



## Original article

## Examining neighborhood poverty-based disparities in HIV/STI prevalence: an analysis of Add Health data



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## ABSTRACT

**Purpose:** The purpose of the study was to estimate the effect of exposure to neighborhood poverty in adolescence on HIV/STI prevalence in early adulthood.

**Methods:** Longitudinal data from three waves of the National Longitudinal Study of Adolescent to Adult Health were analyzed. The primary exposure was living in a high- versus medium/low-poverty neighborhood during wave I. The outcome was having a sexually transmitted infection (STI) or receiving a HIV/STI diagnosis in the past 12 months at wave III. Covariates included sociodemographic, behavioral, and mental health-related factors. Inverse probability weighted marginal structural models were used to estimate neighborhood poverty-based differences in HIV/STI prevalence.

**Results:** The analytic sample comprised 8232 National Longitudinal Study of Adolescent to Adult Health participants. Of these, 16% and 84% resided in high- and medium/low-poverty neighborhoods, respectively. Eleven percent currently had an STI or HIV/STI diagnosis within the prior 12 months. Accounting for measured potential sources of confounding and selection bias, the HIV/STI prevalence difference (95% confidence limits) for those who grew up in high- versus medium/low-poverty neighborhoods was 0.015 (−0.015, 0.045).

**Conclusions:** Strong evidence for neighborhood poverty-based differences in HIV/STI prevalence was not observed. Researchers should continue to investigate the effect of neighborhood-level socioeconomic position measures and, if warranted, identify etiologically relevant exposure periods.

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## Introduction

In the United States, half of all sexually transmitted infections [1] (STIs) and one-fifth of HIV diagnoses [2] occur among adolescents and young adults. Furthermore, diagnosis rates for chlamydia and gonorrhea are highest in this age demographic [3]. The considerable HIV/STI burden experienced by adolescents and young adults drives recent interest in contextual factors, including neighborhood environments, that may influence HIV/STI outcomes in adolescents and young adults [4–10].

Neighborhood-level characteristics such as socioeconomic disadvantage, social cohesion, and sexual norms might influence the occurrence of STIs and/or health-risk behaviors in adolescents or young adults [11–16]. Specifically, neighborhood disadvantage between ages 12 and 16 years has been associated with higher frequency of sexual activity, having more sexual partners, and unprotected sex from ages 18–22 years [14]. Meanwhile, neighborhood differences in crime, availability of services, and sexual networks are potential determinants of disparate background HIV risk in a range of populations [17]. Research also links neighborhood poverty to both sexual behavior [18] and STI outcomes, including repeat chlamydia infection in predominantly young women [19] and trichomoniasis prevalence in young adults [20].

Prior research using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) suggests neighborhood poverty and community socioeconomic disadvantage in childhood influence STI prevalence in young adulthood [6,8]. However, this prior work conditioned on factors (e.g., economic hardship and employment [8]; depression and condom use [6] that were plausibly influenced by neighborhood-level exposures, using methods where such conditioning may have introduced selection bias [21]. Alternative nonstratification-based techniques can minimize confounding and selection bias without blocking pathways of interest or introducing additional selection bias [22]. Thus, we sought to estimate the effect of exposure to neighborhood-level poverty in adolescence on HIV/STI prevalence in young adulthood using a nonstratification-based technique (i.e., marginal structural model) to minimize bias without blocking pathways of interest or introducing additional bias.

## Methods

### Data source

Data from waves I (1994–1995), II (1996), and III (2001–2002) of the Add Health study [23] were used for this secondary data analysis. Add Health is a cohort study investigating how social environments and behaviors in adolescence are linked to health and achievement outcomes. To aid in examining social environments, census-based data (e.g., Census block group-level variables) are included in the Add Health data set.

Systematic unequal probability sampling with implicit stratification of 80 high schools and 52 middle schools was performed to ensure that the Add Health sample is nationally representative with respect to region, urbanicity, school size/type, and ethnicity. To ensure inclusion of minority groups, all of the following racial/ethnic groups were oversampled in wave I based on self-reported data: African-Americans from well-educated families (having a parent with a college degree), Chinese, Cubans, and Puerto Ricans. More detail on the Add Health study design has been provided elsewhere [24,25]. This secondary data analysis was approved by the Brown University Institutional Review Board.

