AIDS: THE MISMANAGEMENT OF AN EPIDEMIC

J. R. THOMPSON

Department of Statistics, School of Social Sciences, Rice University, Houston, TX 77251-1892, U.S.A.
and M.D. Anderson Hospital

Abstract—An argument is made that, so far from being a disease which is unstoppable in its epidemic consequences, AIDS has produced an epidemic, which owes its present virulence to sociological configurations of rather recent existence. Instead of a vigorous attack on the transmission chain of the epidemic, the emphasis of public health policy has been on finding a vaccine and/or a cure of the disease which produces the epidemic. By means of a simple model, it is argued that by simply closing businesses catering to high contact rate anal sex, e.g sexually oriented bathhouses, the American public health establishment might have avoided most of the tragic consequences of the present epidemic.

INTRODUCTION

In the early 1980s, after some decades without the onslaught on the developed nations of a fatal contagious disease with a high rate of occurrence, America became aware, of a mysterious "new" disease, which has been named Acquired Immune Deficiency Syndrome. Some have compared it with some of the epidemics which plagued Europe in an earlier age: cholera, the Bubonic Plague, smallpox. Death totals in the United States alone have been forecast in the millions. A massive program of research into the development of a vaccine and/or a cure has been instituted. By 1987, a number of polls revealed that AIDS was the item of single greatest concern to the American electorate—exceeding nuclear war, takeover by a foreign power or hard economic times. Into the secure Yuppie age has entered a new element, which most citizens had never dreamed could occur—catastrophe by epidemic. We wish to look at a simple model of the epidemic which may give us insight into the causes of the disease, its anticipated course, and an analysis of public health management of the epidemic.

On 13 April 1982, Congressman Henry Waxman [1, p. 144], 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." This statement and similar ones made by other political figures were interpreted by many public health authorities that whatever strategies were to be employed in the war on AIDS, those which interfered with any variety or quantity of homosexual activity were not to be used.

A customary approach to the control of contagious diseases in contemporary America is via medical intervention, either by preventive vaccination or by the use of antibiotics. Historically, sociological control of epidemics has been the more customary method. This has been due, in part, to the fact that vaccines were unknown before the nineteenth century and antibiotics before the twentieth century. But is is interesting to note that for some of the classical diseases which have caused epidemics in the past, e.g. cholera and the Bubonic Plague, the world is still without either effective vaccination or cure. Even when vaccines and/or cures for contagious disease are developed, such developments generally require years of research. Based on millenia of experience, it would appear that sociological control of an epidemic, rather than one based on an anticipated vaccine or cure, should be the first line of defense against a contagious disease, particularly a "new" one.

In the case of some ancient peoples, a large portion of the system of laws dealt with the means of sociological control of epidemics. For example, it should be noted that the 13th, 14th and half of the 15th chapter of Leviticus (131 verses) are dedicated to the sociological control of leprosy. We might contrast this with the fact that the often mentioned dietary (kosher) laws receive only one chapter, the 11th, with a total of 47 verses. In the case of AIDS, we see a disease for which medical control, once a patient has acquired the disease, is even more difficult than for leprosy. However, it does not follow that an incurable disease should produce more virulent epidemics than

a disease like leprosy, which is generally regarded as crippling rather than fatal. AIDS, unlike leprosy, is not transmitted by casual contact. Consequently, we should expect that the transmission chain is much more fragile for AIDS and that relatively painless epidemiological policy can be used to stop an AIDS epidemic.

On 1 December 1987, during an NBC presidential debate, Vice-President George Bush announced that the federal expenditures on AIDS research (i.e. seeking a vaccine or cure) were at levels higher than those either for cancer or heart disease. Unfortunately, a vaccine or a cure is extremely unlikely in the near future. Accordingly, we are confronted with a disease with a 100% fatality record and a per patient medical cost (using the present heroic intervention) in the $100,000/case range. We must ask the question of whether the main thrust of attack on the AIDS epidemic can be deemed optimal or even intelligent.

