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Measures of HIV community viral load and HIV incidence among people who inject drugs

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Abstract

Purpose—To evaluate measures of community HIV viral load (VL) and the association with HIV incidence among people who inject drugs (PWID).

Methods—Data was from the 1986 to 1999 Urban Health Study (UHS) conducted among PWID in the San Francisco Bay Area. Extant measures of community VL use mean VL among HIV+ study participants, not accounting for the proportion of HIV− individuals. We compared the strength of the associations between HIV incidence and the traditionally measured mean community VL and a new prevalence-adjusted community VL, calculated by dividing the sum of VL among HIV+ participants by the total participants irrespective of HIV status.

Results—Mean community VL was not correlated with HIV incidence among this sample of PWID (r_s=0.32, p=0.28). However, prevalence-adjusted community VL was strongly correlated with HIV incidence (r_s=0.69, p=0.009). Nested complimentary log-log linear models indicated that increases in community VL and prevalence-adjusted community VL were both associated with HIV incidence, but prevalence-adjusted community VL was a more sensitive measure (Hazard Ratio[HR]=1.28, p=.038 and HR=3.29, p<.001, respectively).

Conclusions—The effect of community VL on HIV incidence may be stronger than previously reported. Future studies of community VL surveillance should consider accounting for the prevalence of HIV using a prevalence-adjusted community VL measure.

Keywords

HIV; community viral load; viral load; People who Inject Drugs; HIV incidence; HIV epidemiology

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Introduction

In 2004, Fang and colleagues reported that after the initiation of a program offering free highly active antiretroviral (HAART) therapy for HIV, the incidence of HIV in Taiwan decreased by 53% within five years. [1] They suggested that this decrease may have resulted from the reduction in HIV viral load (viral load) in the population after widespread HAART treatment. This led some researchers to propose that community level viral load might be an important marker for population-level HIV transmission [2].

Several studies have examined the relationship between community HIV viral load and HIV seroconversion, including in Vancouver, Canada [3], San Francisco, California [4], and British Columbia, Canada [5, 6]. Each study found that reductions in community HIV viral load corresponded to reductions in HIV incidence in the communities and there has since been a global push to adopt HIV treatment as prevention [7, 8]. HIV "treatment as prevention (TasP)" strategies posit that in order to reduce HIV incidence, public health efforts should focus on HIV treatment among HIV positives to reduce viral load and thereby HIV transmission [9, 10, 11].

Whereas community viral load was intended to be a measure of the potential infectivity and transmission among people living with HIV to the broader community, there are concerns over the definition of community viral load used in these studies. CDC guidelines define community viral load as the mean viral load among *HIV positive* people in a specific population [12]. However, this does not account for the prevalence of HIV in the community, which has been noted by several researchers [13, 14]. We hypothesize that for an accurate assessment of the effect of TasP it is necessary to account for the prevalence of HIV in the community of interest. For illustration, consider a hypothetical Community X consisting of 1,000 people. If only one person in Community X had HIV and that person's viral load was very high, then the calculation for community viral load would be very high. However, chances of HIV transmission in Community X would be low because there is only one person who is HIV positive. If in Community Y, also consisting of 1,000 people, 100 people were HIV positive, but their mean viral load was low, then the community viral load would be low according to the current definition. However, the likelihood of transmission is likely higher in Community Y than in Community X despite a lower mean community viral load, because there are many more people from whom transmission can occur. By including the number of HIV negative people in the denominator of the mean community viral load calculation, we argue that the definition would better reflect community risk and be relevant for monitoring HIV transmission risk and the effectiveness of prevention activities. Despite this concern, studies continue to measure community viral load not accounting for the prevalence of HIV and the CDC guidelines remain unchanged since 2011 [12, 15, 16]. This study seeks to demonstrate the importance of adjusting for HIV prevalence in community viral load measures by comparing the association between community viral load and HIV incidence under two definitions (mean community viral load and prevalence-adjusted community viral load) to evaluate their use in community surveillance and TasP evaluation activities. Using data from the Urban Health Study, a unique dataset from one of the few long-running studies of people who inject drugs (PWID) in the U.S. (conducted from 1986

to 2005), we present an analysis of the utility of community viral load in predicting subsequent HIV incidence in a real-world setting.

