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Abstract

People who inject drugs (PWID) are often members of HIV/AIDS risk networks, where individuals engage in sexual and injection risk behavior. Engagement in HIV care is important for this population. Low socioeconomic status, stigmatization, and lack of access to medical care often complicate successful engagement in the HIV cascade of care for PWID. This study investigates how individual's attitudes about how much control they have over HIV/AIDS risk in their life (i.e., locus of control and self-blame) affect health-seeking behavior in PWID participants and their community members. We applied causal inference methodology to PWID HIV risk networks ascertained from the Social Factors and HIV Risk Study (SFHR) conducted between 1991 and 1993 in Bushwick, Brooklyn, New York. We estimated protective disseminated effects of attitudes toward HIV/AIDS on the health-seeking behaviors of others in the PWID community. In other words, a positive attitude toward controlling HIV/AIDS can improve the health-seeking behavior of other members of the community who report a pessimistic attitude toward HIV/AIDS control. Given this finding, we also discuss potential network interventions to improve health-seeking behavior among both PWID individuals who receive the intervention and others in the PWID network informed by our analysis of disseminated effects.

Keywords: Causal inference, Dissemination, Health attitudes, HIV/AIDS, Injection drug use, Interference, Network intervention, Peer intervention, Risk network

Introduction

HIV/AIDS remains a significant concern among people who inject drugs (PWID) in the United States (Centers for Disease Control and Prevention 2022). PWID are often part of HIV risk networks, where partnerships are defined by sexual or injection risk behaviors, which potentially increase the risk of HIV transmission (Mathers et al. 2008). Compounding the issue, PWID with low socioeconomic backgrounds often struggle with accessing adequate medical care, hindering successful engagement with the HIV care cascade (Ghosh et al. 2017). Thus, PWID are not only at an increased risk of HIV

infection but also face specific barriers that can limit their engagement with HIV care (Ghosh et al. 2017).

Peer interventions have been considered effective for behavioral change to both prevent HIV and support HIV-positive persons, especially in a marginalized population, because peers have additional values for members to successfully introduce behavioral change interventions (Simoni et al. 2011a). A systematic review of the efficacy of peer interventions for people live with HIV/AIDS (PLWHA) suggested a potential effectiveness of peer interventions for improving attitudes and cognitions, HIV knowledge, sexual risk behavior, and substance abuse (Simoni et al. 2011a). However, the empirical evaluation of peer intervention efficacy faces challenges, such as heterogeneities in the target populations, intervention methodologies, and results reported (Simoni et al. 2011a).

One class of interventions attempts to leverage dissemination in a social network for behavioral change (Latkin and Knowlton 2015; Aiello 2017; Curtis et al. 1995). Among a network, dissemination (spillover or interference) can occur when members of a social group modify their behavior based on the traits, beliefs, attitudes, or norms among their social contacts. This can occur when one individual's health attitude affects another individual's health behavior. This dissemination of attitudes may be possible through peer influence, for example, due to the verbalization of attitudes or behavioral modeling (Latkin and Knowlton 2015; De et al. 2007). Although potential HIV transmission networks of PWID have dependence structures associated with sexual and injection risk behaviors, this dependence structure is related to but not synonymous with dissemination in networks. Our work aims to evaluate the dissemination of health attitudes through HIV risk networks under the existence of dependence structures. In earlier work, dissemination was conceptualized as the framework to analyze the dependent events of infectious diseases with four different effects of interest in a two-stage randomized design, where investigators randomly assign a treatment allocation strategy (i.e., vaccination coverage in a community) to each community then assign actual treatment (i.e., vaccine) to participants in each community given the assigned treatment allocation strategy (Halloran and Struchiner 1991; Hudgens and Halloran 2008). In recent work, methods to estimate the four parameters using data from observational studies have been developed (Tchetgen and VanderWeele 2012; Liu et al. 2016).

The efficacy of interventions can be more accurately evaluated by assessing disseminated effects to inform more effective and sustainable solutions for HIV/AIDS prevention among PWID (Benjamin-Chung et al. 2018; Buchanan et al. 2018). Therefore, before introducing a network-based intervention to modify attitudes, an initial step is to disentangle the relationship between the attitudes of PWID and their health-seeking behaviors. Although some earlier studies assessed attitudes toward HIV/AIDS risk (Bandura 1990; Allard 1989), there have been limited studies about the impact of PWID attitudes toward HIV/AIDS risk on their health-seeking behavior. Evaluating the effects of attitudes toward HIV/AIDS risk on PWID's health-seeking behavior can provide substantially new insights for developing more effective and sustainable interventions. This study quantifies the magnitude of PWID's attitudes toward HIV/AIDS risk on their own health-seeking behaviors and the health-seeking behaviors of other individuals in their risk network.

This work extends the original research (Shimada et al. 2023) presented at Complex Networks 2023. In this extended article, we first revisit the analysis conducted by Shimada et al. (Shimada et al. 2023) and scrutinize the observed causal effects, giving further interpretation of observed disseminated effect. Then, based on the estimated causal effects, a further assessment of the network properties of the SFHR PWID network is conducted to inform potentially effective interventions to improve health-seeking behaviors in PWID communities.

Methods

Data collection

The SFHR study was conducted in the Bushwick, Brooklyn, New York between July 1991 and January 1993 (Curtis et al. 1995; Friedman et al. 2006). Data were collected from street-recruited PWID in the Bushwick neighborhood, a low-income area of approximately 100,000 residents with high rates of poverty, injection drug use, and HIV/STI prevalence. The original study enrolled a total of 767 participants and included 3162 dyadic relationships. Dyadic relationships were defined as a participant reporting that he/she had sex or injected drugs with another individual in the previous 30 days, and each participant was asked to name up to 10 contacts (Friedman et al. 2006).

Of the 3162 dyadic links in the original SFHR study, 2498 links recorded were between enrolled and non-enrolled individuals (Friedman et al. 2006). We excluded these non-enrolled individuals from the analysis, including the 2498 edges between enrolled and non-enrolled individuals (Fig. 1). Eighty-two enrolled participants were missing either outcome, exposure, and/or covariate information, so we excluded these individuals from the analysis. After this exclusion, we had 283 participants with no observed relationships with other participants in the network. These 283 isolated participants were also removed from the analysis. The SFHR PWID network for this analysis included 402 participants (i.e., vertices or nodes) with 403 risk connections (i.e., edges) (Fig. 1). Appendix Fig. 1 displays the degree distribution versus the average nearest neighbor degree for 402 participants.