Let us consider an argument, developed by Thompson [2], when the disease was first beginning to produce deaths in the several hundreds. In the model, we have a number of simplifying assumptions. For example, we first disregard time lag effects between exposure to the disease and transformation from susceptible to infective. We assume uniform mixing. Also, we have excluded from consideration the addition of heterosexual and bisexual populations as well as that of i.v. drug users. The size of the target population is, of course, arguable (many will take a sociologically identifiably gay target community of 3,000,000 to be a serious underestimate), and a number of other sizes of the target population could have been presented. However, the inclusion of these complexities and modifications would not significantly change the conclusions of this paper.

First of all, we can determine the probability that a random infective will transmit the disease to a susceptible during a time interval $[t, t + \Delta t]$.

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

where

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

To obtain the expected total increase in the infective population during $[t, t + \Delta t]$, we multiply the above by Y, the number of infectives.

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

For large populations, we can assume, under fairly general conditions, that the expected total change in Y is a very nearly equal to a deterministic Y, i.e.

$$\Delta E(Y) \approx \Delta Y. \tag{3}$$

Letting Δt go to zero, this yields, immediately

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

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

We must also allow for immigration into the susceptible population, λ, and emigration, μ, from both the susceptible and infective populations and for marginal increase in the emigration from the infective population due to AIDS, γ, from sickness and death. Thus we have the improved differential equation model

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

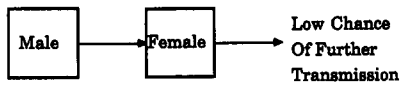$$\frac{dX}{dt} = -\frac{k\alpha XY}{X + Y} + \lambda - \mu X, \tag{5}$$
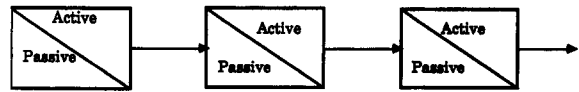
Fig. 1. Heterosexual transmission pattern.

Fig. 2. Homosexual transmission pattern.

where

γ = marginal AIDS death rate;
λ = immigration rate;
μ = emigration rate,

all time rates in months.

For early stages of the disease, $X/(X + Y) \approx 1$. Accordingly, we may write the approximation:

$$\frac{dY}{dt} \approx [k\alpha - \mu - \gamma]Y. \tag{6}$$

This gives us the solution:

$$Y = Y(0)\exp\{[k\alpha - \mu - \gamma]t\}. \tag{7}$$

Now, we shall use some rough estimates for some of the parameters in the equations above. We shall assume that, absent AIDS, the total target population is 3,000,000. We shall assume that an individual stays in this population an average of 15 y (yielding $\mu = 1/(15x\,12) = 0.00556$). We will use as the average time an infective remains infective 10 months (yielding $\gamma = 0.1$). To maintain the population of 3,000,000 (absent AIDS), then we require

$$\frac{dX}{dt} = \lambda - \mu X = 0, \tag{8}$$

or $\lambda = 16,666$. Now, if we combine these figures with early death data from AIDS, we can use the approximation for Y to obtain an estimate for $k\alpha \approx 0.263$. Considering that some values of k which have been variously reported range up to well over 100 per month, we are struck by the low probability of infection per contact (α) between an infective and a susceptible. This rate is exceptionally low, probably well below 0.01. This is very low, when compared to other venereal diseases, where values of this probability in excess of 0.2 are not unusual. *It is this very low probability of transmission of AIDS which distinguishes its epidemiological properties from customary venereal diseases and gives the key to the fragility of the current AIDS epidemic.* The low value of α indicates virus in some quantity is required for transmission of the disease in most case. This means that transmission is much more likely from the active to the passive partner. In Fig. 1, we note that this essentially "deadends" the disease in heterosexual transmission.

In the case of homosexual transmission (Fig. 2), where participants typically play both active and passive roles, the situation is quite different.

In Table 1 we show predicted and observed [3] AIDS figures using the estimated parameters.

Now, using the somewhat smaller $k\alpha$ value of 0.25 and an initial infective population of 2000, Table 2 shows the projections making the assumption that things continue with the parameter values shown in Table 1. "Cumulative deaths" refers to individuals who have passed into a stage of low infectivity.