### Analytic sample

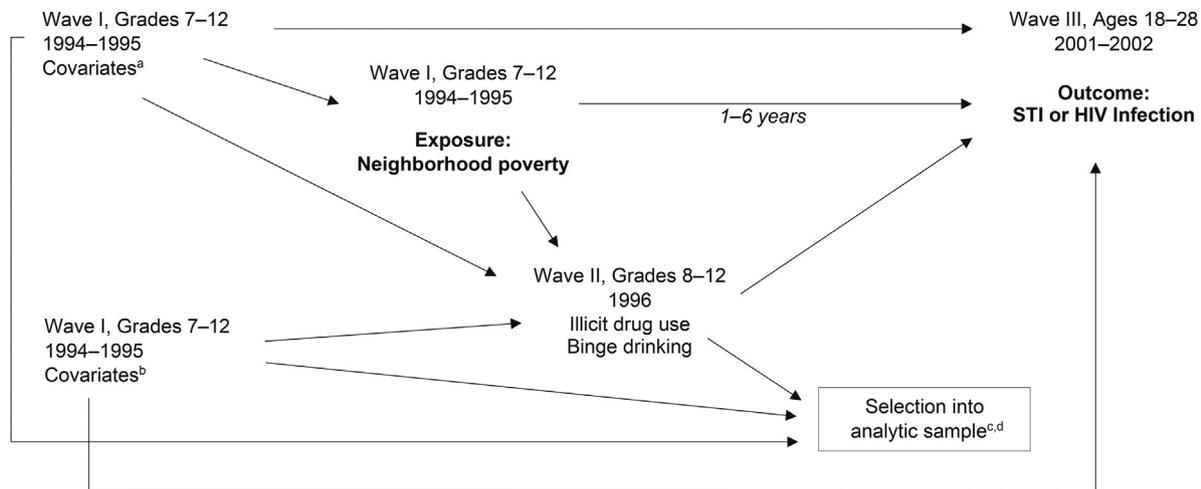
The analytic sample included 8232 Add Health respondents in the restricted-use data set with complete information on the exposure, outcome, and other variables of interest from waves I–III and who met other criteria (Fig. S1). Differences on measured factors between eligible respondents in the final analytic sample (8232 of 18,924, 43.5%) and those excluded for not being interviewed in wave III or having missing outcome data (1954 of 18,924, 10.3%) were accounted for using stabilized inverse probability-of-selection (IPS) weights estimated as a function of covariates assessed during waves I and II [21]. Differences on measured factors between respondents in the final analytic sample and those excluded for not being interviewed for waves II or III or missing covariate (wave II) or outcome data (6286 of 18,924, 33.2%) were accounted for in sensitivity analyses using stabilized IPS weights estimated as a function of covariates assessed during wave I. Missing necessary wave I information on neighborhood or covariates precluded using inverse probability weights to account for differences in measured factors between those included in the final analytic sample and all respondents excluded for reasons other than minority sexual orientation, not being in school, or being in a school without grade levels (9767 of 18,924, 51.6%).

### Conceptual framework

Figure 1 shows a causal diagram for the hypothesized influence of neighborhood poverty in adolescence on STI or HIV prevalence in early adulthood in the Add Health study population. The diagram was informed by the Add Health study design and prior literature [13,14,26–35]. The diagram assumes exposure to neighborhood poverty in adolescence affects later HIV/STI acquisition indirectly via illicit drug use and binge drinking, whereas its effects through all other pathways (e.g., sexual behavior, sexual network characteristics) comprise the direct effect. Illicit drug use and binge drinking explicitly appear on the diagram because unlike other measured factors on the pathway between neighborhood poverty and HIV/STI, drug use and binge drinking are assumed to affect selection into the analytic sample in addition to the outcome, which results in the potential for selection bias [36].

### Exposure

The exposure of interest was neighborhood poverty, ascertained using available Census block group-level information based on a participant's residential address at a single time point during wave I (i.e., grades 7–12). Census block group is the finest available geographic unit with reliable contextual data on socioeconomic position [37]. We examined neighborhood poverty as a binary indicator of living in a high- versus a medium- or low-poverty neighborhood. A secondary analysis examined neighborhood poverty as a three-level variable. High-poverty neighborhoods were those where >23.9% of residents had an income in 1989 below the federal poverty level. Medium-poverty neighborhoods were neighborhoods where between 11.6% and 23.9% of residents were living below the federal poverty level. Low-poverty neighborhoods were neighborhoods where less than 11.6% of residents were living below the federal poverty level. We based poverty cutoffs on Add Health's categorization of neighborhood poverty, consistent with prior work [38–42]. Furthermore, the selected high-poverty threshold is close to the U.S. Census Bureau's "poverty area" definition (20%–40% of residents below the federal poverty line) [43,44].



<sup>a</sup> Covariates include race/ethnicity, US-born, parental education, two-parent household, household receipt of welfare, and alcohol or illegal drug availability in the household.

<sup>b</sup> Covariates include birth year, sex, suicidal ideation, and vaginal sexual intercourse.

<sup>c</sup> Selection weights in the primary analysis were used to weight the analytic sample accounting for potential selection bias induced by excluding observations who were not interviewed or had missing outcome data for Wave III. Weights were estimated as a function of sex, race/ethnicity, US-born status, birth year, alcohol or illegal drug availability in household, suicidal ideation, parental education, two-parent household, household receipt of welfare, and vaginal sexual intercourse in Wave I, as well as illicit drug use and binge drinking in Wave II. Weights were stabilized by the marginal probability of being selected into the analytic sample based on the availability of Wave III data.

<sup>d</sup> Selection weights in the first sensitivity analysis were used to weight the analytic sample accounting for potential selection bias induced by excluding observations who were not interviewed in Waves II or III or had missing data on illicit drug use or binge drinking (Wave II) or HIV/STI (Wave III). Weights were estimated using the same covariates listed in footnote <sup>a</sup> but excluding illicit drug use and binge drinking in Wave II. Weights were stabilized by the marginal probability of being selected into the analytic sample based on the availability of Wave II or III data.

Note: For simplicity, only measured covariates appear in the diagram. Unmeasured covariates are excluded.

**Fig. 1.** Causal diagram for the influence of neighborhood poverty in adolescence on STI or HIV infection in early adulthood within the National Longitudinal Study of Adolescent to Adult Health (Add Health) waves I–III study population, 1994–2002.