Community detection

We identified communities in the SFHR PWID network using a modularity-based community detection approach (Appendix A). A community is defined as a group of participants densely connected with sparser connections to participants outside of the group (Newman 2010). In the SFHR PWID network, these communities included PWID who were more highly connected within the same community but had sparser connections to the PWID in other communities. For the modularity-based method, larger values than expected if connections were randomly assigned suggest the presence of nontrivial community structures in the network (Newman 2010; Kolaczyk and Csárdi 2014). As a result, there are more edges among the participants in a community than between communities in the SFHR network.

Study design (independent variables, outcome, covariates)

We considered two separate exposures to assess attitudes toward HIV/AIDS risk: (1) *HIV/AIDS locus of control (i.e., internal vs. external)* and (2) *blame attributes*

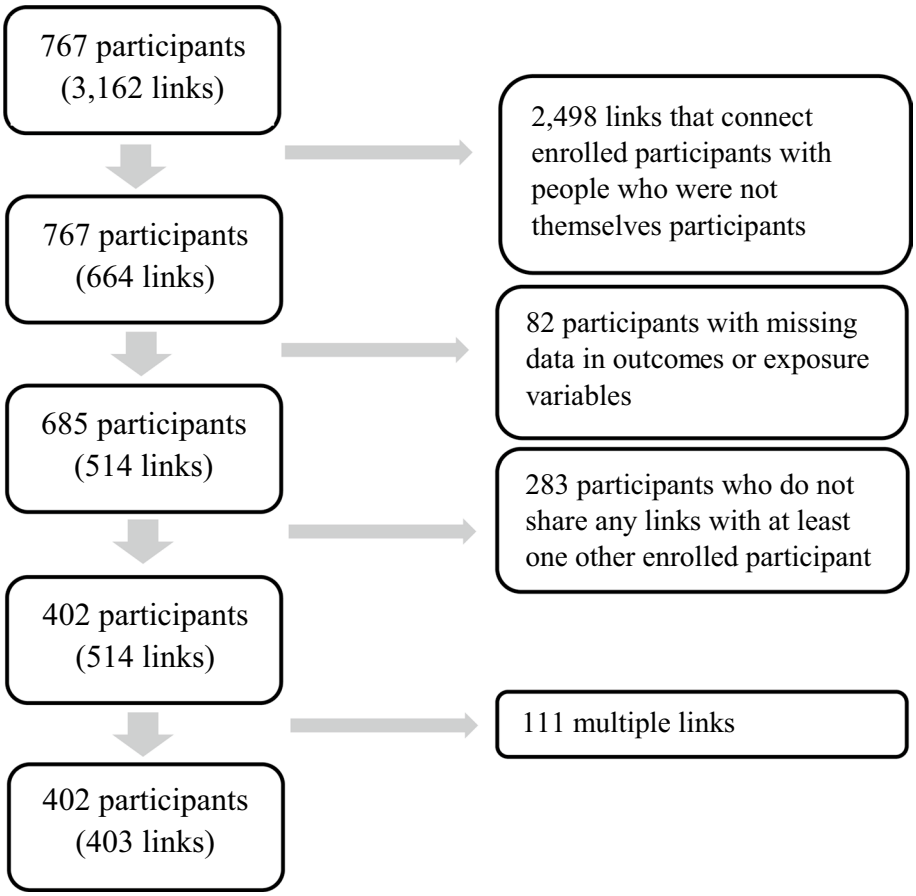

Fig. 1 Sample flowchart of the Social Factors and HIV Risk (SFHR) Study from Bushwick, New York, 1991–1993 (Multiple-risk links (or multiple edges) mean two or more link connections incident to the same pair of nodes. We removed those links to simplify the network in this study. We also defined a link between a pair of nodes as where any participant A names the other participant B, regardless of whether B also names A or not. That is, we do not pay attention to the directionality of links in the SFHR PWID network.)

(self-blame vs. blame others). The locus of control is defined as the degree to which an individual believes they have control over what will happen or has happened to themselves and can be classified into two different types: internal and external (Rotter 1966). Individuals with an internal locus of control (ILOC) attribute the events they experience to factors within their control, while those with an external locus of control (ELOC) attribute events to factors beyond their influence (Blanchard-Fields et al. 2012; Grimes et al. 2004). HIV/AIDS locus of control is defined as an individual belief about how much control one has over its own HIV/AIDS risk. The blame attribute toward HIV/AIDS is defined as an individual *blaming themselves (i.e., self-blame)* or *blaming others or society (i.e., blame others)* for their perceived HIV/AIDS risk. Participants were asked ten questions about their health beliefs to determine their HIV/AIDS locus of control and blame attribute (Table 1). Questions 4, 5, and 9 capture information about PWID's individual blame, and the remaining questions are about PWID's HIV/AIDS locus of control. The reverse scale was used for negatively phrased items. The responses were originally recorded on a Likert scale (strongly agree, agree, somewhat agree, disagree, strongly disagree, don't know,

Table 1 Questions about health beliefs in the Social Factors and HIV Risk (SFHR) Study from Bushwick, New York, 1991–1993

SFHR Questions about Health Beliefs
Q1. It is my own behavior which determines whether I get AIDS or not
* Q2. No matter what I do, if I'm going to get AIDS, I will get AIDS
Q3. I'm in control of whether or not I get AIDS
Q4. My family has a lot to do with whether I get AIDS. **(Blame)
Q5. If I get AIDS, I'm to blame. **(Blame)**
* Q6. Getting AIDS is largely a matter of bad luck
* Q7. No matter what I do, I'm likely to get AIDS
Q8. If I take the right actions, I can avoid getting AIDS
Q9. If I get AIDS it is because of the society we live in. **(Blame)
* Q10. No matter what I do, I'm unlikely to get AIDS

* Item required a reversed scale in calculating the score for health beliefs

** Item required a reversed scale in calculating the score for blame attitudes

refused, and not applicable). To create a binary variable to represent locus of control, for each response out of seven, we assigned the value of 1 if a participant reported ILOC, while the value of -1 was assigned if a participant reported ELOC. If the response was neutral (i.e., don't know), we assigned a value of 0. Adding all values assigned to the responses for health belief related questions, we obtained individual health belief scores ranging from -7 to 7. Then, if one's locus of control score was greater than or equal to three, the participant was assigned as having an ILOC; otherwise, the participant was assigned ELOC. Because traits that consist of both negative and positive aspects can be negatively biased, we selected a positive threshold (Rozin and Royzman 2001). A similar procedure was taken to create a binary variable to represent a participant's blame attribute. We obtained blame scores that ranged from -3 to 3 for individual participants. If a participant's blame score was equal to three, the attribute was assigned as "self-blame"; otherwise, the attribute was "blame others." We verified the internal consistency of the individual responses for the locus of control and blame items with Cronbach's alpha on their original Likert scale.

