INFECTIOUSNESS OF HIV BETWEEN MALE HOMOSEXUAL PARTNERS

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Abstract—To estimate the risk of transmission of HIV per receptive anal sexual contact, 329 homosexually-active men, representing 155 sexual partnerships, were enrolled into a study. Information on HIV infection status and sexual behavior within and outside the primary relationship was collected. Of these 329 men, 24 had AIDS and 31 had ARC. Of the 155 couples, 35 consisted of partners that were both HIV +; 62, of partners that were both HIV −; and 58 were discordant. A binomial model was fit to data obtained in the first visit to estimate per contact risk of HIV transmission. Assuming a constant risk of transmission per sexual contact between infected and uninfected partners, the estimated risk is about 5 to 30 per 1000 receptive anal exposures to ejaculate. Although the average risk of HIV transmission per sexual contact appears to be low, there appears to be great variability in infectivity. To model this variability over time and across individuals, more complex models must be fit to longitudinal studies of sexual partners.

HIV   Sexual transmission   Modeling   Sexual behavior

INTRODUCTION

The major behavioral risk factors in the transmission of the Human Immunodeficiency Virus (HIV) between homosexual/bisexual men include receptive anogenital exposure to infected ejaculate and a large number of sexual partners [1–3]. However, some men with large numbers of receptive anal sexual contacts remain uninfected while others with infrequent contact become infected [4–5]. The degree and duration of infectiousness in patients with AIDS, AIDS-related complex (ARC) and asymptomatic HIV infection are unknown.

Investigators have reported a high degree of variability in the seroprevalence of HIV infection among sexual partners of infected individuals; seroprevalence has varied from 7.9% [6] to 73% [6–17]. Because of the large number of factors that influence transmission, however, inference about infectiousness is difficult. Low seroprevalence among sexual partners may reflect a low number of sexual contacts during periods of infectiousness; high seroprevalence may reflect acquisition of HIV outside the relationship. Moreover, recent studies have suggested that the efficiency of HIV transmission per sexual act is not uniform [18, 19] and individuals who are at more advanced stages of HIV infection, may be more likely to infect their partners. Other immunological factors in the infected partner [13] as well as the presence of concomitant sexually transmitted diseases [6, 16] may also modify the efficiency of HIV transmission per sexual act. In addition, there may be variability in susceptibility to HIV infection, although no evidence for such variability has been found [4].

Although a number of studies have provided estimates of the prevalence of HIV in partners

of infected individuals, none has been large enough to permit characterization of the variability in HIV infectiousness. Grant *et al.* developed a preliminary estimate of infectivity per receptive anal partnership that ignored the variability in duration of partnership as well as in infectivity over time and among individuals; this estimate was 0.102 (95% CI = 0.043–0.160) by assuming that this infectivity is constant [18]. A recent study by Wiley *et al.* provides some evidence, however, that the assumption of constant risk of transmission per contact is not supported by data on female partners of HIV-infected men [19]. Full characterization of infectivity, will require large cohorts observed over long periods of time.

In this report, we estimate the risk of transmission of HIV per sexual contact and the variability in this risk based on self-reported information from sexual partners of men infected with HIV. We first estimate infectivity under the assumption that it is constant across individuals and over time, and then allow for variability between individuals, by assuming that the rate of infectivity for each individual arises from a parametric family of distributions. Since the data on which we report are cross-sectional, they do not permit an analysis of variability over time. Nonetheless, they do permit estimation of the ranges in infectivity; these ranges are developed by fitting a variety of different models and using customary estimates of variance.

METHODS

Selection of study participants

Male index subjects were eligible for enrollment if they were homosexual or bisexual, 18 years of age or older, and resided within the Boston Standard Metropolitan Statistical Area. AIDS and ARC cases were diagnosed according to the Centers for Disease Control (CDC) classification system for AIDS (Group IV subgroups C-1 and D-1) and ARC (Group III, IVA or C-2) [20]. Asymptomatic men were defined as men without AIDS, ARC, malignancy, or other illness lasting more than 1 month. Sexual partners were defined as men who had orogenital, anogenital, oroanal, or manual–rectal contact (fisting) involving an exchange of body fluids and/or penetration with a study subject within the past 30 months. The protocol for this study was approved by the Human Studies

Committees of all participating institutions (BDH&H, FCHC, NEDH, and CDC).

