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Antiretroviral Drug Resistance and Risk Behavior Among Recently HIV-Infected Men Who Have Sex With Men

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Abstract

Objectives—Examine associations among behaviors including substance use during sexual encounters, and transmitted HIV drug resistance in recently HIV-infected men who have sex with men (MSM).

Methods—Between 2002 and 2006, 117 recently HIV-infected MSM completed questionnaires regarding their 3 most recent sexual partners. Serum samples were tested for the presence of genotypic and phenotypic HIV drug resistance. Logistic regression analysis was used to assess the association of substance use, behaviors, and resistance to at least 1 class of HIV drugs.

Results—The mean age of participants was 35 years; 71% identified as white and 19% as Hispanic. Sixty (51%) reported substance use during sexual activity in the past 12 months. A total of 12.5% of 112 had genotypic drug resistance to at least 1 class of antiretroviral medications, and 14% of 117 had phenotypic drug resistance. Substances used during sexual activity associated with phenotypic drug resistance in multivariate models included any substance use (adjusted odds ratio [aOR] = 4.21, 95% confidence interval [CI]: 1.13 to 15.68), polysubstance use (aOR = 5.64, 95% CI: 1.62 to 19.60), methamphetamine (aOR = 4.00, 95% CI: 1.19 to 13.38), 3,4-methylenedioxy-N-methylamphetamine (MDMA)/Ecstasy (aOR = 7.16, 95% CI: 1.40 to 36.59),

and γ -hydroxyl butyrate (GHB) (aOR = 6.98, 95% CI: 1.82 to 26.80). The genotype analysis was similar.

Conclusions—Among these recently HIV-infected MSM, methamphetamine use during sexual activity and use of other substances, such as MDMA and GHB, was associated with acquired drug-resistant virus. No other behaviors associated with acquisition of drug-resistant HIV.

Keywords

HIV antiviral resistance; men who have sex with men; sexual behavior; substance use

The increasing prevalence of drug-resistant strains of HIV has caused concern about the transmission of resistant strains of HIV and the future effectiveness of antiretroviral therapy (ART). Prevalence of primary or transmitted drug resistance has been measured as high as 26% in North America and Western Europe,^{1–4} with evidence from mathematic modeling studies showing the potential for substantial transmission of drug resistance.⁵ Although the prevalence of primary drug resistance is well documented, less is known about mechanisms and risk factors for transmission. Significant public health interest was given to a case of transmitted multiple drug-resistant HIV found in a man with primary HIV infection who reported methamphetamine use, leading to speculation about the role of substance use in the acquisition of resistant HIV.⁶ Additionally, there is empiric evidence associating use of illicit substances, such as methamphetamine, amyl nitrites (poppers), cocaine, and Ecstasy, with high-risk sexual behavior^{7–9} and with incident sexually transmitted infections, including HIV, among men who have sex with men (MSM).^{10–12} Higher rates of substance use and HIV among MSM, compared with the general population, highlight the importance of understanding the potential role of substance use in the transmission of drug-resistant HIV in this population.^{13–16} Although frequent methamphetamine use reported by MSM has been associated with transmitted resistance,¹⁷ the significance of its use during sexual activity remains unclear. Possible mechanisms that may facilitate transmission of resistant virus include reductions in immune function because of substance use, repeated exposure by the same partner, concentration of resistant HIV in certain sexual networks, or specific sexual practices during substance use. This study examines the associations between reported use of substances, including methamphetamine, amyl nitrites, cocaine, γ -hydroxyl-butyrate (GHB), 3,4-methylenedioxy-N-methylamphetamine (MDMA), and marijuana, during sexual encounters, other risk behaviors, and the presence of transmitted HIV drug-resistant strains among a cohort of treatment-naïve recently HIV-infected MSM.

METHODS

The Southern California Acute Infection and Early Disease Research Program (SC-AIEDRP) recruited, enrolled, and collected biologic data on a cohort of recently HIV-infected individuals (infection within the past 12 months), as previously described.^{18,19} Between May 2002 and December 2006, 225 HIV-infected MSM completed questionnaires to assess HIV risk behavior by computer-assisted self-administered interview (CASI). This analysis includes 117 MSM for whom we had HIV resistance data and who responded to detailed questions at baseline about their 3 most recent sexual partners, including partner characteristics, types of sexual activity, and types of substances used just before or during

sexual activity with that partner within 12 months previous to their diagnosis. Participants were asked if they used methamphetamine, volatile nitrites, MDMA, ketamine, GHB, cocaine, marijuana, or other substances when having sexual activity with each of their 3 most recent partners in the past 12 months.