Methods

Selection and Description of Participants

The Urban Health Study was a serial cross-sectional study with an embedded, passive cohort, that recruited PWIDs in three inner-city communities in San Francisco, California from 1986 to 2005, and in Oakland and an adjacent city - Richmond, California, from 1993 to 2002 [17–20]. Participants were recruited in neighborhood settings using targeted sampling methods in communities selected for their high concentrations of PWID [21]. High-risk communities were identified using drug-treatment admission data, police arrest data, direct observation, and ethnographic studies. To be eligible to participate, individuals had to have engaged in injection drug use in the past 30 days and be 18 years of age or older. New respondents were screened for visible signs of recent subcutaneous or intravenous drug use (i.e. tracks, or recently punctured veins). Every 6 months, a new sample was recruited; previous respondents could participate in subsequent surveys irrespective of whether they had continued to inject drugs, but were not actively asked to return to the study. Blood samples were collected at each wave, which enabled HIV testing and allowed the assessment of prevalence cases of HIV from all participants and incident cases of HIV-1 infection from repeat visits of participants [19, 22]. While the study did not require the participants to give their names or addresses, repeat participants were identified through the use of five key identifying data (sex, race/ethnicity, birth date, first two to four letters of mother's maiden name, and state of birth). Data included in these analyses are a subset of the total Urban Health Study and include those who participated in San Francisco from 1987 to 1999 and Oakland and Richmond from 1992 to 1999. Data post 1999 was not included in these analyses due to a reduction in sample size that resulted in low incidence of HIV among repeat participants.

Participants underwent phlebotomy to provide two to four 5ml tubes of blood. One tube went to a lab that tested for HIV-1 antibodies using ELISA. Positive specimens were confirmed as positive through Western blot if the results showed reactive bands at two of the following locations: p24 or gp41; and gp120 or gp160 [18]. The other tubes were separated into serum using a centrifuge and sent to the UCSF AIDS Specimen Bank for banking at −70 degrees Celsius in a freezer. A subset of these specimen was tested for HIV viral load in 2012 (procedure discussed below). The linear range of quantification for the viral load assay was 20–10,000,000 copies/mL. Participants were asked to return for HIV post-test disclosure 2 to 4 weeks after the interview, with return rates over 90% completed. Participants were remunerated $15 to $20 for the initial interview and phlebotomy session, as well as $15 to $20 for the HIV disclosure counseling session.

Sample size per year ranged from 898 to 2,241 (Table 1). Sample size increased in 1992 when additional data collection sites were added in Oakland and Richmond, CA. The sample size for participants who participated in consecutive years, and were therefore eligible for the incidence calculation ranged from 140 in 1987 and 1988 to 1,030 in 1993 and 1994. The median number of participants who participated in consecutive years was 460.5. The number

of people selected for viral load testing at each wave varied considerably. At minimum, 22% of HIV positive individuals were selected for viral load analysis in a given year. At most, 70% of HIV positive individuals were selected for viral load analysis. More information on viral load testing is provided in the *Measures* section.

Statement of IRB/Human Study Assurances

All participants provided informed consent. The consent form was read aloud to each participant, who would initial rather than sign the form to maintain confidentiality. The Institutional Review Boards at the University of California, San Francisco and RTI International approved all study methods.

Measures

Outcome—Annual HIV incidence: A crude annual incidence rate was calculated for HIV infection as the proportion of susceptible (HIV negative) participants who became HIV positive. Respondents who were HIV positive at baseline were excluded from the incidence calculations. Once respondents became HIV positive during the study, they were excluded from the incidence calculation for subsequent years (censored). We calculated incidence for each year for all participants who either a) participated in both waves in the year of interest or b) participated in one of the two semi-annual assessments and participated in the first wave of the next year. If participants were HIV negative for these assessments, they were considered susceptible and included in the denominator. If a participant was HIV positive at one of the two six-month follow-ups, they were counted as an incident case for that year and included in the numerator and denominator. Participants who were HIV negative during the year of interest, but tested positive during the first wave of the following year were counted as an incident case for the year of interest and excluded from calculations for the subsequent years (thereby avoiding being counted twice).

