Prevalence of HIV Infection Among Young Adults in the United States: Results From the Add Health Study

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Objectives. We estimated HIV prevalence rates among young adults in the United States.

Methods. We used survey data from the third wave of the National Longitudinal Study of Adolescent Health, a random sample of nearly 19 000 young adults initiated in 1994–1995. Consenting respondents were screened for the presence of antibodies to HIV-1 in oral mucosal transudate specimens. We calculated prevalence rates, accounting for survey design, response rates, and test performance.

Results. Among the 13 184 participants, the HIV prevalence rate was 1.0 per 1000 (95% confidence interval [CI] = 0.4, 1.7). Gender-specific prevalence rates were similar, but rates differed markedly between non-Hispanic Blacks (4.9 per 1000; 95% CI = 1.8, 8.7) and members of other racial/ethnic groups (0.22 per 1000; 95% CI = 0.00, 0.64).

Conclusions. Racial disparities in HIV in the United States are established early in the life span, and our data suggest that 15% to 30% of all cases of HIV occur among individuals younger than 25 years. (*Am J Public Health.* 2006;96:1091–1097. doi:10.2105/AJPH.2004.054759)

The estimated incidence of both AIDS and HIV in the United States is believed to be fairly stable after having fallen for several years subsequent to the initial crest of the epidemic.[1,2] In contrast, the prevalence of AIDS continues to rise as treatment extends survival rates among individuals with HIV. According to Centers for Disease Control and Prevention (CDC) estimates, more than 174 000 people in the United States were living with HIV (without AIDS) as of December 2003, and another 400 000 were living with AIDS.[1]

On the basis of the age distribution of cases identified through AIDS surveillance systems, estimates suggest that as many as half of all infections occur among individuals younger than 25 years.[3,4] CDC data indicate a relatively balanced gender ratio but a large racial disparity among young people with HIV: 41% of 13- to 24-year-olds reported to the CDC HIV surveillance system in 2001 were young women (no other age group exhibited a larger female share of infection); 56% were non-Hispanic Blacks, as compared with 15% of the general population.[5]

The current data available on HIV prevalence rates among young people are subject to several limitations. CDC surveillance data on HIV prevalence in the United States are steadily improving but remain incomplete, and they are not well suited to estimating prevalence rates among young adults. The CDC surveillance system identifies only those who choose to be tested for HIV and those who fit screening profiles. Surveys indicate that the testing rate outside of the populations at highest risk remains below 50%.[6] Testing also appears to occur late in the course of infection: 20% of patients are diagnosed with HIV and AIDS in the same calendar month and approximately 40% in the same year.[7] Because HIV infection typically precedes AIDS by about 10 years, this suggests that many individuals are infected well before their HIV diagnosis is recorded and that young people may be underrepresented in surveillance data. Finally, the system does not include all states or regions and thus may not provide representative data.

Alternative sources of data on HIV infection among young people include back calculation (imputing dates of infection from dates of AIDS diagnosis) and surveys. Back calculation from incident AIDS cases has become less feasible with the increasingly widespread use of antiretroviral therapy, and the most recent reliable back-calculation estimates are now a decade old.[8] Population-based surveys require very large samples to provide reliable estimates for low-prevalence populations. Data are available from only one such US survey, the National Health and Nutrition Examination Survey (NHANES). NHANES was primarily designed to estimate HIV prevalence rates among adults and thus involved a relatively small sample of young people (approximately 1500 cases).

Wave III of the National Longitudinal Study of Adolescent Health (Add Health), a nationally representative, population-based survey, was designed to provide estimates of HIV prevalence rates among 19- to 24-year-olds in the United States. The third wave of Add Health, conducted in 2001–2002, surveyed approximately 14 000 young adult men and women. These data, which we examined in this study, provide the first general population estimates of the prevalence of HIV by gender and race among US young adults. In addition, Add Health's longitudinal design offered a unique opportunity to prospectively assess the nature of nonresponse in a survey context, in that people who dropped out in later waves had information available from earlier waves that could be used in such assessments.

