AN INFECTIOUS DISEASE MODEL FOR ESTIMATING THE INCIDENCE OF HIV

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KEY WORDS: Transmission model, HIV prevalence, AIDS

ABSTRACT

We present an infectious disease model with the following characteristics:

- Eight disjoint risk groups that are based on transmission via sexual and intravenous drug use in homosexual men, bisexual men, heterosexual men and women.
- Contacts that follow from a homogeneous mixing assumption which takes into account risk group sizes and behavior.
- A disease incubation function based on the results of Bachetti and Moss (1989).
- No infections associated with hemophiliacs, blood product users, children under 13 years of age, and foreign borns.

Simulation experiments were conducted to estimate critical model parameters such as the number of sexual and/or needle sharing contacts needed to explain observed AIDS cases and deaths due to HIV-related illnesses. These estimated parameters were used to make short-term national projections of the incidence of HIV and AIDS.

1. INTRODUCTION

This paper presents a simple model of the AIDS epidemic developed to explore some fundamental relationships among factors that characterize that epidemic. These factors include: (1) needle sharing and sexual contacts between individuals within and among risk groups; (2) the rate of progression from infection with HIV to diagnosis of AIDS; (3) the nature of viral transmission, including infectivity of the virus; and (4) survival rates of persons with AIDS. These factors are imperfectly understood by the scientific community. Until their influence on the epidemic can be evaluated, no reliable projections of the spread of HIV can be developed.

In this study we use the proposed model to estimate risk-group-specific contact (sexual and needle sharing) rates. These estimated rates produce predicted AIDS incidence consistent with observed AIDS incidence as reported to the Centers for Disease Control (CDC) with adjustment for reporting biases. Section 2 of this paper describes the model, Section 3 presents the results of the simulation experiments, and Section 4 interprets the findings and discusses some implications of the results.

2. METHODS

A.Model Logic

Figure 1 is a schematic diagram of the process under investigation. This figure identifies major subpopulation categories tracked by the model for a given risk group. The subpopulations are:

- **the susceptible population**--the population whose behavior places them at risk of HIV infection but who are not infected. $S_i(t)$ is the size of this population at time t for risk group i.
- **the infected population**--those infected with HIV, but not yet displaying clinical manifestations of the AIDS disease. $I_i(t)$ is the size of the infected population at time t for risk group i;
- **the AIDS population**--that segment of the population that has been diagnosed with CDC-defined AIDS. $A_i(t)$ is the size of this population at time t for risk group i;
- **the AIDS death population**--that segment of the population that has died as a consequence of CDC-defined AIDS. $DA_i(t)$ is the size of this population at time t for risk group i.
- **the non-AIDS HIV-related death (NAHD) population**--that segment of the infected population that has died of HIV-related complications not meeting the CDC AIDS definition. $DI_i(t)$ is the size of this population at time t for risk group i.

At the start of the simulated epidemic, the susceptible population is distributed across eight disjoint risk groups (see Table 1). The distribution of susceptibles by risk groups at the beginning of the epidemic is assumed to be the same as the distribution of AIDS cases by risk group as reported to the CDC through March 1989.

