Incidence of Herpes Simplex Virus Type 2 Infection in the United States

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Between the time that two large, national surveys were conducted, the Second National Health and Nutrition Examination Survey (1976–1980) and the Third National Health and Nutrition Examination Survey (1988–1994), prevalence of herpes simplex virus type 2 (HSV-2) infection in the United States increased by 30%. From these survey data, the authors estimated the incidence of HSV-2 infection in the civilian, noninstitutionalized population aged ≥12 years by means of a mathematical model that allowed overall incidence to increase linearly with time but required the shape of the age-specific incidence curve to remain constant. From 1970 to 1985, annual incidence of HSV-2 infection in HSV-2-seronegative persons increased by 82%, from 4.6 per 1,000 (95% confidence interval: 4.2, 5.0) to 8.4 per 1,000 (95% confidence interval: 7.7, 9.1). Incidence in 1985 was higher in women than in men (9.9 vs. 6.9 per 1,000), higher in Blacks than in Whites (20.4 vs. 6.3 per 1,000), and highest in the group aged 20–29 years (14.6 and 22.5 per 1,000 in men and women, respectively). Thus, by 1985, approximately 1,640,000±150,000 persons (730,000 men and 910,000 women) were being infected annually with HSV-2. Am J Epidemiol 2001;153:912–20.

Herpes simplex virus type 2 (HSV-2) is the most frequent cause of genital herpes in the United States and can infect the skin, anorectum, oral mucosa, eyes, and central nervous system (1–3). Infection in immunocompromised hosts results often in severe local disease and occasionally in disseminated disease. Infection during pregnancy, particularly around the time of delivery (4–7), places the infant at risk for neonatal herpes, a frequently devastating disease with high mortality and a high rate of permanent neurologic impairment among survivors (8, 9). This virus is spread through intimate contact and is often unrecognized; infection is commonly asymptomatic, and viral shedding may occur intermittently for years in the absence of symptoms (2, 10, 11).

Since the early 1970s, public awareness and concern about genital herpes have increased considerably, in part because of the impression that the burden of HSV-2-related disease has been rising (12). From the late 1960s to the mid-1990s, the annual number of outpatient visits for genital herpes in the United States increased steadily from 20,000 to over 150,000 (13). A recent national survey revealed that the prevalence of antibody to HSV-2 in US residents aged 12 years or older had increased from 16.4 percent in the late 1970s to 21.9 percent in the early 1990s (14, 15). The increase in prevalence was greatest in White teenagers and young adults.

There are no nationally representative data on the incidence of HSV-2 infection in the United States. Direct measurement of incidence is difficult because infection is most often asymptomatic and unrecognized. Furthermore, genital herpes infection is not reportable in most states. Determining seroconversion rates in a large, representative cohort would be possible but costly. A more practical approach is to estimate the past incidence of infection from existing seroprevalence data by means of “catalytic modeling” (16–20). We applied a variation of this methodology to data from the Second National Health and Nutrition Examination Survey (NHANES II) and the Third National Health and Nutrition Examination Survey (NHANES III) to estimate the incidence of HSV-2 infection in the general population as well as the risk of infection during pregnancy.
MATERIALS AND METHODS

Data sources

NHANES II and NHANES III were conducted from 1976 to 1980 and from 1988 to 1994, respectively (21, 22). Both surveyed multistage, stratified, clustered samples of the civilian, noninstitutionalized population to provide nationally representative prevalence estimates for a variety of health measures and conditions. From among participants aged 12 years or older from whom serum samples were available, a sample of 16,691 participants (3,597 from NHANES II and 13,094 from NHANES III) was tested for the presence of antibody to herpes simplex virus type 1 (HSV-1) and HSV-2 (15). Sera from both surveys were tested in a single laboratory by using an antigen specific to HSV-2 (glycoprotein gG-2) (23, 24). In persons with culture-proven, recurrent genital herpes, the sensitivity and specificity of this assay are more than 98 and 99 percent, respectively (24). The prevalence of HSV-2 antibody in these samples has been described previously (14, 15).

General description of the incidence models

Mathematical details of the models are given in the Appendix. What follows is a general description of how the models function.