METHODS

Design and Participants

Add Health was designed to assess the health status of adolescents and explore the causes of their health-related behaviors. Three waves of data were collected: wave I during September 1994 through December 1995, wave II during April through August 1996, and wave III during August 2001 through April 2002. The primary sampling unit for the

original survey was the school, and the study design ensured that the sample was representative of US schools with respect to region of the country, urbanism, school type, ethnicity, and school size. The wave I representative sample comprised 18 924 participants; targeted oversampling ensured substantial representation of non-Hispanic Blacks, Hispanics, Asian Americans, and Native Americans (for additional details on the study design, see http://www.cpc.unc.edu/projects/addhealth).

The wave III sample consisted of 14 322 wave I participants located and reinterviewed during the fieldwork period (75.7% response rate). The great majority of respondents were interviewed at home; if this was not possible, every attempt was made to interview them in their current location. For example, to the extent that permission was obtained by local authorities and prison officials and confidentiality of the interview could be ensured, respondents were interviewed at military bases, penitentiary facilities, and other institutions. Before beginning, the interviewer described the wave III interview and obtained consent for participation. Completion of the interview was not linked to a requirement to provide a biospecimen. At the end of the interview, respondents were asked to independently consent to and provide (with an incentive payment of $10) an oral mucosal transudate (OMT) specimen for HIV testing.

Here we report prevalence estimates stratified according to self-reported gender and race. We collapsed race into 2 categories: non-Hispanic Black and other. This unfortunate limitation was necessitated by the small number of HIV-positive cases observed and the reporting rules designed to protect respondent confidentiality. Respondents who reported "Black or non-Hispanic Black" as their only or primary race and did not report Hispanic origin were classified as non-Hispanic Black.

Specimen Collection and Testing

The OraSure HIV-1 Oral Fluid Specimen Device (OraSure Technologies, Bethlehem, Pa) was used in collecting OMT specimens. The collection device was then placed in the vial supplied with the kit and shipped to the laboratory facility in a plastic bag. The Oral Fluid Vironostika HIV-1 Microelisa System (Organon Teknika Corp, Durham, NC) was used in testing eligible OMT samples for the presence of HIV-1 antibodies.

OMT specimens with a negative enzyme-linked immunosorbent assay (ELISA) result after initial testing were reported as negative, as were specimens that were reactive on the initial run but for which both duplicate repeat tests were negative and specimens repeatedly reactive according to ELISA but demonstrating no bands after Western blot testing. OMT specimens that were repeatedly reactive according to ELISA and met the criteria for a positive Western blot were reported as positive. OMT specimens that were repeatedly reactive after ELISA testing but did not demonstrate a banding pattern meeting the criteria for a positive Western blot were reported as indeterminate.

Statistical Analyses

Use of a diagnostic test in a survey context gives rise to 2 sources of uncertainty in estimates: measurement error because of test inaccuracy and potential sample bias. The OMT assay is a highly accurate test, with a reported sensitivity of 98.80% and a specificity of 99.86%.[9] Because the performance of the test is not known with certainty, we combined estimates from the one published study[9] on its performance with the results from Add Health to estimate prevalence, sensitivity, and specificity jointly using maximum likelihood analyses and standard adjustments for test performance. We used bootstrap resampling to calculate 95% confidence intervals[10] based on quantiles from 1000 resamples.[11]

As a national survey involving a probability sample, Add Health was designed to be used for general population inference, but some limitations should be noted. For example, the original wave I sample frame consisted only of 7th- through 12th-grade adolescents included on school registers. Also, the response rate in wave I was 78.9%, and nonrespondents in wave I were not eligible for future waves (although nonrespondents in later waves were). The impact of the school enrollment criterion on a wide range of risk-related behaviors appeared to be relatively small,[12] and, as a result of the longitudinal design, the wave III sample included several hundred high school dropouts. However, the adolescents at highest risk (e.g., runaways and homeless youth) were still not likely to be included in the survey. Our results should not be extrapolated to this group, but its absence is unlikely to have influenced our general population estimates.