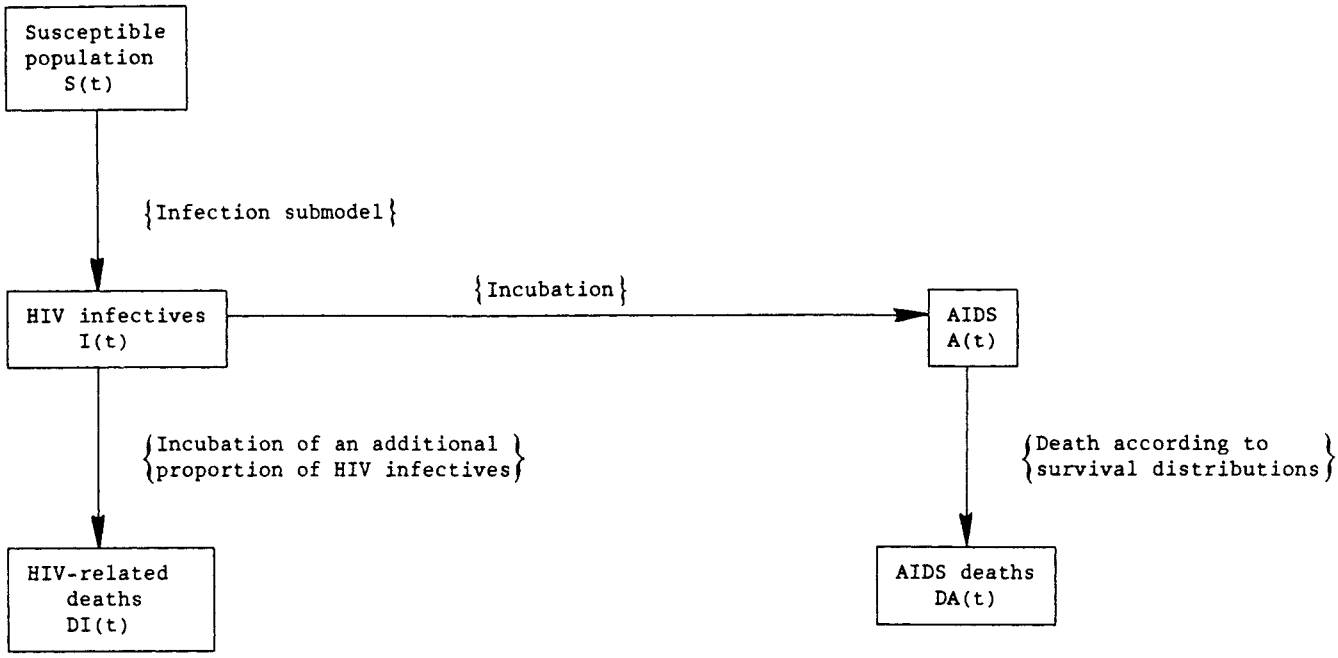
Table 1. Risk Group Definitions

1. Homosexual males who do not use IV drugs (HM)
2. Homosexual males who use IV drugs (HMI)
3. Bisexual males who do not use IV drugs (BM)
4. Bisexual males who use IV drugs (BMI)
5. Other males who do not use IV drugs (OM)
6. Other males who use IV drugs (OMI)
7. Females who do not use IV drugs (FM)
8. Females who use IV drugs (FMI)

We assume one infected person in each risk group at the beginning of the epidemic, T_o. The model estimates risk-group-specific infection rates that reproduce (within a specified level of accuracy) the AIDS incidence data adjusted for (1) reporting delays; (2) change in case definition; and (3) underreporting (see Section C.4). Infection rates can be expressed as a function of the proportion of safe contacts (i.e., those involving needle cleaning or protected intercourse), the average number of contacts per susceptible and the probability of infectivity given contact. Infection rates are estimated for each time interval. Changes in IV drug use and/or sexual behavior over time are reflected in changes in the infection rates.

Using these new estimates, the model then estimates HIV incidence in each interval according to the infection submodel defined

Figure 1. Population Progression for a Given Risk Group as Characterized by Model



below. New infectives are incubated to AIDS diagnosis and the number of new AIDS cases during each interval is recorded.

Persons with AIDS are assigned to groups by type of initial diagnosis (KS, PCP, or other). A diagnosis-specific survival function is applied to estimate the number of persons with AIDS dying during specific time intervals.

In summary, the model produces as infection rate estimates those values which lead to estimated AIDS incidence approximately equal to observed, adjusted AIDS incidence. HIV incidence is also estimated using these infection rates. In addition, deaths among AIDS cases and among HIV infectives are estimated within given time intervals. These death estimates are based on mortality experience assumptions for the two groups.

B.Mathematical Description

Equations (1) to (5) (corresponding to the five states identified in Figure 1) describe the model dynamics of the ith risk group. Although equations are presented as differential equations, they are implemented as difference equations.