All serum samples from baseline were sequenced by population-based nucleotide sequencing of the whole pro-tease gene (codons 1 to 99) and codons 1 to 305 of the reverse transcriptase gene. Sequencing was performed locally (Viroseq v.2.0; Celera Diagnostics, Alameda, CA) or centrally (GeneSeq HIV; Monogram Biosciences, Inc., South San Francisco, CA). Genotypic resistance (GR) was determined using the Stanford algorithm, with resistance defined as a score of 60 or greater with regard to nonnucleoside reverse transcriptase inhibitors (NNRTIs), nucleoside reverse transcriptase inhibitors (NRTIs), protease inhibitors (PIs), amino acid revertants at codon 215, or any combination of these types of resistance. Drug susceptibility testing was performed at baseline using a recombinant virus assay (PhenoSense HIV; Monogram Biosciences, Inc.).²⁰ Reduced drug susceptibility was defined as a fold change (FC) in the 50% inhibitory concentration (IC₅₀) over the biologic cutoff (99th percentile of the FC distributions among wild-type viruses).²¹ For the purpose of study analyses, an FC of 10 was used to indicate high-level reduced susceptibility for each drug. Phenotypic resistance (PR) data were available for all 117 participants, whereas GR data were only available for 112 (96%) participants.

Associations between phenotypic or genotypic drug resistance and risk behaviors were evaluated using *t* tests, the Wilcoxon rank sum test, Mantel-Haenszel χ^2 methods, and the Fisher exact test, and multivariate logistic regression analysis was used to assess the association of behaviors reported and resistance to at least 1 class of HIV drugs. An a priori model was specified, and all analyses were conducted using SAS software, version 9.1 (SAS Institute Inc., Cary, NC).

RESULTS

Among the recently HIV-infected MSM, the mean age was 35 years, most identified as white (71%) or Hispanic (19%), and nearly half had completed college (47%). The median number of sexual partners in the 12 months before the interview was 25 (mean = 53; interquartile range [IQR]: 10 to 50), with a median of 7 anonymous partners (mean = 20) and 3 single-time partners (mean = 7). All participants reported sex with men in the past year, and 5 (4%) reported sex with men and women. The median total number of unprotected sex acts across the 3 most recent partners was 5, with a mean of 47 and a range of 0 to 516 (IQR: 2 to 33), with 10 people reporting 100 or more unprotected sex acts. Sixty MSM (51%) reported substance use during sexual activity in the past 12 months with at least 1 of their 3 most recent partners, with 35% reporting use of multiple substances. Methamphetamine was the most commonly used drug reported during sexual activity with the 3 most recent partners (34%), followed by volatile nitrites (29%), marijuana (24%), and GHB (19%).

Genotypic drug resistance to at least 1 class of anti-retroviral medications was identified in 14 (12.5%) of 112 participants, and phenotypic drug resistance was identified in 16 (14%) of

117 participants. Differences in the prevalence of resistance by test type are partly explained by 2 additional cases of PR detected among those for whom genotype testing data were not available. In addition, NNRTI polymorphism resulting in susceptibility changes that just reached the threshold of major reduced susceptibility showed no major GR mutations in 2 cases. However, the overall level of agreement between genotypic and phenotypic drug resistance was excellent ($\kappa = 0.87$).

Drug resistance was found in a greater percentage of MSM who reported substance use during sexual activity with a partner in the past 12 months as compared with those who did not report substance use, with a significant difference seen among those reporting polysubstance use (use of more than 1 substance with the same partner), GR (54% vs. 30%; $P = 0.09$), and PR (63% vs. 31%; $P = 0.01$) (Table 1). When phenotype findings were limited to those who also had genotype data available ($n = 112$), the differences in PR and polysubstance use still remained (57% vs. 30%; $P = 0.04$). More than half of those reporting methamphetamine use had evidence of resistant virus as compared with 30% of nonusers (GR: $P = 0.08$; PR: $P = 0.04$). More resistance was also found among those who reported the use of MDMA and GHB. Comparisons of those with drug resistance with those with wild-type virus revealed no differences in terms of demographics, behavioral factors, or characteristics of the 3 most recent partners, including number of sexual encounters in the past 12 months, duration of sexual activity, having a main partner, having unprotected anal intercourse (UAI), or having a known positive partner. Substance users were not different from those who did not use substances by age, race/ethnicity, having 1 or more of their 3 most recent partners being HIV-seropositive, exchanging money or goods for sex, or average duration of sex. However, a greater percent of substance users than those not using substances reported UAI (92% vs. 78%; $P = 0.03$), use of erectile dysfunction medication in the past 12 months (55% vs. 35%; $P = 0.03$), group sex in the past 12 months (83% vs. 64%; $P = 0.03$), more total partners (median = 30 vs. 15; IQR: 12 to 50 vs. 8 to 40; $P = 0.015$), and more anonymous sex partners (median: 15 vs. 3, IQR: 2 to 31 vs. 0 to 15; $P = 0.002$).