Data collection

At enrollment all participants were interviewed to obtain data on demographic characteristics, occupational and travel histories, sexual behaviors with study participants and with partners not enrolled in the study, and drug and alcohol use. A complete medical history was obtained for the 5 years before study enrollment and a complete clinical examination was performed.

Laboratory studies

All participants were evaluated with a complete blood count (CBC) and lymphocyte subset determination, performed using specific monoclonal antibodies (Ortho Diagnostics, Raritan, N.J.). Antibodies to HIV were determined by use of an enzyme immunoassay (EIA: Abbott Laboratories, Chicago, Ill.). Western blot confirmations were performed on all sera initially positive by EIA and on a sample of other sera initially positive by previously described methods [21].

Enrollment

Twenty-four AIDS patients as well as 119 asymptomatic men (either infected with HIV or at risk of infection) agreed to participate as index participants. Each index participant who enrolled in the study attempted to enroll at least one partner. The majority of the index participants were recruited from the Fenway Community Health Center in Boston; the remainder were recruited from the New England Deaconess Hospital. A detailed description of enrollment is available from Seage *et al.* [22] Of the 174 index participants, 42 (24.1%) were unable to enroll a partner, 114 (65.5%) enrolled one partner, 13 (7.5%) enrolled two partners and 5 (2.9%) enrolled three partners. Therefore, a total of 132 index participants enrolled a total of 155 sexual partners in the study.

Demographic characteristics

The average age of the cohort members was 32.8 years. There were no significant trends of HIV infection observed with education (mean = 16.0 years), income (median = U.S.$10,000–$19,999), race (95% were white), length of time residing in metropolitan Boston (mean = 144 months), or marital status (10% were married at some time).

Statistical methods

Logistic regression was used to determine the factors that were related to HIV infection. Analyses were done separately for participants whose study partners were or were not infected. The factors we considered included: (1) number of sexual contacts with the study partner, (2) proportion of those contacts that were anal receptive, (3) number of steady partners (more than 3 sexual contacts) outside the primary relationship, (4) number of non-steady partners (3 or fewer sexual contacts) outside this relationship, and (5) T4 and T8 lymphocyte counts (cells/mm^3). The date of infection is not known for the infected study partners, therefore it is not known how many of the sexual exposures with infected partners took place after infection. Because condom use was reported by less than 1% of study participants at time of enrollment, such use could be ignored in our analysis. To get ranges for the risk of transmission per exposure, we assume different values for the maximum number of contacts that have taken place after infection, and the bounds were 50, 100, and 1000. These values correspond to a belief that only the last 50, 100 or 1000 exposures to an infected partner took place after his infection with HIV.

The relationship between the log odds of being infected with HIV was modeled as a linear function of the factors listed above:

$$\log \Pr(\text{HIV}+)/\Pr(\text{HIV}-) = \sum_i k_i x_i$$

where k_i is the regression coefficient, x_i is the factor of interest, and HIV+ or HIV− refers to the presence or absence of HIV infection. If the risk of transmission of HIV per sexual exposure to an infected partner were constant, the log odds of infection with HIV would have the following relationship to number of contacts, n:

$$\log [\Pr(\text{HIV}+)/\Pr(\text{HIV}-)]$$
$$= \log[1 - (1-p)^n] - n \log(1-p) \quad (1)$$

where n is the number of exposures to an infected study partner, and p is the probability of transmission per exposure. Thus, under the assumption of constant risk of transmission per contact, the coefficient k_i referring to the number of contacts with an infected study partner is the slope of a linear approximation to equation (1). This also applies to the relationship between HIV infection and the number of non-steady partners.

To estimate the risk of HIV transmission per exposure to an infected partner, this risk assumed to be constant at each exposure. In addition, each outside partner was assumed to have an equal risk of transmitting HIV. Because the time of infection of the study participants is not known, it is not possible to determine how many of the exposures took place after infection of the index partner. To determine a range of values for risk per exposure, three analyses were performed in which the total number of exposures was truncated at 50, 100 and 1000. Partners of infected individuals may themselves have been infected by exposures outside the primary relationship. Thus, there are four different possible configuration of infection status for each set of partners. The likelihood contribution and sample size of partnerships of each type are shown below.

