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 used 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. \( D_i(t) \) 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. \( D_{NAHD}(t) \) 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 group 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

<table>
<thead>
<tr>
<th>Risk Group Definitions</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Homosexual males who do not use IV drugs (RM)</td>
<td></td>
</tr>
<tr>
<td>2. Homosexual males who use IV drugs (HMI)</td>
<td></td>
</tr>
<tr>
<td>3. Bisexual males who do not use IV drugs (BM)</td>
<td></td>
</tr>
<tr>
<td>4. Bisexual males who use IV drugs (BMI)</td>
<td></td>
</tr>
<tr>
<td>5. Other males who do not use IV drugs (OM)</td>
<td></td>
</tr>
<tr>
<td>6. Other males who use IV drugs (OMI)</td>
<td></td>
</tr>
<tr>
<td>7. females who do not use IV drugs (FM)</td>
<td></td>
</tr>
<tr>
<td>8. females who use IV drugs (PMI)</td>
<td></td>
</tr>
</tbody>
</table>

We assume one infected person in each risk group at the beginning of the epidemic, \( T_0 \). 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

\[
\frac{dS_i(t)}{dt} = \delta S_i(T_o) - (\mu_i + \lambda_i(t))S_i(t) \tag{1}
\]

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

2. Infectives

\[
\frac{dI_i(t)}{dt} = \lambda_i(t)S_i(t) - (\gamma_i(t) + \theta_i(t) + \mu_i)I_i(t) \tag{2}
\]

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, \( \mu_i \).

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

\[
\gamma_i(t) = \lambda_i(t) - \rho_i \int_{T_o}^{t} \lambda_i(s)S_i(s)f_i(t|s)ds \bigg/ I_i(t) \tag{3}
\]

where

- \( \lambda_i(s)S_i(s) \) = the number of new infections in risk group i at time s
- \( \rho_i \) = the proportion of HIV infectives in the ith risk group that progress to AIDS rather than non-AIDS HIV related death
- \( f_i(t|s) \) = 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:

\[
\theta_i(t) = (1 - \rho_i) \int_{T_o}^{t} \lambda_i(s)S_i(s)g_i(t|s)ds \bigg/ I_i(t) \tag{4}
\]

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

\[
\frac{\partial A_i(t)}{\partial t} = \gamma_i(t) I_i(t) - A_i(t) \lambda_i(t),
\]

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 \( \pi_{ik}\) be the probability of being diagnosed with the \( k \)th AIDS type and \( d_k(t | s) \) be the probability of death at time \( t \) given diagnosis at time \( s \) for the \( k \)th AIDS type. With this notation, the death rate is defined as:

\[
\lambda_i(t) = \sum_k \pi_{ik} \int_{T_0}^T \gamma_i(s) I_i(s) d_k(t | s) ds / \lambda_i(t)
\]

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

\[
\frac{\partial A_i(t)}{\partial t} = \lambda_i(t) A_i(t)
\]

5. Non-AIDS HIV Deaths

\[
\frac{\partial I_i(t)}{\partial t} = \beta_i(t) I_i(t)
\]

AIDS deaths and non-AIDS HIV deaths depend on their respective death rates, \( \lambda_i(t) \) and \( \beta_i(t) \).

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 \( i \)th risk group, \( P_i(t) \) is the rate of partner acquisition for the \( i \)th 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 \( i \)th group at time \( t \).

The proportion of total contacts that occur with members of the \( j \)th risk group is denoted \( \beta_j(t) \). Under the homogeneous mixing assumption,

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

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 \( j \)th risk group for an individual in the \( i \)th group.

Under the assumption of homogeneous mixing, the probability of a given contact occurring with an infected member of the \( j \)th 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 \( i \)th group and an infective in the \( j \)th group produces an HIV infection. Then, the rate of new infections per susceptible at time \( t \) is:

\[
\lambda_i(t) = \alpha_i(t) \sum_j q_j(t) H_{ij}.
\]

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, \( T_0 \), 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 underdiagnosis 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.,

\[ \mu_1 = \mu_2 = \ldots = \mu_8 = 0.025. \]

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

\[ \delta_1 = \delta_2 = \ldots = \delta_8 = 0.025. \]

This per capita recruitment rate is always applied to the initial population size, \( S_i(T_0) \).

7. AIDS Deaths
The death rates, \( \{\lambda_i(t)\} \), 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 (Rochenberg 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 Scenario 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 (Hij)