## Outcome

Based on the existing literature [28], the primary outcome was receipt of an HIV/STI diagnosis in the past 12 months or current STI at a single time point in wave III (i.e., ages 18–28). This outcome was operationalized as a binary variable indicating either (1) biologically confirmed current infection with chlamydia, gonorrhea, or *Trichomonas vaginalis*, as indicated by urine specimen testing or (2) self-reported diagnosis in the past 12 months by a medical professional of chlamydia, gonorrhea, *T. vaginalis*, syphilis, genital herpes, genital warts, human papillomavirus, pelvic inflammatory disease, cervicitis or mucopurulent cervicitis, urethritis, vaginitis, another STI, or HIV/AIDS.

## Covariates

Covariates were selected based on Figure 1 and included the self-reported factors collected in wave I that are listed in the footnote of Figure 1, as well as self-reported illicit drug use and binge drinking collected in wave II. The wave I survey did not include questions on anal or oral sex. Illicit drug use at wave II captured self-reported use of marijuana, inhalants, cocaine, illegal drugs, or injection drugs between waves I and II. Binge drinking in wave II ascertained whether an individual had five or more drinks in a row on at least one day within the past 12 months. Covariates were included in all relevant models as categorical variables using one or more indicator(s).

## Statistical analysis

Measured characteristics of included and excluded respondents were examined and compared using  $\chi^2$  tests based on the survey-weighted data. The survey weights were provided by Add Health

and correspond to the wave I sampling design, including oversampling of specific groups. To apply the below mentioned approach by Miglioretti and Heagerty [45], we did not use the poststratification variable in any of the models. Per Add Health documentation [46], omitting this variable would be expected to produce negligible changes in the standard errors.

Next, wave I survey-weighted (unadjusted) and stabilized inverse probability weighted (IPW) marginal structural models [22,29,47] specified with the identity link function and binomial distribution were used to obtain prevalence differences and 95% confidence limits (CLs) for the relationship between neighborhood poverty in adolescence and HIV/STI prevalence in early adulthood. All unadjusted and stabilized IPW marginal structural models that examined the relationship of interest accounted for clustering by middle or high school, which was the primary sampling unit for waves I–III, and by Census tract or block numbering area, using an approach by Miglioretti and Heagerty [45] that accounts for the non-nested clustering of the data. Census block groups were assumed to be nested within Census tracts or block numbering areas [48]. All stabilized IPW marginal structural models minimized sources of confounding and selection bias related to the earlier described measured covariates.

Stabilized IPW marginal structural models that examined the relationship between neighborhood poverty and HIV/STI prevalence were fit using a combined weight. The combined weight was derived by multiplying stabilized inverse probability-of-neighborhood-and-selection weights by the wave I grand sample weights [49]. The stabilized inverse probability-of-neighborhood-and-selection weights were calculated by multiplying stabilized inverse probability-of-neighborhood weights and the earlier mentioned stabilized IPS weights. The stabilized inverse probability-of-neighborhood weights and stabilized IPS weights were estimated in the wave I survey-weighted data as a function of

**Table 1**

Individual- and neighborhood-level characteristics of respondents by inclusion in final analytic sample, National Longitudinal Study of Adolescent to Adult Health, waves I–III 1994–2002

Individual-level characteristics, wave I <sup>†</sup> Grades 7–12, 1994–1995	Included (n = 8232)	Excluded <sup>†</sup> (n = 1954)	P-value <sup>‡</sup>
Biological sex, n (%)			
Male	3796 (47.2)	1065 (56.3)	<.001
Female	4436 (52.8)	889 (43.7)	
Race/ethnicity, n (%)			
White	4820 (73.3)	1070 (67.2)	.002
Black	1508 (11.8)	389 (15.5)	
Hispanic	1205 (9.9)	350 (12.7)	
Asian	579 (3.7)	113 (3.2)	
Other (American Indian, multirace)	120 (1.4)	32 (1.4)	
U.S. born, n (%)			
Born within the United States	7622 (94.9)	1753 (92.6)	<.001
Born outside the United States	610 (5.1)	201 (7.4)	
Birth year, n (%)			
1974–1976	198 (2.1)	37 (1.4)	<.001
1977	834 (8.0)	239 (10.9)	
1978	1861 (18.0)	453 (18.6)	
1979	1801 (19.1)	478 (21.4)	
1980	1536 (20.7)	355 (20.2)	
1981	1202 (19.2)	251 (18.8)	
1982 and later	800 (12.9)	141 (8.6)	
Two-parent household, n (%)			
Two-parent household	5181 (65.1)	1016 (53.8)	<.001
Single-parent or other household	3051 (34.9)	938 (46.2)	
Maternal education, n (%)			
Less than high school	1208 (13.7)	316 (17.0)	.013
High school degree or equivalent	2823 (37.1)	684 (37.8)	
More than high school, no college degree	1613 (20.4)	403 (20.8)	
College degree	2588 (28.8)	551 (24.4)	
Paternal education, n (%)			
Less than high school	1300 (15.1)	326 (16.3)	.120
High school degree or equivalent	2784 (35.4)	689 (37.7)	
More than high school, no college degree	1456 (17.7)	350 (17.8)	
College degree	2692 (31.8)	589 (28.2)	
Receipt of welfare by father or mother, n (%)			
Yes	640 (7.2)	215 (11.7)	<.001
No	7592 (92.8)	1739 (88.3)	
Alcohol easily available in household, n (%)			
Yes	2543 (31.0)	566 (28.8)	.211
No	5689 (69.0)	1388 (71.2)	
Illegal drugs easily available in household, n (%)			
Yes	222 (2.5)	51 (2.7)	.732
No	8010 (97.5)	1903 (97.3)	
Suicidal ideation in past year, n (%)			
Yes	1043 (12.9)	233 (12.1)	.479
No	7189 (87.1)	1721 (87.9)	
Has ever had vaginal sexual intercourse, n (%)			
Yes	2554 (27.6)	669 (33.3)	<.001
No	5678 (72.4)	1285 (66.7)	
Neighborhood-level characteristics, wave I <sup>†</sup> Grades 7–12, 1994–1995			
Proportion persons in Census tract living below poverty level, n (%)			
Low (<11.6%)	5194 (62.0)	1188 (57.7)	.023
Medium (11.6%–23.9%)	1641 (21.8)	401 (21.7)	
High (>23.9%)	1397 (16.2)	365 (20.7)	
Individual-level characteristics, wave II <sup>†</sup> Grades 8–12, 1996			
Illicit drug use since wave I interview, n (%)			
Yes	2113 (25.6)	536 (28.8)	
No	6119 (74.4)	1418 (71.2)	.031