Poststratification weights were developed by the Add Health research team to correct for cumulative differences in survey participation according to race, gender, and original grade level.[13] We used these weights to adjust for unbiased nonresponse in wave III. If survey participation or specimen provision was less likely among groups with higher rates of HIV infection, however, our data would still underestimate true prevalence rates. This is the most challenging type of nonresponse to address in survey contexts, but the longitudinal design of Add Health provided a unique opportunity to do so by comparing the characteristics of respondents who did and did not provide specimens in the third wave of the study. More than 99% of those who did not provide specimens in wave III had information available on HIV-related risk behaviors in earlier survey waves. We used this information and a sensitivity analysis to examine the potential effects of biased nonresponse on our prevalence estimates.

We used SPSS[14] in conducting descriptive analyses. We calculated prevalence estimates and confidence intervals and made sensitivity projections using the R statistical package.[15]

RESULTS

Study Population

In all, 14 322 individuals were interviewed in the wave III survey (75.7% of the eligible wave I sample), and specimens from 13 184 of the interviewed participants were available for HIV testing (92.1% of wave III participants, 69.7% of the eligible wave I sample). About one quarter of the interview nonresponse in wave III was because of individuals refusing to take part (n=1109; 6%); the remaining nonrespondents (n=3493; 18%) could not be located or were unable to participate for other reasons. Among those who participated, 769 (5.4%) refused to be tested for HIV, and 369 (2.7%) of the specimens obtained could not be processed owing to shipping or laboratory problems. Response rates were slightly higher among young

TABLE 1—Demographic Characteristics of the Add Health Wave III Sample: 2001–2002 (n = 14 322)

	No.	Unweighted %[a]	Weighted %[b]
Gender			
Men	6767	47.2	50.8
Women	7555	52.8	49.2
Race/ethnicity			
White	7741	54.0	67.5
Hispanic	2340	16.3	11.8
Non-Hispanic Black	3042	21.2	16.0
Asian American	1026	7.2	3.7
Native American	136	0.9	0.8
Age, y			
18–19	1453	10.1	12.7
20–21	4123	28.8	32.3
22–23	5520	38.5	32.1
24–25	3101	21.7	21.7
≥26	125	0.9	1.2
Region			
West	3685	25.7	17.7
Midwest	3328	23.2	28.7
South	5506	38.5	40.2
Northeast	1790	12.3	13.4

[a]Percentage in the study sample.
[b]Percentage in the target US population.

women than young men and varied modestly by race (range: 72%–78%). Although the overall age range was 18 to 28 years, 93% of the participants were between 19 and 24 years old. Table 1 shows the demographic breakdown of the sample.

HIV Prevalence

Fifteen specimens tested positive for HIV, 8 were indeterminate, and the remainder were negative. The indeterminate cases appeared to be randomly distributed in the sample and were removed from further analyses. After correction for the sensitivity and specificity of the Orasure test, the estimated overall HIV prevalence was about 1 per 1000 (Table 2), with slightly higher rates among young men and a striking racial disparity: of the 15 positive tests, 12 were among non-Hispanic Black respondents. These results correspond to estimated prevalence rates of 4.9 per 1000 among non-Hispanic Blacks and 0.2 per 1000 in the remaining population, a 20-fold gap. Overall, the estimated number of HIV-infected individuals in this age group (projected with the Add Health sample weights) is 21 400 (17 300 non-Hispanic Blacks and 4100 members of the remaining population).