The outcome was defined as the receipt of the SFHR HIV test result. The HIV test was conducted as a part of the SFHR study, and the participants could receive the result after their interview. Therefore, there was a temporal ordering between the exposures and the outcome. Receipt of HIV test results for each participant was recorded as a binary variable (i.e., 1 if "Yes" and 0 otherwise).

Participants' demographic other characteristics were summarized with descriptive statistics (Table 2). We also created a binary variable indicating participants' knowledge of their HIV/AIDS status before the SFHR study. We assumed that a participant knows their HIV/AIDS status if they were ever told they had HIV/AIDS or if they were tested at least once before SFHR and obtained the last test result; otherwise, we assumed the participant did not know their HIV/AIDS status before SFHR. In the final model of the analysis to adjust for confounding, the following variables were included as covariates: medical payment method (some insurance, paid by self, or other), pre-SFHR knowledge of HIV/AIDS status (Yes vs. No), sex (Male vs. Female), race (White vs. Non-White), age

Table 2 Participants' characteristics in the Social Factors and HIV Risk (SFHR) Study from Bushwick, New York, 1991–1993 (n = 402)

Characteristics[a]	Number of Participants (%)
Age, Mean (SD)	35 (6.9)
Young Adult (19–39 years old)	290 (72)
Middle Aged (40–65 years old)	112 (27)
Sex	
Male	287 (71)
Female	115 (29)
Race/ethnicity	
White	153 (38)
Non-White[b]	249 (62)
Highest education	
Less than high school graduation	258 (64)
High school or more	143 (36)
Work status	
No job	364 (91)
Some work	37 (9)
Where currently live	
In your own apartment or house	116 (29)
Someone else's apartment or house	192 (48)
Homeless/other	94 (23)
Medical expense payment method[c] Pay myself	83 (21)
Some insurance	262 (65)
Other	57 (14)
Ever told that you have AIDS/HIV	27 (7)
Ever tested for HIV	234 (58)
Number of HIV tests ever taken, Mean (SD)	1.9 (1.2)
Pick up your HIV test results last time[d]	164 (70)
HIV/AIDS status known before SFHR study[e]	188 (47)
HIV positive	162 (41)
HB core antibody positive	244 (75)
AIDS	20 (5)

[a] There were missing observations in highest education and work status. In the HIV positive, HB core antibody positive, and AIDS variables, there were six, seventy-seven, and seven missing data, respectively

[b] Non-White category includes Black/African American (n = 105), Latino/Hispanic (n = 141), Native American (n = 2), and Other (n = 1)

[c] Some insurance included Medicaid, Medicare/Social security, employment health plan, and Community organization/agency. Pay myself includes family member, friends and no one as well as pay myself

[d] The percentage in the right column in this question was calculated based on 234 people who answered YES to the previous Ever-tested question

[e] This pre-SFHR knowledge of HIV/AIDS status variable is created based on the information from ever told, ever tested, number of HIV test ever taken, and pick-up the last HIV test results

(40–65 vs. 19–39), and the interactions of sex and age, and race and medical payment method to allow the model to be more flexible. For the 96 communities of PWID, we computed the observed distributions of the proportion of PWID reporting ILOC/self-blame in a community (i.e., observed coverage).

Causal inference framework under the presence of dissemination (including assumptions)

A two-stage randomized design allows for exchangeability (i.e., no unmeasured confounding) at both the individual- and community-levels. Coverage is defined as the probability of treatment or exposure in a community (denoted by $\alpha, \alpha\prime$ where $\alpha < \alpha\prime$). In our context, coverage is the prevalence rate of internal locus of control within a SFHR PWID community, and this measure may differ from the observed percentage of internal locus of control among participants in that community. In the presence of dissemination, potential outcomes are indexed by the exposure of the individual and also the proportion of exposures of other members in the same community. Causal inference in this setting requires several identifying assumptions. Two assumptions are related to the structure of dissemination: *partial interference* and *stratified interference*. The partial interference assumption means that individual's locus of control can possibly affect the receipt of HIV testing results of other individuals in the same community but does not affect others outside of that community (Hudgens and Halloran 2008; Tchetgen and VanderWeele 2012; Saul and Hudgens 2017; Sobel 2006). The stratified interference assumption means that an individual's potential outcome depends only on his/her own locus of control and also the proportion of those community members reporting ILOC (Hudgens and Halloran 2008; Tchetgen and VanderWeele 2012). We also make the following three assumptions: (i) conditioning on a vector of pre-exposure covariates (e.g., medical payment method, pre-SFHR knowledge of HIV/AIDS status, sex, race, age, and the interactions of sex and age, and race and medical payment method), the vector of locus of control exposures for a community is independent of community-level potential outcomes (i.e., *community-level exchangeability*), (ii) for each pre-exposure covariate, there is a positive probability of each level of the community-level exposure (i.e*., community-level positivity*), and (iii) the exposure is well defined, which means an individual either has ILOC or ELOC and if there are other versions of locus of control, we assume that they are irrelevant to the causal effects of interest. We assume there is no misclassification of attitudes towards HIV/AIDS; that is, every participant correctly reports his/her attitudes in the study, and this exposure accurately captures the underlying attitudes. We also assume the model for the exposure weights is correctly specified (e.g., correct functional forms of covariates), and there is no homophily; that is, individuals in the network are not forming HIV risk connections based on some unobserved variables also associated with their health-seeking behaviors (McPherson et al. 2001), and missing outcomes, exposures, or covariates are missing completely at random (MCAR) (Little and Rubin 2019). The assumption of no homophily can be plausible in the SFHR study because it is unlikely that PWID formed risk connections based on whether they would receive their HIV test results. In this setting, the partial interference assumption can also be valid if there is little dissemination between the communities in the network. In the SFHR PWID network, there were more edges among participants within a community than between communities, supporting this assumption.