Any visibility of a degenerative and fatal disease in the proportion range of a few percent of the target population will almost certainly cause members of that population to consider modifying

Table 1. AIDS cases

Date	Actual	Predicted
May 82	255	189
Aug. 82	475	339
Nov. 82	750	580
Feb. 83	1150	967
May 83	1675	1587

Table 2. Projections of AIDS with $k\alpha = 0.25$

Year	Cum. deaths	Fraction infective
1	6434	0.004
2	42,210	0.021
3	226,261	0.107
4	903,429	0.395
5	2,003,633	0.738
10	3,741,841	0.578
15	4,650,124	0.578
20	5,562,438	0.578

their membership in it. In the days of the plague in Western Europe, one could attempt to leave centers of congested population. It would appear likely that AIDS will cause a diminution of λ and k and an increase of μ. We note that the period for doubling the number of AIDS cases in the United States in 1982 was essentially 5 months, but has increased to a year at present [4].

Let us consider, for example, the effect of diminishing k. We note that in the early stages of the disease, an equilibrium value of $k\alpha = 0.1056$ is obtained. At this value, with all other parameters held constant, the total "cumulative deaths" after 20 y is 47,848 with a fraction of infectives quickly reaching 0.000668. Now, let us suppose that fear reduces k to 20% of 0.25, by the use of condoms and some restraint in activity. Then, Table 3 shows that the disease quickly retreats into epidemiological insignificance.

But let us suppose that a high contact fraction, p, retains a $k\alpha$ value τ times that of the less active population. Our model becomes

$$\frac{dY_1}{dt} = \frac{k\alpha X_1(Y_1 + \tau Y_2)}{X_1 + Y_1 + \tau(Y_2 + X_2)} - (\gamma + \mu)Y_1,$$

$$\frac{dY_2}{dt} = \frac{k\alpha\tau X_2(Y_1 + \tau Y_2)}{X_1 + Y_1 + \tau(Y_2 + X_2)} - (\gamma + \mu)Y_2,$$

$$\frac{dX_1}{dt} = -\frac{k\alpha X_1(Y_1 + \tau Y_2)}{X_1 + Y_1 + \tau(Y_2 + X_2)} + (1 - p)\lambda - \mu X_1,$$

$$\frac{dX_2}{dt} = -\frac{k\alpha\tau X_2(Y_1 + \tau Y_2)}{X_1 + Y_1 + \tau(Y_2 + X_2)} + p\lambda - \mu X_2. \tag{9}$$

Let us examine conditions under which the epidemic is not sustainable if, starting with no infectives, a small number of infectives is added. We note that if $Y_1 = Y_2 = 0$, the equilibrium values for X_1 and X_2 are $(1 - p)(\lambda/\mu)$ and $(p)(\lambda/\mu)$, respectively. Then, expanding the right-hand sides of the two equations in a Maclaurin series, we have (using lower case symbols for the perturbations from 0),

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

$$\frac{dy_2}{dt} = \frac{k\alpha\tau p}{1 - p + \tau p}y_1 + \left[\frac{k\alpha\tau^2 p}{1 - p + \tau p} - (\gamma + \mu)\right]y_2. \tag{10}$$

The solutions to a system of the form

$$\frac{dy_1}{dt} = ay_1 + by_2,$$

$$\frac{dy_2}{dt} = cy_1 + dy_2, \tag{11}$$

are given by

$$y_1(t) = c_1 \exp(r_1 t) + c_2 \exp(r_2 t)$$

$$y_2(t) = c_1 \frac{r_1 - d}{c} \exp(r_1 t) + c_2 \frac{r_2 - d}{c} \exp(r_2 t), \tag{12}$$

where

$$r_1 = \frac{a + d + \sqrt{(a + d)^2 - 4(ad - bc)}}{2},$$

$$r_2 = \frac{a + d - \sqrt{(a + d)^2 - 4(ad - bc)}}{2}. \tag{13}$$

In order that y_1 and y_2 go to zero, and hence the epidemic be not sustained, we require r_1 and r_2

Table 3. Projections of AIDS with $k\alpha = 0.05$

Year	Cum. deaths	Fraction infective
1	1751	0.00034
2	2650	0.00018
3	3112	0.00009
4	3349	0.00005
5	3471	0.00002
10	3594	0.000001

