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High-intensity cannabis use associated with lower plasma HIV-1 RNA viral load among recently-infected people who use injection drugs

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Abstract

Introduction and Aims—Cannabis use is common among people who are living with HIV/AIDS. While there is growing pre-clinical evidence of the immunomodulatory and anti-viral effects of cannabinoids, their possible effects on HIV disease parameters in humans is largely unknown. Thus, we sought to investigate the possible effects of cannabis use on plasma HIV-1 RNA viral loads among recently-seroconverted illicit drug users.

Design and Methods—We used data from two linked longitudinal observational cohorts of people who use injection drugs. Using multivariable linear mixed-effects modeling, we analysed the relationship between pVL and high-intensity cannabis use among participants who seroconverted following recruitment.

Results—Between May, 1996 and March, 2012, 88 individuals seroconverted after recruitment and were included in these analyses. Median pVL in the first 365 days among all seroconverters was 4.66 log10 c/mL. In a multivariable model, at least daily cannabis use was associated with 0.51 log10 c/mL lower pVL ($\beta = -0.51$, Standard Error = 0.170, p-value = 0.003).

Discussion—Consistent with the findings from recent *in vitro* and *in vivo* studies, including one conducted among lentiviral-infected primates, we observed a strong association between cannabis use and lower pVL following seroconversion among illicit drug-using participants.

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Conclusion—Our findings support the further investigation of the immunomodulatory or anti-viral effects of cannabinoids among individuals living with HIV/AIDS.

Keywords

Plasma HIV-1 RNA viral load; cannabis; cannabinoids; HIV infection; disease progression

INTRODUCTION

Despite the development of highly-active antiretroviral therapy (HAART), people who use illicit drugs continue to experience high levels of preventable HIV/AIDS-related morbidity and mortality (1). To date, studies from a wide variety of settings indicate that people who use illicit drugs (DU) have lower rates of HAART initiation (2), are less likely to achieve virological suppression (3), and experience higher rates of mortality (4).

Beyond the barriers to optimal HAART access and adherence faced by people who use drugs, there are also concerns about the possibility of deleterious direct effects of specific illicit drugs on HIV disease progression (5). A number of studies have identified links between common psychoactive agents, including cannabis, heroin and cocaine, and relevant immunologic or virologic parameters (6–10). For example, morphine was found to promote, in a dose-dependent fashion, the replication of HIV-1 in a culture of human peripheral blood mononuclear cells (9). Similarly, long-term cocaine administration was associated with immune system impairment in a murine model of retroviral infection (7). However, among people living with HIV/AIDS (PLWHA) in the pre-HAART era, the evidence on the relationship between illicit drugs and HIV disease progression was contradictory and, in the HAART era, disease course is largely driven by patterns of exposure to combination antiretroviral therapy (5).

High levels of cannabis use are reported by people living with HIV/AIDS, in attempts to ameliorate the side-effects of antiretroviral therapy as well as recreationally (11,12). Although many jurisdictions are reforming legal prohibitions to facilitate licit access to so-called medical marijuana, the scientific evidence base for cannabinoids is limited and their effect on HIV disease parameters such as plasma HIV-1 RNA viral load (pvL) is largely unknown. However, there is a growing body of literature from pre-clinical studies identifying immunomodulatory and anti-viral capacities of cannabinoids (13–15). Recently, Molina *et al*. used simian immunodeficiency virus (SIV)-infected rhesus macaques, a model system for lentiviral infection, to experimentally test the possible effects of delta-9-tetrahydrocannabinol (Δ^9-THC), the primary psychoactive constituent of cannabis (16). Animals exposed to chronic administration of Δ^9-THC prior to and following SIV infection exhibited lower plasma SIV-RNA viral loads and lengthened survival. In this study, we sought to replicate these findings in humans by retrospectively analyzing data from individuals newly-infected with HIV in order to investigate the possible effects of cannabis on pVL.

METHODS

Data for these analyses was accessed from the Vancouver Injection Drug User Study (VIDUS) and the AIDS Care Cohort to evaluate Exposure to Survival Services (ACCESS), two linked prospective observational cohorts based in Vancouver, Canada. These studies have previously been described in greater detail elsewhere (17,18). Briefly, VIDUS is an ongoing prospective cohort of HIV-negative people who use injection drugs (IDU), while ACCESS is an ongoing prospective cohort of HIV-positive people who use illicit drugs (DU). Both studies, which operate out of the same facility, began recruitment in May, 1996 and focused on the city's Downtown Eastside (DTES) neighbourhood, a post-industrial area with high rates of poverty, illicit drug use and HIV infection. Individuals are eligible for inclusion if they are aged ≥ 18 years and have used illicit drugs via injection (VIDUS) or any illicit drug other than cannabis (ACCESS) in the previous month and can provide written informed consent.