Exposure—Viral Load: Due to resource constraints, viral load testing was conducted on only one sample per HIV positive respondent even if the respondent participated in more than one wave of the study. Viral load for each HIV positive respondent was measured from the first HIV positive test of the respondent, which means we eliminated potential retention biases. Viral load for each HIV positive UHS participant (n=1,375) was determined from serum samples stored at the University of California San Francisco AIDS Specimen Bank. Samples were tested at the Duke Human Vaccine Institute Immunology and Virology Quality Assessment Center using the FDA approved Roche COBAS Ampliprep/COBAS Taqman 48 system. Briefly, 1-milliliter of sample was added to the assay system. Viral RNA was extracted and reverse-transcribed. The assay then amplified and detected a portion of the HIV-gag gene. Results were given as the number of viral copies per milliliter. Each run of samples included three kit controls, high positive, low positive, and a negative. In addition, with each run we performed an internal control that is then tracked over many runs for within laboratory result variation monitoring. Of the 1,375 samples tested, 126 samples (9.1%) did not yield usable results due to failure of the sample to amplify through PCR, failure to detect virus, or viral copies below the quantifiable range for the assay. This rate of sample failure is to be expected given the systematically lower viral copy counts observed in serum versus plasma samples [23]. Nonetheless, comparisons of viral load within results

determined from the same bodily fluid are valid. The range of log10 copies of virus/mL for the 1,151 respondents included in these analyses (1987 to 1999) was 2.3 to 6.45 with a mean of 4.33.

The Prevalence-Adjusted Community Viral Load is a variant of the calculation of community viral load used in previously published studies. In the seminal paper by Das and colleagues [4], mean community viral load was an algebraic mean calculated only among HIV positive individuals and did not account for the proportion of negative persons in the population. In our analyses, the conceptual formula for calculating alternate community viral load was the sum of the viral load from HIV positive individuals divided by the total number of HIV negative and positive people at that wave, thus accounting for the number of people with an effective viral load of zero. In the UHS viral load was not measured for all HIV positive individuals at every wave. For calculating the alternative community viral load we took the sum of available viral load data at each wave and divided it by the total of the number of HIV positive individuals with viral load data for that wave, plus the number of HIV negative participants that maintained the HIV prevalence ratio at each wave. Henceforth, when we refer to the calculation used by Das and colleagues we use the term *mean community viral load* and the calculation that accounts for the number of HIV negative individuals as *prevalence-adjusted community viral load*.

Other Measures—HIV Prevalence: HIV prevalence in the UHS sample was calculated as the proportion of respondents who tested positive out of all respondents participating in that wave. UHS data collection waves occurred in 6 month increments. For these analyses, this data was aggregated into annual measures. For respondents who participated in both waves of a year, if the respondent tested HIV positive at either wave they were considered HIV positive for that year.

HIV Treatment: Self-reported participation in current HAART HIV treatment data (yes/no) was assessed beginning in 1997 when HAART became available. Treatment data were aggregated into annual measures in the same manner described above for HIV prevalence.

Analyses

First, HIV viral load (both measures), prevalence, incidence, and treatment were graphically plotted across survey years. Similar to Montaner et al.,[6] we employed simple Spearman rank correlations to examine the point in time correlation between viral load and HIV incidence and the correlation of viral load to the following year. Finally, proc glimmix (SAS®) was used to fit a complimentary log-log linear model with random intercept, to evaluate the incident hazard-ratio associated with increases in log (10) viral load. The model was nested within person to account for repeated measures prospective data and allowed for data missing between waves (i.e. if someone participated in waves 1, 3, and 5, the data was not excluded). Analyses were conducted in SAS® software version 9.3 [24]. All tests were two-tailed with cut-off value of $p<.05$.

Although much has changed in the HIV epidemic since the data used in these analyses were collected (1986 to 1999), the hypothesis that both the number of people infected and their

level of infectivity impact HIV incidence rates can be strongly tested in this cohort and the results are likely to be applicable across periods of the epidemic.

Results

Sample Characteristics

HIV prevalence ranged from 10% to 17% from 1987 to 1999 (Figure 1). HIV incidence dropped sharply from a high of 32 cases per 1000 in 1987 to under 10 cases per 1000 from 1992 forward. HIV incidence among repeat participants ranged from 0.3% to 3.15%. The prevalence of current HIV treatment never rose above 32% in this sample. Community viral load (c per ml) peaked in 1994 at just over 30%, and dropped to less than 15% by 1999. Prevalence-adjusted community viral load was below 5% throughout the observation period.