The models estimated force of infection (25), which, for the purposes of this paper, is considered synonymous with annual incidence in the susceptible (i.e., seronegative) population. Force of infection was always modeled independently for males and females and for three race/ethnic groups: non-Hispanic Whites, non-Hispanic Blacks, and others (including Hispanics, Native Americans, Asians, and Pacific Islanders), referred to in this paper as Whites, Blacks, and others, respectively.

The past force of infection was modeled iteratively. At the beginning of each iteration, the modeled force of infection from the previous iteration was used to calculate what the prevalence of antibody to HSV-2 would be at the time of NHANES II and NHANES III (the “modeled prevalence”). The modeled prevalence was then compared with the actual prevalence according to the two surveys, and the force of infection was adjusted by using mathematical algorithms. This process was repeated until further adjustments resulted in no further improvement of the goodness of fit of the model.

The age-specific force-of-infection curve was determined by using the intercensal estimate of the civilian, noninstitutionalized population in that year. To estimate the risk of seroconversion during pregnancy, force of infection in women was weighted by using the 1996 US natality data set (27).

RESULTS

The three models produced similar estimates of age-, race-, and gender-specific force of infection. In all instances, differences between the models were small relative to the estimated standard errors. Therefore, only those results from the third model, which best fit the data, are presented here. As with the others, this model fit the observed seroprevalence well, especially that from the much larger NHANES III survey (figure 1).

Choice of time-dependent parameters

The estimated force of infection in any year varied with the placement of \( y_0 \) (figure 2). Nonetheless, the annual force of infection prior to the 1970s (4–5 per 1,000) and in the mid-1980s (8–9 per 1,000) depended little on the choice of \( y_0 \) (figures 2 and 3). Therefore, this paper provides results for only two periods: “before 1970” and 1985.

The goodness of fit was relatively insensitive to the choice of \( y_0 \) as long as it was placed between 1970 (weighted deviance, 2,091) and 1980 (weighted deviance, 2,083). For this reason, \( y_0 \) was not chosen solely on the basis of statistical considerations. Rather, 1970 was chosen because published data showed that outpatient visits for genital herpes began to rise in about 1970 (12, 13, 28).

The model fit best when the annual rates of increase in force of infection were set to 7, 2, and 9 percent of the pre-1970 baselines for Whites, Blacks, and others, respectively. Within each race/ethnic group, the estimated rates of increase, as a percentage of the baseline force of infection, were equal for males and females.
Force of infection, incidence, and prevalence of HSV-2 infection

The estimated annual force of infection increased from 4.6 per 1,000 (95 percent confidence interval (CI): 4.2, 5.0) before 1970 to 8.4 per 1,000 (95 percent CI: 7.7, 9.1) in 1985, a relative increase of 82 percent (figure 4). During this time, increases in prevalence of HSV-2 infection would have lagged behind increases in force of infection. In the overall population (aged 0 years or older), prevalence would have risen from 13.6 percent to 15.7 percent between 1970 and 1985, a relative increase of only 16 percent. Taking into account the increasing seroprevalence, the incidence averaged over the entire US population (i.e., the combined HSV-2-seronegative and -seropositive population) would have increased from 4.0 per 1,000 (95 percent CI: 3.7, 4.3) to 7.1 per 1,000 (95 percent CI: 6.5, 7.6) between 1970 and 1985, a relative increase of 78 percent.

The age-specific force of infection in 1985 increased with age to a peak of 14.6 and 22.5 per 1,000 in men and women aged 20–29 years and then declined to 2.6 and 3.4 per 1,000 in men and women aged 50 years or older (table 1). In all age groups, the force of infection was higher in Blacks than in Whites and higher in women than in men (figure 5). The disparity between men and women was greatest for adolescents and young adults and least at the extremes of ages.

In 1985, the estimated number of new HSV-2 infections in the US civilian, noninstitutionalized population was 1,670,000 (95 percent CI: 1,520,000, 1,820,000; table 2). A total of 56 percent of the infections were estimated to have occurred in women and 44 percent in men. The largest proportion of infections occurred in those aged 20–29 years (38 percent), followed by persons aged 30–39 years (25 percent), and those aged 12–19 years (17 percent).