Table 4. Projections of AIDS with $k\alpha = 0.05$, $\tau = 5$, $p = 0.10$

Year	Cum. deaths	Fraction infective
1	2100	0.0005
2	4102	0.0006
3	6367	0.0007
4	9054	0.0008
5	12,274	0.0010
10	40,669	0.0020
15	105,076	0.0059
20	228,065	0.0091

to be negative. Substituting in equation (13) from equations (10), we note that this is achieved if

$$\frac{k\alpha}{\gamma + \mu} < \frac{\tau p + 1 - p}{\tau^2 p + 1 - p}. \tag{14}$$

For a population with $\tau = 1$ or $p = 0$, we note that the average contact-transmission rate is simply $k\alpha$ and the disease is not sustained if

$$k\alpha < \gamma + \mu. \tag{15}$$

For $\tau > 1$ and $p > 0$, however, the average contact-transmission rate is $k\alpha[(1 - p) + \tau p]$.

One measure of enhancement of marginal increase in disease sustainability due to a subpopulation exhibiting greater sexual activity than the average is given by the ratio Q of the overall contact-transmission average required to sustain the disease in the homogeneous case divided by that for a population with a relatively high activity subpopulation:

$$Q = \frac{1 - p + \tau^2 p}{(1 - p + \tau p)^2}. \tag{16}$$

In Fig. 3, we exhibit a graph of Q for various activity multipliers τ and proportions p of the more active subpopulation.

Let us now generalize the system in equations (9) to include time delays from exposure to infectivity

$$\frac{dW_1}{dt} = \frac{k\alpha X_1(Y_1 + \tau Y_2)}{X_1 + Y_1 + \tau(Y_2 + X_2)} - (\beta + \mu)W_1,$$

$$\frac{dW_2}{dt} = \frac{k\alpha\tau X_2(Y_1 + \tau Y_2)}{X_1 + Y_1 + \tau(Y_2 + X_2)} - (\beta + \mu)W_2,$$

$$\frac{dY_1}{dt} = \beta W_1 - (\mu + \gamma)Y_1,$$

$$\frac{dY_2}{dt} = \beta W_2 - (\mu + \gamma)Y_2,$$

$$\frac{dX_1}{dt} = -\frac{k\alpha X_1(Y_1 + \tau Y_2)}{X_1 + Y_1 + \tau(Y_2 + X_2)} + (1 - p)\lambda - \mu X_1,$$

$$\frac{dX_2}{dt} = -\frac{k\alpha\tau X_2(Y_1 + \tau Y_2)}{X_1 + Y_1 + \tau(Y_2 + X_2)} + p\lambda - \mu X_2. \tag{17}$$
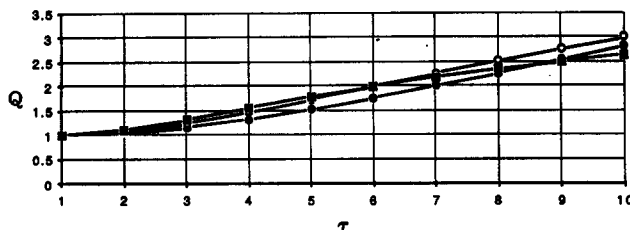

Fig. 3. Multipliers of sustainability (-●- $p = 0.05$; -○- $p = 0.10$; -■- $p = 0.20$).

Here, we use the symbol W to indicate an intermediate individual who has been infected by AIDS but is not yet able to transmit the disease to others. The Y populations are the individuals who are able to transmit the disease to others. Transformation from the W state to the Y state is at rate β. By going through a lengthy perturbation argument, using Laplace transforms, we find that in the case of time delay between transmission and infectivity shown in equations (17), the epidemic is not sustained if

$$\frac{k\alpha}{\gamma + \mu} < \frac{\tau p + 1 - p}{\tau^2 p + 1 - p} \frac{(\beta + \mu)(\mu + \gamma)}{\beta}. \tag{18}$$

The enhancement factor due to heterogeneity of contact rate is precisely that given in equation (16). Thus Fig. 3 applies even when we add the more realistic assumption of a time delay between transmission and infectivity.