Association of community Viral Load measures with HIV Incidence

Simple spearman rank correlation of community viral load indicated that mean viral load among HIV positive individuals was not correlated with HIV incidence for the same year (r_s=0.32, p=0.28; Table 2) or HIV incidence in the following year (r_s=0.20, p=0.527). Prevalence-adjusted community viral load was positively associated with the concurrent year's HIV incidence (r_s=0.69, p=0.009) and HIV incidence in the following year (r_s=0.76, p=0.004).

The nested complimentary log-log analyses showed that every point increase in log(10) community viral load was associated with a 28% increase in annual HIV incidence (p=0.038; Table 2) and every point increase in log(10) prevalence-adjusted community viral load was associated with a 229% increase in annual HIV incidence (p<.001).

Discussion

Community viral load is being treated as a marker for HIV infectivity at the population level and has been used as a basis for promoting TasP [4, 8, 25]. A commentary in *Clinical Infectious Diseases* notes "The single most important indicator that HIV treatment and public health programs should be concerned about today is viral load, which is key to both transmission and disease progression [26]." However, only a few studies have examined the relationship between community viral load and HIV incidence; furthermore, none of the identified studies accounted for the proportion of HIV negative individuals in the community viral load calculation [3, 4, 6]. We found that among PWID, each log increase in prevalence-adjusted community viral load (accounting for the proportion of HIV negative people in the community) was associated with over a 3-fold increase in the subsequent rate of HIV incidence. However, the community viral load solely calculated as a mean among individuals with HIV was only weakly associated (1.28-fold increase) with the subsequent rate of HIV infection. These results suggest it is important to incorporate the persons who are HIV negative into the calculations for community viral load. Prior studies which used mean community viral load may have underestimated the relationship of community viral load to community level HIV incidence. Our findings support the need for further research into measures of transmission risk, such as those conducted by Kelley et al. [13], which examines alternate methods for calculating risk in community HIV surveillance activities

and recent work by Solomon and colleagues, which found that another measure of viral load which took HIV prevalence into account (viremia prevalence) was more strongly related to HIV incidence than traditional community viral load [27].

One consideration when interpreting these findings is that the majority of this dataset predates HIV anti-retroviral treatment (ART), which was first made available in 1996 in the U.S. Even after the introduction of HAART, less than one-third of our HIV positive study participants reported currently taking such medications. This was likely due to many important factors during this period (1996–1999), including guidance about when to initiate HAART, the complexity of HAART regimens, stigma against PWID, mistrust between providers and PWID, lack of easy access to treatment facilities, and denial about HIV status [28–33]. The HIV epidemic among PWID in the San Francisco Bay Area was stemmed by reducing HIV incidence and keeping HIV prevalence at moderate levels in large part through more traditional HIV prevention initiatives such as needle and syringe programs and HIV testing and counseling efforts [17, 18, 34]. Additional research is needed to assess the relationship between prevalence-adjusted community viral load and HIV incidence from 2000 on, as it would provide a picture during a period of much higher HAART uptake.

Prior evaluations of measuring community viral load have focused the argument around sexual transmission, with evidence that reduction in viral load due to HAART results in a reduction in transmission via a reduction in the viral load in seminal and vaginal fluid [14, 35]. However, our analyses examined transmission irrespective of method of transmission (injection drug use or sexual risk behaviors), thereby remaining agnostic about the relative risk for sexual transmission and injection transmission. By focusing on a population with high injection drug use, our results suggest that prevalence-adjusted community viral load is an important HIV transmission risk indicator for the community, independent of route of transmission.

This study has several potential limitations to be considered when interpreting its results. First, this study was a repeated cross-sectional study with passive retention methods, which means that we had low retention rates that may have biased explanatory and outcome variables. However, previous analyses have shown that HIV incidence among repeaters in our study from 1987 to 1998 was similar to those who did not return [19]. Second, the participants were not randomly selected, as this is not feasible in community-based studies of PWID due to injection drugs being illegal and stigmatized. This may have led to self-selection bias. Third, we only had funds to conduct viral load testing on the first HIV positive serum sample from each participant, but none from follow-up waves. This likely meant that we were less likely to include participants who were on HAART, as first time UHS participants may have only just learned that they were HIV positive, thus the viral load cannot be assumed to generalize to those receiving antiretroviral treatment. Fourth, the definition of community may be a matter of debate. San Francisco has distinct areas, which may be geographically or culturally different and thereby alter or prevent interactions between residents. Sample size considerations prevent the evaluation of these neighborhood-level effects in the UHS data, but should be considered in future research. Lastly, the data are from 1986 to 1999, and the epidemiology of HIV among PWID has likely changed significantly in the 18 years since these data were collected. However, the main concepts