In seronegative women, the risk of acquiring HSV-2 during a 9-month pregnancy increased from 9.1 per 1,000 pregnancies (95 percent CI: 8.1, 10.2) before 1970 to 16.7 per 1,000 pregnancies (95 percent CI: 14.6, 18.9) in 1985. This risk was greatest in the group aged 20–29 years (18.5 per 1,000, 95 percent CI: 15.3, 21.7). Because 80.6 percent of all women giving birth in 1985 would have been seronegative for HSV-2 infection, the overall rate of HSV-2 infection during pregnancy among all pregnant women would have been 13.5 per 1,000 (95 percent CI: 12.0, 15.0) in that year.


DISCUSSION

Using a novel adaptation of a method to model infection rates from seroprevalence data, we estimated that the annual incidence of new HSV-2 infections in uninfected persons in the United States in the mid-1980s was approximately 8.4 per 1,000. Rates were higher in women than in men, higher in Blacks than in other race/ethnic groups, and, in general, highest during the third decade of life.

The results of this model are consistent with other published estimates of incidence in European and North American populations. In a prospectively studied cohort of Swedish girls born in 1958 and 1959, HSV-2 seroconversion occurred at annual rates of 5, 24, and 23 per 1,000 from ages 13 to 18, 17 to 22, and 21 to 29 years, respectively (29). In a cohort of US university students in the mid-1980s, the annual seroconversion rate was 20 per 1,000 (30, 31). In pregnant women in Washington State and Birmingham, Alabama, 16 and 20 per 1,000 seronegative women, respectively, seroconverted over the course of their pregnancy (7, 31), corresponding to respective annual rates of 21 and 27 per 100,000. During two recent HSV-2 vaccine trials, groups of women and men at increased risk for infection seroconverted at rates of 68 and 44 per 1,000, respectively (32). Among women attending an antenatal clinic in London, United Kingdom, in 1980 and 1981, Ades et al. (19) estimated seroconversion rates of 2.4, 5, and 20 per 1,000 pregnancies in Asians, Whites, and Blacks, respectively, on the basis of a model that assumed a constant rate of acquisition.

The peak incidence in persons aged 20–29 years is consistent with studies in the United States and the United Kingdom showing that outpatient visits for genital herpes reach their maximum in this age group (12, 27, 33). This is older than the peak age for notifications in the United States of acute bacterial, sexually transmitted diseases such as
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FIGURE 2. Dependence of the overall force of infection (annual incidence in the susceptible (i.e., seronegative) population) of herpes simplex virus type 2 on year 0 (yr), 1960–1990. The four lines show the estimated force of infection in the US population for four different values of yr.

We estimate that the rate of HSV-2 seroconversion per 1,000 pregnancies in 1985 was 16.7, which, given the 3.76 million US livebirths that year (37) and the estimated prevalence of HSV-2-susceptible mothers (81 percent, data not shown) implies that 51,000 women would have become newly infected with HSV-2 during their pregnancy in that year. The prevalence of antibody to HSV-1 in HSV-2-negative women would have been 39 percent (data, not shown, from NHANES III); thus, 20,000 would have represented primary infections (i.e., HSV-2 infections in women without prior HSV infection), and 31,000 would have represented nonprimary first infections (i.e., HSV-2 infections in women with prior HSV-1 infections). Children of these women would be at the highest risk for neonatal infection if they were born during the time window between infection and HSV-2 seroconversion. If viral shedding during this window lasts 11.4 days in primary infection and 6.8 days in nonprimary first infection (38), and a constant risk of acquisition of HSV-2 throughout pregnancy is assumed (7), then 1,600 neonates would be at risk each year as a result of new maternal HSV-2 infections. Approximately 37 percent of these (6, 7), or 600, would become infected. Neonatal HSV-1 infection would account for approximately half as many cases (6–8), bringing the total to 900 neonatal herpes infections annually as a result of primary and nonprimary first maternal infections. This figure is consistent with another estimate of 700–2,300 cases of neonatal herpes annually in the United States (39) and is higher than the rate of neonatal herpes observed in King County, Washington, in the early 1980s (11.9 per 100,000 births, corresponding to 450 cases in a birth cohort of 3.76 million) (40).