To investigate whether biased nonresponse affected these estimates, we examined data from earlier waves of the study. We used 4 indicators of male same-sex attraction or activity and a question on injection drug use from wave II. We examined each question independently and as a cumulative sum. We found no significant differences between responders and nonresponders. We also constructed an index designed to capture 2 key elements of general sexual exposure: lifetime number of sex partners, reported from the first 2 waves (including both same- and opposite-sex partners), and gross variations in local HIV prevalence rates. On this index, number of partners was multiplied by a prevalence factor of 1 (rural), 2 (suburban), or 5 (urban), reflecting urban–rural prevalence differences in CDC AIDS surveillance statistics.[16]

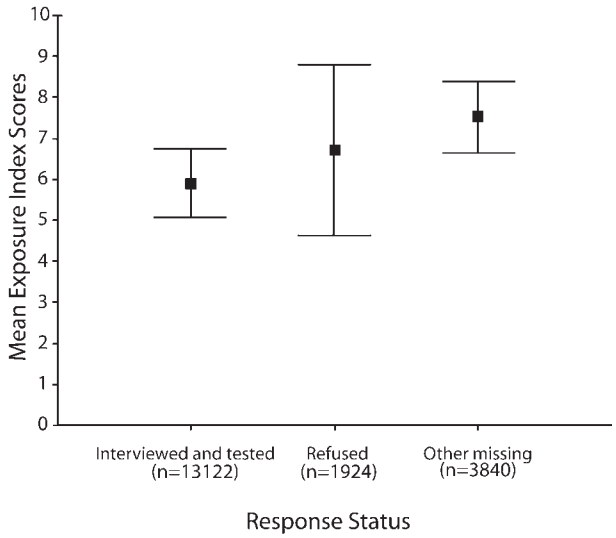
Participants who provided specimens in wave III and participants who refused (either the interview or the test) did not differ significantly in their level of potential sexual exposure to HIV (Figure 1). Potential exposure was significantly higher among those who were not included for reasons other than refusal ("other reasons" in Figure 1). The mean difference, although statistically significant, was only 21%. As an example of its potential impact, the prevalence among nonrespondents would have to have been 5 times higher than that among respondents to double our population estimate. A prevalence differential on the order of the 21% exposure difference observed here would have virtually no effect on our estimate.

We compared these findings with published estimates from other large, nationally representative data sources. The only other representative household-based survey in which HIV status was ascertained was

TABLE 2—Estimated HIV Prevalence Rates Among Young Adults: United States, 2001–2002

	Estimated Prevalence (per 1000) (95% CI)	Estimated No. of Cases (95% CI)
Gender		
Men	1.06 (0.25, 2.19)	11 954 (2 803, 24 471)
Women	0.87 (0.26, 1.63)	9 432 (2 787, 17 631)
Race/ethnicity		
Non-Hispanic Black	4.92 (1.84, 8.70)	17 275 (5 810, 30 477)
Other	0.22 (0.00, 0.64)	4 112 (0, 11 724)
Overall	0.97 (0.40, 1.70)	21 387 (8 688, 37 532)

Note. CI = confidence interval.



Note. Shown are means and 95% confidence intervals (CIs) for the wave I and wave II cumulative sexual exposure indexes for 3 groups of Add Health survey participants: those who provided a usable specimen in wave III, those who participated in earlier waves but refused either the interview or the HIV test in wave III, and those who participated in earlier waves but either were not interviewed for other reasons in wave III or provided an unusable specimen. Number of sexual partners (both male and female) was multiplied by a prevalence differential factor for location (1 = rural, 2 = semiurban, 5 = urban) to estimate sexual exposure (see "Results" for details).

FIGURE 1—Add Health wave I and wave II cumulative sexual exposure index differentials, by response status in wave III.

NHANES. Both NHANES III (1988–1994) and the more recent NHANES rolling survey (1999–2002) involved wider age ranges than Add Health for the tested samples (18–59 and 18–49 years, respectively).[17,18] The data for the rolling survey can be broken down to the 19- to 24-year age range, but this limits the sample to about 1500 respondents and only one case involving an HIV-positive test; thus, we used the full sample in our comparison. The most recent back-calculation estimates based on AIDS incidence data cover an age range closer to that of Add Health (18–22 and 23–27 years), but these estimates date to 1993.[19]

Estimates from the CDC surveillance system allow for the closest match with the present data, with a similar age range and time (20–24 years in 2001) as well as an age range selection allowing for a comparison with NHANES (20–59-year-olds).[5] At that time, the system excluded a number of important areas, including California, and had included New York for only 1 year. The CDC system is designed to measure incident rather than prevalent cases of HIV, so we constructed the standard proxy for prevalent HIV infections by summing cumulative HIV infections and AIDS cases and subtracting cumulative AIDS deaths. Because information on degree of population coverage was not available, we did not attempt to calculate prevalence rates from the CDC data.

