HHS Public Access

Author manuscript

Am J Cardiol. Author manuscript; available in PMC 2023 February 15.

Published in final edited form as:

Am J Cardiol. 2022 February 15; 165: 46–50. doi:10.1016/j.amjcard.2021.10.051.

Relation of Cannabis Use to Elevated Atherosclerotic Cardiovascular Disease Risk Score

Travis M. Skipina, MD[a], **Nikhil Patel, MD**[a], **Bharathi Upadhya, MD**[b], **Elsayed Z. Soliman, MD**[b]

[a]Department of Internal Medicine, Wake Forest School of Medicine, Winston Salem, NC, USA

[b]Cardiovascular Medicine Section, Department of Internal Medicine, Wake Forest School of Medicine, Winston Salem, NC, USA

Abstract

We hypothesized that cannabis use is associated with cardiovascular disease (CVD) risk factors. This could explain the reported link between cannabis and cardiovascular events including stroke and myocardial infarction. This analysis included 7,159 participants (age 37.8±12.4 years, 48.6% male, 61.5% Caucasian) from the National Health and Nutrition Examination Survey years 2011–2018. Cannabis use was defined by self-report. Participants with a history of stroke or myocardial infarction were excluded. Composite CVD risk was assessed using the ACC/AHA 10-year Atherosclerotic Cardiovascular Risk (ASCVD) Score. Participants were classified based on their ASCVD risk levels as: low (<5.0%), borderline (5.0–7.4%), intermediate (7.5–19.9%), and high (≥20.0%). Multinomial logistic regression was used to examine the association between cannabis use and ASCVD risk category using low-risk ASCVD category as the reference level. About 63.9% (n=4,573) of participants had ever used cannabis. Ever cannabis use was associated with 60% increased odds of high-risk ASCVD score [Odds Ratio (OR) and 95% Confidence Interval (95% CI): 1.60 (1.04–2.45), p-value=0.03]. We also observed a dose-response relationship between increased use of cannabis and a higher risk of ASCVD. Those reporting ≥2 uses per month had 79% increased odds of high-risk ASCVD score [OR (95%CI): 1.79 (1.10–2.92), p-value=0.02] and those reporting ≥1 use per day had 87% increased odds of high-risk ASCVD score [OR (95%CI): 1.87 (1.16–3.01), p-value<0.001]. In conclusion, cannabis use is associated with elevated CVD risk. Individuals using cannabis should be screened for CVD risk, and appropriate risk reduction strategies should be implemented.

Corresponding Author: Travis Milan Skipina, MD, Department of Internal Medicine, Wake Forest School of Medicine, 1 Medical Center Boulevard, Winston Salem, NC, USA. Tel: +1 336 716 5039; fax: +1 336 716 4995; tskipina@wakehealth.edu.

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Conflicts of Interest Statement: The authors whose names are listed certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

Keywords

Cannabis; Cardiovascular risk factors; ASCVD; atherosclerosis; cardiometabolic disease

Despite improvements in atherosclerotic risk factor modification and treatment of clinical atherosclerotic cardiovascular disease (ASCVD), recreational drug use remains one of the key modifiable risk factors[1,2]. With a growing prevalence of cannabis use in the young population[3] in the setting of widespread legalization, the importance of risk stratification is paramount. The connection between cannabis use and ASCVD has several proposed pathophysiological mechanisms including thrombosis[4], vasospastic and tachycardia-mediated oxygen supply/demand mismatch[5,6], hyperadrenergic state[7,8], and oxidative stress/endothelial injury[9], all of which are frequently implicated with atherosclerotic cardiovascular events. We hypothesized that cannabis use is associated with CVD risk factors. We tested this hypothesis using a composite of CVD risk factors assessed using the American College of Cardiology/American Heart Association 10-year ASCVD risk score in the National Health and Nutrition Examination Survey (NHANES).

Methods:

The NHANES is a survey of the U.S. population. Data in NHANES were collected from 2011–2018 through a series of interviews and evaluations at an independent examination center[10].