These rates of seroconversion per pregnancy are predicated on similar forces of infection in pregnant and nonpregnant women. However, pregnant women may engage in sexual intercourse less often, may be less likely to change partners, and may have other characteristics that put them at lower risk.
FIGURE 3. Dependence of the age-specific force of infection (annual incidence in the susceptible (i.e., seronegative) population) of herpes simplex virus type 2 on year 0 (yr0), United States. Age-specific estimates of force of infection showed the least dependence on yr0 in the time period before yr0 and in the mid-1980s. The estimated force of infection in the late 1970s (not shown) and in the 1990s was much more dependent on the choice of yr0.

for HSV-2 infection relative to nonpregnant women. Conversely, other factors, such as decreased condom use or increased partner changing, could increase this risk.

Persons citing our results should understand their limitations. As with any model, the estimates and confidence intervals are conditional on the assumptions of the model. The confidence intervals, for example, do not account for uncertainties in yr0 and the rate of increase over time or for uncertainties about the dynamics of this increase. Also, estimates of incidence in the oldest age groups should be considered less reliable than those in younger age groups, since the prevalence in the former can be greatly affected by the incidence in younger age groups. Thus, the prevalence in older age groups may be subject to cohort effects not taken into account by the model. Furthermore, recent studies have documented that HSV-2 antibody levels in infected persons occasionally fall below the level of detection (41). Such seroreversion would cause our model to underestimate incidence.

We chose to model the increase in force of infection over time as a simple linear function; our model included data from only two points in time and thus did not allow a more complex analysis. In reality, the increase was almost certainly nonlinear and may have varied by age group. For this reason, and because the estimates of incidence in the late 1980s depended greatly on the value chosen for yr0, it would be a mistake to extrapolate our results beyond the mid-1980s. Furthermore, since the incidence of gonorrhea, syphilis, chlamydia, and primary human immunodeficiency virus infection has probably decreased since the early 1990s (42–44), the upward trend in the incidence of HSV-2 infection may also have reversed, although condom use, which may account for much of the decline in the incidence of sexually transmitted disease, is probably less effective in preventing HSV-2 transmission.

The estimated 82 percent increase in incidence from 1970 to 1985 is considerably greater than the 16 percent relative increase in prevalence we estimated to have occurred during this time or the 34 percent relative increase in HSV-2 seroprevalence between the time that NHANES II and NHANES III were conducted (15). Trends in the overall seroprevalence of infectious diseases generally lag behind trends in incidence and may not reflect them accurately. For example, if the incidence of HSV-2 infection increased substantially in the 1970s and 1980s, as we believe it did, then overall seroprevalence may currently be increasing even if incidence has stabilized or is decreasing slightly. Factors other than increasing incidence could have contributed to the observed increase in HSV-2 seroprevalence, although their effects would likely have been very small: a decreasing age at acquisition or a decreasing mortality rate among
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FIGURE 4. Estimated annual force of infection (annual incidence in the susceptible (i.e., seronegative) population) of herpes simplex virus type 2, United States, 1960–1990.

TABLE 1. Estimated force of infection,* prevalence, and risk of infection with herpes simplex type 2 virus during pregnancy before 1970 and in 1985, United States