1. Susceptibles

$$\frac{\partial S_i(t)}{\partial t} = \delta_i S_i(T_o) - \left(\mu_i + \lambda_i(t)\right) S_i(t) \qquad (1)$$

The susceptible population in the ith risk group increases according to the recruitment rate, δ_i [always applied to the initial population $S_i(T_o)$], and decreases due to the attrition rate, μ_i, and the rate of HIV infection, $\lambda_i(t)$.

2. Infectives

$$\frac{\partial S_i(t)}{\partial t} = \lambda_i(t) S_i(t) - \left(\gamma_i(t)+\theta_i(t)+\mu_i\right) I_i(t) \qquad (2)$$

The infected population in risk group i increases according to the rate of HIV infection, $\lambda_i(t)$, and decreases according to the rates of progression to AIDS, $\gamma_i(t)$, non-AIDS HIV deaths, $\theta_i(t)$, and non-HIV related deaths, μ_i.

The rate of progression to AIDS, $\gamma_i(t)$, is defined as:

$$\gamma_i(t) = \rho_i \int_{T_o}^{t} \lambda_i(s) S_i(s) f_i(t|s)\, ds \Big/ I_i(t)$$

where

$\lambda_i(s)S_i(s)$ = the number of new infections in risk group i at time s

ρ_i = the proportion of HIV infectives in the ith risk group that progress to AIDS rather than non-AIDS HIV related death

and

$f_i(t|s)$ = the density function for progression to AIDS at time t given infection at time s for risk group i.

The rate of progression to NAHD is:

$$\theta_i(t) = \left(1 - \rho_i\right) \int_{T_o}^{t} \lambda_i(s) S_i(s) g_i(t|s)\, ds \Big/ I_i(t)$$

where

$g_i(t|s)$ = the probability distribution for progression to NAHD at time t given infection at time s for risk group i.

3.AIDS Cases

$$\frac{\partial A_i(t)}{\partial t} = \gamma_i(t)I_i(t) - \Lambda_i(t)A_i \qquad (3)$$

The AIDS population in risk group i increases according to the rate of progression to AIDS, $\gamma_i(t)$, and decreases according to the death rate, $\Lambda_i(t)$. The death rate $\Lambda_i(t)$ depends on the distribution of initial diagnoses. Let M_{ik} be the probability of being diagnosed with the kth AIDS type and $d_k(t|s)$ be the probability of death at time t given diagnosis at time s for the kth AIDS type. With this notation, the death rate is defined as:

$$\Lambda_i(t) = \sum_k M_{ik} \int_{T_o}^{t} \gamma_i(s)I_i(s)d_k(t|s)ds \Big/ A_i(t)$$

where $\gamma_i(s)I_i(s)$ is the number of new persons with AIDS in risk group i at time s.

4. AIDS Deaths

$$\frac{\partial DA_i(t)}{\partial t} = \Lambda_i(t)A_i(t) \qquad (4)$$

5. Non-AIDS HIV Death

$$\frac{\partial DI_i(t)}{\partial t} = \theta_i(t)I_i(t) \qquad (5)$$

AIDS deaths and non-AIDS HIV deaths depend on their respective death rates, $\Lambda_i(t)$ and $\theta_i(t)$.

6. New Infections (Homogeneous Mixing)

If $C_i(t)$ is the average number of contacts per partner (sexual or needle sharing) for a susceptible in the ith risk group, $P_i(t)$ is the rate of partner acquisition for the ith group, and

$$\alpha_i(t) = C_i(t)P_i(t)$$

then $\alpha_i(t)$ represents the rate of contacts for an individual in the ith group at time t.

The proportion of total contacts that occur with members of the jth risk group is denoted $\beta_j(t)$. Under the homogeneous mixing assumption, $\beta_j(t)$ depends on the ratio of the size of the jth group to the total, i.e.,

$$\beta_j(t) = \frac{N_j(t)}{\sum_k N_k(t)}$$

where

$$N_j(t) = S_j(t) + I_j(t) + A_j(t).$$

The product $\alpha_i(t)\beta_j(t)$ represents the rate of contacts with members of the jth risk group for an individual in the ith group.