Models

We are interested in four different causal effects in the presence of dissemination (Hudgens and Halloran 2008; Tchetgen and VanderWeele 2012) (Fig. 2). In the following,

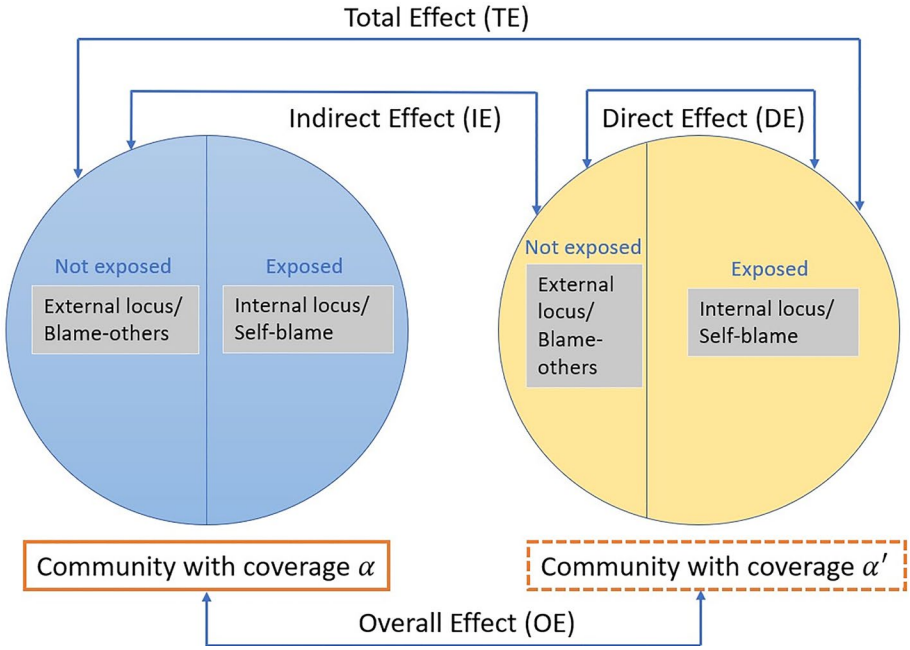

Fig. 2 Schematic diagram of causal estimation in the presence of dissemination adapted from Halloran and Struchiner (1991)

Y_{ij}, A_{ij}, X_{ij} represent the observed outcome of receipt of SFHR HIV testing result, attitude status, and covariate vector of the j^{th} individual in community i, respectively. Also, $\boldsymbol{A_i}$ and $\boldsymbol{X_i}$ are vectors of exposures (i.e., locus of control/blame attributes) and covariate matrices for members within community i, respectively. In our setting, the coverage $\alpha or \alpha\prime$ is defined as the probability of PWID reporting ILOC/self-blame in a community. The potential outcome of the j^{th} individual in community i depends on the exposure of that individual and the proportion of exposure of others in community i.

The following notation represents four different causal effects of interest (Hudgens and Halloran 2008). Please see Appendix B for complete details about the estimands and estimators. The population-level estimands and estimators discussed herein pertain specifically to the study population (i.e., PWID in Bushwick, New York) rather than the broader and hypothetical underlying population. The *direct* effect, which compares population-level average potential outcomes under the exposure ELOC/blame others to ILOC/self-blame under a coverage level α of ILOC, is defined as

$$\overline{DE}(\alpha) = \overline{Y}(a = 0; \alpha) - \overline{Y}(a = 1; \alpha), \tag{1}$$

where our reference group is those with ILOC/self-blame. The *indirect* (or disseminated) effect, which compares population-level average potential outcomes under ELOC/blame others who belong to communities with different coverage levels of ILOC ($\alpha, \alpha\prime$ where $\alpha < \alpha\prime$), is defined as

$$\overline{IE}(\alpha, \alpha\prime) = \overline{Y}(a = 0; \alpha) - \overline{Y}(a = 0; \alpha\prime), \tag{2}$$

the *total* (or composite) effect, which is the sum of direct and indirect effects and can be interpreted as the maximum impact of exposure at population-level, is defined as

$$\overline{TE}(\alpha, \alpha\prime) = \overline{Y}(a = 0; \alpha) - \overline{Y}(a = 1; \alpha\prime), \tag{3}$$

the *overall* effect, which is interpreted as a population-level marginal effect of attitude exposures (i.e., ILOC and ELOC/self-blame and blame others) in the comparison between communities with different coverage levels ($\alpha, \alpha\prime$ where $\alpha < \alpha\prime$), is defined as

$$\overline{OE}(\alpha, \alpha\prime) = \overline{Y}(\alpha) - \overline{Y}(\alpha\prime). \tag{4}$$

Then, population-level inverse probability weighted (IPW) estimators of four different causal effects are represented by:

$$\widehat{DE}(\alpha) = \widehat{Y}^{ipw}(a = 0; \alpha) - \widehat{Y}^{ipw}(a = 1; \alpha), \tag{5}$$

$$\widehat{IE}(\alpha, \alpha\prime) = \widehat{Y}^{ipw}(a = 0; \alpha) - \widehat{Y}^{ipw}(a = 0; \alpha\prime), \tag{6}$$

$$\widehat{TE}(\alpha, \alpha\prime) = \widehat{Y}^{ipw}(a = 0; \alpha) - \widehat{Y}^{ipw}(a = 1; \alpha\prime), \tag{7}$$

$$\widehat{OE}(\alpha, \alpha\prime) = \widehat{Y}^{ipw}(\alpha) - \widehat{Y}^{ipw}(\alpha\prime), \tag{8}$$

where $\widehat{Y}^{ipw}$ is the average of community-level average estimated potential outcomes. When quantifying these parameters in an observational study, community-level propensity scores can be estimated using the information of individual-level covariates in a mixed effects logit model with a random effect for correlation in each community. Then, the inverse of this estimated propensity score is used as an exposure weight in the estimator of interest (i.e., a contrast of estimated average community-level potential outcomes) (Tchetgen and VanderWeele 2012). We used robust variance estimators to construct 95% Wald-type confidence interval for the effects (Perez-Heydrich et al. 2014).

Software

We used SAS 9.4 (Cary, NC; https://www.sas.com/) for data preparation and R version 3.4.4 (https://cran.r-project.org/) for the visualization and analysis. For the estimation in the presence of interference, we used the "inferference" package in R (https://cran.r-project.org/package=inferference) (Saul and Hudgens 2017), which implements the IPW estimation method for observational studies (Tchetgen and VanderWeele 2012; Perez-Heydrich et al. 2014).

Results

Descriptive statistics

Among the 402 participants, the mean age was 35 years and about 70% were male. Most participants had less than a 12th-grade education (64%), were unemployed (91%), and had some insurance for medical expenses (65%). Based on the history of HIV testing and the receipt of the last test result, (i.e., knowledge of HIV/AIDS status), 47% knew their own HIV/AIDS status before the SFHR study (Table 2). In addition, 41% were HIV infected, 5% had developed AIDS, and 75% were positive for Hepatitis B (HB) (Table 2). Out of the total 402 participants 19% (n = 77) picked up their SFHR HIV testing results.

Consistency of composite measure

Calculating Cronbach's alpha provides insight into the internal consistency of a composite measure for internal locus of control (ILOC)/external locus of control (ELOC) and self-blame/blame others. The values of Cronbach's alpha for health belief and blame questions were 0.77 and 0.47, respectively, which indicates an acceptable internal consistency in health belief questions; however, the internal consistency for blame questions suggests less reliability for this domain.

