The United States AIDS Epidemic in First World Context [1], [2]

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Abstract The incidence of AIDS cases per hundred thousand in the United States is several times that in the rest of the First World (between five and eight depending on which countries are included in the First World). There does not appear to be much evidence of other First World countries "catching up" to the AIDS rate of the United States. An argument is made that it is the relative ineffectiveness of the American public health management of the epidemic which has been the root cause of the high American incidence. Consideration is given to the possibility that the United States may be "Country Zero," i.e., the possibility that persons infected by HIV in the United States may have been essential to maintain the AIDS epidemic in other First World countries.

Keywords AIDS, bathhouses, control, international.

1 Introduction

Early on in the AIDS epidemic, Thompson [2] developed a model demonstrating that a small subpopulation, with unusually high rates of sexual activity, in the American gay community could serve to drive the endemic across the epidemiological threshhold into a full epidemic. This effect was not due to the attending total increase of sexual activity in the gay community, but rather to the effect of the small subpopulation [3], [4], [5]. From a public health standpoint, the model indicated that it would be appropriate for centers of high contact anonymous sex, such as the gay bathhouses, to be closed. Except for short periods of time in random locations, such closings have not been implemented by the US public health authorities. Thompson has drawn comparisons with the ineffectiveness of US public health policy in the American polio epidemic of the 1940s [4]. In the case of the polio epidemic, it was fairly clear that closings of public swimming pools and cheap Saturday afternoon matinees were indicated, but public health officials did not wish to take the static for such actions.

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[2] Reprinted from *Advances in Mathematical Population Dynamics: Molecules, Cells and Man* (1998), Axelrod, Arino & Kimmel, eds., World Scientific Publishing Company, pp. 345-354

More recent work by Thompson and Go [6] indicates that once the percentage of HIV infectives in a local gay population reaches 40%, the benefit of such closings is probably marginal. There is little doubt that such a rate has been reached already in a number of United States urban centers, though probably not in all. At any rate, American public health policy in the management of the AIDS epidemic has proved catastrophic. Most of the money spent to date in AIDS research comes from American sources. Nevertheless, we continue to have much the highest AIDS rate per hundred thousand in the First World, and recent WHO statistics reveal that the AIDS rate in the United States is several times that of the rest of the First World. The City of Houston alone has more AIDS cases than in all of Canada (which has more than ten times Houston's population). A recent doctoral dissertation by West [7], shows that the weakened resistance of HIV infectives makes them more susceptible to tuberculosis. Since tuberculosis is an aerosol borne disease, West investigated the possibility that number of infections into the nonHIV population could have dramatic public health consequences. Fortunately, his work indicates that this is unlikely, with a worst case scenario of under ten thousand additional TB cases per year for the entire nation. And, his model indicates that modest additional funding to tuberculosis treatment centers might bring the marginal increase in TB due to HIV infectives to no more than a few hundred.

Notions that most persons engaging for long times in high risk behavior will be eliminated by the epidemic seem to be unfortunately accurate in the aggregate. However, notions that the epidemic simply will end as a result of the removal by the disease of these infectives from the population may not be correct. It is entirely possible that, absent a cure or a vaccine, AIDS could have devastating effects in the United States until well into the next century.

2 Current AIDS Incidences

In the matter of the present AIDS epidemic in the United States, a great deal of money is being spent on AIDS. However, practically nothing in the way of steps for stopping the transmission of the disease is being done (beyond education in the use of condoms). Indeed, powerful voices in the Congress speak against any sort of government intervention. On April 13, 1982, Congressman Henry Waxman [1] stated in a meeting of his Subcommittee on Health and the Environment, "I intend to fight any effort by anyone at any level to make public health policy regarding Kaposi's sarcoma or any other disease on the basis of his or her personal prejudices regarding other people's sexual preferences or life styles." (It is significant to note that Representative Waxman, whose district includes Beverly Hills, has been one of the most strident voices in the fight to stop smoking, considering rigorous measures acceptable to end this threat to human health.) We do not even have a very good idea as to what fraction of the target population in the United States is HIV positive, and anything approaching mandatory testing is regarded by American political leaders as an unacceptable infringement of civil liberties.

In Table 1, we show AIDS figures (cumulative except for first quarter of 1995) for representative First World Countries.

Table 1. First World AIDS Figures.