Participants <18 years of age or with a history of myocardial infarction or stroke were excluded from the analysis. Those with any missing data regarding cannabis use, ASCVD components, and any other variables analyzed were excluded. Age, gender, race, tobacco smoking status, and cannabis use were defined by self-report. Ever cannabis users answered affirmative to the question, “Have you ever, even once, used marijuana or hashish?” Users who reported using cannabis at least once per month for a period of at least 12 months answered affirmative to the question, “Have you ever smoked marijuana or hashish at least once a month for more than a year?” Current users listed an integer in response to the question, “During the last 30 days, on how many days did you use marijuana or hashish?” Light users were defined as those who reported using ≤4 days per month; heavy users were defined as those who reported using ≥5 days per month. These metrics have been used in prior studies of NHANES[11,12].

Composite cardiovascular risk was assessed using the American College of Cardiology/American Heart Association 10-year ASCVD score[13]. The ASCVD score is a validated risk assessment tool that estimates cardiovascular risk using a pooled cohort equation that uses age, race, gender, cholesterol, blood pressure, and comorbidities to provide a 10-year estimate of risk for major cardiovascular events[13]. ASCVD scores were reported as percentages. When modeled categorically, the following risk intervals were used: low (<5.0%), borderline (5.0–7.4%), intermediate (7.5–19.9%), and high (≥20.0%).

Diabetes was defined as use of an antihyperglycemic medication, fasting serum glucose ≥126 mg/dL, or hemoglobin A1c ≥6.5%. Hypertension was defined as use of an

antihypertensive medication or systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥80 mmHg according to American Heart Association/American College of Cardiology guidelines[14]. Hyperlipidemia was defined as total cholesterol ≥200 mg/dL, serum triglycerides ≥150 mg/dL, low-density lipoprotein ≥190 mg/dL, or use of lipid-lowering medications. Obesity was defined as body mass index ≥30 kg/m^2.

Population characteristics were compared based on ever cannabis use. Continuous variables were reported as mean ± standard deviation. Categorical variables were reported as frequency and percentage. A chi-square test was used to compare categorical variables and a student's t-test was used to compare continuous variables.

Multinomial logistic regression was used to estimate the association between cannabis use and ASCVD score when ASCVD score was modeled categorically; the low risk ASCVD score category was used as the reference level. Since ASCVD score is calculated using age, gender, ethnicity, diabetes status, hypertension status, tobacco smoking status, and hyperlipidemia, these variables were not adjusted for in the regression models. Associations were also measured in subgroups stratified by race, gender, hypertension, obesity, and hyperlipidemia. In the subgroup analyses, a linear regression model was used to test for interaction with the addition of the interaction term between ever cannabis use and subgroup stratification.

All statistical analyses were conducted using RStudio version 1.3.1093 (Boston, MA) and p-values were considered significant if <0.05.

Results:

In this analysis, there were 7,159 participants (age 37.8±12.4 years, 48.6% male, 61.5% Caucasian) after exclusions. About 63.9% (n=4,573) of participants had ever used cannabis. Population characteristics stratified by cannabis use status are shown in Table 1. Participants who had ever used cannabis were more likely to be male, white, and be current tobacco users. Never cannabis users were more likely to have obesity and diabetes. Ever cannabis users had higher mean ASCVD risk scores.

Table 2 shows the results of the multinomial logistic regression. Ever cannabis use was associated with increased odds of borderline [OR (95% CI): 1.51 (1.23–1.84, p<0.001)], intermediate [OR (95% CI): 1.49 (1.26–1.78, p<0.001)], and high [OR (95% CI): 1.60 (1.04–2.45, p=0.03)] ASCVD scores (reference group: low ASCVD score). Current cannabis use was associated with increased odds of borderline [OR (95% CI): 1.32 (1.01–1.72, p=0.04)] and intermediate [OR (95% CI): 1.37 (1.09–1.73, p=0.007)] ASCVD scores (reference group: low ASCVD score). Current cannabis use was not associated with increased odds of high [OR (95% CI): 1.41 (0.80–2.51, p=0.24)] ASCVD scores (reference group: low ASCVD score).