Index	Partner	Likelihood contribution	Sample size
HIV−	HIV−	$(1-po)^{no}$	124
HIV−	HIV+	$(1-po)^{no}(1-pi)^{ni}$	59
HIV+	HIV−	$[1-(1-po)^{no}]$	59
HIV+	HIV+	$[1-(1-po)^{no}(1-pi)^{ni}]$	70

where po is the risk of transmission for each partner outside the study relationship, pi is the risk of transmission for each exposure to an infected primary partner, no is the number of outside partners and ni is the number of exposures to an infected primary partner. The full likelihood is the product of the likelihoods for each infection configuration.

Since some infected participants may have infected their partners after a small number of contacts while others fail to infect their partners after hundreds of contacts, a second analysis based on a different assumption was performed. This analysis assumes that the risk of transmission per exposure was constant within each partnership, i.e. p_j for the jth partnership, but that the p_j are random variables, sampled from a beta distribution:

$$\Pr(p_j) = \Gamma(a+b)/[\Gamma(a)\Gamma(b)]\, p_j^{a-1}(1-p_j)^{b-1}.$$

Since time of infection is now known, the distribution of p_j will be affected not only by the true infectivity, but also by the proportion of exposures that took place after infection. For this analysis, only participants who had at least 5 anal-receptive exposures to an infected study partner were included. There were 27 seronegative and 44 seropositive individuals with infected partners who met this criteria. Subsequent analysis of longitudinal studies of transmission will make it possible to estimate

this distribution without confounding, since the time interval of infection will be known.

RESULTS

I. Analysis of factors related to HIV infection

Results of logistic regression analysis. Among 129 men with infected study partners, only the number of receptive anal exposures, truncated at 100, was significantly associated with infection at the 0.05 level (Table 1). T helper and T suppressor cell counts were available for 99 infected study partners of these 129 men. No association between these counts and HIV transmission could be inferred. Although mean T helper count was slightly lower among infected men with infected partners than among infected men with uninfected partners (411 compared to 479), this difference was not statistically significant. There was virtually no difference in the means of T suppressor count between these two groups (566 compared to 578). When included in the logistic regression model, T cell subsets of the study partner were not predictive of infection with HIV ($p > 0.2$). Nor was a statistically significant association noted between infection with HIV and number of steady or non-steady partners; although uninfected men tended to have more of the former and fewer of the latter.

Among 158 participants with uninfected study partners, the only variable associated with HIV infection at the 0.05 level was proportion of sexual contacts with the study partner that was anal receptive (Table 2). The directions of the associations between HIV infection and number of steady and non-steady partners was

the same as before, but these associations approached statistical significance at the 0.05 level. Although the magnitude of associations between HIV infection and sexual behavior varied, depending on whether the exposures were truncated at 100 and 1000, the directions of these associations did not. For example, number of receptive anal exposures to an infected partner was much more strongly associated with infection at the lower truncation level. This may result from the fact that the loglinear approximation to the relationship between odds of infection and number of contacts would be less accurate when the number of contacts is large.

II. Estimation of risk of transmission per exposure

Three sets of analysis were performed, in each of which parameters were estimated by the method of maximum likelihood. First, the parameters *po* and *pi* were estimated jointly using all of the data. Second, *po* was estimated using only information on individuals with uninfected partners. Third, upper bounds of *pi* were estimated by setting *po* to 0.

The risk of infection per outside partner was estimated to be 0.075($\pm$0.018) when only participants with uninfected primary partners were included in the analysis, but 0.095 ($\pm$0.018) when the full cohort was used (Table 3). The risk per contact ranged from 0.008 ($+$0.003) when the number of exposures was truncated at 1000 and the possibility of being infected outside the relationship was allowed, to 0.032 ($\pm$0.010) when individuals were assumed to have been infected from the primary partner and the number of contacts was truncated at 50.

Table 1. Factors related to HIV infection for participants with infected study partners

Variable	Regression coefficient	*p*-Value
Number of exposures to infected partner bounded by 100		
No. of receptive anal exposures with study partner	−0.0144	0.01
No. steady partners	0.157	0.21
No. non-steady partners	−0.014	0.23
Unbounded number of contacts		
No. of receptive anal exposures with study partner	−0.0019	0.12
No. steady partners	0.178	0.15
No. non-steady partners	−0.014	0.25

The number of T helper cells and T suppressor cells in an infected study partner were not predictive of a participant's infection status. Regression coefficients associated with these counts were 0.00097 and −0.00019; *p* values were greater than 0.2 in both cases.