Year	USA	UK	Canada	Australia	Denmark
1989	141443	3426	4342	1891	544
1990	182004	4596	5462	2524	741
1991	251811	5425	7164	3348	925
1992	323872	6907	8624	4078	1109
1993	388434	8516	9914	4785	1347
1994	401789	9865	10391	5075	1549
1995*	64562	1609	1290	707	238
*First Quarter 1995 New Cases					

In Figure 1, we graph AIDS ratios per hundred thousand for the United States divided by those for various countries. We note how the cumulative ratios are nearly the same as those as the ratios of new cases per hundred thousand for the first quarter of 1995. The information in Figure 1 stands on its own. Clearly, the AIDS rates in the United States are much higher than those in other First World Countries. (Other countries not included on the chart include France with a rate one third that of the USA, Holland with a rate one seventh that of the USA, and Japan with a rate less than one two hundredth that of the USA.) And the same is true whether cumulative or new cases are considered. This pattern has been clear for more than a decade. The United States spends more on AIDS research and treatment than the rest of the world combined. But public health and other political officials in the United States have disdained to take any rational action based on our comparative dysfunctionality in the management of the AIDS epidemic. A model based explanation for America's high AIDS rate was given as long ago as 1984 [2].

3 Modeling The "Bathhouse Effect"

To develop a model for AIDS could, quite easily, involve hundreds of variables. At this time, we have neither the data nor the understanding to justify such a model for AIDS. Then, one could argue, as some have, that we should disdain modeling altogether until such time as we have sufficient information. But to give up modeling until "all the facts are in" would surely push us past the time where our analyses would be largely irrelevant. There is every reason to hope that at some future time we will have a vaccine and/or a treatment for AIDS. This was the case, for example, with polio. Do we now have the definitive models for polio? The time for developing models of an epidemic is during the period when they might be of some use. The model below was first presented in 1984 [2]. (A more general version was presented in 1989 [5], but the insights are the same when the simpler model is used.) We shall begin with a classical contact formulation:

$$P(\text{transmission from infective in } [t, t+\Delta t)) = k\alpha\Delta t \frac{X}{X+Y}, \tag{1}$$

where

k = number of contacts per month;
α = probability of contact causing AIDS;
X = number of susceptibles;
Y = number of infectives.

We shall then seek the expected total increase in the infective population during $[t, t+\Delta t)$ by multiplying the above by the total number of infectives.

$$\Delta E(Y) = YP(\text{transmission in } [t, t+\Delta t) \approx \Delta Y. \tag{2}$$

Letting $\Delta t \to 0$, we have

$$\frac{dY}{dt} = \frac{k\alpha XY}{X+Y}, \tag{3}$$

$$\frac{dX}{dt} = -\frac{k\alpha XY}{X+Y}. \tag{4}$$

There are other factors which must be added to the model such as immigration into the susceptible population, λ, and emigration, μ, from both the susceptible and infective populations, as well as a factor, γ, to allow for marginal increase in the emigration from the infective population due to sickness and death. Thus, we have the improved differential equation model

$$\frac{dY}{dt} = \frac{k\alpha XY}{X+Y} - (\gamma + \mu)Y, \tag{5}$$

$$\frac{dX}{dt} = -\frac{k\alpha XY}{X+Y} + \lambda - \mu X. \tag{6}$$

Let us now proceed away from the situation where it is assumed that all persons in the gay population have equal contact rates to one where there are two populations, the majority, less sexually active, but with a minority (e.g., bathhouse visitors) with greater activity than that of the majority. In the following, we shall use the subscript "1" to denote the majority, less sexually active portion of the target (gay) population, and the subscript "2" to denote the minority, sexually very active portion (the part which engages in high frequency anonymous anal intercourse, typically at bathhouses). The more active population will be taken to have a contact rate τ times that of the rate k of the majority portion of the target population. The fraction of the more sexually active population will be taken to be p.

$$\begin{aligned}
\frac{dY_1}{dt} &= \frac{k\alpha X_1(Y_1+\tau Y_2)}{X_1+Y_1+\tau(Y_2+X_2)} - (\gamma+\mu)Y_1; \qquad (7)\\
\frac{dY_2}{dt} &= \frac{k\alpha\tau X_2(Y_1+\tau Y_2)}{X_1+Y_1+\tau(Y_2+X_2)} - (\gamma+\mu)Y_2;\\
\frac{dX_1}{dt} &= -\frac{k\alpha X_1(Y_1+\tau Y_2)}{X_1+Y_1+\tau(Y_2+X_2)} + (1-p)\lambda - \mu X_1;\\
\frac{dX_2}{dt} &= -\frac{k\alpha\tau X_2(Y_1+\tau Y_2)}{X_1+Y_1+\tau(Y_2+X_2)} + p\lambda - \mu X_2.
\end{aligned}$$

where

k = number of contacts per month;
α = probability of contact causing AIDS;
λ = immigration rate into sexually active gay population;
μ = emigration rate from sexually active gay population;
γ = marginal emigration rate from sexually active gay population due to sickness and death;
X = number of susceptibles;
Y = number of infectives.