In both studies, at the baseline and every biannual study visit thereafter, participants respond to an interviewer-administered questionnaire on illicit drug use patterns and related issues, are examined by a study nurse and provide blood for serologic analyses. All VIDUS participants are tested for HIV infection at each six-month follow-up. Baseline HIV-negative individuals who seroconvert during follow-up are transferred from the VIDUS to the ACCESS study. Both the VIDUS and ACCESS studies have been approved by the University of British Columbia/Providence Healthcare Research Ethics Board.

In this study, we included all individuals who tested negative for HIV infection at the baseline VIDUS visit and then seroconverted to HIV infection as indicated by a positive and confirmed test either through the study or from a healthcare provider. We estimated the date of seroconversion as the mid-point between the date of the last negative antibody test and the first positive antibody test. We excluded individuals who did not have ≥ 1 interview in ACCESS within 365 days of the estimated date of seroconversion.

Information on HIV serostatus and illicit drug use gathered through the interview and examination process is augmented by data on HIV/AIDS clinical monitoring and antiretroviral therapy (ART) held by the British Columbia Centre for Excellence in HIV/AIDS (BCCfE), as described in detail elsewhere (19). Briefly, the BCCfE has provided ART and related care free of charge to all individuals living with HIV/AIDS in British Columbia by government mandate since 1992. Through a confidential linkage to BCCfE data, a complete retrospective and prospective clinical profile, including data on all ART dispensations and the results of every plasma HIV-1 RNA viral load (pVL) tests conducted in the province of BC is available for each study participant. In this study, we excluded all individuals who did not undergo ≥ 1 pVL test within 365 of the estimated date of seroconversion. We also censored individuals from the date of the first dispensation of any antiretroviral therapy during the first 365 days following the estimated date of seroconversion.

Using this analytic sample, we tested the hypothesis that high-intensity cannabis use was (i.e., ≥ daily use) associated with lower pVL independent of possible confounding factors.

Our outcome of interest was all pVL measurements taken during the first 365 days following the estimated date of seroconversion. These were obtained through the confidential linkage detailed above and included all measurements conducted through the study as well as any conducted outside of the study setting, for example, by a participant's personal physician. The Roche Amplicor Monitor assay was used to determine pVL from participant blood samples (Roche Molecular Systems, Pleasanton, California, United States.)

The primary explanatory variable was cannabis use in the six month period prior to the interview, dichotomized as ≥ daily vs. < daily. We also included secondary explanatory variables that we hypothesized might be associated with both cannabis use and pVL, such as: Age (per year increase); sex (female vs. male); Caucasian ancestry (yes vs. no); any injection drug use in the past six months (yes vs. no); any non-injection drug use in the past six months (defined as the use of any illicit drug other than cannabis via a non-parenteral route; yes vs. no), and any alcohol use. Because we have previously observed poorer housing status to be associated with higher pVL as well as well as malnutrition, we also included homelessness (yes vs. no). All of these variables save sex and ancestry were time-updated and refer to the six month period prior to the interview.

As a first step, we built a boxplot to visually compare all pVL measurements stratified by high-intensity cannabis use. Next, we used contingency tables including Odds Ratios (OR) and *p*-values to investigate the distribution of all explanatory variables stratified by the median of the first pVL observation from each participant. To model the relationship between pVL and cannabis use while accounting for multiple observations per participant, we systematically fit a series of linear mixed effects models with random intercepts and random slopes, as in previous longitudinal analyses of pVL. All models included the primary explanatory variable; to some we added terms for one-knot b-splines or natural spline fit to the time since estimated seroconversion. Models also included Gaussian or autoregressive correlation matrices. We selected the final model form through an examination of each model's Aikaike Information Criterion. Using this form, we fit models for the outcome and each explanatory variable and a final multivariable model including all explanatory variables. All statistical analyses were conducted using R version 2.15.1 (R Foundation for Statistical Computing, Vienna, Austria.)

RESULTS

Between May, 1996 and March, 2012, 149 individuals who were HIV-negative at their VIDUS baseline interview later tested positive for HIV infection. Of these, 88 (62%) individuals completed at least one ACCESS interview and had ≥ 1 pVL observation within the 365 days following the estimated date of seroconversion and therefore were included in these analyses. Individuals included did not differ from those excluded by gender, age or ancestry (all $p > 0.05$.)