Coverage by community

There were 85 connected components, and one of them formed a giant component, including 199 participants and 275 risk connections (Fig. 3). Using a modularity-based community detection approach (Appendix A), we found 12 communities in the giant component (Fig. 4) and defined a total of 96 communities in the observed network. Among all 403 risk connections, 56 were between communities (14%), and 347 were within communities (86%). The average number of participants in a community was 4.2 participants (ranging from 2 to 35 participants). Given the 96 communities of PWID, the observed distributions of the proportion of PWID reporting ILOC/self-blame in a community (i.e., observed coverages) are shown in Supplemental Figures Appendix Fig. 2. The distribution of coverage of HIV/AIDS self-blame attribute had wider variation than that of ILOC. To ensure enough communities at each coverage level, we focused our causal inference analysis on coverages of 50%, 70% and 99%. The interconnection between attitudes and the outcome in the SFHR PWID network was visualized in Fig. 3. Reporting ILOC was associated with 87% (95% CI 0.85–4.11) higher odds of receipt of HIV test results (Appendix Tables 1 and 2).

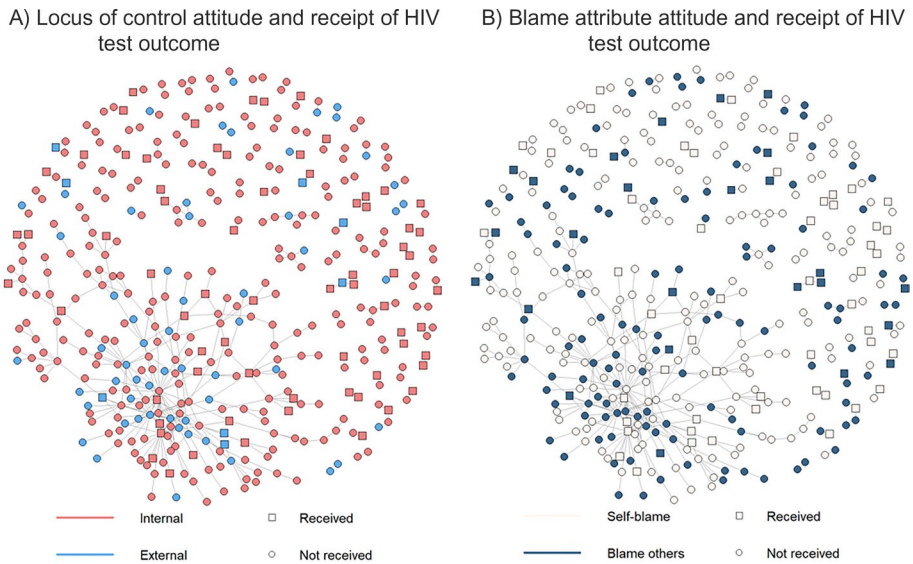

Fig. 3 The Social Factors and HIV Risk Study (SFHR) PWID network for the analysis with 402 nodes and 403 links. A dyadic link is defined as a participant reporting that he/she had sex or injected drugs with another individual in the previous 30 days (light gray lines). The term "Received" indicates the act of a participant picking up the SFHR HIV test result, while "Not received" denotes the absence of such an action

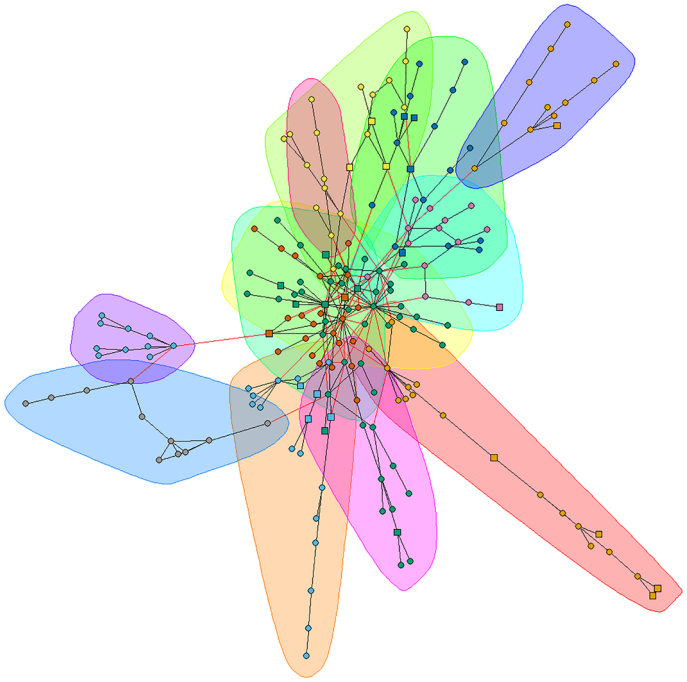

Fig. 4 Community detection among nodes in the giant component in the Social Factors and HIV Risk (SFHR) Study PWID network. Edges between communities determined by the modularity-based community detection method are shaded in red. There were 12 communities in the giant component of the SFHR PWID network

Network properties and variables of interest

Within the previously identified 96 communities, degree and subgraph centrality were calculated for each participant given their own community. Subgraph centrality measures how central or important a particular participant (i.e., node) is within a network by counting the number of closed loops that start at that participant, but it gives less importance to longer loops. Therefore, the more loops a participant is part of, especially shorter ones, the higher its subgraph centrality. Subgraph centrality is considered more discriminative for the nodes of a network than other centrality measures such as degree, betweenness, closeness or eigenvector centrality (Estrada and Rodriguez-Velazquez 2005). Among all 402 participants, medians of degree and subgraph centrality were 1.00 and 1.75, respectively. Overall, the results imply participants with ELOC are more likely to be deeply embedded in a community, while participants with ILOC are more periphery (subgraph centrality medians: 2.20 for ELOC and 1.70 for ILOC, not shown in Table 3). In summarizing degree and subgraph centrality by HIV/AIDS locus of control (i.e., belief) and the receipt of the SFHR HIV test result, participants with ELOC and receipt of the test result had a slightly higher degree (median = 2.00, Table 3) compared to participants in other categories. Furthermore, participants with ELOC and receipt of the test result also had a higher subgraph centrality of 2.47 (Table 3). On the other hand, participants with ILOC and receipt of the test result tended to have a slightly lower subgroup centrality (median = 1.59, Table 3). A similar summary was also created by blame attribute and the receipt of the SFHR HIV test result, but the degree and subgraph centrality were similar across categories.

