THE AIDS EPIDEMIC’S INFLUENCE ON NEW YORK CITY’S GAY SEXUAL CONTACT RATE FROM AN ANALYSIS OF GONORRHEA INCIDENCE

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(Received April 1990)

Communicated by E. Y. Rodin

INTRODUCTION

As public awareness of the mortal danger of the AIDS epidemic increased, the frequency of acts of unprotected anal intercourse by members of the bisexual and homosexual (gay) risk group began to decrease. The number of new unprotected sexual contacts per person per unit time at any time \( t \) is known as the contact rate and will be denoted by \( C(t) \). The decrease in the contact rate began to manifest itself in an anal/rectal gonorrhea incidence rate that has dropped by a factor of 20. Since a decreasing contact rate also means the slowing of the growth of the AIDS epidemic, knowledge of the time variation of the contact rate \( C(t) \) is essential if the calculation of the future AIDS incidence in the gay risk population is to be feasible. Since the biological parameters characterizing the AIDS infection, such as the infectivity, latency period, and incubation period probability density curve, are still poorly understood, it is useful to extract as much information as possible from a more familiar and rapidly responding STD such as gonorrhea.

This paper extends the analysis of a previous work on this subject [1] to cover the gonorrhea epidemic in New York City. In reference [1] we adapted the heterosexual gonorrhea model of Hethcote and Yorke [2] to the gay risk population, and illustrated our results by using them to analyze the gonorrhea epidemic in San Francisco. For convenience, the appendix gives a brief summary of our model results in reference [1].

The gonorrhea incidence statistics used in this paper were generously supplied by Drs. Polly Thomas and Rita Hindin of the New York City Department of Health [3]. The data provided consists of the annual number of gonorrhea cases seen in New York City health clinics in each year broken down by gonorrhea type. This data makes it possible to compare the relative impact knowledge of the AIDS epidemic has had on the contact rates of the gay and heterosexual risk populations in New York City. We find that a drop of 35% or less in the gay contact rate is sufficient to account for the observed decrease in the anal gonorrhea incidence data for New York City. By contrast, the data demonstrates that the heterosexual risk population has yet to respond to the growing danger of AIDS.

RESULTS

The annual anal gonorrhea incidence \( R(t) \) (number of cases seen annually) is a function of time \( t \). The incidence rate data for New York City is shown in Fig. 1, where the annual number in a given year is plotted in the middle of the year. The rates have dropped from nearly 2000 cases annually in 1981 to about 100 in 1988, the most recent year for which data is complete. Fitting
the parameterized function given in equation (10a) to this data using a least squares fit yields the model parameters given in equation (11) in the appendix. As noted in the appendix, the function chosen to parameterize the drop in the incidence rate $R(t)$ is an exponential decay identical to that which describes radioactive nuclear decay. This makes it possible to characterize the incidence rate $R(t)$ by its half-life $T_{0.5}$ in the region $t > t_A$, where the AIDS epidemic had a measurable effect on the gonorrhea incidence rate. The connection between the half-life parameter and the parameter $k$ is given by equation (10b). From the best fit parameters, the drop in incidence rate began on March 7, 1983 (the precision of the data is such that this date is only approximately known).

$$R(t) \ (y^{-1})$$

![Graph](image)

**Fig. 1. New York City annual male anal gonorrhea incidence rate $R(t)$**

The annual incidence rate obtained from the model fit is also plotted in Fig. 1. Although this 3-parameter fit is quite good, the actual anal gonorrhea incidence rate for New York City did not monotonically decrease in the region $t > t_A$. All boroughs experienced an increase in the incidence rate $R(t)$ in the year 1985 over what it was in 1984. This relative increase can be readily seen in Fig. 1, and the effect of the spur on the fit is to increase the value of the half-life parameter $T_{0.5}$.

In this paper we will assume that the sole cause of the drop in the anal gonorrhea incidence rate $R(t)$ for times $t > t_A$ is a drop in the contact rate $C(t)$. Thus, equations (8) and (9) in the appendix can be iterated to calculate the time dependent drop in New York City's effective contact rate $C_e(t)$ defined as the product of the contact rate $C(t)$ and the infectivity per relationship $f$. During the steady state period, when $t < t_A$ and the AIDS epidemic had yet to have an impact on the gonorrhea incidence rate, the steady state value for the number of gays infected with anal gonorrhea is denoted by $I^*$. If the size of the gay core population is denoted by $N$, then the steady state prevalence $p^*$ is defined by $p^* = I^*/N$. The steady state effective contact rate $C_0^* \equiv fC^*$ is a function of the prevalence $p^*$ and can be calculated using equation (4). The steady state prevalence for San Francisco was known to be $p^* = 0.08$. The prevalence for New York City is not known. However, we have taken the San Francisco value as typical of an urban gay population and used it to compute the effective contact rate $C_0^*$ for New York City. We also repeated our calculations using a higher prevalence value of $p^* = 0.2$ to investigate the effects of under reporting of cases. The results of both of these calculations are shown in Fig. 2.