<table>
<thead>
<tr>
<th>Age group (years) and gender</th>
<th>Force of infection (per 1,000)</th>
<th>Prevalence (%)</th>
<th>Risk of infection during pregnancy (per 1,000 seronegative women)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before 1970 Value</td>
<td>95% CI†</td>
<td>Before 1970 Value</td>
</tr>
<tr>
<td>All</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>3.7</td>
<td>3.2, 4.2</td>
<td>6.9</td>
</tr>
<tr>
<td>Female</td>
<td>5.5</td>
<td>5.1, 5.9</td>
<td>9.9</td>
</tr>
<tr>
<td>0–11</td>
<td>1.1</td>
<td>0.7, 1.6</td>
<td>2.2</td>
</tr>
<tr>
<td>Male</td>
<td>1.2</td>
<td>0.8, 1.5</td>
<td>2.1</td>
</tr>
<tr>
<td>Female</td>
<td>4.2</td>
<td>3.3, 5.2</td>
<td>7.7</td>
</tr>
<tr>
<td>12–19</td>
<td>7.9</td>
<td>7.1, 8.6</td>
<td>13.8</td>
</tr>
<tr>
<td>Male</td>
<td>4.2</td>
<td>3.3, 5.2</td>
<td>7.7</td>
</tr>
<tr>
<td>Female</td>
<td>7.9</td>
<td>7.1, 8.6</td>
<td>13.8</td>
</tr>
<tr>
<td>20–29</td>
<td>7.8</td>
<td>6.7, 8.8</td>
<td>14.6</td>
</tr>
<tr>
<td>Male</td>
<td>12.4</td>
<td>10.8, 14.0</td>
<td>22.5</td>
</tr>
<tr>
<td>Female</td>
<td>4.2</td>
<td>3.3, 5.2</td>
<td>7.7</td>
</tr>
<tr>
<td>30–39</td>
<td>6.5</td>
<td>4.5, 8.5</td>
<td>12.6</td>
</tr>
<tr>
<td>Male</td>
<td>9.1</td>
<td>7.4, 10.8</td>
<td>17.3</td>
</tr>
<tr>
<td>Female</td>
<td>2.2</td>
<td>1.2, 3.3</td>
<td>4.0</td>
</tr>
<tr>
<td>40–49</td>
<td>3.3</td>
<td>2.1, 4.5</td>
<td>6.1</td>
</tr>
<tr>
<td>Male</td>
<td>2.2</td>
<td>1.2, 3.3</td>
<td>4.0</td>
</tr>
<tr>
<td>Female</td>
<td>3.3</td>
<td>2.1, 4.5</td>
<td>6.1</td>
</tr>
<tr>
<td>≥50</td>
<td>1.5</td>
<td>0.6, 2.4</td>
<td>2.6</td>
</tr>
<tr>
<td>Male</td>
<td>1.8</td>
<td>0.7, 2.9</td>
<td>3.4</td>
</tr>
<tr>
<td>Female</td>
<td>2.2</td>
<td>1.2, 3.3</td>
<td>4.0</td>
</tr>
</tbody>
</table>

* Annual incidence in the susceptible (i.e., seronegative) population.
† CI, confidence interval estimated by using balance repeat replicates, assuming that the values for yr and m (the year in which force of infection was assumed to begin increasing and the annual rate of increase relative to the baseline) are known.
FIGURE 5. Force of infection (annual incidence in the susceptible (i.e., seronegative) population) of herpes simplex virus type 2 in Blacks and Whites, by age group and gender, United States, 1985. The error bars represent 95% confidence intervals estimated by using balanced repeat replication. Force of infection in the youngest age group was close to 0, with wide confidence intervals, and is not shown. Force of infection in the older age groups should be considered less reliable, for reasons stated in the Discussion section of the text.

populations at high risk for HSV-2 infection would both have caused prevalence to increase.

The next NHANES survey is now in progress and will soon provide new estimates of the seroprevalence of HSV-2 in the United States. These data can be incorporated into our model as they become available, making the results more robust. Data from teenagers and young adults will be especially important, since prevalence among these persons is most sensitive to recent changes in incidence and is thus more useful for delineating trends. If the current trend of increasing prevalence is to be reversed, strong prevention efforts will be needed as well as new technologies, such as vaccines.

REFERENCES


TABLE 2. Estimated number of new herpes simplex type 2 virus infections* in the United States, 1985

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>No. of new infections</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–11</td>
<td>90</td>
<td>60, 110</td>
</tr>
<tr>
<td>12–19</td>
<td>290</td>
<td>250, 330</td>
</tr>
<tr>
<td>20–29</td>
<td>640</td>
<td>570, 720</td>
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<tr>
<td>30–39</td>
<td>410</td>
<td>330, 490</td>
</tr>
<tr>
<td>40–49</td>
<td>100</td>
<td>60, 130</td>
</tr>
<tr>
<td>≥50</td>
<td>140</td>
<td>60, 210</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>730</td>
<td>630, 840</td>
</tr>
<tr>
<td>Female</td>
<td>940</td>
<td>860, 1,010</td>
</tr>
<tr>
<td>Total</td>
<td>1,670</td>
<td>1,530, 1,820</td>
</tr>
</tbody>
</table>

* No. of new infections and 95% confidence interval (CI) are given in thousands.
APPENDIX