**Table 1 (continued)**

Individual-level characteristics, wave I <sup>†</sup> Grades 7–12, 1994–1995	Included (n = 8232)	Excluded <sup>†</sup> (n = 1954)	P-value <sup>‡</sup>
Binge drinking since wave I interview, n (%)			
Yes	2277 (28.9)	589 (31.7)	
No	5955 (71.1)	1365 (68.3)	.148
Individual-level characteristics, wave III <sup>†</sup> Ages 18–28, 2001–2002			
HIV/STI in past year, n (%)			
Yes	979 (11.2)	n/a	n/a
No	7253 (88.8)		
Biologically confirmed STI <sup>§</sup> , n <sup>  </sup> (%)			
Yes	434 (5.3)	n/a	n/a
No	6812 (94.7)		

n/a = not applicable.

\* Counts and column percentages presented. Counts are unweighted, whereas percentages were calculated based on wave I survey weights that account for the complex survey design.

† 1954 respondents excluded because of not being interviewed or missing outcome data for wave III.

‡  $\chi^2$  analysis “included” versus “excluded” based on weighted counts.

§ Gonorrhea, chlamydia, or *Trichomonas vaginalis* as assessed by urinalysis.

|| n = 7246.

the following covariates: race/ethnicity, U.S.-born status, parental education, living in a two-parent household, household receipt of welfare, alcohol or illegal drug availability in the household, birth year, sex, vaginal sexual intercourse, and suicidal ideation at wave I, plus illicit drug use and binge drinking at wave II.

A secondary analysis examined the influence of neighborhood poverty as a three-level variable (low-, medium-, and high-poverty neighborhood). We also conducted two sensitivity analyses: (1) an analysis using alternative stabilized selection weights to account for differences in measured factors between included respondents and respondents excluded because of not being interviewed for waves II or III or having missing covariate (wave II) or outcome data, as described previously, and (2) an analysis using only biologically confirmed STI (chlamydia, gonorrhea, or *T. vaginalis*) as the outcome variable.

Logistic regression models were used to estimate all stabilized inverse probability weights. Estimated stabilized inverse probability weights were evaluated based on their distributions as recommended by Cole and Hernán [50] and appeared to be well behaved (Tables S1–S4). Statistical analyses were performed in SAS Version 9.4 (SAS Institute, Inc., Cary, NC). A two-sided  $\alpha = 0.05$  was specified, and 95% CLs were calculated.

## Results

### Characteristics

Individual- and neighborhood-level characteristics of 8232 respondents in the final analytic sample and 1954 respondents with complete information for waves I and II but who were excluded for missing wave III information are presented in Table 1. Add Health respondents in the final analytic sample were mostly white, U.S.-born, and in grades 7–10 at wave I. Approximately 16%, 22%, and 62% of analytic sample participants lived in high-, medium-, and low-poverty neighborhoods in wave I, respectively. At wave II (grades 8–12), 25.6% reported having used illicit drugs since wave I, and 28.9% reported binge drinking within the past 12 months; by wave III (ages 18–28), 11.2% either had an STI or reported a recent HIV/STI diagnosis. Evidence supporting differences in

**Table 2**

Survey and stabilized inverse probability weighted prevalence differences for HIV/STI diagnosis in early adulthood, comparing high- to medium/low-poverty neighborhoods in adolescence, National Longitudinal Study of Adolescent to Adult Health, waves I–III 1994–2002 ( $n = 8232$ )<sup>\*</sup>

Analysis of binary poverty	Prevalence difference in HIV/STI diagnosis	95% CL
Primary analysis		
Unadjusted	0.096	0.055, 0.137
IPW MSM <sup>†,‡</sup>	0.015	−0.015, 0.045
First sensitivity analysis		
IPW MSM <sup>†,§</sup>	0.026	−0.013, 0.065

CL = confidence limits; IPW MSM = inverse probability weighted marginal structural model.

<sup>\*</sup> 8232 is the unweighted sample size, whereas survey and inverse probability weighted populations were used for analyses.

<sup>†</sup> Accounts for potential confounding bias related to race/ethnicity, U.S.-born status, two-parent household, parental education, household receipt of welfare, and alcohol or illegal drug availability in household (wave I) via inverse probability weighting. The inverse probability weights were stabilized by the marginal probability of the observed neighborhood poverty level within the analytic sample.

<sup>‡</sup> Accounts for potential selection bias related to sex, race/ethnicity, U.S.-born status, two-parent household, parental education, household receipt of welfare, alcohol or illegal drug availability in household, birth year, suicidal ideation, vaginal sexual intercourse (wave I), and illicit drug use and binge drinking at wave II via inverse probability weighting. The inverse probability weights were stabilized by the marginal probability of being selected into the analytic sample based on the availability of wave III data.