<table>
<thead>
<tr>
<th>Transmitters</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HM</td>
<td>HMI</td>
<td>BM</td>
<td>BMI</td>
<td>OM</td>
<td>OMI</td>
<td>FM</td>
<td>FMI</td>
</tr>
<tr>
<td>HM</td>
<td>MM</td>
<td>MM</td>
<td>MM</td>
<td>MM</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>HMI</td>
<td>MM</td>
<td>MMN</td>
<td>MM</td>
<td>MMN</td>
<td>0</td>
<td>N</td>
<td>0</td>
<td>N</td>
</tr>
<tr>
<td>BM</td>
<td>MM</td>
<td>MM</td>
<td>MM</td>
<td>MM</td>
<td>0</td>
<td>0</td>
<td>FM</td>
<td>FM</td>
</tr>
<tr>
<td>BMI</td>
<td>MM</td>
<td>MMN</td>
<td>MM</td>
<td>MMN</td>
<td>0</td>
<td>N</td>
<td>FM</td>
<td>FMN</td>
</tr>
<tr>
<td>OM</td>
<td>0</td>
<td>N</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>FM</td>
<td>FM</td>
</tr>
<tr>
<td>OMI</td>
<td>0</td>
<td>N</td>
<td>0</td>
<td>N</td>
<td>0</td>
<td>N</td>
<td>FM</td>
<td>FMN</td>
</tr>
<tr>
<td>FM</td>
<td>0</td>
<td>N</td>
<td>MF</td>
<td>MF</td>
<td>MF</td>
<td>MF</td>
<td>MFN</td>
<td>0</td>
</tr>
<tr>
<td>FMI</td>
<td>0</td>
<td>N</td>
<td>MF</td>
<td>MFN</td>
<td>MF</td>
<td>MF</td>
<td>0</td>
<td>N</td>
</tr>
</tbody>
</table>

where

MM = .0071 = male to male sexual infectivity (Wiley 1987)
N = .010 = shared needle infectivity (Des Jarlais 1989)
FN = .0014 = male to female sexual infectivity (Wiley 1987)
MF = .0014 = female to male 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

<table>
<thead>
<tr>
<th>Year</th>
<th>HM P</th>
<th>HM O</th>
<th>BM P</th>
<th>BM O</th>
<th>OM P</th>
<th>OM O</th>
<th>FMI P</th>
<th>FMI O</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>431</td>
<td>434</td>
<td>46</td>
<td>46</td>
<td>8</td>
<td>8</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>1982</td>
<td>1056</td>
<td>1076</td>
<td>167</td>
<td>171</td>
<td>39</td>
<td>40</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>1983</td>
<td>3001</td>
<td>3001</td>
<td>264</td>
<td>263</td>
<td>166</td>
<td>167</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>1984</td>
<td>6311</td>
<td>6370</td>
<td>885</td>
<td>894</td>
<td>280</td>
<td>278</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>1985</td>
<td>11586</td>
<td>11583</td>
<td>1362</td>
<td>1258</td>
<td>485</td>
<td>485</td>
<td>84</td>
<td>84</td>
</tr>
<tr>
<td>1986</td>
<td>17598</td>
<td>17706</td>
<td>2021</td>
<td>2061</td>
<td>759</td>
<td>755</td>
<td>160</td>
<td>161</td>
</tr>
<tr>
<td>1987</td>
<td>22557</td>
<td>22557</td>
<td>2755</td>
<td>2228</td>
<td>915</td>
<td>919</td>
<td>372</td>
<td>373</td>
</tr>
<tr>
<td>1988</td>
<td>22284</td>
<td>22461</td>
<td>2486</td>
<td>1978</td>
<td>892</td>
<td>891</td>
<td>593</td>
<td>592</td>
</tr>
</tbody>
</table>

**I Diff.** 0.43 13.17 1.93 0.38 1.93 0.42 0.66 0.54

---

### Scenario 2

<table>
<thead>
<tr>
<th>Year</th>
<th>HM P</th>
<th>HM O</th>
<th>BM P</th>
<th>BM O</th>
<th>OM P</th>
<th>OM O</th>
<th>FMI P</th>
<th>FMI O</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>342</td>
<td>343</td>
<td>36</td>
<td>36</td>
<td>23</td>
<td>23</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>1982</td>
<td>839</td>
<td>851</td>
<td>133</td>
<td>135</td>
<td>62</td>
<td>63</td>
<td>31</td>
<td>32</td>
</tr>
<tr>
<td>1983</td>
<td>2392</td>
<td>2375</td>
<td>371</td>
<td>366</td>
<td>382</td>
<td>369</td>
<td>133</td>
<td>132</td>
</tr>
<tr>
<td>1984</td>
<td>5041</td>
<td>5041</td>
<td>707</td>
<td>707</td>
<td>883</td>
<td>892</td>
<td>224</td>
<td>220</td>
</tr>
<tr>
<td>1985</td>
<td>9157</td>
<td>9166</td>
<td>1107</td>
<td>996</td>
<td>1700</td>
<td>1704</td>
<td>385</td>
<td>384</td>
</tr>
<tr>
<td>1986</td>
<td>14002</td>
<td>14012</td>
<td>1610</td>
<td>1631</td>
<td>2825</td>
<td>2819</td>
<td>605</td>
<td>597</td>
</tr>
<tr>
<td>1987</td>
<td>17857</td>
<td>17851</td>
<td>2181</td>
<td>1763</td>
<td>3933</td>
<td>3959</td>
<td>728</td>
<td>727</td>
</tr>
<tr>
<td>1988</td>
<td>17894</td>
<td>17775</td>
<td>1948</td>
<td>1565</td>
<td>4091</td>
<td>3936</td>
<td>705</td>
<td>705</td>
</tr>
</tbody>
</table>

**I 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.

I Difference = percent difference between predicted and observed incidence.
REFERENCES