Mathematical Details of the Models

All models assumed that infection always resulted in seroconversion, seroreversion was rare, and infection was not associated with increased mortality. Although the models did not take into account the effects of prior HSV-1 infection on HSV-2 acquisition, the prevalence of HSV-1 did not change between NHADES II and NHADES III; therefore, any effects from HSV-1 would have been stable during this time. The models assumed that force of infection, \( \lambda \), was both age and time dependent and could be expressed as a product of two functions:

\[
\lambda(a, yr) = f(a) \times g(yr)
\]

in which \( f(a) \) was the baseline age-specific force of infection at age \( a \) and \( g(yr) \) was the time-dependent multiplier in year \( yr \). Estimated prevalence at any given age \( A \) in the year of a survey, \( yr_s \), \( P(A, yr_s) \), was estimated as

\[
P(A, yr_s) = 1 - \exp\left[-\int_0^A \lambda(a, yr) da\right]
\]

in which \( yr = yr_s - A + a \).
We modeled \( f(a) \) separately with three different functions, which were chosen because they allowed force of infection to increase with age, then decrease, and finally to level off at older ages. In all models, several parameters were modeled by regression analysis, including the age at which force of infection peaked, the force of infection at this peak, and the force of infection in the oldest age groups.

The first model assumed that infection did not occur before age 10 years and that the baseline force of infection was constant with respect to age in persons aged 50 years or older. Baseline force of infection between ages 10 and 50 years was described by a polynomial,

\[
f(a) = \beta_0 + \beta_1 a + \beta_2 a^2 + \beta_3 a^3 + \ldots
\]

in which the \( \beta \)s were coefficients that could be estimated in a generalized linear model.

The second model assumed that

\[
f(a) = (\lambda_{\text{max}} - \lambda_{\text{late}} \cdot \text{STEP}(a_{\text{peak}})) \cdot \exp(-\delta(a - a_{\text{peak}})^2) + \lambda_{\text{late}} \cdot \text{STEP}(a_{\text{peak}})
\]

in which \( \lambda_{\text{max}} \) was the maximum baseline force of infection, \( a_{\text{peak}} \) was the age at which this maximum occurred, \( \delta \) was a parameter inversely related to the width of the age range at highest risk for infection, \( \lambda_{\text{late}} \) was the baseline force of infection at older ages, and \( \text{STEP}(a_{\text{peak}}) \) was a Heavyside function equal to 0 before \( a_{\text{peak}} \) and 1 thereafter. The four parameters were estimated by using nonlinear regression analysis after specifying a large grid of starting values.

In the third model, the mean baseline force of infection was taken from \( K \) different submodels. In each submodel, \( f(a) \) was modeled as a step function with a finite number of steps of width \( K \) years, followed by a final step of variable width. The first step began at age 0 years in the first submodel, age 1 year in the second model, age 2 years in the third model, and so on. In the \( n \)th step of the \( k \)th submodel, \( f(k,a) = \beta_{k,n} \). After each of the \( K \) submodels was fit independently to the data, \( f(a) \) was taken as the mean of the \( \beta \)s that included that particular age. \( K \) was varied from 6 to 20 to find the minimum value that produced a smooth, age-specific, force-of-infection curve.

In all models, \( g(\text{yr}) \) was assumed to be 1.0 prior to a certain year, \( \text{yr}_0 \), and to increase linearly thereafter by \( m \) percent of the baseline. \( \text{yr}_0 \) and \( m \) were determined empirically by varying \( \text{yr}_0 \) from 1940 to 1988, determining the value of \( m \) that produced the best fit of the model for each \( \text{yr}_0 \), and then charting goodness of fit by \( \text{yr}_0 \).

The modeling was performed with SAS software (SAS Institute, Cary, North Carolina). The models were fit with either PROC GENMOD, a generalized linear modeling procedure (first and third models), or PROC NLIN, a nonlinear modeling procedure (second model), such that the estimated prevalence, \( P(A,\text{yr}) \), matched the actual prevalence observed in the two surveys. Goodness of fit was assessed by the weighted deviance (minus two times the logarithm of the unconditional binomial likelihood ratio, weighted with the normalized survey weight). Variance was estimated by using Faye’s variation of the balance repeat replicate method (45). Replicate weights were calculated with WESVARPC software (WESTAT, Rockville, Maryland).