Table 3 Degree and subgraph centrality for each participant in its community by health belief and receipt of HIV test result

	Min	Q1	Median	Q3	Max
Degree					
External (ELOC) x Not receipt	1.00	1.00	1.00	2.00	12.00
External (ELOC) x Receipt	1.00	1.00	2.00	2.25	3.00
Internal (ILOC) x Not receipt	1.00	1.00	1.00	2.00	15.00
Internal (ILOC) x Receipt	1.00	1.00	1.00	2.00	13.00
Subgraph centrality					
External (ELOC) x Not receipt	1.54	1.54	2.10	3.52	25.50
External (ELOC) x Receipt	1.54	1.70	2.47	2.98	5.73
Internal (ILOC) x Not receipt	1.54	1.54	1.75	2.62	55.20
Internal (ILOC) x Receipt	1.54	1.54	1.59	2.71	44.80

Estimated effect sizes

Results for both unadjusted and adjusted estimates are displayed in Table 4. The IPW estimates of the population-level causal effects of ELOC on receipt of SFHR HIV test results are displayed in Table 4. Adjusted estimates, i.e., those that controlled for baseline covariates, were comparable to unadjusted estimates but had slightly narrower confidence intervals. For the direct effect estimates, communities with 50% and 70% coverages had estimates of similar magnitude for locus of control on the receipt of HIV testing results. In other words, in a community with 50% coverage of ILOC, we would expect 13 more participants to receive their HIV test result per 100 individuals under ILOC exposure compared to ELOC. Interestingly, if a community has the highest coverage of 99%, the estimated direct effect was the smallest among the three coverage groups: $\widehat{DE}(99) = -0.10$ (95% CI: $-0.26, 0.05$). A significant indirect effect was estimated comparing 50% and 70% ILOC coverage communities: $\widehat{IE}(50, 70) = -0.03$ (95% CI: $-0.06, -0.01$). That is, we would expect 3 more individuals with ELOC to receive their HIV test result in a 70% ILOC coverage community compared to a community with only 50% ILOC coverage. The largest total effect estimate was for the comparison 50% and 70% ILOC coverage communities, and 50% and 99% ILOC coverage communities: $\widehat{TE}(50, 70) = \widehat{TE}(50, 99) = -0.17$ (95% CIs: $-0.27, -0.08$ for 50% versus 70%; $-0.28, -0.07$ for 50% versus 99%). In other words, we expect 17 more participants to receive their HIV test result per 100 individuals if they reported ILOC in 70% ILOC coverage communities compared to individuals who reported ELOC in a 50% ILOC coverage community. Finally, the overall effect estimates indicate that the likelihood of receipt of HIV testing results increases as community coverage increases. For example, we expect that 11 more individuals will receive their HIV test results if a community has 99% ILOC coverage compared to a community with only 50% coverage (95% CI: 0.03, 0.18). None of the estimates for the causal effect of blame on the probability of receiving HIV test results were statistically significant. However, Cronbach's alpha for the blame attribute was quite low (0.77 for belief, and 0.47 for blame) indicating a possible lack of internal consistency for this measure. Similarly, the distribution of observed coverage for this attribute had more variation than that observed for ILOC/ELOC.

Table 4 Unadjusted and adjusted estimated risk differences (RDs) with corresponding 95% confidence intervals (95% CIs) of causal effects of locus of control (external vs. internal) and blame (others vs. self) on likelihood of receiving SFHR HIV test results among 402. Coverage is defined as the probability of internal locus of control in a community. Baseline covariates are included in a community-level propensity score

Effect	Coverage	Unadjusted		Adjusted	
	(α%, αr%)	RD	95% CI	RD	95% CI
Locus of control (external vs. internal)					
Direct	(50, 50)	−0.15	(−0.23, −0.06)	−0.13	(−0.23, −0.03)
Direct	(70, 70)	−0.14	(−0.25, −0.04)	−0.14	(−0.25, −0.02)
Direct	(99, 99)	−0.10	(−0.26, 0.06)	−0.10	(−0.26, 0.05)
Indirect	(50, 70)	−0.04	(−0.07, −0.01)	−0.03	(−0.06, −0.01)
Indirect	(50, 99)	−0.07	(−0.16, 0.02)	−0.07	(−0.15, 0.00)
Indirect	(70, 99)	−0.03	(−0.10, 0.04)	−0.04	(−0.09, 0.01)
Total	(50, 70)	−0.18	(−0.27, −0.10)	−0.17	(−0.27, −0.08)
Total	(50, 99)	−0.17	(−0.28, −0.07)	−0.17	(−0.28, −0.07)
Total	(70, 99)	−0.13	(−0.25, −0.01)	−0.14	(−0.26, −0.02)
Overall	(50, 70)	−0.07	(−0.10, −0.04)	−0.06	(−0.09, −0.04)
Overall	(50, 99)	−0.10	(−0.18, −0.01)	−0.11	(−0.18, −0.03)
Overall	(70, 99)	−0.03	(−0.10, 0.04)	−0.04	(−0.10, 0.01)
Blame (others vs. self)					
Direct	(50, 50)	−0.04	(−0.16, 0.08)	−0.06	(−0.17, 0.04)
Direct	(70, 70)	−0.04	(−0.16, 0.09)	−0.04	(−0.15, 0.07)
Direct	(99, 99)	−0.08	(−0.25, 0.10)	−0.07	(−0.23, 0.10)
Indirect	(50, 70)	−0.00	(−0.05, 0.04)	−0.01	(−0.05, 0.03)
Indirect	(50, 99)	0.03	(−0.08, 0.14)	0.02	(−0.07, 0.12)
Indirect	(70, 99)	0.04	(−0.04, 0.11)	0.03	(−0.03, 0.10)
Total	(50, 70)	−0.04	(−0.16, 0.09)	−0.05	(−0.16, 0.06)
Total	(50, 99)	−0.04	(−0.20, 0.12)	−0.04	(−0.19, 0.10)
Total	(70, 99)	−0.04	(−0.20, 0.12)	−0.04	(−0.18, 0.11)
Overall	(50, 70)	−0.01	(−0.05, 0.04)	−0.00	(−0.04, 0.03)
Overall	(50, 99)	−0.02	(−0.14, 0.09)	−0.01	(−0.12, 0.09)
Overall	(70, 99)	−0.02	(−0.10, 0.06)	−0.01	(−0.08, 0.07)

Sensitivity analysis

Some participants (i.e., nodes) were excluded from the analysis due to the missing outcome, exposure, and/or covariate information. The community detection results could be sensitive to the removal of nodes from the network; therefore, a sensitivity analysis was performed by first conducting community detection, then removing participants with missing information and eliminating isolated participants. As a result of the sensitivity analysis, the number of communities and participants in the SFHR network changed (94 communities with 425 participants). However, the results for the estimated causal effects of interest were comparable to the main analysis (results not shown).