We observed a dose-response relationship between increased use of cannabis and a higher risk of ASCVD. Reported cannabis use at least once per month for ≥1 year was associated with increased odds of borderline [OR (95% CI): 1.73 (1.38–2.16, p<0.001)], intermediate [OR (95% CI): 1.96 (1.62–2.37, p<0.001)], and high [OR (95% CI): 1.87 (1.16–3.00,

p=0.01)] ASCVD scores (reference group: low ASCVD score). Reported cannabis use ≥2 times per month was associated with increased odds of borderline [OR (95% CI): 1.78 (1.43–2.23, p<0.001)], intermediate [OR (95% CI): 1.90 (1.56–2.30, p<0.001)], and high [OR (95% CI): 1.79 (1.10–2.92, p=0.02)] ASCVD scores (reference group: low ASCVD score). Reported cannabis use ≥1 time per day was associated with increased odds of borderline [OR (95% CI): 1.72 (1.38–2.16, p<0.001)], intermediate [OR (95% CI): 1.94 (1.60–2.35, p<0.001)], and high [OR (95% CI): 1.87 (1.16–3.01, p<0.001)] ASCVD scores (reference group: low ASCVD score).

Table 3 summarizes the results of the multinomial regression when stratified by race, gender, hypertension, obesity, and hyperlipidemia. Results were consistent among sub-groups analyzed. Table 4 shows ASCVD scores stratified by cannabis consumption frequency. Table 5 shows cannabis consumption stratified by hyperlipidemia status.

Discussion:

In this cross-sectional analysis, cannabis use was significantly associated with a composite of CVD risk factors estimated using the 10-year ASCVD risk score. In addition, we also observed a dose-response relationship between increased use of cannabis and a higher risk of ASCVD. These results were consistent in subgroups stratified by demographics and comorbidities.

Cannabis is the most commonly used drug of abuse in the United States[3]. The use of cannabis and its derivatives continues to increase as states approve the legalization of these products for both medicinal and recreational use[15]. ASCVD, which comprises ischemic heart disease, ischemic cerebrovascular disease, and peripheral arterial disease, is a disease no longer restricted to older age. Recent epidemiological data have shown an upsurge in the incidence of all major types of ASCVD in young and middle-aged adults[16]. Understanding the potential cardiovascular risk linked to cannabis use is critical for planning preventive strategies[17].

The effects of cannabis use on the various risk factors of ASCVD are inconsistent. Early reports showed that marijuana use causes tachycardia, peripheral vasodilation, and elevation in both systolic and diastolic blood pressures (BP) when supine[18]. Tachycardia is believed to be a result of increased sympathetic nervous system activity after marijuana use[18]. It is postulated that marijuana-induced sympathetic stimulation increases myocardial oxygen demand. This, along with a decreased oxygen supply due to carboxyhemoglobin formation from inhalation of products of combustion in marijuana cigarettes decreases exercise capacity[19]. In a recent report from the NHANES, recent cannabis use was associated with an increased systolic BP. However, no association was detected between lifetime history of cannabis use and BP levels[20]. Among elderly adults, one prospective cohort study demonstrated a reduction in both diastolic and systolic BP after three months of medical cannabis treatment[21]. Similar inconsistent relationships appear in research at the intersection of cannabis, obesity, and diabetes. Physiologically, preclinical evidence suggests central activation of cannabinoid-1 (CB1) receptors promotes hyperphagia which would plausibly suggest an association with obesity[22]. Furthermore, CB1 receptors activation

in liver increases de-novo fatty acid synthesis, decreases lipolysis, and induces insulin resistance[23]. Despite this backdrop of physiologic evidence, epidemiological studies suggest lower obesity prevalence, lower biomarker levels of impaired glucose metabolism, and lower diabetes prevalence[11,24]. In a meta-analysis, these inverse relationships remain replicable[25] and this “protective paradox” persists. However, a major limitation of these findings is that they were all conducted among various cross-sectional field surveys. Lastly, high-quality evidence regarding the effect of cannabis on lipoproteins remains sparse and inconsistent. In numerous translational studies, cannabinoids have been shown to modulate the immune system, alter lipid metabolism, and affect endothelial cells[26]. This is important because inflammatory cytokines, oxidized low-density lipoprotein and macrophages play key roles in pathogenesis of atherosclerosis[26]. Despite these findings, smoking cannabis appears to have marginal effects in a favorable direction with respect to triglycerides and HDL-C in epidemiological studies[27]. Interestingly, Steffens et al. showed a decrease in progression of atherosclerotic lesions in murine models after oral administration of low-dose THC[28].