Given the fundamental differences in the sources of data we used in our comparisons, the degree of consistency in the total numbers of cases estimated among young adults was surprising (Table 3). Both the back-calculation and CDC estimates for this population were within the confidence interval for the Add Health estimate, although the Add Health estimate itself was about 50% lower. The NHANES estimates, derived from a substantially wider age range, were naturally much higher than those of Add Health, but they were similar in magnitude to the CDC estimate for that age range.

Group-specific estimates were mixed: These estimates were consistent for women and non-Hispanic Blacks but less consistent for men and individuals from other racial/ethnic groups. The Add Health, back-calculation, and CDC estimates for women were very close, in terms of both numbers of cases and prevalence rates. This was the case as well among non-Hispanic Blacks. Among men, however, the Add Health estimates were about half the comparable back-calculation and CDC estimates, and the number of cases estimated for individuals in the Add Health "other" race/ethnicity category was less than one quarter the number obtained from the other data sources. In Add Health, the race gap in HIV prevalence rates was estimated to be approximately 20:1 (Table 3). The next closest estimate, about 10:1, was derived from the NHANES rolling survey, and the back-calculation estimate for a decade earlier was approximately 5:1.

DISCUSSION

The Add Health results suggest that the prevalence rate of HIV among young adults in the United States is approximately 1 per 1000. Rates of infection are slightly higher among young men than among young women, and there are large differences between non-Hispanic Blacks and members of other racial/ethnic groups. The Add Health estimates were lower than those of other data sources for men and for members of racial/ethnic groups other than non-Hispanic Black.

How well did this survey capture the true prevalence rate among young adults? Although the number of HIV cases in the Add Health sample was too small to support a multivariate analysis of HIV determinants, the substantial amount of data available from the large overall sample allowed for evaluation of possible biases and sources of error in estimates of prevalence rates.

Diagnostic test performance characteristics, for example, were unlikely to have compromised the findings reported here. Given the low overall prevalence rate in this sample, any bias in estimates would be dominated by the specificity (rather than the sensitivity) of the HIV test, and the effect would be to inflate prevalence estimates with false positives. The Add Health estimates, however, do not appear to be too high, suggesting that the test's performance had little impact.

TABLE 3—Comparisons of Add Health HIV Prevalence Estimates With Estimates From Other Data Sources

	Add Health: 2001–2002 (19–24-Year-Olds[a]) (95% Confidence Interval)	Back Calculation: 1993		CDC Surveillance: 2001		NHANES: 1988–1994 (18–59-Year-Olds)	NHANES: 1999–2002 (18–49-Year-Olds)
		18–22-Year-Olds	23–27-Year-Olds	20–24-Year-Olds	20–59-Year-Olds		
No. of cases							
Total	21 387 (8 688, 37 532)	33 100	116 000	36 252	537 106	461 000	540 060
Male	11 954 (2 803, 24 471)	22 100	88 500	23 494	413 153	368 000	402 002
Female	9 432 (2 787, 17 631)	11 000	27 500	12 758	123 953	94 000	138 058
Non-Hispanic Black	17 275 (5 810, 30 477)	16 000	49 200	18 179	238 193	189 000	319 387
Other race/ethnicity	4 112 (0, 11 724)	17 100	66 800	18 073	298 913	273 000	220 672
Prevalence per 1000							
Total	0.97 (0.40, 1.70)	1.98	6.54	. . .	. . .	3.20	4.26
Male	1.06 (0.25, 2.19)	2.60	9.92	. . .	. . .	5.20	6.40
Female	0.87 (0.26, 1.63)	1.34	3.12	. . .	. . .	1.30	2.16
Non-Hispanic Black	4.92 (1.84, 8.70)	6.42	19.99	. . .	. . .	11.00	21.37
Other race/ethnicity	0.22 (0.00, 0.64)	1.20	4.37	. . .	. . .	2.11	1.97

Note. CDC = Centers for Disease Control and Prevention; NHANES = National Health and Nutrition Examination Survey.
[a]The full sample age range was 18 to 28 years, but 93.2% of the participants were 19 to 24 years of age.