Discussion

We assessed the impact of attitudes among PWID toward HIV/AIDS risk on their own and the health-seeking behaviors of others in their HIV risk network community. The results indicate that PWID with the internal locus of control were more likely to receive their HIV testing result, regardless of the internal locus status of other individuals in their community, possibly because individuals who report more control over their health outcomes may be motivated to engage in behaviors to improve their health. Our results also indicate that PWID with the external locus of control in a community with a high coverage of internal were more likely to receive their HIV testing results compared to a lower coverage community. This means that PWID may engage in protective health behaviors due not only to their own internal motivation but also via reinforcement from others with internal locus in their community. This suggests an intervention that could modify the HIV/AIDS locus of control has the potential to improve the health-seeking behavior of treated individuals as well as their sexual and drug use contacts. Moreover, an intervention aimed at increasing coverage from 50 to 70% could yield a comparable impact to an intervention aimed at increasing coverage to 99%. Such a finding might offer a considerable advantage in terms of resource allocation and effort by setting data-informed benchmarks for intervention coverage. Though attitudes were not a public health intervention in the SFHR study, in terms of social psychology, attitudes could be modified and eventually affect people's behavior (Festinger 1962; Batson et al. 1997, 2002; Latkin et al. 2003). Furthermore, even small changes in health-seeking behavior could have a larger impact over long durations of time, particularly with infectious diseases in networks through changes in social norms (Curtis et al. 1995; Friedman et al. 2006).

Prospective interventions

Given the observed findings from causal inference and network property analyses, we discuss potentially effective interventions to modify the HIV/AIDS locus of control among PWID. Estimated disseminated effects indicated that PWID with the external locus of control could have a protective health-seeking behavior via community reinforcement from those with an internal locus of control. Intuitively, this kind of reinforcement is more likely to happen when an opinion leader exists in a community to build and spread the community's social norms. A previous study found that PWID can change their HIV risk behaviors by the change in social norms related to those risk behaviors (Latkin et al. 2013). We also observed that participants with the external locus of control were more central in a community as measured by network properties. Given this, popular opinion leader (PoL) intervention can be a potentially effective peer-led intervention in the SFHR PWID network setting. We anticipate that PoL works well in a natural setting such as their own community (Simoni et al. 2011b) because intervention would be more likely to be accepted by community members if delivered by a peer, especially in marginalized populations such as PWID. There was no peer-driven intervention with an opinion leader in each community in the SFHR PWID network; however, focusing intervention delivery on participants with the external locus of control and relatively high community centrality would be a promising strategy to leverage the intervention

impact via a protocol-based intervention that both alters an individual's health beliefs and amplifies the possible impact through dissemination to others in the community.

Limitations

The validity and reliability of the exposure variables as measures of individual attitudes toward HIV/AIDS risk could be further studied. The internal consistency for blame questions suggests less reliability and the coverage of blame attribute had more variability. Future research could address the refinement of both the blame attribute measurement and appropriate coverage thresholds. The development of carefully constructed standardized questionnaires to assess PWID's attitudes toward HIV/AIDS risk could improve this study. There may be missing individuals and connections, rendering the observed network likely different from the full underlying network, and this limits the conclusion of our results to the observed SFHR PWID network. Future work could be developed to improve the ascertainment of edges (Friedman et al. 2018) and address missingness and sampling bias in network-based studies (Kim and Leskovec 2011). Another limitation is that this study dataset is dated and the health-seeking behavior of PWID could have changed since the SFHR study was conducted in the early 1990s. However, this work provides insights into attitudes during an emerging HIV epidemic.

Future directions for research

First, in this study, the disseminated effect of attitudes was defined as one directional. That is, one individual's exposure affects other's outcome. However, there could be different mechanisms that explain the disseminated effect and future studies could be conducted to better assess this with multiple follow-up visits. A more realistic treatment allocation strategy that allows for correlation of exposure assignment in observational studies could be employed instead of the counterfactual Bernoulli individual group assignment strategy assumption (Barkley et al. 2020; Papadogeorgou et al. 2019). Lastly, the variation in community size could also be considered by using improved estimators such as cluster- and individual-weighted estimators (Basse and Feller 2018). By understanding attitudes among PWID toward HIV/AIDS on their health-seeking behavior, future interventions could be more effective and sustainable in preventing HIV transmission and improving the HIV continuum of care among PWID.

Conclusion

This study reports a novel application of existing causal inference methodology for clustered observational network data to evaluate the dissemination of health attitudes among an observed HIV/AIDS risk network of people who inject drugs. Furthermore, a potentially effective intervention to improve health-seeking behaviors in PWID communities is discussed based on estimated causal effects and the SFHR PWID network properties. Our study provides new insights for developing more effective and sustainable interventions by evaluating the impact of attitudes among PWID toward HIV/AIDS risk on health-seeking behaviors in their risk network.

Appendix A: Community detection

A community (or cluster) is defined as a group of vertices densely connected, with an only sparser connection to other groups of vertices (Newman 2010). Hierarchical clustering is one of the most common methods for community detection (Newman 2010; Kolaczyk and Csárdi 2014). In this method, the closest or most similar vertices are combined to form a community with a measure of similarity or connection strength between vertices based on the network structure (Newman 2010). We employed the modularity-based community detection method (Kolaczyk and Csárdi 2014). This community detection method is not only a simple and most commonly used method but also the sparseness of our SFHR PWID network for analysis can benefit from the modularity-based method to find communities in the network. Define $f_{ij} = f_{ij}(\boldsymbol{C})$ as the fraction of edges in the original network that connects vertices in a community i with vertices in community $j = 1, \ldots, K$ and $i \neq j$. Given this, the modularity of $\boldsymbol{C}$ is defined by

$$mod(\boldsymbol{C}) = \sum_{k=1}^{K} \left[f_{kk}(\boldsymbol{C}) - f_{kk}^{*}\right]^2 \tag{A.1}$$

where f_{kk} is the fraction of edges which connect vertices within the same community k in G, and f_{kk}^{*} is the expected value of f_{kk} under a random edge assignment. Modularity is large when there is a more substantial connection among some vertices than expected, and this suggests the presence of a nontrivial community structure in the network. In practice, the community detection in our PWID network was conducted with "fastgreedy.community" algorithm in "igraph" package in R.