Our results corroborate recent findings linking cannabis use to premature ASCVD independent of traditional atherosclerotic risk factors or concomitant use of other drugs[2]. Despite the known synergism between tobacco use and other illicit drugs such as cocaine and cannabis[29], our study adds to a growing body of evidence suggesting the importance of facilitating a discussion among young cannabis users regarding their risk for development of ASCVD. Since we excluded those with a history of CVD, our findings also have implications from a preventive cardiology standpoint.

Our study suffers from numerous limitations. The cross-sectional design is subject to temporality and residual confounding biases. Also, cannabis use was defined by self-report and is vulnerable to both reporting and recall bias, so the prevalence may have been underestimated. We have various categories of cannabis use history, but we do not have data regarding the dosage, route of administration, or periods of abstinence. Further, we do not have data regarding the type of cannabis used. Cannabis is a diverse genus that comes in a wide variety of potencies[30]. Therefore, the differential composition of cannabis may also influence its mechanism of ASCVD. Despite these weaknesses, our study demonstrates a novel link between cannabis use and ASCVD. Other strengths include a racially diverse population and large sample size.

Cannabis use is associated with elevated ASCVD risk score after adjusting for traditional atherosclerotic risk factors.

Acknowledgements:

There were no further contributions to this project beyond those of the listed authors.

Funding Support:

Research reported in this publication was supported by the National Center for Advancing Translational Sciences of the National Institutes of Health under Award Number UL1TR001420 (Soliman). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

References

1. Berry JD, Dyer A, Cai X, Garside DB, Ning H, Thomas A, Greenland P, Van Horn L, Tracy RP, Lloyd-Jones DM. Lifetime risks of cardiovascular disease. N Engl J Med 2012;366:321–329. [PubMed: 22276822]
2. Mahtta D, Ramsey D, Krittanawong C, Al Rifai M, Khurram N, Samad Z, Jneid H, Ballantyne C, Petersen LA, Virani SS. Recreational substance use among patients with premature atherosclerotic cardiovascular disease. Heart 2021;107:650–656. [PubMed: 33589427]
3. The CBHSQ Report. Rockville (MD), 2013.
4. Dahdouh Z, Roule V, Lognone T, Sabatier R, Grollier G. Cannabis and coronary thrombosis: what is the role of platelets? Platelets 2012;23:243–245. [PubMed: 21806494]
5. Sanchez Artiles AE, Awan A, Karl M, Santini A. Cardiovascular effects of cannabis (marijuana): A timely update. Phytother Res 2019;33:1592–1594. [PubMed: 30761633]
6. Jones RT. Cardiovascular system effects of marijuana. J Clin Pharmacol 2002;42:58S–63S. [PubMed: 12412837]
7. Singh A, Saluja S, Kumar A, Agrawal S, Thind M, Nanda S, Shirani J. Cardiovascular complications of marijuana and related substances: a review. Cardiol Ther 2018;7:45–59. [PubMed: 29218644]
8. Ghosh M, Naderi S. Cannabis and cardiovascular disease. Curr Atheroscler Rep 2019;21:21. [PubMed: 30980200]
9. Patel RS, Kamil SH, Bachu R, Adikey A, Ravat V, Kaur M, Tankersley WE, Goyal H. Marijuana use and acute myocardial infarction: a systematic review of published cases in the literature. Trends Cardiovasc Med 2020;30:298–307. [PubMed: 31439383]
10. Plan and operation of the third national health and nutrition examination survey, 1988–94. series 1: programs and collection procedures. Vital Health Stat 1 1994:1–407.
11. Rajavashisth TB, Shaheen M, Norris KC, Pan D, Sinha SK, Ortega J, Friedman TC. Decreased prevalence of diabetes in marijuana users: cross-sectional data from the national health and nutrition examination survey (NHANES) III. BMJ Open 2012;2:e000494.
12. Smit E, Crespo CJ. Dietary intake and nutritional status of US adult marijuana users: results from the third national health and nutrition examination survey. Public Health Nutr 2001;4:781–786. [PubMed: 11415485]
13. Goff DC Jr., Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Gibbons R, Greenland P, Lackland DT, Levy D, O'Donnell CJ, Robinson JG, Schwartz JS, Shero ST, Smith SC Jr., Sorlie P, Stone NJ, Wilson PW, Jordan HS, Nevo L, Wnek J, Anderson JL, Halperin JL, Albert NM, Bozkurt B, Brindis RG, Curtis LH, DeMets D, Hochman JS, Kovacs RJ, Ohman EM, Pressler SJ, Sellke FW, Shen WK, Smith SC Jr., Tomaselli GF, American college of cardiology/american heart association task force on practice G. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the american college of cardiology/american heart association task force on practice guidelines. Circulation 2014;129:S49–73. [PubMed: 24222018]
14. Whelton PK, Carey RM, Aronow WS, Casey DE Jr., Collins KJ, Dennison Himmelfarb C, DePalma SM, Gidding S, Jamerson KA, Jones DW, MacLaughlin EJ, Muntner P, Ovbiagele B, Smith SC Jr., Spencer CC, Stafford RS, Taler SJ, Thomas RJ, Williams KA Sr., Williamson JD, Wright JT Jr., 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: executive summary: a report of the american college of cardiology/american heart association task force on clinical practice guidelines. Hypertension 2018;71:1269–1324. [PubMed: 29133354]
15. Wilkinson ST, Yarnell S, Radhakrishnan R, Ball SA, D'Souza DC. Marijuana legalization: impact on physicians and public health. Annu Rev Med 2016;67:453–466. [PubMed: 26515984]
16. Andersson C, Vasan RS. Epidemiology of cardiovascular disease in young individuals. Nat Rev Cardiol 2018;15:230–240. [PubMed: 29022571]
17. Hasin DS, Kerridge BT, Saha TD, Huang B, Pickering R, Smith SM, Jung J, Zhang H, Grant BF. Prevalence and correlates of DSM-5 cannabis use disorder, 2012–2013: findings from the national