As can be seen from Fig. 2, if the steady state prevalence $p^*$ was 0.08 (0.2), a drop of only 30% (47%) in the steady state contact rate $C^*$ is sufficient to account for the recorded drop in New York City's anal gonorrhea incidence rate by the end of 1988. For a steady state prevalence
value of $p^* = 0.08$, the value of the steady state effective contact rate is $C^*_e = 25.5y^{-1}$, which, according to the result in equation (5), is sufficient to maintain a gonorrhea epidemic. By the end of 1988, however, the effective contact rate drops to the value of $17.9y^{-1}$, which is below the critical effective contact rate of $18.25y^{-1}$ necessary to sustain the gonorrhea epidemic, according to the result in (5). Although this drop is significant insofar as ending the gonorrhea epidemic, it is wholly inadequate insofar as the AIDS epidemic is concerned, since the HIV infection is life-long. For an initial prevalence of 0.2, the effective contact rate falls from $37.5y^{-1}$ to $20y^{-1}$. For both assumed prevalences, the drops are similar to those predicted for San Francisco.

New York City’s heterosexual gonorrhea data are strikingly different from the anal gonorrhea data described above. The male and female annual gonorrhea rates, excluding anal and oral, are plotted in Fig. 3. Since the overwhelming majority of these cases are not gays, these rates are a good measure of the heterosexual gonorrhea incidences. Clearly, these incidences do not show a trend towards decreasing values as the danger of the AIDS epidemic became known to the heterosexual risk population. As a matter of fact these gonorrhea incidence rates rose dramatically in 1986 from what it was in previous years. This indicates that the heterosexual risk population in New York City has not responded to the growing danger of AIDS.

REFERENCES

3. Polly Thomas, M.D., Director, and Rita Hindin, Ph.D., Epidemiologist, AIDS & HIV Surveillance, Department of Health, 125 Worth St., N.Y., N.Y. 10013.

APPENDIX

Modeling the Gonorrhea Infection in the Gay Risk Population

Males with gonorrhea can either be symptomatic or asymptomatic. About 90% of male gonorrhea cases are asymptomatic, having the relatively short average duration of infection of $T_S = 8$ days. The remaining 10% of the cases are asymptomatic and have the relatively long average duration of infection of $T_L = 128$ days. The subscripts refer to “short” and “long” duration, respectively. The average duration of infection for the male risk (core) population $N$ as a whole is then $T = 20$ days.
At any time $t$ the gay core population of size $N(t)$ consists of a symptomatic part $N_S(t)$ and an asymptomatic part $N_L(t)$. The 3 populations $N(t)$, $N_S(t)$, and $N_L(t)$ can be divided into infected and uninfected parts. Thus, $N(t) = I(t) + U(t)$, $N_S(t) = I_S(t) + U_S(t)$, and $N_L(t) = I_L(t) + U_L(t)$, where $I(t) = I_S(t) + I_L(t)$ and $U(t) = U_S(t) + U_L(t)$.

We assume that the symptomatic and asymptomatic core populations have similar behavioral characteristics on the average. Specifically, we will assume that both populations have the same unprotected contact rate $C(t)$ and mean transmission probability per unprotected relationship $f$.

The equations for the symptomatic incidence $R_S(t)$ and asymptomatic incidence $R_L(t)$ are given by

$$R_j(t) = C(t)fI(t)[N_j(t) - I_j(t)]N_j^{-1}, \quad j = S, L. \quad (1)$$

The symptomatic and asymptomatic infected populations obey the following equations:

$$\frac{dI_j(t)}{dt} = R_j(t) - \Delta_j, \quad j = S, L. \quad (2)$$

**The Steady State and the Period Before 1982**

Under steady state conditions the population sizes, incidence rates, and contact rate all remain constant. From the gonorrhea incidence rate for New York City that appears in Figure 1, the New York City anal gonorrhea epidemic was in an approximate steady-state in the period before 1982 when knowledge of the mortal danger of AIDS was not widespread. The gonorrhea incidence rate remained essentially constant until some critical time $t_A$, when gay awareness of the AIDS epidemic began to produce a measurable decrease in these incidence rates. The separate data for each borough, which has not been plotted here, shows a similar time course. We will denote all steady state quantities with a starred superscript $\ast$. Thus, for example, the (constant) incidence rates in the steady state period will be denoted by $R_j = R_j(t_A)$, with $j = S$ or $L$. Since the left-hand side of equation (2) vanishes during the steady state period, we have

$$R_j = I_j^\ast/T_j, \quad j = S, L. \quad (3)$$

The prevalence during the steady state $p^\ast$ is defined as $p^\ast = I^\ast/N$. Using equations (1) and (3), it is straightforward to prove the following useful identity which relates the prevalence $p^\ast$ to the steady state infectious contact rate $C^\ast$:

$$1 = \frac{C^\ast fN_SN_S^{-1}}{C^\ast f + T_S^{-1}} + \frac{C^\ast fN_LN_L^{-1}}{C^\ast f + T_L^{-1}}. \quad (4)$$

Since $p^\ast \geq 0$, there is a critical value for the contact rate $C^\ast_{cr}$ below which a gonorrhea epidemic cannot be sustained in a steady state. Setting $p^\ast = 0$ in equation (4), we find that the critical contact rate satisfies

$$C^\ast_{cr} = \frac{1}{fT} = \frac{18.25}{f} \text{ per year} > 18.25 \text{ per year}.$$
Thus, with an annual contact rate below $18.25\, y^{-1}$, a gonorrhea epidemic will die out.

**Modeling the Drop in the Gonorrhea Incidence Rate**

In the following analysis we will assume that the size of the gay risk population $N$ remains constant. We further assume that the same fraction of the core population is symptomatic (asymptomatic) for the period $t < t_A$ as for the period $t > t_A$. In the time period $t > t_A$ most of our model quantities become time dependent as depicted in equations (1) and (2). Now the gonorrhea incidence rate $R(t)$ is a slowly varying function with respect to time changes on the order of $\Delta t = 1$ day. In the time period $t > t_A$ we break up the time scale $t$ into daily increments by setting

$$t \equiv (t_A + k)\text{days}, \quad k = 0, 1, 2, \ldots.$$  

(6)

Adding the equations in (2) together then gives

$$R(t_A + k) = fC(t_A + k)I(t_A + k)\left[1 - \frac{I(t_A + k)}{N}\right].$$  

(7)

We approximate this equation by writing it as

$$R(t_A + k) = fC(t_A + k)I(t_A + k - 1)\left[1 - \frac{I(t_A + k - 1)}{N}\right].$$  

(8)

We now assume that the same fraction of the core population is symptomatic (asymptomatic) for $t > t_A$ as for $t < t_A$. Approximating equations (1) and (2) as difference equations, we then get

$$I_S(t_A + k) = I_S(t_A + k - 1) - \frac{I_S(t_A + k - 1)}{T_S} + fC(t_A + k)I(t_A + k - 1)\left[0.9 - \frac{I_S(t_A + k - 1)}{N}\right].$$  

(9)

A similar equation is obtained for $I_L$. Using equation (8), these equations for $I_S$ and $I_L$ can be iterated to yield $C_E(t) \equiv fC(t), I_S(t),$ and $I_L(t)$ starting from known initial values at $t_A$ and the reported incidence curve.

**Determining the Gonorrhea Incidence Function $R(t)$**

As a suitable parameterization of the gonorrhea incidence data for New York City we have chosen the following function:

$$R(t) = \begin{cases} R^*, & t \leq t_A, \\ R^* \exp\{-k(t - t_A)\}, & t > t_A. \end{cases}$$  

(10a)

It is convenient to relate the parameter $k$ to the elapsed time $T_{0.5}$ required for the incidence rate to fall by 50%, so

$$k T_{0.5} = \ln 2.$$  

(10b)

The parameterized incidence function $R(t)$ requires the values of 3 parameters to determine it: $R^*$, $k$ (or $T_{0.5}$), and $t_A$. These parameters were determined by fitting this function to the annual gonorrhea incidence statistics for New York City using a least-square fit program and a VAX computer. The computed values for the parameters are as follows:

$$R^* = 1740.5 y^{-1},$$

$$t_A = 83.2 \equiv \text{March 7, 1983},$$

$$T_{0.5} = 2.04 y,$$

$$k = 0.3404 y^{-1}.$$  

(11)