Table 2. Factors related to HIV infection for participants with uninfected
partners

Variable	Regression coefficient	p-Value
Number of contacts bounded by 100		
No. of exposures	0.00035	0.69
Proportion anal receptive	−0.89	0.07
No. steady partners	0.26	0.07
No. non-steady partners	−0.022	0.13
Unbounded number of exposures		
No. of exposures	0.0006	0.29
Proportion anal receptive	−0.86	0.03
No. steady partners	0.278	0.06
No. non-steady partners	−0.021	0.13

III. Estimating the distribution of infectivity across partners

Figure 1 shows the distribution of p_j under the assumption that the risk of transmission from non-study partners was 0.075 per partner, i.e. the rate that was estimated from infected individuals with uninfected study partners. When 0.095 was used instead (this was the estimated risk of transmission using all of the partners), the results changed only slightly. The estimated beta coefficients were $a = 0.35$ and $b = 6.5$; the estimated mean risk of transmission was 0.051. An estimated 48% of the infected partners have a risk of transmission that is less than 0.02 per exposure; 80% have a risk that is less than 0.10. When the number of contacts was truncated at 100, the mean risk per contact decreases to 0.038% ($a = 0.55$; $b = 14$). This reduction results from limiting the number of contacts that

Table 3. Estimate of infectivity per anal receptive contact

(1) Data: full cohort

Upper bound of no. of exposures	$po \pm 2SE$	$pi \pm 2SE$
50	0.095 (±0.022)	0.017 (±0.007)
100	0.095 (±0.020)	0.013 (±0.005)
1000	0.095 (±0.020)	0.008 (±0.003)

Assumption: transmission occurs either from an outside partner or from exposure to a primary partner. (These exposures may be truncated at 50, 100, or 1000).

(2) Data: participants with uninfected primary partners

po	$(\pm 2SE)$
0.075	(±0.018)

Assumption: transmission occurred only from contacts outside primary relationship

(3) Data: full cohort

Upper bound of no. of exposures	pi $(\pm 2SE)$
50	0.032 (±0.010)
100	0.020 (±0.006)
1000	0.013 (±0.004)

Assumption: transmission occurred only from primary partner.

an uninfected partner can have with an infected partner without becoming infected. After truncation, 51% of partners have a risk that is less than 0.02 per exposure and 91% have a risk that is less than 0.10. Both sets of parameters lead to distributions with fairly high dispersion. This reflects the fact that 12 men reported more than 100 receptive anal exposures to their partners' ejaculate without seroconverting (the model would predict only 1), while 5 men may have seroconverted after 10 or fewer exposures to an infected study partner and fewer than 3 partners outside this relationship. Note that the effect of truncation is to reduce the average risk of transmission per exposure in this analysis whereas it increased the estimated risk per exposure in the previous model.

The reason for the higher estimates of infectivity than those described above is that the unusual observations, i.e. uninfected participants who reported many exposures to infected partners and partners who may have transmitted HIV after a small number of contacts, are not so influential on these results as on the analysis that assumes the same risk of transmission for all partners. Although the parameters of the beta distribution cannot be estimated with great precision from these data, they suggest that infectivity is highly variable among infected individuals; precise quantification of this variability must await analysis of longitudinal data.

DISCUSSION

Research on partners of HIV infected people reveals a great deal of variability in rates of infection. This variability may reflect the mode of infection as well as stage on disease of the index case. Rates of infection range from 9.5% (2 of 21) among female partners of HIV infected symptomatic hemophiliacs [8], to 18% (10 of 55)
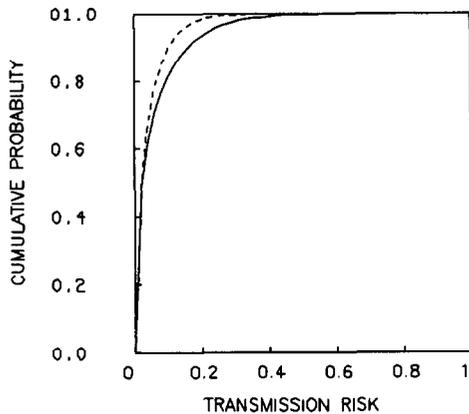
Fig. 1. Cumulative distribution of risk of HIV transmission
per sexual contact. —— Number of exposures truncated at
1000; – – – number of exposures truncated at 100.

