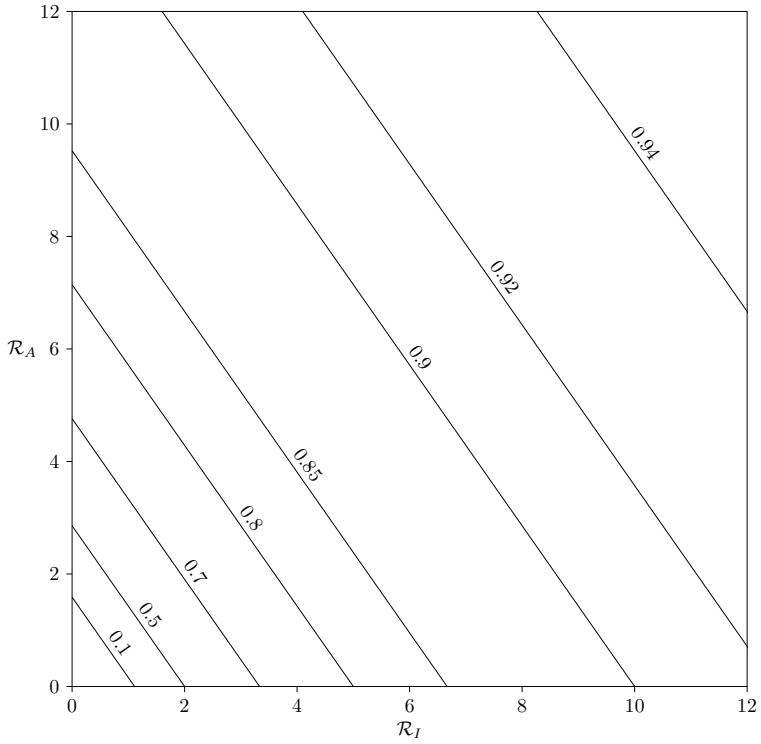

FIGURE 5. Level curves of (26) for $\varepsilon = 0.3$, $q = 0$. The labels above each curve indicate the value of p for which $\mathcal{R}_c = 1$. Thus, disease control is achieved in the triangular region below the line.

as we expect, R_A is much larger then unity, withdrawl can be very effective. Thus, early identification of HIV- progression is important.

In terms of dimensionless parameters, $\mathcal{R}_c$ is

$$\mathcal{R}_c = (1-p)(\mathcal{R}_I + \mathcal{R}_A(1-q)(1-\varepsilon)). \tag{25}$$

Note that $\mathcal{R}_c = \mathcal{R}_I$ in the special case that $R_A << R_I$ (or $q = 1$) and $p = 0$. This would be the case if AIDS symptoms develop rapidly as the viral load increases so that infectious individuals in the second stage withdraw from contact, due to morbidity or death, quickly. The reproduction number is also very sensitive to the ratio $\mathcal{R}_A/\mathcal{R}_I$ especially given the fact that the values of these two parameters are uncertain.

The critical preventability necessary to end an epidemic is calculated by setting $\mathcal{R}_c$ equal to 1 and re-writing (25) as follows:

$$p_c = 1 - \frac{1}{\mathcal{R}_I + \mathcal{R}_A(1-q)(1-\varepsilon)}. \tag{26}$$

Level curves for this relation are straight lines shown in Figure 5.

We now consider two scenarios for individual withdrawal, q, and look at the effects on p_c: first we assume that every individual in the second infectious stage withdraws from sexual activity ($q = 1$) to obtain

$$p_c = 1 - \frac{1}{\mathcal{R}_I}.$$

The assumed condom preventability range of $0.6 < p < 0.9$ then corresponds to a range $2.5 < \mathcal{R}_I < 10$. Estimates for $\mathcal{R}_I$ are in the low end of this range, and thus condoms could be sufficient to drive $\mathcal{R}_c$ below unity, resulting in the disease-free equilibrium becoming globally asymptotically stable. However, this is most likely unattainable as it would be extremely difficult, if not impossible, to have every individual in the second infectious stage withdraw from sexually activity [28]. If we assume that $q = 0$ so that no infectious individuals withdraw we obtain

$$p_c = 1 - \frac{1}{\mathcal{R}_I + \mathcal{R}_A(1-\epsilon)} = 1 - \frac{1}{\mathcal{R}_0}.$$

Thus, if $\mathcal{R}_0 > 6$, as is likely, then $p_c > 0.84$, and it is unlikely that condoms would be sufficient in halting the epidemic.

It can be seen from Figure 5 that with no infectious individuals withdrawing from sexual activity ($q = 0$), a preventability value of approximately 93% would be necessary to ensure $\mathcal{R}_c$ was below unity assuming a worst-case scenario for $\mathcal{R}_I$ and $\mathcal{R}_A$. This figure also predicts that if the current estimates of transmission are high then condoms could be effective as these values would be included in the triangular region for $p_c \leq 0.9$.

5. **Discussion and conclusion.** The purpose of this paper was to investigate the effectiveness of condoms as a control strategy for HIV. We considered a model where a fraction of individuals would withdraw from contact. Although our model is not as complex as other models (i.e., does not consider migration patterns, different contact rates for males and females, different classes for males and females, etc), it does provide us with an easier analysis to obtain $\mathcal{R}_c$ and a critical preventability for control of an HIV epidemic by condom use.

We developed a simple preventability model for control of an HIV epidemic by condom use. The inclusion of q is a necessary parameter to model the contact rate of AIDS individuals given the reduction in incidence levels and number of contacts as discussed in [29, 30]. Also, given the health factors that AIDS-infected individuals face, such as becoming bedridden the inclusion of q for individual withdrawal is a necessary component of the contact rate. As opposed to previous contact rates, having a fraction of the AIDS population withdraw from sexual contact should give more accurate contact rates compared to not including it. We showed that the disease-free equilibrium is globally asymptotically stable whenever $\mathcal{R}_c \leq 1$ and examined the sensitivity of $\mathcal{R}_I$ and $\mathcal{R}_A$ to p and q. We also showed that similar sizes for the peak of the epidemic were obtained using standard and mass action incidence for a full range of parameter space. This implies that one can use the simpler mass action incidence as a first approximation to the peak of the epidemic.

We finally showed that condoms are effective in lowering the number of disease outbreaks and the size of the epidemic. However, assuming that no infectious individuals withdraw from sexual activity ($q = 0$) requires a preventability level of approximately 97% to control the epidemic. Even with some individuals withdrawing from contact, a preventability value in the range of 85 to 93% would be necessary to ensure the disease could not invade the population. This is presumably due to the large range of values for $\mathcal{R}_I$ as q only reduces $\mathcal{R}_A$. Thus, in order for individual withdrawal to be effective it would require individuals in the first infectious stage

who are in the very early symptomatic stages to withdraw as well. We conclude condoms are not sufficient to halt the epidemic unless transmission probabilities are at the lower range of current estimates. A two group model should be studied in more detail to determine if targeted condom use of high risk contacts is feasible as it has been suggested by [19]. It is thought if condoms are introduced into a high-risk group before the disease spreads to the low risk groups then condoms can be sufficient to halt the epidemic. However, if condoms are introduced too late into the high-risk group then condoms alone will not be sufficient.

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