Random sampling variability was also unlikely to have played a major role in the findings described here because, although the number of cases observed was small, the overall sample size was quite large. If the true prevalence rate were actually 2 in 1000 (twice our estimate), the odds of drawing a sample such as ours at random would be approximately 1000 to 1. Factoring in the probable geographic clustering of HIV and the full initial clustering of the wave I sample, the odds might rise to 2 in 100. The cluster sampling effect was likely to be smaller, however, in that nearly 70% of participants had moved since their first interview.

This leaves the question of systematic biases in the sample population. Several empirical indicators suggest that the Add Health wave III sample was not systematically biased with respect to potential HIV exposure. These indicators include longitudinal evidence that wave III participants were similar to nonparticipants on a broad range of HIV-related risk behaviors during their teenage years, the fact that rates of other sexually transmitted infections in the wave III sample were consistent with existing estimates,[20,21] and the fact that the estimates for non-Hispanic Blacks—the sample subgroup most likely to be affected by underrepresentation of high-risk incarcerated populations—were comparable to those derived from other data sources.

School dropouts were not included in the original sample frame, but those who dropped out of school between wave I (1995) and wave III (10 years later) were followed, and thus several hundred dropouts were included in the sample described here. Finally, although our sample slightly overrepresented the South and underrepresented the other three study regions, the effect on our overall prevalence estimates was likely to be small. Recent CDC data suggest that the South and Northeast are the epicenters of HIV infection among young people.[22] If this is true, then the present regional imbalance might have had little impact, because the Add Health sample percentage in these 2 regions combined (50%) was very close to the population percentage (49% in the 2000 census).

The preceding does not mean that nonresponse in the Add Health survey was entirely unbiased with respect to HIV. Other mechanisms might have been at work, and the previous wave information used here was gathered 5 years earlier and thus may not have reflected respondents' risk status at wave III. Taken together, however, our data suggest that if the Add Health wave III sample estimates for men in the "other" race/ethnicity category are too low, the mechanism is specific to HIV status and race. On balance, our results indicate that population-based surveys can be used to obtain estimates of national HIV prevalence rates, even in low-prevalence settings. As back calculation becomes less reliable and HIV testing methods become simpler, population-based survey data can become a reliable means of HIV surveillance.

Beyond surveillance, the findings reported here have 2 implications for understanding the HIV epidemic in the United States. First, both the Add Health–NHANES comparison and the CDC estimates shown in Table 3 imply that there are roughly 20 times more cases among 20- to 59-year-olds than among individuals younger than 25 years. The size of this differential is not consistent with the widely cited estimate that half of all infections occur among those younger than 25 years.[3]

If we take Table 3's Add Health and CDC estimates among young adults as a constant baseline and project them forward, assuming an exponential 12-year average survival period (12 years is the value used in the Rosenberg estimate[8]), then a rough approximation of the equilibrium number (i.e., the point at which the number of infections among those younger than 25 years and the number

among those aged 20–59 years are constant) of individuals 20 to 59 years old who contracted HIV before the age of 25 years would be 80 000 to 160 000 (according to the Add Health and CDC estimates, respectively). Comparing this estimate with the CDC total of about 540 000 HIV infections among 20- to 59-year-olds (Table 3) suggests that only 15% to 30% of infections are acquired before the age of 25 years. Raising the average survival time to 30 years would increase the estimated percentage of individuals contracting HIV before 25 years of age to 20% to 45% at equilibrium.