<sup>§</sup> Accounts for potential selection bias related to all factors mentioned in footnote<sup>†</sup> via inverse probability weights excluding illicit drug use and binge drinking at wave II because illicit drug use and binge drinking information was not available for all participants used to estimate the corresponding selection weights. The inverse probability weights were stabilized by the marginal probability of being selected into the analytic sample based on the availability of wave II or III data.

sociodemographic and behavioral factors between individuals excluded from the final analytic sample for missing wave III information and included respondents was observed.

#### Marginal structural models in primary analyses

In the unadjusted analysis, the difference in HIV/STI prevalence (95% CLs) was 0.096 (0.055, 0.137), comparing respondents who resided in high-poverty neighborhoods to those in medium/low-poverty neighborhoods (Table 2). On accounting for confounding and selection bias, the corresponding quantities for the marginal structural model primary analysis were 0.015 (−0.015, 0.045) (Table 2).

#### Marginal structural models in secondary and sensitivity analyses

A secondary analysis treating neighborhood poverty as a three-level exposure found similar results to the primary analysis that accounted for confounding and selection bias. Specifically, in secondary analyses accounting for confounding and selection bias using a marginal structural model, the difference in HIV/STI prevalence was 0.018 (−0.013, 0.049) and 0.014 (−0.013, 0.042) comparing respondents who resided in high- versus low-poverty and medium- versus low-poverty neighborhoods, respectively (Table 3). Findings were similar in the sensitivity analysis that used alternative selection weights (Table 2) or biologically confirmed STI as the outcome variable (Table S5), compared with the findings from the primary marginal structural model analysis.

## Discussion

Our unadjusted findings suggest that those residing in poorer neighborhoods in adolescence are more likely to have an STI or HIV in early adulthood. However, accounting for potential confounding and selection bias indicated that there was not strong evidence to suggest that neighborhood poverty in adolescence influenced HIV/

**Table 3**

Survey and stabilized inverse probability weighted prevalence differences for HIV/STI diagnosis in early adulthood as a function of three-level neighborhood poverty in adolescence, National Longitudinal Study of Adolescent to Adult Health, waves I–III 1994–2002 ( $n = 8232$ )<sup>\*</sup>

Secondary analysis	Prevalence difference in HIV/STI diagnosis	95% CL
Unadjusted		
High versus low poverty	0.103	0.062, 0.144
Medium versus low poverty	0.025	−0.002, 0.052
IPW MSM <sup>†,‡</sup>		
High versus low poverty	0.018	−0.013, 0.049
Medium versus low poverty	0.014	−0.013, 0.042

CL = confidence limits; IPW MSM = inverse probability weighted marginal structural model.

<sup>\*</sup> 8232 is the unweighted sample size, whereas survey and inverse probability weighted populations were used for analyses.

<sup>†</sup> Accounts for potential confounding bias related to race/ethnicity, U.S.-born status, two-parent household, parental education, household receipt of welfare, and alcohol or illegal drug availability in household (wave I) via inverse probability weighting. The inverse probability weights were stabilized by the marginal probability of the observed neighborhood poverty level within the analytic sample.

<sup>‡</sup> Accounts for potential selection bias related to sex, race/ethnicity, U.S.-born status, two-parent household, parental education, household receipt of welfare, alcohol or illegal drug availability in household, birth year, suicidal ideation, vaginal sexual intercourse (wave I), and illicit drug use and binge drinking at wave II via inverse probability weighting. The inverse probability weights were stabilized by the marginal probability of being selected into the analytic sample based on the availability of wave III data.

STI prevalence in young adulthood. Neither restricting the outcome to biologically confirmed chlamydia, gonorrhea, or *T. vaginalis* nor considering alternative weights for selection into the analytic sample qualitatively altered findings that accounted for confounding and selection bias.

Our findings that account for potential confounding and selection bias conflict somewhat with prior work conducted in Add Health [6–8]. Whereas our study did not find strong evidence that neighborhood poverty in adolescence influenced HIV/STI prevalence in young adulthood, Ford and Browning [8] found that exposure to concentrated neighborhood poverty in adolescence (wave I) was positively associated with chlamydia infection during young adulthood (odds ratio = 1.23, 95% CL: 1.06, 1.42). Likewise, Wickrama et al. [6] found evidence of a positive association between higher values on a childhood community disadvantage index and a greater number of recent HIV/STI diagnoses reported in young adulthood, interpreting their finding as the direct effect of community disadvantage operating through pathways other than the sexual, behavioral, and psychological mediators included in their model. In a study that agreed qualitatively with our findings, grouping adolescents by school as a proxy for their community membership, Harling et al. [7] did not find strong evidence that structural inequality and relative deprivation during wave I were associated with STIs in waves II or III after adjusting for individual- (e.g., race/ethnicity) and school-level (e.g., urbanicity) factors unlikely to have been influenced by the exposures.

Observed discrepancies across studies may emanate from different operational definitions of neighborhood-level exposures or HIV/STI outcomes [6,8]. We created a combined HIV/STI variable in our primary analysis, using both self-reported HIV/STI and biologically confirmed STI. Ford and Browning [8] modeled urinalysis-detected chlamydia infection; Wickrama et al. [6] used the log-transformed count of self-reported HIV/STI diagnoses. The studies by Ford and Browning [8] and Wickrama et al. [6] conditioned on variables temporally subsequent to the poverty or disadvantage exposure measured in wave I and which may have been influenced by the exposure, for example, economic hardship and employment (wave III) [8] or depression (wave II) and condom use (wave III) [6]. Such conditioning may have introduced selection bias [22].