Separate multivariate models for each substance use variable controlled for age, race/ethnicity, total number of sex partners in the past 12 months, at least 1 of the 3 most recent partners reported as being HIV-seropositive, and previous exposure to antiretroviral medications ($n = 3$). Associations with primary phenotypic drug resistance were found for use of the following during sexual activity: any substance use (adjusted odds ratio [aOR] = 4.21, 95% confidence interval [CI]: 1.13 to 15.68), polysubstance use (aOR = 5.64, 95% CI: 1.62 to 19.60), methamphetamine (aOR = 4.00, 95% CI: 1.19 to 13.38), MDMA/Ecstasy (aOR = 7.16, 95% CI: 1.40 to 36.59), and GHB (aOR = 6.98, 95% CI: 1.82 to 26.80) (see Table 1). Similar trends were noted in the genotype analysis, with only GHB and MDMA/Ecstasy not reaching significance, although the smaller sample size may have limited the power to detect these associations. None of the other behavioral covariates were associated with resistant virus in non-aORs or aORs.

The association between substance use and resistance was also examined by CD4 cell count and viral load (VL). No differences were observed in the CD4 cell count and VL status of those who used substances compared with those who did not. The mean CD4 count was 563 cells/ μ L for substance users as compared with 515 cells/ μ L for nonusers (range: 226 to 1344

cells/ μ L and 254 to 1006 cells/ μ L, respectively; P value for difference = 0.16). The mean VL was 4.5 $\log_{10}$ copies/mL (range: 2.3 to 6.1 $\log_{10}$ copies/mL) for substance users and 4.6 $\log_{10}$ copies/mL (range: 2.6 to 6.0 $\log_{10}$ copies/mL; P value for difference = 0.54). In addition, no differences were observed in CD4 cell count and VL status among those who had resistance at baseline as compared with those who did not have resistance. For those with PR compared with those without resistance, the mean CD4 count was 527 cells/ μ L (range: 226 to 949 cells/ μ L) versus 541 cells/ μ L (250 to 1344 cells/ μ L; P value for difference = 0.78) and the mean VL was 4.6 $\log_{10}$ copies/mL versus 4.6 $\log_{10}$ copies/mL (P value for difference = 0.94). The same pattern was noted for GR, with no difference by CD4 cell count or viral load level.

DISCUSSION

Our study confirms another report¹⁷ and provides greater insight indicating that for MSM, there is an association between use of methamphetamine and transmitted drug-resistant HIV. We provide further evidence that this association persists if methamphetamine use occurs during sexual activity and suggest that use of other substances during sex, such as MDMA and GHB, may also be associated with acquired drug-resistant virus. Unlike previous studies that demonstrated associations between methamphetamine use in general and transmitted drug resistance, we found an association between the use of methamphetamine and other substances during sexual activity while accounting for differences in sexual practices and level of activity. Given the relatively high levels of substance use observed among MSM,^{13,14,16} methamphetamine, MDMA, and GHB may play an important role in the transmission of HIV drug resistance in this population, suggesting that resistant viruses may circulate among networks of MSM who use illicit drugs.

Our analysis included a number of behavioral factors not previously examined as possible mechanisms for higher efficiency of transmission of resistant virus among substance users; for example, the level of sexual exposure. This was measured by the number of protected and unprotected sexual encounters for the 3 most recent partners, existence of a main partner, and duration of sexual activity. None of these measures of amount of sexual exposure were associated with having resistant virus. We also analyzed differences in CD4 cell counts and HIV-1 RNA levels by drug resistance and substance abuse and found no differences for any of these comparisons. Factors that were possibly related to sexual networks were also examined. When 1 of the 3 most recent partners was met in a social setting, such as with friends, at work, or in their neighborhood, participants were less likely to report substance use with sexual activity (17% vs. 26%), whereas those reporting substance use reported more anonymous partners over the previous 12 months. This suggests that drug users may be more likely to have sex with partners unlinked from more socially based networks in which many MSM meet sexual partners. In addition to a higher likelihood of high-risk sexual behavior among drug users, partners in these sexual networks of drug users come from a much larger pool and enhance mixing across sexual networks.

It has been suggested that individuals acquire resistant virus from partners who are also drug users and who have developed resistance as a result of poor adherence to their HIV medication.¹⁷ Although we were unable to examine substance and medication use of sex

partners directly, the percentage reporting HIV-seropositive partners was not different among those who reported substance use as compared with those who did not report substance use. We also tried to examine whether MSM with resistant virus were more likely to report sexual partners who were also drug users; however, there was too much missing data to test this statistically. Moreover, partner reports of partner behavior have been previously shown to be unreliable.²² Therefore, the possibility that individuals acquire resistant virus from partners who are substance users remains difficult to demonstrate empirically and was beyond the limits of this study.

