



BACKGROUND

- Cannabis affects many gastrointestinal processes via the endocannabinoid system, which plays an important role in fatty liver diseases (Cohen & Neuman, 2020).
- Liver steatosis is common among people living with human immunodeficiency virus (HIV) and hepatitis C virus (HCV), and represents a significant risk factor for progression to liver fibrosis, cirrhosis, and hepatocellular carcinoma (Stevenson, 2016; Torgensen, 2019).
- There is conflicting evidence regarding the impact of cannabis use on liver steatosis in people living with HIV and/or HCV infections (Barre, 2021; Nordmann, 2018).
- We assessed the relationship between the use of cannabis and hepatic steatosis in a cohort of people living with and without HIV and HCV.

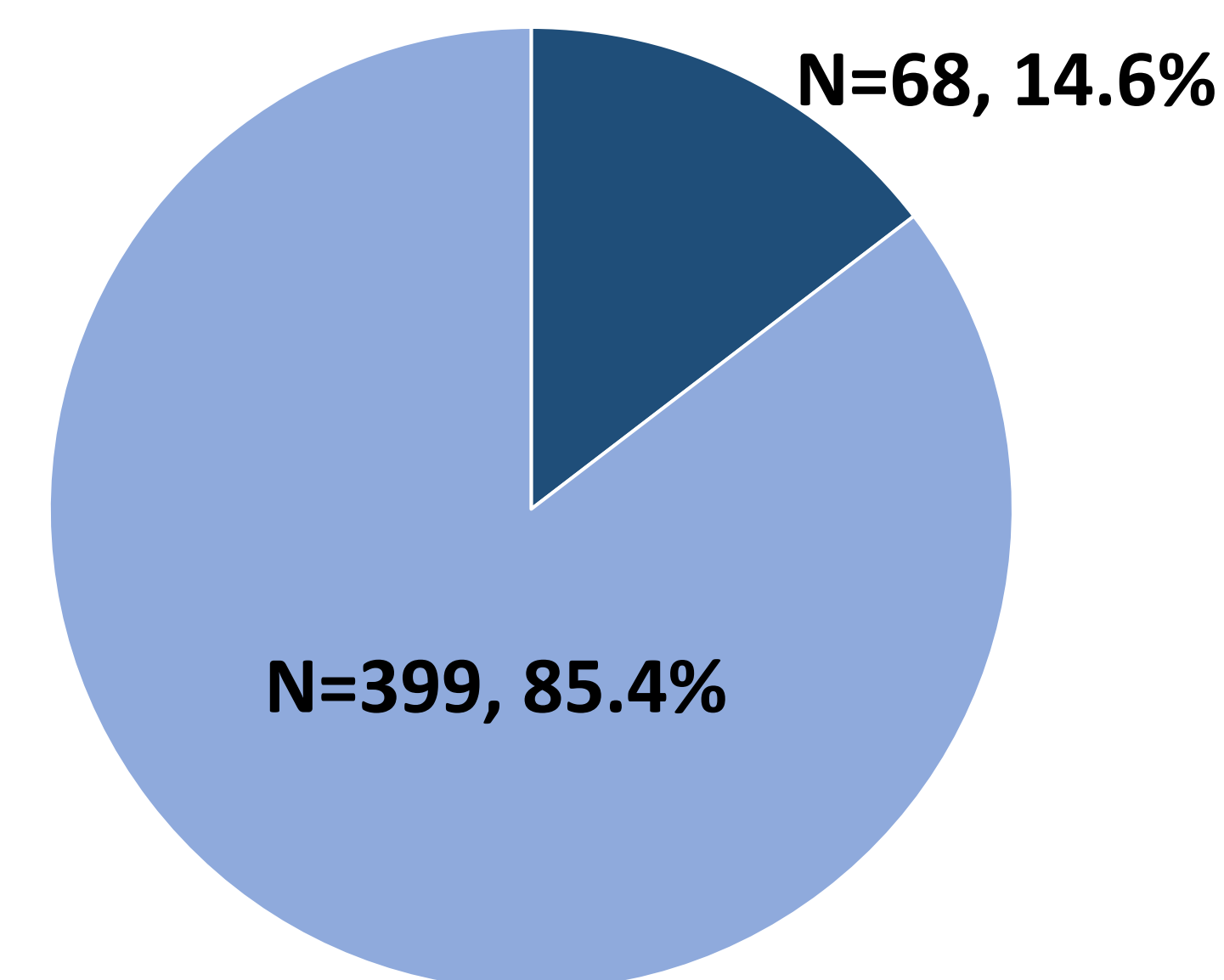
METHODS

- Cross-sectional analysis of data from the Miami Adult Studies on HIV (MASH) cohort.
- The use of cannabis in the past 30 days was self-reported.
- Hepatic steatosis (liver fat content > 5%) was assessed with magnetic resonance imaging-derived proton density fat-fraction (MRI-PDFF). Contraindications for MRI included claustrophobia, ferromagnetic implants, and poor fit due to body size (waist circumference >55 inches/140 cm).
- HIV/HCV status was confirmed with medical records with signed release of medical information.
- Univariate and multivariable binary logistic regressions were performed for hepatic steatosis. Variables with significant associations to hepatic steatosis (P < 0.05) in univariate analyses were selected for the multivariable model.
- This study was approved by the Florida International University Institutional Review Board and all participants provided written consent for participation in the study, release of medical information, and MR assessments.

The use of cannabis may reduce the risk of hepatic steatosis in HIV/HCV-uninfected and HIV mono-infected, but not in HCV-infected individuals.

RESULTS

- There were 467 participants of whom 38.5% were HIV mono-infected, 13.7% were HCV mono-infected, and 12.4% were HIV/HCV co-infected. All people living with HIV were on antiretroviral treatment (ART).
- Sixty-eight participants (14.6%) had hepatic steatosis.