Under the assumption of homogeneous mixing, the probability of a given contact occurring with an infected member of the jth group is:

$$q_j(t) = \frac{I_j(t) + A_j(t)}{N_j(t)} .$$

Define H_{ij} as the probability that a given contact between a susceptible in the ith group and an infective in the jth group produces an HIV infection. Then, the rate of new infections per susceptible at time t is:

$$\lambda_i(t) = \alpha_i(t) \sum_j \beta_j(t)q_j(t)H_{ij} .$$

C.A priori Parameter Values

A number of critical parameter values are needed to project the future epidemic. None of these is accurately known. They include:

- the start date of the epidemic
- the size of the risk-taking population by risk category
- the HIV infection rates by risk category
- the incubation period distribution by risk category
- mortality rate of AIDS patients.

We discuss below the values for these parameters that we have selected based on current research.

1. Start of the Epidemic

Significant numbers of individuals were infected as early as 1977 (Des Jarlais et al. 1989). To be conservative we have assumed the start date, T_o, to be three years earlier, i.e., January 1, 1974. Although there is evidence of earlier isolated unreported AIDS cases (Selik et al. 1984; Evatt et al. 1985), all results reported here are based on the 1974 starting date.

2. Population at Risk

The size of the population at risk was obtained from Wells et al. (1988). They report a national estimate of 7 million people who practice those behaviors that place them at risk of HIV infection.

The distribution of the susceptible population across risk groups adjusted for non-AIDS HIV deaths was based on the distribution of AIDS cases among risk groups in the AIDS surveillance data.

3. Incubation Function

The incubation distribution used in this study was based on the non-parametric function reported by Bachetti and Moss (1989). This function was estimated using data on three cohorts of homosexual men in San Francisco and reported AIDS incidence. It estimates rates of progression to AIDS for the first 10 years after infection. Beyond 10 years, we have assumed a constant hazard (equal to the hazard during the tenth year). This assumption yielded an incubation function from which approximately 98 percent of the cases are expected to progress to AIDS within 30 years of infection.

4. AIDS Cases

AIDS incidence was estimated by risk group and year of diagnosis adjusted for reporting delays, definition changes, and underreporting. A description of the procedures used to adjust for

reporting delays and definition change biases is presented in Hamill et al. (1989). In addition, the recommendations of the USGAO (1989) were used to adjust those estimates for under-diagnosis and underreporting for reasons other than those considered in Hamill et al.

5. Attrition Rates

Attrition is defined as loss of susceptible and infected individuals due to mortality unrelated to HIV infection. In our model, a mortality rate based on an average survival of 40 years from entry into the epidemic was used for all risk groups, i.e.,

$$\mu_1 = \mu_2 = \ldots = \mu_8 = .025.$$

6. Susceptible Recruitment Rates

Recruitment rates are region, age, and risk-group-specific. It is assumed that the recruitment rate equals initial attrition rate, i.e., the sizes of the susceptible populations would have remained constant in the absence of the AIDS epidemic. Constant recruitment is maintained at this level when

$$\delta_1 = \delta_2 = \ldots = \delta_8 = .025.$$

This per capita recruitment rate is always applied to the initial population size, $S_1(T_o)$.

7. AIDS Deaths

The death rates, $\{\lambda_1(t)\}$, are based on constant hazards specific to the types of initial AIDS diagnoses and the proportion of persons with each diagnosis type. The influence of antivirals and other therapies is not included. For our study, AIDS death rates are adjusted for type of clinical manifestation of the syndrome. Three types were defined: KS only (approximately 10% of the total cases), PCP only (55% of total cases), and all other cases. The average survival from time of diagnosis has been calculated as 1.5 years, .87 years, and 1.01 years, respectively (Rothenberg et al. 1987).

8. Non-AIDS HIV Deaths

There have been recent citations regarding non-AIDS HIV (NAHD) deaths (Stoneburner et al. 1988; USGAO 1989): the extent and potential influence of these deaths are too significant to ignore. In our model, the non-AIDS HIV death rate was based on reported results of one non-AIDS death for each reported AIDS case in an IV drug user (Des Jarlais 1989). To include non-AIDS HIV deaths in the model, we assume the same incubation distribution for NAHD as for AIDS. This implies that persons with non-AIDS HIV disease progress to NAHD as rapidly as other infecteds progress to AIDS.