related to how we calculate community viral load (i.e. both the number of people infected – HIV prevalence - and their level of infectivity – viral load among those infected - impact HIV incidence rates) are as relevant today as they were during this study period.

Despite the limitations, this study contributes to the limited extant research assessing the relationship among community viral load and HIV incidence among PWID. Moreover, it confirms the importance of including HIV prevalence in the calculation of community viral load. In our study, prevalence-adjusted community viral load was much more strongly associated with HIV incidence than traditional community viral load among HIV positive individuals (the calculation promoted by CDC [12]), suggesting that previous studies may have underestimated the predictive utility of community viral load.

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References

1. Fang CT, et al. Decreased HIV transmission after a policy of providing free access to highly active antiretroviral therapy in Taiwan. J Infect Dis. 2004; 190(5):879–85. [PubMed: 15295691]
2. Castel AD, et al. Use of the community viral load as a population-based biomarker of HIV burden. AIDS. 2012; 26(3):345–353. [PubMed: 22008660]
3. Wood E, et al. Longitudinal community plasma HIV-1 RNA concentrations and incidence of HIV-1 among injecting drug users: prospective cohort study. BMJ. 2009; 338:b1649. [PubMed: 19406887]
4. Das M, et al. Decreases in community viral load are accompanied by reductions in new HIV infections in San Francisco. PloS one. 2010; 5(6):e11068. [PubMed: 20548786]
5. de Vos AS, et al. Treatment as prevention among injecting drug users; extrapolating from the Amsterdam cohort study. AIDS. 2014; 28(6):911–918. DOI: 10.1097/QAD.0000000000000190 [PubMed: 24401639]
6. Montaner JS, et al. Association of highly active antiretroviral therapy coverage, population viral load, and yearly new HIV diagnoses in British Columbia, Canada: a population-based study. Lancet. 2010; 376(9740):532–539. [PubMed: 20638713]
7. Hull MW, Wu Z, Montaner JS. Optimizing the engagement of care cascade: a critical step to maximize the impact of HIV treatment as prevention. Curr Opin HIV AIDS. 2012; 7(6):579–586. [PubMed: 23076123]
8. Smith K, et al. HIV-1 treatment as prevention: the good, the bad, and the challenges. Curr Opin HIV AIDS. 2011; 6(4):315. [PubMed: 21646878]
9. Cohen MS, et al. Prevention of HIV-1 Infection with Early Antiretroviral Therapy. N Engl J Med. 2011; 365:493–505. [PubMed: 21767103]
10. Granich RM, et al. Universal voluntary HIV testing with immediate antiretroviral therapy as a strategy for elimination of HIV transmission: a mathematical model. Lancet. 2009; 373(9657):48–57. [PubMed: 19038438]
11. Granich RM, et al. Highly active antiretroviral treatment as prevention of HIV transmission: review of scientific evidence and update. Curr Opin HIV AIDS. 2010; 54:298–304.
12. Centers for Disease Control and Prevention. Guidance on Community Viral Load: A Family of Measures, Definitions, and Method for Calculation. Centers for Disease Control and Prevention; Atlanta, GA: 2011.
13. Kelley CF, et al. Measuring population transmission risk for HIV: an alternative metric of exposure risk in men who have sex with men (MSM) in the US. PloS one. 2012; 7(12):e53284. [PubMed: 23285274]