Our study, too, has limitations that may explain the observed discrepancies. Our approach (i.e., using stabilized IPW marginal structural models) requires that all factors related to confounding and selection bias, such as bias stemming from study exclusions, are accounted for; measurement error is negligible; all fitted models are correctly specified; and positivity holds [22,50]. However, unmeasured factors related to confounding and/or selection bias (e.g., experiencing homelessness [17]) may exist and could have impacted our findings. In addition, we could not account for potential selection bias related to exclusions for missing wave 1 exposure or covariate data.

As most of our data were self-reported, nonnegligible measurement error may exist, especially for factors involving recall of distant events or behaviors potentially considered less socially desirable. Measurement error may also affect our classification of neighborhood poverty, captured at a single time point, and may not accurately reflect respondents' exposure in adolescence. Assuming our measure did accurately reflect respondents' exposure in adolescence, neighborhood poverty in young adulthood may be the etiologically relevant time period. Furthermore, in using the proportion of individuals below the 1989 poverty line as our exposure, we focused on a single dimension of neighborhood socioeconomic position. Composite measures combining information from multiple factors (e.g., unemployment rate, female-headed households, poverty concentration), such as measures used in two of the previously discussed Add Health studies [6,8], may better capture the overall context at the neighborhood level, potentially at the expense of obscuring which constituent factors drive HIV/STI prevalence disparities.

Although the outcome variable, HIV/STIs, included both self-report and biological specimens, only three STIs (chlamydia, gonorrhea, and *T. vaginalis*) in the full restricted-use Add Health sample were ascertained using biological specimen testing. Therefore, our outcome may have missed prevalent STIs. STIs and HIV ascertained based on self-reported diagnoses—including chlamydia, gonorrhea, and *T. vaginalis*, which were assessed via both self-report and urinalysis—are subject to measurement error. However, sensitivity analyses restricted to results from solely biological specimens yielded generally similar results as the primary analysis.

Despite these limitations, our study had several strengths. The study population was nationally representative. The availability of longitudinal data enabled measurement of neighborhood poverty before the assessment of HIV/STIs and likely temporally before the occurrence of the infections of interest, particularly in analyses using only biologically confirmed STIs. Finally, we used complex longitudinal survey data and modern causal inference methods to appropriately estimate quantities of interest, which, to our knowledge, has been carried out infrequently for research questions pertaining to neighborhood effects on HIV/STI outcomes between adolescence and young adulthood in this context.

In summary, our study did not find strong evidence to support the influence of exposure to neighborhood poverty in adolescence on HIV/STI prevalence in young adulthood. Although we did not find strong evidence to support such an influence using marginal structural models that minimize confounding and selection bias without introducing additional selection bias or blocking pathways of interest, neighborhood poverty has been strongly associated with sexual network position [51]. This strong association suggests that neighborhood poverty still may play a role in the overall risk environment. However, in an analysis of adolescents' reported number of sexual partners, Carlson et al. [52] showed that composite measures such as neighborhood disadvantage may conflate the effects of distinct place characteristics; the same might be true for composite HIV/STI outcomes. Although neighborhoods and

sexual networks are closely intertwined—and together produce environments that place adolescents and young adults at risk for HIV/STI acquisition—exposure in adolescence to neighborhood poverty itself may not exert a strong influence on prevalence of HIV/STI in young adulthood. Thus, examining the role of neighborhood-level factors in HIV/STI outcomes among adolescents and young adults remains worthy of further investigation.

Given the limitations of existing work, such further investigations should use methods that minimize confounding and selection bias without introducing additional selection bias or blocking pathways of interest [22]. Future work should also identify which indicators of neighborhood socioeconomic position (e.g., disadvantage or poverty), if any, contribute to HIV/STIs in adolescents and young adults. If neighborhoods are deemed to impact HIV/STI outcomes, future studies should also aim to pinpoint the most etiologically relevant time windows for neighborhood-level exposures. Finally, because intervening directly on community and neighborhood environments can present unique difficulties [53], reducing rates of HIV/STIs among adolescents and young adults may require identifying intermediates that can be more feasibly intervened on.