of female partners of symptomatic male trans-
fusion recipients [10], to 45% (51 of 114) among
female partners of AIDS and ARC i.v. drug
users (IVDU) [11]. The reasons for this varia-
bility are not well understood. Several studies
have suggested that asymptomatic persons
are less likely to transmit HIV to their sexual
partners than symptomatic persons [12–14].
Osmond et al. found that gay men who had
AIDS or ARC were more likely to infect their
partners than were asymptomatic seropositives
[23]. However, in comparing these groups one
must be careful to note that the durations of the
relationships and rates of sexual activity also
vary. More efficient HIV transmission may also
be a function of other immunologic parameters
[22] as well as concomitant sexually transmitted
diseases. Nonetheless, in our study, the ability
to infect a partner did not appear to be related
to immune status.

A recent cross-sectional study of behavioral
practices among homosexual couples found that
the majority of homosexual partners of AIDS
and ARC patients were HIV antibody positive
on screening [24]. It is difficult to estimate per
contact risk from such studies, because the
participants often had substantial numbers of
contacts outside the relationship. Unlike our
study, however, the study by Coates et al. did
not include comparison groups of healthy,
uninfected or asymptomatic, HIV-infected
homosexuals. Understanding the infectiousness
of asymptomatic HIV infected individuals is
especially important since it is estimated that
1–1.5 million Americans may fall in this
category [25].

Our analyses were based on data from an
ongoing study of HIV transmission between

male homosexual partners. Two analyses were
performed: one in which the risk of transmission
was assumed to be constant per sexual contact,
and a second in which this risk was assumed to
be randomly distributed. Even under the as-
sumption that the risk per exposure is constant,
the range in estimates of this risk is large: from
0.008 (± 0.003) when sexual contacts outside
the primary relationship are included and all
exposures with an infected partner are assumed
to have taken place after infection to 0.032
(± 0.010) when the primary partner is assumed
to be the sole transmitter of HIV infection and
only the last 50 of the exposures to an infected
partner are assumed to have taken place after
infection.

Regardless of the assumptions made, there
was evidence of lack of fit of the binomial model
to the study data. In fitting the model, each
individual can be assigned a probability of
being seropositive depending on his number
of exposures to his study partner and number of
outside partners. One way to assess fit is to
compare the observed and predicted numbers of
seropositive individuals among those who have
seropositive partners. Table 4 gives the observed
and predicted HIV serostatus for such in-
dividuals, broken down by the number of anal
receptive exposures to the study partner. These
predicted values are computed with the number
of exposures truncated at 1000; an individual
is predicted to have a given HIV status if his
probability of having that status is greater than
0.5. This table shows that the binomial model
underestimates the number of individuals who
would be infected after a few contacts and
overestimates the number who would be in-
fected after many contacts. One explanation for
this lack of fit is that infectiousness varies
considerably over individuals.

Because of the poor fit of the binomial
model, we fit a second model that allowed the
per contact risk of transmission to vary over
individuals. The results of fitting this model
imply a broad range of risk of transmission of
HIV to the partners of seropositive men: 50%
of the partners of infected men have a sero-
conversion risk per exposure of less than 0.02;
10–20% of infected partners have a greater than
0.10 risk of seroconversion per sexual exposure.
Interpretation of these results is complicated by
the lack of information about time of infection
of the index case as well as by the possibility
of infection outside the relationship. Nonethe-
less, after controlling to the maximum degree

Table 4. Observed and predicted* HIV serostatus by number of exposurest to infected partners

Number of exposures:	0–10		11–50		51–100		>100		Total
Predicted status:	+	–	+	–	+	–	+	–	
Observed status:									
HIV+	5	21	2	8	11	1	22	0	70
HIV–	2	39	2	4	0	0	11	1	59
Total	7	60	4	12	11	1	33	1	129

*An individual is predicted to have a given serostatus if his probability of having that status is grater than 0.5 as calculated by the binomial model with untruncated number of partners.
tThis number refers to the untruncated number of anal exposures to a study partner.

possible for these effects, our analysis indicated that HIV-infected individuals may vary considerably in their ability to infect a partner; these results imply that heterogeneity in infectivity must be included in any attempt to model the dynamics of the AIDS epidemic.

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