14. Miller WC, et al. Community viral load as a measure for assessment of HIV treatment as prevention. Lancet Infect Dis. 2013; 13(5):459–464. [PubMed: 23537801]
15. Landis RC, et al. HIV Viral Load Trends in Six Eastern Caribbean Countries Utilizing a Regional Laboratory Referral Service: Implications for Treatment as Prevention. PloS one. 2015; 10(4):e0125435. [PubMed: 25923741]
16. Chakraborty H, et al. HIV community viral load trends in South Carolina. International journal of STD & AIDS. 2016 0956462416642349.
17. Bluthenthal RN, et al. The effect of syringe exchange use on high-risk injection drug users: a cohort study. AIDS. 2000; 14(5):605–611. [PubMed: 10780722]
18. Kral AH, et al. Sexual transmission of HIV-1 among injection drug users in San Francisco, USA: risk-factor analysis. Lancet. 2001; 357(9266):1397–1401. [PubMed: 11356437]
19. Kral AH, et al. Trends in human immunodeficiency virus seroincidence among street-recruited injection drug users in San Francisco, 1987–1998. Am J Epidemiol. 2003; 157(10):915–922. [PubMed: 12746244]
20. Kral AH, et al. Comparing Respondent-Driven Sampling and Targeted Sampling Methods of Recruiting Injection Drug Users in San Francisco. J Urban Health. 2010; 87(5):839–850. [PubMed: 20582573]
21. Watters JK, Biernacki P. Targeted sampling: options for the study of hidden populations. Soc Probl. 1989; 36(4):416–430.
22. Moss AR, et al. HIV seroconversion in intravenous drug users in San Francisco, 1985–1990. AIDS. 1994; 8(2):223–31. [PubMed: 8043227]
23. Lew J, et al. Determinations of levels of human immunodeficiency virus type 1 RNA in plasma: Reassessment of parameters affecting assay outcome. J Clin Microbiol. 1998; 36(6):1471–1479. [PubMed: 9620364]
24. SAS Institute Inc. SAS/STAT® 9.3. SAS Institute Inc; Cary, NC: 2011.
25. Kirk G, et al. Decline in community viral load strongly associated with declining HIV incidence among IDU. 18th Conference on Retroviruses and Opportunistic Infections. 2011
26. De Cock KM. Plus ca change… antiretroviral therapy, HIV prevention, and the HIV treatment cascade. Clin Infect Dis. 2014; 58(7):1012–4. [PubMed: 24429442]
27. Herbeck J, Tanser F. Community viral load as an index of HIV transmission potential. The Lancet HIV. 2016; 4(3):e152–e154.
28. Bassetti S, et al. Why is highly active antiretroviral therapy (HAART) not prescribed or discontinued? Swiss HIV Cohort Study. J Acq Immun Def Synd. 1999; 21(2):114–119.
29. Clarke S, et al. Assessing limiting factors to the acceptance of antiretroviral therapy in a large cohort of injecting drug users. HIV Medicine. 2003; 4(1):33–37. [PubMed: 12534957]
30. Dieffenbach CW, Fauci AS. Universal voluntary testing and treatment for prevention of hiv transmission. JAMA. 2009; 301(22):2380–2382. [PubMed: 19509386]
31. O'Connor PG, Selwyn PA, Schottenfeld RS. Medical care for injection-drug users with human immunodeficiency virus infection. N Engl J Med. 1994; 331(7):450–9. [PubMed: 8035842]
32. Sherer R. Adherence and antiretroviral therapy in injection drug users. JAMA. 1998; 280(6):567–568. [PubMed: 9707152]
33. Strathdee SA, et al. Barriers to use of free antiretroviral therapy in injection drug users. JAMA. 1998; 280(6):547–549. [PubMed: 9707146]
34. Bluthenthal RN, et al. Approval of syringe exchange programs in California: Results from a local approach to HIV prevention. Am J Public Health. 2008; 98(2):278–283. [PubMed: 17538068]
35. Attia S, Egger M, Müller M, Zwahlen M, Low N. Sexual transmission of HIV according to viral load and antiretroviral therapy: systematic review and meta-analysis. AIDS. 2009; 23(11):1397–1404. [PubMed: 19381076]