Our analysis has several limitations. The relatively small sample size, particularly in terms of the GR profile, limited our ability to detect differences across groups or to examine potential interaction effects. The behavioral information collected in this study was based on self-report and is subject to the inherent biases associated with this method of data collection, although this was minimized by our use of CASI. The questionnaire focused on risks around sexual activity; therefore, we did not measure such risk behaviors as substance use in general or depression that may be associated with the practice of these behaviors. Finally, our analysis may not be representative of other at-risk populations, because our study sample was limited to MSM in the southern California area. Nonetheless, the cohort is one of the largest of recently HIV-infected individuals and uniquely provides detailed and extensive behavioral and biologic data.

Clearly, heavy use of substances, especially methamphetamine, at the time of sexual activity increases practice of behaviors associated with acquiring HIV. Furthermore, continued substance use among those infected has been shown to reduce adherence and increase the likelihood of the development of resistance.²³ Therefore, the need for prevention programs to reduce abuse of illicit substances so as to control the spread of HIV and to enhance the health of those infected is compelling and urgent. However, the role of substance use in the acquisition of transmitted resistant virus is not clear. This and a previous study were conducted in urban centers in California in which drug use and sexual behavior patterns among MSM are relatively similar.¹⁷ For instance, use of such substances as heroin and cocaine may be more prevalent among MSM in the eastern United States, whereas methamphetamine use may be more prevalent among those on the West Coast.^{13,24} There is a need for more studies that examine behavioral factors associated with acquired HIV drug resistance in settings with different patterns of substance use. Finally, studies are needed to examine physiologic differences between those who acquire transmitted resistant virus and those exposed to similar risk factors who do not.

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TABLE 1

Association Between Substance Use With Sex in the Past Year and HIV Drug Resistance Among MSM With Acute and Early HIV Infection

Variable	GR		No GR		GR*		aOR (95% CI) [†]
	n	(%)	n	(%)	Non-aOR (95% CI)	aOR (95% CI)	
Total	14	(12.5)	98	(87.5)	—	—	—
Any substance use	9	(64.3)	46	(46.9)	2.04	(0.64 to 6.51)	3.17 (0.85 to 11.86)
Polysubstance use	8	(57.1)	30	(30.6)	3.02	(0.96 to 9.47)	4.26 (1.19 to 15.26)
Methamphetamine	8	(57.1)	28	(28.6)	3.33	(1.06 to 10.48)	4.63 (1.27 to 16.91)
Ecstasy	3	(21.4)	6	(6.1)	4.18	(0.91 to 19.13)	4.44 (0.81 to 24.23)
Poppers	5	(35.7)	28	(28.6)	1.40	(0.43 to 4.51)	—
GHB	5	(35.7)	16	(16.3)	2.85	(0.84 to 9.62)	3.94 (0.98 to 15.84)
Cocaine	1	(7.7)	8	(8.2)	0.87	(0.10 to 7.49)	—
Marijuana	5	(35.7)	21	(21.4)	2.04	(0.62 to 6.73)	—
Vicodin	1	(7.1)	4	(4.1)	1.81	(0.19 to 17.44)	—

Variable	PR		No PR		PR*		aOR (95% CI) [†]
	n	(%)	n	(%)	Non-aOR (95% CI)	aOR (95% CI)	
Total	16	(13.7)	101	(86.3)	—	—	—
Any substance use	11	(68.8)	49	(48.5)	2.34	(0.76 to 7.21)	4.21 (1.13 to 15.68)
Polysubstance use	10	(62.5)	31	(30.7)	3.76	(1.26 to 11.27)	5.64 (1.62 to 19.60)
Methamphetamine	9	(56.3)	31	(30.7)	2.90	(0.99 to 8.50)	4.00 (1.19 to 13.38)
Ecstasy	4	(25.0)	6	(5.9)	5.28	(1.30 to 21.41)	7.16 (1.40 to 36.59)
Poppers	6	(37.5)	28	(27.7)	1.56	(0.52 to 4.71)	—
GHB	7	(43.8)	15	(14.8)	4.46	(1.44 to 13.80)	6.98 (1.82 to 26.80)
Cocaine	1	(6.2)	9	(8.9)	0.68	(0.08 to 5.77)	—
Marijuana	6	(37.5)	22	(21.8)	2.16	(0.71 to 6.58)	—
Vicodin	1	(6.2)	4	(4.0)	1.62	(0.17 to 15.46)	—

* GR models based on a sample size of 112; PR models based on a sample size of 117.

[†] aORs represent separate models; each model is adjusted for age, race, total number of sex partners in the past 12 months, HIV-positive partner, previous exposure to antiretroviral medications, and specific drug variable noted.