We note how dramatically the presence of a small subpopulation with significantly higher sexual activity than that of the majority of the group enhances sustainability of the epidemic. Consequently, we note the very real possibility that, had the public health authorities in the various municipalities in the United States simply revoked, early on, the licenses of bathhouses and other establishments which facilitate very high contact rate activity of anal intercourse, the disease of AIDS might never have reached epidemic proportions in this country. Even more intriguing is the question as to whether absent the liberalized licensing regulations of such establishments promulgated in the late 1970s and early 1980s, AIDS would ever have developed in significant numbers. It is interesting to note that many leaders of the gay community requested bathhouse closings from the early days of the epidemic but were generally rebuffed by the municipal public health authorities until mid-1984 or later, when fear induced avoidance of such establishments had already contributed to their decline [1, pp. 328, 431, 441-443, 454, 464].

To examine from a slightly different angle the effect of a subpopulation with much greater activity than the rest of the group, in Table 4, we consider the case where $k\alpha = 0.05$, $\tau = 5$, and $p = 0.1$. Note that here 90% of the population has reduced its contact rate to that which, in Table 3, produced a rapid disappearance of the disease.

We notice how the presence of even a small subpopulation with a high contact rate can stop the demise of the epidemic. But, if this proportion becomes sufficiently small, without further increase in the activity multiplier, then the disease is removed from an epidemic to an endemic situation, as we see in Table 5 with $p = 0.05$ and all other parameters the same as Table 4.

The dramatic effect of a small high contact rate subpopulation may be considered in the case where 90% of the population has a $k\alpha$ of 0.02 and 10% has a $k\alpha$ of 0.32. This gives a population with an overall $k\alpha$ of 0.05. If this low value is maintained uniformly across the population, then we have seen that the disease quickly dies out. But consider the situation in Table 6 where the mix is given as above.

Thus, it is quite clear that, with the same overall group average contact rate, a population exhibiting homogeneous activity is much more resistant to sustaining an epidemic than one with a small high contact rate subpopulation.

It is now clear that the presence of AIDS in the human population is of long standing. We have AIDS-producing virus in United States human blood specimens going back at least to 1969, in specimens from human blood in Zaire at least as far back as 1959 [5]. Furthermore, we now have virological evidence [6] that AIDS is of ancient origin. At least in the case of AIDS, the virologist's maxim that all diseases are old diseases seems validated.

Table 5. Projections of AIDS with $k\alpha = 0.05$, $\tau = 5$, $p = 0.05$

Year	Cum. deaths	Fraction infective
1	1917	0.00043
2	3272	0.00033
3	4344	0.00027
4	5228	0.00022
5	5971	0.00019
10	8263	0.00008
15	9247	0.00003
20	9672	0.00002

Table 6. Projections of AIDS with $k\alpha = 0.02$, $\tau = 16$, $p = 0.1$

Year	Cum. deaths	Fraction infective
1	2184	0.0007
2	6536	0.0020
3	20,583	0.0067
4	64,157	0.0197
5	170,030	0.0421
10	855,839	0.0229
15	1,056,571	0.0122
20	1,269,362	0.0182

Since AIDS is not a new disease, then we ought to ask what has changed in order than an endemic disease has now reached epidemic proportions. After all, we should recall that Belgian colonial troops went back and forth from Zaire to Belgium for nearly a hundred years. If there was ever a significant outbreak of AIDS in Belgium during the colonial epoch, no one has discovered the fact. It seems most likely that the reason is that the high contact rates which characterize some segments of the homosexual communities which exist in some American cities have never occurred before in the history of the world.

Naturally there is concern as to whether AIDS can be sustained heterosexually in the United States. The main argument in support of such a possibility is the fact that the concurrent Central African AIDS epidemic is apparently almost completely a heterosexual phenomenon. How much of the African transmission is due to nonsexual modes, e.g. local health officials dispensing medical care with unsterilized needles and surgical instruments and how much is due to untreated lesions from other venereal diseases dramatically enhancing the possibility of female to male transmission (an essential mode if the epidemic is to be sustained heterosexually), is a matter of conjecture. But we know that economic hard times have seriously stressed the quality of medicine in many African countries, and the two modes of transmission mentioned above may explain the heterosexual AIDS edpidemic there. If so, it is very likely that the AIDS epidemic in Africa is the indirect consequence of conditions of economic decline which are not likely to be matched anywhere in the United States. Of the 45,425 adult American cases of AIDS listed by the CDC on 16 November 1987, slightly less than 4% were effected by heterosexual transmission [7]. Of the 42,208 male cases, less than 2% were heterosexually transmitted. Interestingly, of the 3217 female cases, 29% were the result of heterosexual transmission (the rest being by drug use and blood transfusion). The argument that female-to-male transmission is unlikely except in special circumstances like those mentioned above still squares with the data for five years into the epidemic. The indications are that conditions do not exist in the United States which can produce a stand-alone heterosexual epidemic. However, any failure of the American public health community to provide effective health care delivery to stop epidemics of other venereal diseases than AIDS in communities of the disadvantaged could facilitate a heterosexual AIDS epidemic in such communities.