We note that even with a simplified model such as that presented here, we appear to be hopelessly overparameterized. There is little chance that we shall have reliable estimates of all of: $k, \alpha, \gamma, \mu, \lambda, p, \tau$. One of the techniques sometimes available to the modeller is to express the problem in such a form that most of the parameters will cancel. For the present case, we will attempt to determine the $k\alpha$ value necessary to sustain the epidemic for the heterogeneous case when the number of infectives is very small.

If $Y_1 = Y_2 = 0$, then the equilibrium values for X_1 and X_2 are $(1-p)(\lambda/\mu)$ and $p(\lambda/\mu)$, respectively. Expanding the right-hand sides of (7) in a Maclaurin series, we have (using lower case symbols for the perturbations from 0),

$$\begin{aligned}\frac{dy_1}{dt} &= \left[\frac{k\alpha(1-p)}{1-p+\tau p} - (\gamma+\mu)\right] y_1 + \frac{k\alpha(1-p)}{1-p+\tau p} y_2 \\ \frac{dy_2}{dt} &= \frac{k\alpha\tau p}{1-p+\tau p} y_1 + \left[\frac{k\alpha\tau^2 p}{1-p+\tau p} - (\gamma+\mu)\right] y_2.\end{aligned} \tag{8}$$

Summing, we have

$$\frac{dy_1}{dt} + \frac{dy_2}{dt} = \left[\frac{k\alpha(1-p)+k\alpha\tau}{1-p+\tau p} - (\gamma+\mu)\right] y_1 + \left[\frac{k\alpha\tau^2 p + k\alpha(1-p)}{1-p+\tau p} - (\gamma+\mu)\right] y_2 \tag{9}$$

In order for the coefficient of y_2 to be negative, we require:

$$k\alpha < (\gamma+\mu)\left[\frac{1-p+\tau p}{\tau^2 p + 1 - p}\right] = k^*\alpha \tag{10}$$

In order for the coefficient of y_1 to be negative, we require

$$k\alpha < (\gamma+\mu) \tag{11}$$

Since $\tau \geq 1$, the condition in (10) will guarantee that the inequality in (11) will be satisfied as well. Now, in the homogeneous contact case (i.e., $\tau = 1$), we note that for the epidemic not to be sustained we require the condition in equation (11), i.e.,

$$k_H\alpha < (\gamma+\mu) \tag{12}$$

For the heterogeneous contact case with k^*, the average contact rate is given by

$$\begin{aligned}k_{ave}\alpha &= p\tau(k^*\alpha) + (1-p)(k^*\alpha) && (13)\\ &= \frac{[p\tau + (1-p)]^2}{\tau^2 p + 1 - p} && (14)\end{aligned}$$

So, dividing the sustaining $k_H\alpha$ by the sustaining value for the heterogeneous contact rate, we have

$$Q = \frac{1-p+\tau^2 p}{(1-p+\tau p)^2} \tag{15}$$

We note that we have been able here to reduce the parameters necessary for consideration from seven to two. This is fairly typical for model based approaches: the dimensionality of the parameter space may be reducible in answering specific questions. It is shown elsewhere [5] than the addition of time delay effects between infection and infectiousness to the model still yields precisely the enhancement factor shown in Figure 2. In Figure 2, we note a plot of this "bathhouse enhancement factor." Note that the addition of the bathhouses to the transmission picture had roughly the same effect as if all the members of the target population had doubled their contact rate. And remember that the picture has been corrected to discount any increase in the overall contact rate which occurred as a result of the addition of the bathhouses. In other words, the enhancement factor is totally due to heterogeneity. Here, τ is the activity multiplier for the sexually very active gay subpopulation and p is the proportion of the total sexually active gay population which it constitutes. We note that *for the same total number of sexual contacts across the gay population*, the presence of a small high activity subpopulation can have roughly the same effect as if the entire gay population had doubled its sexual activity. It is this heterogeneity effect which I have maintained for a decade as the cause of AIDS getting over the threshhold of sustainability in the United States.