These 88 individuals contributed 184 pVL observations during the study period. The median of all pVL observations was 4.66 log10 c/mL (inter-quartile range [IQR] = 4.11 – 5.08.) As shown in Figure 1, median pVL was 0.55 log10 c/mL lower during periods of at least daily cannabis use compared to others (4.73 vs. 4.18, $p = 0.003$.) In the cross-sectional analyses of

all explanatory variables stratified by the value of the first pVL observation (> 4.7 vs. ≤ 4.7 log10 c/mL) shown in Table 1, there was no significant difference observed between individuals reporting at least daily cannabis use (Odds Ratio = 0.34, 95% Confidence Interval = 0.08 – 1.45, *p*-value = 0.184.) At least daily cannabis use was associated with lower pVL (β = −0.44, SE = 0.170, *p*-value = 0.010) in a bivariate linear mixed effects model with no spline term and a Gaussian correlation matrix. In a multivariable linear mixed effects model, at least daily cannabis use was independently associated with lower pVL (β = −0.51, SE = 0.170, *p*-value = 0.003) after adjustment for age, gender, ancestry, homelessness, alcohol use, injection drug use and non-injection drug use.

DISCUSSION

In this study, we observed significantly lower pVL among people reporting at least daily cannabis use in the first year following HIV seroconversion. This difference persisted in a multivariable statistical model in which high-intensity cannabis use was associated with 0.51 lower log10 c/mL pVL after adjustment for possible confounders.

We are aware of only two studies that have assessed the relationship between exposure to cannabis and pVL (20,21). In 2003, Abrams *et al*. observed no significant differences in pVL among 67 HIV-positive patients randomly assigned to smoke marijuana, ingest a 2.5-capsule of dronabinol (Δ^9-THC) or ingest a placebo capsule three times daily before meals for 21 days (21). More recently, Ghosn *et al*. found that cannabis use during sexual intercourse was significantly associated with higher likelihoods of elevated seminal plasma viral load in an observational study of 157 men who have sex with men on successful combination antiretroviral therapy (20). Unlike our study among ART-naïve individuals, both studies were conducted among individuals engaged on ART. Also, Ghosn *et al*. did not adjust their multivariable results for ART adherence, allowing for the possibility that the observed association was the result of the neuropsychological effects of cannabis use on adherence to treatment.

As our results were derived from an observational study where exposure to cannabis was not randomly assigned, we cannot exclude the possibility that the observed association was the result of unmeasured confounding or some other form of error. However, the results were robust to adjustment by possible confounders and, in addition, we do not believe individuals differentially reported cannabis use based on their pVL levels. Although an abundance of caution should be exercised whenever inferring similarities between data generated in primates and human participants, the observed association is consistent with the findings of Molina *et al*. from their experiment involving chronic exposure to Δ^9-THC among rhesus macaques experimentally infected with SIV (16). In that study, monkeys exposed to Δ^9-THC exhibited lower viral loads in plasma and cerebrospinal fluids, greater retention of body mass, attenuated inflammation and lengthened survival compared to placebo.

The current findings should be evaluated in light of a growing body of evidence generated from pre-clinical settings on the structure and function of the endocannabinoid receptor system and its possible role in HIV disease. Cannabinoids, including Δ^9-THC, bind to receptors expressed by cells in the nervous and immune systems (22) and, in addition to

their well-known psychoactive effects, have been shown to have immunosuppressive and anti-inflammatory properties (22–25). These may be the result of cannabinoid-mediated changes in immunologic functioning through pathways including the production of pro-inflammatory cytokines and lymphocytes (25,26). In individuals infected with HIV, the creation and maintenance of chronic inflammatory states is correlated with increased viral replication driven by cytokines such as TNF-α. In addition to these immunomodulatory pathways, a direct antiviral effect of cannabinoids has been proposed (6,27). One experiment showed WIN55,212-2, a synthetic cannabinoid receptor agonist, suppressed replication of HIV-1 in microglia, the major cell type productively infected in the human nervous system (6).

To conclude, we retrospectively analyzed longitudinal cohort data from individuals who use injection drugs and were recently infected with HIV. In a multivariate model controlling for possible confounders, at least daily cannabis use was associated with 0.51 log10 c/mL lower plasma HIV-1 RNA viral load. We believe this is the first study to describe a possibly beneficial effect for cannabinoids on HIV disease progression among humans. Our results support further investigation of the possible virological and immunological aspects of cannabinoid exposure among people living with HIV/AIDS.