Appendix B: Methods for dissemination

In this Appendix, we provide additional details on the estimands and estimators based on those proposed by Tchetgen Tchetgen and Vandweele (Tchetgen and VanderWeele 2012). To define the potential outcomes, we assume a Bernoulli individual group allocation strategy under which each individual has an independent attitude status (i.e., ILOC/self-blame attribute) and probability of that status is denoted by α (Tchetgen and VanderWeele 2012). To note, we are not assuming that the observed locus of control is an independent Bernoulli random variable; however, this distribution of exposure is used to define the counterfactuals. We assume that the counterfactual attitude status follows a Bernoulli allocation. When there are communities K, each of the communities has n_i individuals for $i = 1, 2, \ldots, K$. The probability of community i's attitude vector is

$$\pi_i(\boldsymbol{A_i}; \alpha) = \prod_{j=1}^{n_i} \alpha^{A_{ij}} (1-\alpha)^{1-A_{ij}} \tag{B.1}$$

so, the probability of community i's attitude vector which excludes the jth individual is

$$\pi_i\left(\boldsymbol{A_{i,-j}}; \alpha\right) = \prod_{k=1, k\neq j}^{n_i} \alpha^{A_{ik}} (1-\alpha)^{1-A_{ik}}. \tag{B.2}$$

When an individual has an attitude $a \in \{a = 0, a = 1\}$ with probability (or coverage) α, his/her average potential outcome is denoted by

$$\bar{Y}_{ij}(a;\alpha) = \sum_{\mathrm{a}_{i,-j} \in \mathcal{A}(n_i-1)} Y_{ij}\left(a,\ _{i,-j}\right)\pi_i\left(\mathrm{a}_{i,-j};\alpha\right). \tag{B.3}$$

The marginal individual average potential outcome is defined by

$$\bar{Y}_{ij}(\alpha) = \sum_{\mathrm{a}_i \in \mathcal{A}(n_i)} Y_{ij}(\mathrm{a}_i)\pi_i(\mathrm{a}_i;\alpha). \tag{B.4}$$

With this notation, a community-level average potential outcome is

$$\overline{Y}_i(a;\alpha) = \frac{1}{n_i}\sum\nolimits_{j=1}^{n_i} \overline{Y}_{ij}(a;\alpha). \tag{B.5}$$

Then, a population-level average potential outcome with a certain coverage α is

$$\overline{Y}(a;\alpha) = \frac{1}{K}\sum\nolimits_{i=1}^{K}\left\{\frac{1}{n_i}\sum\nolimits_{j=1}^{n_i}\overline{Y}_{ij}(a;\alpha)\right\} = \sum\nolimits_{i=1}^{K}\overline{Y}_i(a;\alpha). \tag{B.6}$$

As in the case of the marginal individual average potential outcome, we can express the population average potential outcome with

$$\overline{Y}(\alpha) = \frac{1}{K}\sum\nolimits_{i=1}^{K}\left\{\frac{1}{n_i}\sum\nolimits_{j=1}^{n_i}\overline{Y}_{ij}(\alpha)\right\}. \tag{B.7}$$

To estimate causal effects in the presence of dissemination, we used an inverse probability weighting (IPW) estimator (Tchetgen and VanderWeele 2012; Liu et al. 2016) assuming dissemination only within a community identified by a community detection method. In practice, however, the true propensity scores are often unknown, and one can use the following model:

$$f_{\boldsymbol{A_i}|\boldsymbol{X_i}}(\boldsymbol{A_i}|\boldsymbol{X_i};\theta_x,\theta_s) = \int\prod_{j=1}^{n_i} h_{ij}(b_i;\theta_x)^{A_{ij}}\left\{1-h_{ij}(b_i;\theta_x)\right\}^{1-A_{ij}} f_b(b_i;\theta_s)db_i \tag{B.8}$$

where $h_{ij}(b_i;\theta_x) = \Pr\left(a_{ij}=1|x_{ij},b_i,\theta_x\right) = logit^{-1}\left(x_{ij}\theta_x + b_i\right)$ is a propensity score for the j^{th} individual in community i, and $f_b(\cdot;\theta_s)$ is the density of a community-specific random effect b_i which assumed to follow a normal distribution with mean 0 and variance θ_s (Friedman et al. 2018).

With the estimated community-level propensity score, the IPW estimator for community-level average potential outcomes are calculated by

$$\widehat{Y}_i^{ipw}(a,\alpha) = \frac{\sum_{j=1}^{n_i}\pi_i\left(\boldsymbol{A}_{\boldsymbol{i},-\boldsymbol{j}};\alpha\right)I\left(A_{ij}=a\right)Y_{ij}}{n_i f_{\boldsymbol{A_i}|\boldsymbol{X_i}}\left(\boldsymbol{A_i}|\boldsymbol{X_i};\widehat{\theta}\right)}, \tag{B.9}$$

and the marginal potential outcomes are

$$\widehat{Y}_i^{ipw}(\alpha) = \frac{\sum_{j=1}^{n_i} \pi_i(\boldsymbol{A_i}; \alpha) I(\boldsymbol{A_i}; a) Y_{ij}}{n_i f_{\boldsymbol{A_i}|\boldsymbol{X_i}}\left(\boldsymbol{A_i}|\boldsymbol{X_i}; \widehat{\theta}\right)}. \tag{B.10}$$

Abbreviations

AIDS	Acquired immunodeficiency syndrome
CI	Confidence interval
DE	Direct effect
ELOC	External locus of control
HB	Hepatitis B
HIV	Human immunodeficiency virus
IE	Indirect effect
ILOC	Internal locus of control
IPW	Inverse probability weighted
LOC	Locus of control
MCAR	Missing completely at random
NIDA	National Institute on Drug Abuse
NIH	National Institutes of Health
OE	Overall effect
PLWHA	People live with HIV/AIDS
PWID	People who inject drugs
RD	Risk difference
SD	Standard deviation
SFHR	Social Factors and HIV Risk Study
STI	Sexually transmitted infection
TE	Total effect

Supplementary Information

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Supplementary Material 1.

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Author contributions
Ayako Shimada conducted a literature review, performed the statistical analysis, and contributed manuscript writing. Ashley Buchanan and Natallia Katenka oversaw the methodology and statistical analysis and contributed manuscript writing. Benjamin Skov performed the sensitivity analysis and contributed manuscript writing. Gabrielle Lemire contributed to the methodology and manuscript writing. Stephen Kogut and Samuel Friedman provided substantive guidance for the application of statistical methods and interpretation of the results and contributed to manuscript writing. Samuel Friedman also helped contextualize and explain the SFHR data and study. All authors read and approved the final manuscript.

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Availability of data and materials
A public use version of this dataset is available at https://www.icpsr.umich.edu/web/NAHDAP/studies/22140.

Declarations

Ethics approval and consent to participate
This study was reviewed and approved by the University of Rhode Island Institutional Review Board.

Consent to participate
No additional data collection was required for this project. SFHR obtained consent from participants.

Competing interests
The authors declare no competing interests.

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