For a fixed value of τ, we have that Q is maximized when

$$p = \frac{1}{1+\tau} \tag{16}$$

For this value of p, we have

$$Q = \frac{(1+\tau)^2}{4\tau} \tag{17}$$

4 Heterogeneity Effects In The Mature Epidemic

The AIDS epidemic in the United States has long passed the point where perturbation threshhold approximations can be used assuming a small number of infectives. One might well ask the question as to whether bathhouse closings at this late date would have any benefit. To deal with this question, we unfortunately lose our ability to disregard five of the seven parameters, and must content ourselves with picking reasonable values for those parameters. A detailed analysis is given in [6]. Here we shall content ourselves with looking at the case where the contact rate before the possible bathhouse closings is given by

$$(k\alpha)_{overall} = (1 - p + \tau p)(\gamma + \mu). \tag{18}$$

Furthermore, we shall take $\mu = 1/(180 \text{ months})$ and $\lambda = 16{,}666$ per month. (We are assuming a target population, absent the epidemic, of roughly 3,000,000.) For a given fraction of infectives in the target population of π, we ask what is the ratio of contact rates causing elimination of the epidemic for the closings case divided by that without closings. Such a picture is given in Figure 3. It would appear that as long as the proportion of infectives is no greater than 40% of the target population, there would be a benefit from bathhouse closings. Unfortunately, in most large cities in the United States, this infectivity rate may well have been exceeded already.

5 Is The United States Country Zero?

At the beginning of the AIDS epidemic, much energy was expended to find "Patient Zero," the individual who was responsible for bringing the disease to America. Epidemiologically, such witch hunts make little sense. But a search for a pool of infectives from whom the disease can leak into other societies makes a great deal of sense. As we trace the possible paths by which infectives can have transmitted the HIV virus to persons from other First World countries, the alarming possibility presents itself that it is the United States which is "Country Zero," that is, without the enormous pool of infectives in the United States, it may well be that most of the cases in other First World Countries would never have occurred. During the last decade, American politicians have sought to impose a myriad of restrictions on immigration in order to avoid the importation of the HIV virus. It may turn out that it is other countries who should worry about the importation of HIV from the United States.

A major purpose for the creation of epidemiological models is to present public health authorities with control mechanisms which can be used for minimizing the number of persons infected. There does not appear to have been much interest on the part of United States public health officials to use models for this purpose in the case of the AIDS epidemic. Possibly as a consequence of this lack of interest, there have been few models created toward the purpose of control. Already in the United States, more cases of AIDS have been recorded than the number of Americans killed in World War II. When questions are raised by the general public as to the utility of research funding to national needs, American biometry has much to answer for in the case of its performance in the United States AIDS epidemic.

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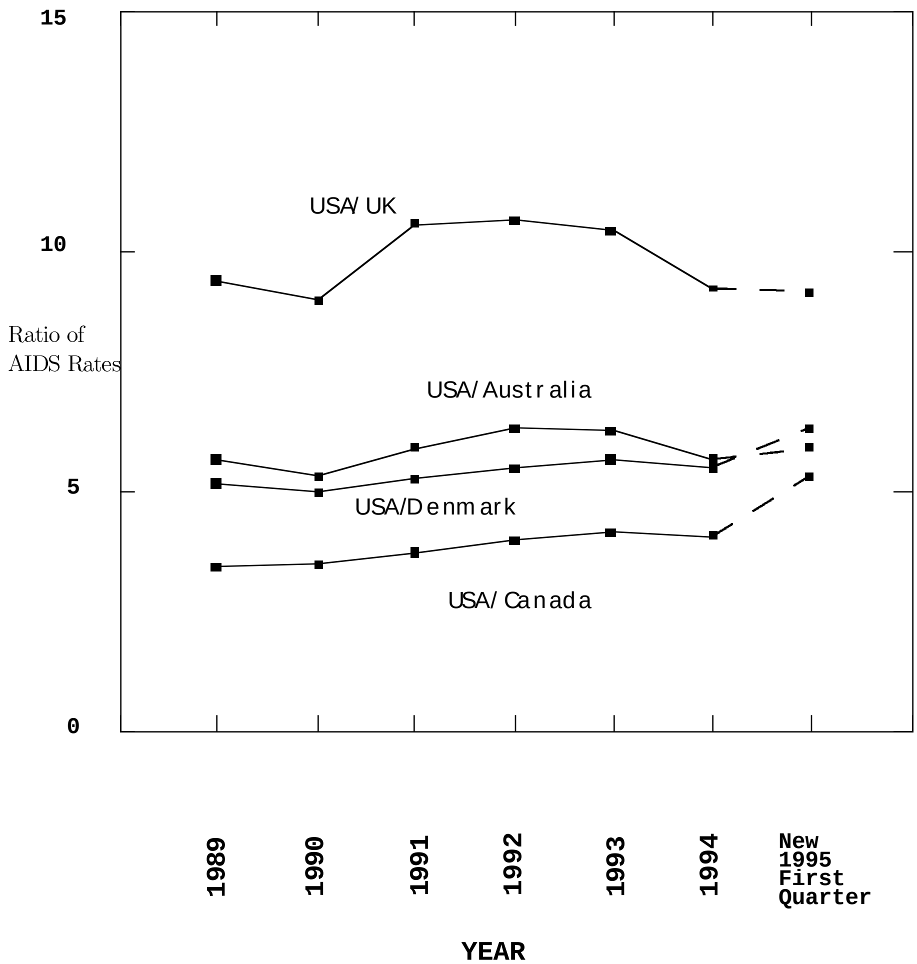