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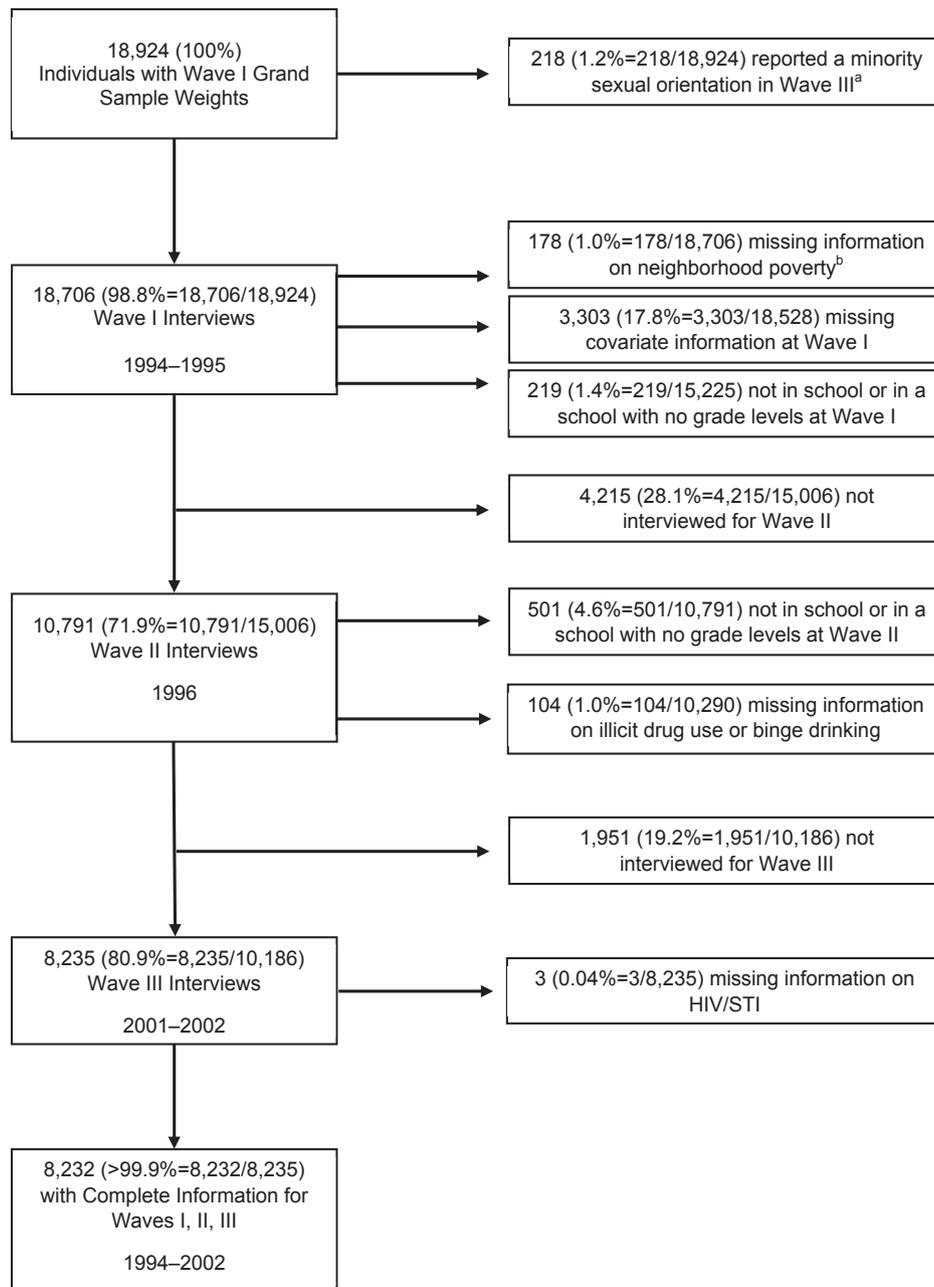
Authors' contributions: A.R.Z. contributed to the conception and design of the study, acquisition and interpretation of the data, and critical revision of the manuscript. J.W.A. contributed to the conception and design of the study, analysis and interpretation of the data, and drafting and critical revision of the manuscript. J.R.G. contributed to analysis and interpretation of the data and the drafting and critical revision of the manuscript. B.D.L.M. contributed to the conception and design of the study, interpretation of the data, and critical revision of the manuscript. C.J.H. contributed to the conception and design of the study, acquisition and interpretation of the data, and drafting and critical revision of the manuscript. Funders did not influence data analysis or interpretation of results. All authors approve the final version of the manuscript, agreeing to be accountable for all aspects of the work and attesting both to its accuracy and integrity. No person meeting the criteria for authorship has been omitted from the author list.

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## Appendix A



**Figure S1.** Exclusion criteria applied to identify the analytic sample of 8232 respondents from the National Longitudinal Study of Adolescent to Adult Health (Add Health) waves I–III, 1994–2002. <sup>a</sup>Given that the relationship between contextual factors and HIV/STI risk may vary by sexual orientation due to differences in behavioral/network-based<sup>1–3</sup> risk factors, we excluded 218 respondents who reported a minority sexual orientation in wave III, due to an insufficient sample size to make appropriate inferences among these individuals. <sup>b</sup>This 178 includes 10 respondents who were excluded because of having an unstable neighborhood poverty estimate due to small sample sizes in data derived from census questionnaires that were administered only to a subset of residents, and 168 respondents who were excluded for missing neighborhood poverty information due to a missing or insufficient geocode.

**Table S1**

Distribution of stabilized inverse probability weights in primary and secondary analyses examining relationship between neighborhood poverty and HIV/STI prevalence, National Longitudinal Study of Adolescent to Adult Health, waves I–III 1994–2002, ( $n = 8232$ )

Weights	Mean (SD)	Median (quartiles) <sup>a</sup>	Minimum, maximum
Primary analysis (binary poverty)			
Neighborhood weights	1.026 (0.571)	0.905 (0.877, 1.033)	0.181, 10.810
Selection weights	1.010 (0.092)	0.990 (0.945, 1.050)	0.853, 1.808
Neighborhood-and-selection weights	1.040 (0.628)	0.902 (0.829, 1.069)	0.183, 13.634
Secondary analysis (three-level poverty)			
Neighborhood weights	1.069 (0.887)	0.858 (0.757, 1.093)	0.187, 21.617
Selection weights	1.010 (0.092)	0.990 (0.945, 1.050)	0.853, 1.808
Neighborhood-and-selection weights	1.088 (1.006)	0.867 (0.727, 1.128)	0.188, 26.524

Distributions based on stabilized inverse probability weights estimated in the survey-weighted population but prior to multiplying stabilized inverse probability weights by the wave I grand sample weights. No additional survey-weighting was performed when calculating stabilized weight distributions.