- Multivariate logistic regression showed that BMI (kg/m²; adjusted odds ratio [aOR] 1.11, 95% CI 1.06-1.17, P <0.001) and cannabis use (aOR 0.48, 95% CI 0.26-0.91, P=0.024) were significant independent predictors of hepatic steatosis after adjustment for covariates (race/ethnicity, BMI, smoking, and cocaine use).
- Cannabis use was associated with reduced risk of hepatic steatosis in both obese (relative risk [RR] 0.47, 95% CI 0.22-0.98, P=0.044) and non-obese (RR 0.37, 95% CI 0.14-0.94, P=0.038) participants.
- Bivariate logistic regression analysis stratified by HIV/HCV status was used to study the relationship of BMI and cannabis with hepatic steatosis in HIV and/or HCV. The use of cannabis was associated with lower odds for hepatic steatosis in the HIV/HCV-uninfected and HIV mono-infected groups, but not in HCV-infected groups (Table 1).
- BMI increased the risk of liver steatosis in all groups except in the HCV mono-infected group; shown in Table 1.



■ Liver steatosis ■ No steatosis

CONCLUSIONS

- The use of cannabis may reduce the risk of hepatic steatosis in HIV/HCV-uninfected and HIV mono-infected, but not in HCV-infected individuals.
- The analyses showed this association both in obese and non-obese participants. These findings add to a growing body of conflicting evidence on the impact of cannabis use on hepatic steatosis. Studies are needed to elucidate lifestyle and biologic mechanisms for the beneficial effect of cannabis use observed in this study.

ADDITIONAL KEY INFORMATION

Principal investigator:
Marianna K. Baum, PhD (baumm@fiu.edu)

No conflicts of interest declared.

This work was supported by the National Institute on Drug Abuse [U01DA040381]. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Table 1. Bivariate logistic regression for hepatic steatosis (> 5% fat) stratified by HIV/HCV status

Covariate	HIV/HCV uninfected (n=168)				HIV mono-infected (n=180)			
	Adjusted OR*	95% CI Lower	95% CI Upper	P	Adjusted OR*	95% CI Lower	95% CI Upper	P
BMI, kg/m ²	1.08	1.01	1.16	0.036	1.16	1.07	1.25	<0.001
Cannabis	0.18	0.05	0.64	0.008	0.40	0.16	0.98	0.047
	HCV mono-infected (n=64)				HIV/HCV co-infected (n=58)			
BMI, kg/m ²	1.03	0.85	1.25	0.730	1.14	1.01	1.29	0.041
Cannabis	0.99	0.13	7.49	0.989	1.55	0.36	6.56	0.555

*Adjusted for race/ethnicity, BMI, smoking, and cocaine use (found significantly associated with hepatic steatosis in univariate analysis).