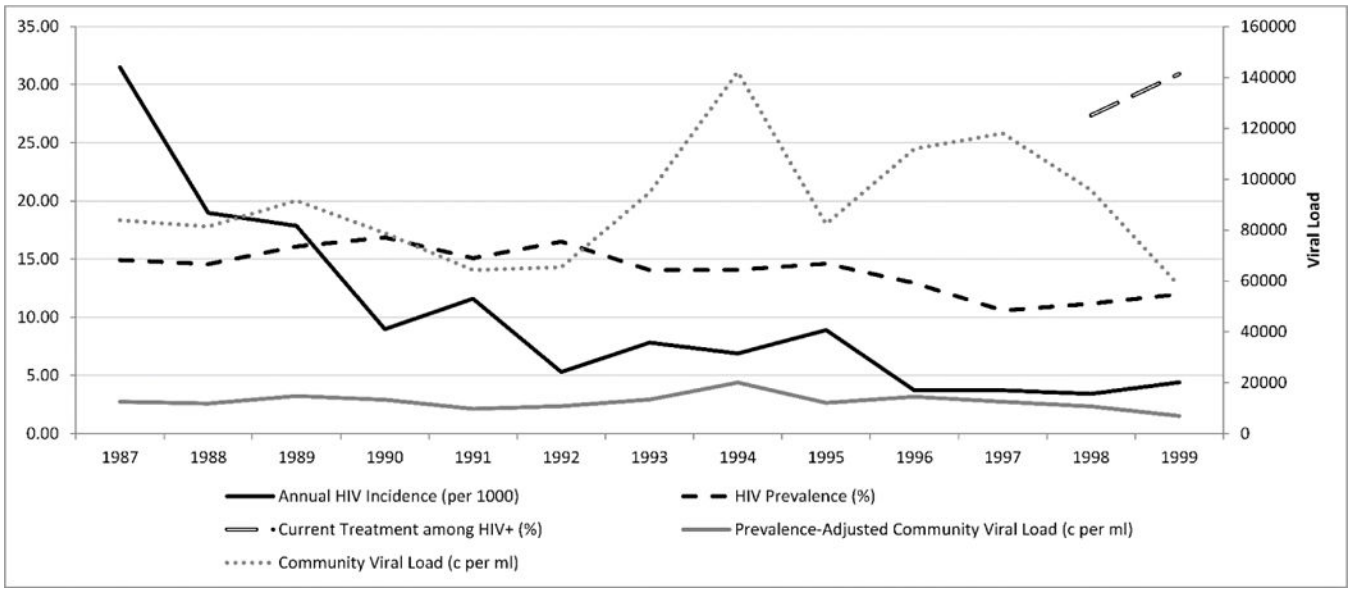

Figure 1.

HIV Prevalence, Incidence, Treatment, and Viral Load among People Who Inject Drugs in San Francisco Bay Area from 1987 to 1999.

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Table 1

UHS Sample Size Characteristics by Year.

Year	Total N	N (%) participated in next year	N HIV− (%)	N HIV+ (%)	N HIV+ Selected for Viral Load (%)
1987	1106	N/A	941 (85.08)	165 (14.92)	77 (46.67)
1988	1085	140 (12.7)	927 (85.44)	158 (14.56)	110 (69.62)
1989	1001	152 (14.0)	840 (83.92)	161 (16.08)	108 (67.08)
1990	882	217 (21.7)	733 (83.11)	149 (16.89)	89 (59.73)
1991	898	257 (29.1)	762 (84.86)	136 (15.14)	35 (25.74)
1992[1]	1929	290 (32.3)	1610 (83.46)	319 (16.54)	197 (61.76)
1993	2054	927 (48.1)	1765 (85.93)	289 (14.07)	107 (37.02)
1994	1990	1030 (50.1)	1710 (85.93)	280 (14.07)	88 (31.43)
1995	1883	929 (46.7)	1609 (85.45)	274 (14.55)	71 (25.91)
1996	1910	731 (38.8)	1662 (87.02)	248 (12.98)	115 (46.37)
1997	1794	878 (46.0)	1604 (89.41)	190 (10.59)	50 (26.32)
1998	1903	780 (43.5)	1684 (88.49)	219 (11.51)	49 (22.37)
1999	1924	631 (31.2)	1693 (87.99)	231 (12.01)	55 (23.81)

[1]Second city added to data collection. N/A= Not Applicable

Table 2

Spearman Rank Correlations and Hazard Ratios of Viral load and HIV Incidence

	Community Viral Load	Prevalence-Adjusted Community Viral Load
Spearman Rank Correlation: Same Year		
r_s	0.32	0.692
p-value	0.28	0.009
Spearman Rank Correlation: Following year		
r_s	0.2	0.762
p-value	0.53	0.004
Hazard Analysis		
Hazard Ratio	1.28	3.29
p-value	0.038	<.001