SD = standard deviation.

<sup>a</sup> First and third quartiles reported.

**Table S2**

Distribution of stabilized inverse probability weights in sensitivity analysis using alternative selection weights in model examining relationship between neighborhood poverty as a binary variable and HIV/STI prevalence, National Longitudinal Study of Adolescent to Adult Health, waves I–III 1994–2002, ( $n = 8232$ )

Weights	Mean (SD)	Median (quartiles) <sup>a</sup>	Minimum, maximum
Neighborhood weights	1.026 (0.571)	0.905 (0.877, 1.033)	0.181, 10.810
Selection weights	1.060 (0.586)	0.856 (0.798, 0.960)	0.696, 6.412
Neighborhood-and-selection weights	1.090 (0.971)	0.792 (0.697, 1.118)	0.154, 26.991

Distributions based on stabilized inverse probability weights estimated in the survey-weighted population but prior to multiplying stabilized inverse probability weights by the wave I grand sample weights. No additional survey-weighting was performed when calculating stabilized weight distributions.

SD = standard deviation.

<sup>a</sup> First and third quartiles reported.

**Table S3**

Distribution of stabilized inverse probability weights in sensitivity analysis using biologically confirmed chlamydia, gonorrhea, or *Trichomonas vaginalis* in model examining relationship between neighborhood poverty as a binary variable and STI prevalence, National Longitudinal Study of Adolescent to Adult Health, waves I–III 1994–2002, ( $n = 7246$ )

Weights	Mean (SD)	Median (quartiles) <sup>a</sup>	Minimum, maximum
Neighborhood weights	1.027 (0.571)	0.901 (0.874, 1.035)	0.184, 9.656
Selection weights	1.017 (0.096)	1.002 (0.948, 1.068)	0.834, 1.648
Neighborhood-and-selection weights	1.047 (0.613)	0.911 (0.825, 1.093)	0.180, 10.480

Distributions based on stabilized inverse probability weights estimated in the survey-weighted population but prior to multiplying stabilized inverse probability weights by the wave I grand sample weights. No additional survey-weighting was performed when calculating stabilized weight distributions.

SD = standard deviation.

<sup>a</sup> First and third quartiles reported.

**Table S4**

Distribution of stabilized inverse probability weights in sensitivity analysis using alternative selection weights and biologically confirmed chlamydia, gonorrhea, or *Trichomonas vaginalis* in model examining relationship between neighborhood poverty as a binary variable and STI prevalence, National Longitudinal Study of Adolescent to Adult Health, waves I–III 1994–2002, ( $n = 7246$ )

Weights	Mean (SD)	Median (quartiles) <sup>a</sup>	Minimum, maximum
Neighborhood weights	1.027 (0.571)	0.901 (0.874, 1.035)	0.184, 9.656
Selection weights	1.064 (0.576)	0.867 (0.800, 0.978)	0.678, 5.080
Neighborhood-and-selection weights	1.095 (0.930)	0.796 (0.696, 1.127)	0.152, 22.136

Distributions based on stabilized inverse probability weights estimated in the survey-weighted population but prior to multiplying stabilized inverse probability weights by the wave I grand sample weights. No additional survey-weighting was performed when calculating stabilized weight distributions.

SD = standard deviation.

<sup>a</sup> First and third quartiles reported.

**Table S5**

Survey and stabilized inverse probability weighted prevalence differences for biologically confirmed chlamydia, gonorrhea, or *Trichomonas vaginalis* infection in early adulthood as a function of binary neighborhood poverty in early adolescence, National Longitudinal Study of Adolescent to Adult Health, waves I–III 1994–2002, ( $n = 7246$ )<sup>a</sup>

Analysis	Prevalence difference	95% CL
Biologically confirmed STI sensitivity analysis		
Unadjusted	0.078	0.048, 0.109
IPW MSM <sup>b,c</sup>	0.019	–0.005, 0.042
First sensitivity analysis and biologically confirmed STI sensitivity analysis		
IPW MSM <sup>b,d</sup>	0.035	0.000, 0.069

CL = confidence limits; IPW MSM = inverse probability weighted marginal structural model.

<sup>a</sup> 7246 is the unweighted sample size, while survey and inverse probability weighted populations were used for analyses.

<sup>b</sup> Accounts for potential confounding bias related to race/ethnicity, US-born status, two-parent household, parental education, household receipt of welfare, and alcohol or illegal drug availability in household (wave I) via inverse probability weighting. The inverse probability weights were stabilized by the marginal probability of the observed neighborhood poverty level within the analytic sample.

<sup>c</sup> Accounts for potential selection bias related to sex, race/ethnicity, US-born status, two-parent household, parental education, household receipt of welfare, alcohol or illegal drug availability in household, birth year, suicidal ideation, vaginal sexual intercourse (wave I), and illicit drug use and binge drinking at wave II via inverse probability weighting. The inverse probability weights were stabilized by the marginal probability of being selected into the analytic sample based on the availability of wave III data.

<sup>d</sup> Accounts for potential selection bias related to all factors mentioned in footnote <sup>c</sup> via inverse probability weights excluding illicit drug use and binge drinking at wave II, because illicit drug use and binge drinking information were not available for all participants used to estimate the corresponding selection weights. The inverse probability weights were stabilized by the marginal probability of being selected into the analytic sample based on the availability of wave II or III data.

## Appendix A References

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