9. Infectivity

The probability of infection in a single contact depends on the mode of transmission. Table 2 identifies seven distinct modes of transmission associated with various pairings between risk groups and presents the values that were used in all simulations.

3. RESULTS

A.Simulation Scenarios

The model was run under two scenarios. Under Scenario 1 the NAHD subpopulation was set at zero. That is, those HIV-positive IV drug users who contracted an opportunistic infection which is not classified as AIDS (such as tuberculosis) and who died before they contracted an opportunistic infection which is classified as AIDS (such as P. carinii pneumonia) were not included as a separate subpopulation in the model. The AIDS case data were adjusted for reporting delays, change in case definition, and underreporting as described in Hamill et al. (1989). Then an additional upward adjustment of 39 percent was made to account for NAHDs and underreporting due to reasons suggested in the USGAO report (1989), but not included in the adjustment described in Hamill et al.

Under Scenario 2 one NAHD was assumed for every AIDS case in an IV drug user. The AIDS case data were adjusted according to Hamill et al. as under Scenario 1. The additional upward adjustment for underreporting was only 10 percent, however, because NAHDs were included as subpopulation in the model. These assumptions will have the effect of projecting more AIDS cases overall than under Scenario 1, for in this case the NAHDs can infect others who will in turn progress to AIDS in subsequent intervals.

B.Estimation of Contact Rates

The risk-group-specific and time-specific contact rate parameter values were varied until predicted AIDS incidence was within a specified tolerance (two percent) of observed AIDS incidence. For a few groups, negative contact rates were generated. These values were reassigned a value of zero and the tolerance was relaxed. Predicted and observed AIDS incidence are presented in Table 3.

C.Estimates of AIDS Cases

The results of Scenarios 1 and 2 for 1990 and 1993 are presented in Table 4. They include estimates of AIDS cases, AIDS cases plus NAHD, Total Infecteds (Alive) and Ever Infected cases. All estimates are cumulative from 1981 through 1990 or 1993, as indicated. A comparison of estimates from Scenarios 1 and 2 gives a range for projections. For example, the estimate of Ever Infected in 1990 ranges between 716,000 and 806,000. The difference between Total Infected (Alive) and Ever Infected cases is due to deaths from AIDS and other causes. The columns labelled "8 Risk Groups in Table 1" present estimates produced by the model. Since the model does not include pediatric, transfusion, foreign born, and hemophilia risk groups, model estimates were increased by 10.5 percent in order to produce national estimates. These are shown in columns labelled "Total."

4. DISCUSSION

The major feature of the RTI model is that risk-group-specific contact rates were estimated as those values which adequately predict AIDS incidence data. The estimation procedures can provide indications of problems in the form of implausible or improbable endogenous estimates. These problems may lie either with model assumptions or selected parameter values. For example, negative rates were estimated for homosexuals who use IV drugs. (These rates were subsequently set to zero.) In other cases (involving a non-Bachetti and Moss incubation

Table 2. Transmitter to Recipient Infectivity Probability Matrix $\{H_{ij}\}$

Recipients	Transmitters							
	HM	HMI	BM	BMI	OM	OMI	FM	FMI
HM	MM	MM	MM	MM	0	0	0	0
HMI	MM	MMN	MM	MMN	0	N	0	N
BM	MM	MM	MM	MM	0	0	FM	FM
BMI	MM	MMN	MM	MMN	0	N	FM	FMN
OM	0	N	0	0	0	0	FM	FM
OMI	0	N	0	N	0	N	FM	FMN
FM	0	N	MF	MF	MF	MF	MFN	0
FMI	0	N	MF	MFN	MF	MF	0	N

where

MM = .0071 = male to male sexual infectivity (Wiley 1987)
N = .010 = shared needle infectivity (Des Jarlais 1989)
FN = .0010 = female to male sexual infectivity (Wiley 1987)
MF = .0014 = male to female sexual infectivity (Peterman et al. 1988)
MMN = .0086 = the average of male to male sexual infectivity and shared needle infectivity (unweighted average)
FMN = .0055 = average of female to male sexual infectivity and shared needle infectivity (unweighted average)
MFN = .0051 = average of male to female sexual infectivity and shared needle infectivity (unweighted average).