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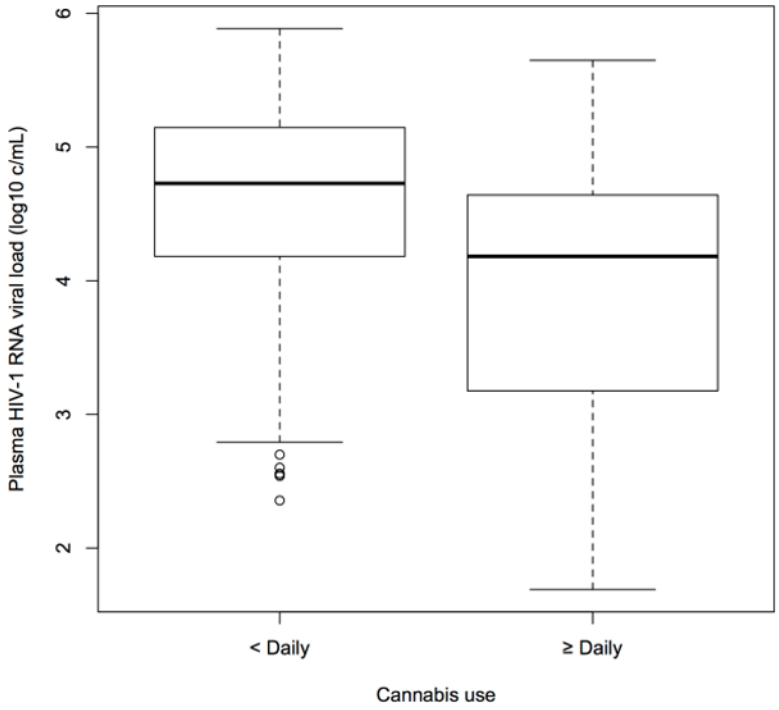

Figure 1.

Boxplot of plasma HIV-1 RNA viral load observations stratified by cannabis use among 88 people who use illicit drugs with recent HIV infection

Table 1

Characteristics of 88 people who use injection drugs with recent HIV infection stratified by first plasma HIV-1 RNA viral load (pVL) observation (≤ 4.7 log10 c/mL vs. > 4.7)

Characteristic	**pVL ≤ 4.7 42 (47.7) n (%)**	**pVL > 4.7 46 (52.3) n (%)**	**OR[1]**	**95% CI[2]**	***p*-value**
Cannabis use					
< Daily	35 (83.3)	43 (93.5)	1.00		
≥ Daily	7 (16.7)	3 (6.5)	0.34	0.08 – 1.45	0.184
Age (per 10 years)					
Median (IQR)	3.6 (2.9 – 4.0)	3.6 (2.9 – 4.5)	1.00	0.89 – 1.14	0.955
Gender					
Male	21 (50.0)	28 (60.9)	1.00		
Female	21 (50.0)	18 (39.1)	0.64	0.28 – 1.50	0.391
Ancestry					
Non-Caucasian	22 (52.4)	18 (39.1)	1.00		
Caucasian	20 (47.6)	28 (60.9)	1.71	0.73 – 3.99	0.284
Homeless					
No	37 (88.1)	42 (91.3)	1.00		
Yes	5 (11.9)	4 (8.7)	0.70	0.18 – 2.82	0.731
Injection drug use					
No	3 (7.1)	3 (6.5)	1.00		
Yes	39 (92.9)	43 (93.5)	1.10	0.21 – 5.79	0.908
Non-injection drug use					
No	11 (26.2)	20 (43.5)	1.00		
Yes	31 (73.8)	26 (56.5)	0.46	0.19 – 1.14	0.090
Alcohol use					
No	17 (40.5)	25 (54.3)	1.00		
Yes	25 (59.5)	21 (45.7)	0.57	0.25 – 1.33	0.193

[1]Odds Ratio;

[2]95% Confidence Interval

Table 2

Bivariable and multivariable analyses of factors associated with plasma HIV-1 RNA viral load (copies/mL, per log10) among 88 individuals who use injection drugs recently infected with HIV-1

Characteristic	Bivariable			Multivariable		
	β	SE[1]	p-value	β	SE[1]	p-value
Cannabis use (≥ Daily vs. < daily)[2]	−0.44	0.170	0.010	−0.51	0.170	0.003
Age (per year older)	0.01	0.011	0.193	0.01	0.011	0.419
Gender (Female vs. male)	−0.33	0.177	0.064	−0.20	0.183	0.284
Ancestry (Caucasian vs. non)	0.37	0.176	0.034	0.35	0.180	0.056
Homelessness (Yes vs. no)[2]	0.30	0.186	0.102	0.44	0.191	0.023
Injection drug use (Yes vs. no)[2]	−0.35	0.274	0.202	−0.45	0.273	0.103
Non-injection drug use (Yes vs. no)[2]	−0.01	0.124	0.960	0.02	0.135	0.863
Alcohol use (Yes vs. no)[2]	−0.13	0.133	0.323	−0.17	0.145	0.232

[1] Standard Error;

[2] Refers to six-month period prior to interview