Figure 1. Comparative National Incidences of AIDS.

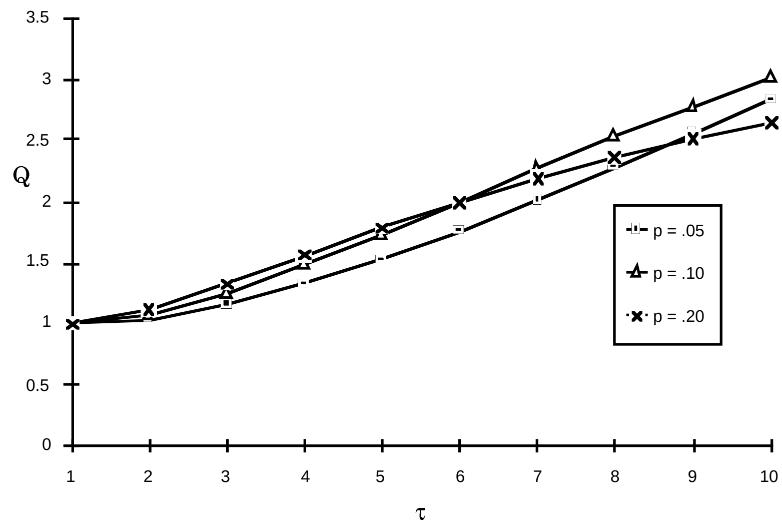

Figure 2. Effect Of High Activity Subpopulation

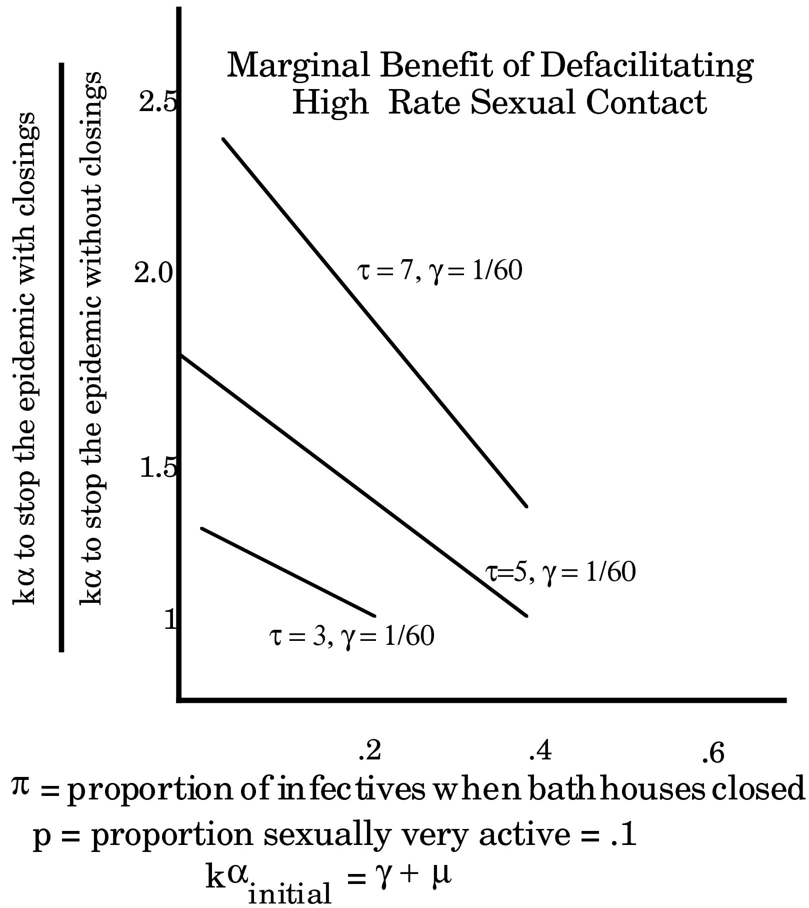

Figure 3. Bathhouse Closing Effects.