function and a lower population at risk estimate), estimated contact rates were so high that the susceptible population was exhausted leading to a large discrepancy between predicted and observed incidence. In these situations, the initial size of the susceptible population may be underestimated, the probabilities of infection given contact may be underestimated, and/or the homogenous mixing assumption may be unrealistic and lead to overestimates of the number of contacts. In general, these discrepancies are reflections of the limitations in modeling an epidemic about which so little is known.

A number of other factors affect the fit and the validity of model projections. These include:

- **Data**--The projections reported here are based on national data. The use of the model with data at the city or Standard Metropolitan Statistical Area (SMSA) level would provide better explanatory capabilities. In addition, substantial undercounting exists in the CDC surveillance data. Although we have attempted to adjust for this along the lines suggested by the USGAO, reliable information on AIDS incidence is not available.
- **Incubation Function**--The incubation function is based on data for homosexual men from San Francisco. This function may not be appropriate for other risk groups and/or other regions. Since no data exist upon which to base an estimate of the incubation period distribution beyond ten years, a constant hazard is assumed after ten years. This assumption, however, may not be appropriate. Moreover, no effect of antiviral treatment was included in the model although evidence is accumulating which indicates that such treatment extends the incubation period.
- **Infectivity**--The homogeneous mixing model assumes constant infectivity throughout the infectious period. If this assumption is, in fact, not valid then model results may not provide adequate estimates of infection rates.
- **Contacts vs. Partnerships**--Many studies have demonstrated the significant role of partnership formation as a determinant of new infections (Anderson 1988; Dietz 1989). Our model ignores partnership formation and estimates new infections as a consequence of the average number of contacts (sexual or needle sharing).

In general, models can be useful for exploring relationships among variables and projecting the future course of events. For the model described above and for AIDS models in general, many uncertainties exist and there is a scarcity of reliable data upon which to establish the validity of an AIDS model.

The RTI model provides national projections for four years into the future using the contact rates estimated for 1988. Because of limitations inherent in the modeling process and lack of crucial information regarding the epidemic, this model is more useful for examining the dynamics of the epidemic than for making projections.

Table 3. Predicted and Observed AIDS Incidence
Scenario 1

Year	HM		HMI		BM		BMI		OM		OMI		FM		FMI	
	P	O	P	O	P	O	P	O	P	O	P	O	P	O	P	O
1981	431	434	46	46	29	29	8	8	3	4	79	79	4	4	30	31
1982	1056	1076	167	171	78	79	39	40	3	1	365	379	19	19	81	83
1983	3001	3001	464	463	461	466	166	167	4	3	1123	1123	55	54	322	324
1984	6311	6370	885	894	1116	1127	280	278	41	42	2249	2250	144	147	586	587
1985	11586	11583	1362	1258	2152	2153	485	485	84	67	4065	4035	398	396	1064	1063
1986	17598	17706	2021	2061	3539	3563	759	755	160	161	5938	5942	781	784	1665	1661
1987	22557	22557	2755	2228	4955	5003	915	919	372	373	7993	7916	1147	1143	2414	2439
1988	22284	22461	2486	1978	5219	4973	892	891	593	592	8571	8571	1369	1380	2648	2635
% Diff.	0.43		13.17		1.93		0.38		1.93		0.42		0.66		0.54	