In both survival scenarios, however, approximately 35 years is required to reach equilibrium, and before that the estimated percentage of individuals contracting HIV before the age of 25 years is substantially lower. This difference in estimates could reflect real changes in the age distribution of HIV incidence in the past 10 years, or it might be the result of changes in measurement of HIV and AIDS. Given that it has implications for targeting prevention efforts, this topic deserves more study.

Second, our findings show that the well-known racial disparities in HIV infection in the United States are established early in the life span. Although our estimate of a 20-fold prevalence gap between non-Hispanic Blacks and members of other racial/ethnic groups appears high relative to the back-calculation estimates shown in Table 3, it is consistent with the 19-fold estimate based on CDC data on incident cases in 2000.[5] It also parallels the large racial differentials evident in the prevalence rates of other sexually transmitted infections: In 2000, rates of gonorrhea were 24 times higher among non-Hispanic Black youths than among White youths, rates of syphilis were 30 times higher,[23] and rates of chlamydia[23] and herpes simplex virus type 2[24] exhibited significant (although smaller) racial disparities.

Race is likely to be a proxy for a number of factors that influence risk exposure, including discrepancies in health care access, differences in sexual behavior, genetic differences, and the structure of partnership networks. The relative importance of each of these factors in determining racial disparities in HIV prevalence rates is gradually becoming clear. The stability of race differentials has been shown to persist after control for socioeconomic status[25] and number of sexual partners,[26] remaining strong even in the case of young people who enter juvenile detention facilities[27] and inject drugs.[28]

This evidence suggests that, although poverty and behavioral differentials may matter, they do not account alone for the race disparities observed in HIV prevalence rates. There has been no proposed mechanism by which genetic differences might influence susceptibility or infectivity, and there is no current evidence pointing to such a mechanism. The mechanism of sexual networks, by contrast, is both conceptually clear and empirically established: People are infected by their partners, who in turn are infected by their partners. "Networks" is simply the term that refers to these direct and indirect connections that sustain transmission.[29]

Recent studies have shown that sexual networks tend to display a pattern of "assortative mixing," segregating along race, class, and geographic lines. Among non-Hispanic Blacks, race, poverty, and geographic clustering result in higher prevalence rates of sexually transmitted diseases among potential partners,[30] raising the probability of exposure to infection.[31] When partnerships overlap in time—a pattern labeled "concurrency"—the connectivity of a sexual network increases, and transmission of sexually transmitted infections is amplified.[32–34] Concurrency is more common among non-Hispanic Blacks than among other groups,[35,36] so this may further amplify the spread of infection in these relatively segregated networks.

In summary, although the prevalence of HIV among young adults in the United States appears to be relatively low, the burden is inequitably distributed by race. The cumulative body of research over the past decade suggests that the pervasive racial differentials in rates of sexually transmitted infections are induced by the structure of partnership networks: Assortative mixing segregates networks according to race, geography, and class, concentrating the effects of both economic disadvantage and concurrent partnerships.

The implications for HIV research and prevention are clear. We need to better understand the ways in which networks influence the transmission dynamics of HIV, given that small differences in behavior can have large (nonlinear) effects on network connectivity. Moreover, because people's risk depends on the behavior of their partners, we need to better understand the relational context of HIV risk behavior; individual knowledge and attitudes are only part of the solution. Unless and until a sterilizing HIV vaccine becomes available, reducing disparities in HIV prevalence rates—both within and between countries—requires a better understanding of the links between individual behavior, partnership dynamics, and transmission networks. ■

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Contributors

M. Morris contributed to the analytic strategy, descriptive tabulations, and the writing of the article. M.S. Handcock contributed to statistical analysis and programming for estimates of prevalence. W.C. Miller and M.M. Hobbs provided statistical consultation on test performance and interpretation. C.A. Ford designed the biomarker survey and notification protocol. J.L. Schmitz supervised the Orasure OMT laboratory testing. M.S. Cohen selected the biomarkers. K.M. Harris was the deputy director for Add Health wave III data management. J.R. Udry was the study director for Add Health.

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Human Participant Protection

This study was approved by the institutional review boards of the University of North Carolina and the

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