One possibility with AIDS is that there is a "Typhoid Mary" phenomenon. This means that a significant fraction of the infectives remain infective for long periods of time. To see the effects of such a phenomenon, let us suppose $k\alpha = 0.05$, but 50% of those who contract the disease have a period of infectivity of 100 months instead of only 10 (see Table 7).

Such a disastrous scenario is, naturally, made much worse as we increase the fraction of those with the long sexually active life expectancy. For example, if this proportion is 90%, we have Table 8.

If the Typhoid Mary phenomenon were an actuality, then the effect of AIDS could be catastrophic indeed. (Note that no presence of an exceptionally active subpopulation is necessary to cause this catastrophic scenario.) However, this would imply that AIDS was a new disease, since such a phenomenon would have sustained the epidemic in an earlier time. As pointed out above, we have evidence that AIDS is an old disease. Furthermore, a significant Typhoid Mary phenomenon would appear to work against the aforementioned continual lengthening of the doubling time for observed cases of the disease.

Table 7. Projections of AIDS with $k\alpha = 0.05$ and half of the infectives with $\gamma = 0.01$

Year	Cum. deaths	Fraction infective
1	1064	0.00066
2	1419	0.00075
3	2801	0.00089
4	3815	0.00110
5	5023	0.00130
10	16,032	0.00330
15	44,340	0.00860
20	115,979	0.02210

Table 8. Projections of AIDS with $k\alpha = 0.05$ and 90% of the infectives having $\gamma = 0.01$

Year	Cum. deaths	Fraction infective
1	457	0.0094
2	1020	0.0013
3	1808	0.0020
4	2943	0.0028
5	4587	0.0041
10	32,911	0.0260
15	194,154	0.1441
20	776,146	0.4754

CONCLUSIONS

Among the developed nations, the United States has much the highest incidence of AIDS. On the basis of cases per 100,000 population, the American rate is four times that of Canada, six times that of Denmark (which recently legalized homosexual marriages) and 12 times that of the United Kingdom (where gays have, on the average, eight different partners per year). Is the United States simply demonstrating AIDS incidence rates which will be matched by the other developed nations at a later time? I doubt it.

American public health authorities have been, in my view, far more sympathetic than those in other countries to the legalization of establishments, such as gay bathhouses, which facilitate high incidence anonymous anal intercourse. That a small subpopulation with a contact rate much higher than that of the rest of the target group (even if the overall contact rate of the group is held constant) should have such a dramatic effect on facilitating the crossing of the epidemic threshold is, perhaps, surprising. It surprised me when I developed the model in 1983. I still stand by its conclusions in 1989.

Some will object to the model as an oversimplification. Some workers in AIDS epidemiology feel that a truly accurate model will not be available for some years. But while we wait for the truly accurate model, the AIDS epidemic in the United States is running its course. In point of fact, many generalizations of the model are relatively simple. For example, if one wishes to use number of partners rather than number of contacts as the driving force behind the epidemic, the model can still be used. Other modifications, such as two separated epochs of high infectivity for each infective would require more work. I doubt if the conclusions concerning the bathhouses would be changed. When we utilize a model, as has been done here, not to make absolute extrapolations, but rather to make comparisons under two different scenarios, we have taken an important step in "robustification".

That AIDS has presented American public health with a new set of problems is undoubted. But a public health system which cannot deal effectively with new situations leaves much to be desired.

In the long run, members of a target population will tend to take the necessary steps to lessen their participation in an epidemic. But it is the function of a public health establishment to shortcircuit the painful path dependent on experience of members of the target population. Had public health authorities vigorously attacked the transmission chain early on by shutting down high contact inducing establishments, the present AIDS holocaust in the American gay community might well have been avoided.

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