Scenario 2

Year	HM		HMI		BM		BMI		OM		OMI		FM		FMI	
	P	O	P	O	P	O	P	O	P	O	P	O	P	O	P	O
1981	342	343	36	36	23	23	7	7	2	3	62	63	3	3	24	24
1982	839	851	133	135	62	63	31	32	2	1	293	300	15	15	65	66
1983	2392	2375	371	366	382	369	133	132	3	2	902	889	44	43	258	256
1984	5041	5041	707	707	883	892	224	220	33	33	1780	1781	115	117	468	464
1985	9157	9166	1107	996	1700	1704	385	384	67	53	3214	3193	314	314	840	842
1986	14002	14012	1610	1631	2825	2819	605	597	128	128	4690	4703	616	620	1312	1315
1987	17857	17851	2181	1763	3933	3959	728	727	295	295	6273	6265	904	904	1924	1931
1988	17894	17775	1968	1565	4091	3936	705	705	470	469	6817	6783	1088	1092	2101	2086
% Diff.	0.26		13.40		1.56		0.55		1.89		0.41		0.41		0.47	

P = predicted AIDS incidence; O = observed (adjusted) AIDS incidence.
% Difference = percent difference between predicted and observed incidence.

REFERENCES

ANDERSON, R. M. (1988), "The Epidemiology of HIV Infection: Variable Incubation Plus Infectious Periods and Heterogeneity in Sexual Activity, **Journal of the Royal Statistical Society**, A, 151, Part 1, 66-93.

BACHETTI, P. AND MOSS, A. R. (1989), "Incubation Period of AIDS in San Francisco," **Nature**, 338:251-253.

DES JARLAIS, D. C.; FRIEDMAN, S. R.; NOVICK, D. M.; SOTHERAN, J. L.; THOMAS, P.; YANCOVITZ, S. R. MILDVAN, D.; WEBER, J; KREEK, M. J., MASLANSKY, R.; BARTELME, S.; SPIRA, R.; AND MARMOR, M. (1989), "HIV-1 Infection Among Intravenous Drug Users in Manhattan, New York City, from 1977 through 1987," **Journal of the American Medical Association**, 261;7:1088-1012.

DES JARLAIS, D. C. (1989), personal communication.

DIETZ, K. (1989), **The Role of Pair Formation in the Transmission Dynamics of HIV**, presented at the 1989 Joint Statistical Meetings, American Statistical Association, Washington, D. C., August 6-10, 1989.

EVATT, B. L., GOMPERTS, E. D., MCDOUGAL, J. S. RAMSEY, B. B. (1985), "Coincidental Appearance of LAV/HTLV-III Antibodies in Hemophiliacs and the Onset of the AIDS Epidemic," **The New England Journal of Medicine**, 312;8:483-486.

HAMILL, D. N., COOLEY, P. C., READE-CHRISTOPHER, S. J., VAN DER HORST, C. M. (1989), **Estimating AIDS Incidence: Adjusting Case Reports for Changes in Definition and Reporting Delays**, Working Paper No. 2 (supported by the National Institute on Drug Abuse).

ROTHENBERG, R., WOELFEL, M., STONEBURNER, R., MILBERG, J., PARKER, R., AND TRUMAN, B. (1987), "Survival with the Acquired Immunodeficiency Syndrome: Experience with 5,833 Cases in New York City," **The New England Journal of Medicine**, 317(21), 1297-1302.

SELIK, R. M. HAVERKOS, H. W., CURRAN, J. W. (1984), "Acquired Immune Deficiency Syndrome (AIDS) Trends in the United States, 1978-1982," **American Journal of Medicine**, 76:493-500.

STONEBURNER, R. L., DES JARLAIS, D. C., BENEZRA, D., GORELKIN, L., SOTHERAN, J. L., FRIEDMAN, S. R., SCHULTZ, S., MARMOR, M., MILDVAN, D., MARKANSKY, R. (1988), "A Larger Spectrum of Severe HIV-1-Related Disease in Intravenous Drug Users in New York City," **Science**, 242:916-919.

UNITED STATES GENERAL ACCOUNTING OFFICE (1989), **AIDS Forecasting: Undercount of Cases and Lack of Key Data Weaken Existing Estimates.** Report to Congressional Requesters. GAO/PEMD-89-13.

WELLS, J. A., WILENSKY, G. R., VALLERON, A., BOND, G., SELL, R. L., AND DEFILIPPES, P. (1988), **Population Prevalence of AIDS High Risk Behaviors in France, the United Kingdom and the United States.** Presented at the Vth International Conference on AIDS, Montreal, Canada, June 4-9.