epidemiologic survey on alcohol and related conditions-III. Am J Psychiatry 2016;173:588–599. [PubMed: 26940807]

18. Beaconsfield P, Ginsburg J, Rainsbury R. Marihuana smoking. cardiovascular effects in man and possible mechanisms. N Engl J Med 1972;287:209–212. [PubMed: 4402574]
19. Aronow WS, Cassidy J. Effect of marihuana and placebo-marihuana smoking on angina pectoris. N Engl J Med 1974;291:65–67. [PubMed: 4599385]
20. Alshaarawy O, Elbaz HA. Cannabis use and blood pressure levels: united states national health and nutrition examination survey, 2005–2012. J Hypertens 2016;34:1507–1512. [PubMed: 27270185]
21. Abuhasira R, Haviv YS, Leiba M, Leiba A, Ryvo L, Novack V. Cannabis is associated with blood pressure reduction in older adults - a 24-hours ambulatory blood pressure monitoring study. Eur J Intern Med 2021;86:79–85. [PubMed: 33483174]
22. Seeley RJ, Woods SC. Monitoring of stored and available fuel by the CNS: implications for obesity. Nat Rev Neurosci 2003;4:901–909. [PubMed: 14595401]
23. Silvestri C, Di Marzo V. The endocannabinoid system in energy homeostasis and the etiopathology of metabolic disorders. Cell Metab 2013;17:475–490. [PubMed: 23562074]
24. Penner EA, Buettner H, Mittleman MA. The impact of marijuana use on glucose, insulin, and insulin resistance among US adults. Am J Med 2013;126:583–589. [PubMed: 23684393]
25. Alshaarawy O, Anthony JC. Cannabis smoking and diabetes mellitus: results from meta-analysis with eight independent replication samples. Epidemiology 2015;26:597–600. [PubMed: 25978795]
26. Singla S, Sachdeva R, Mehta JL. Cannabinoids and atherosclerotic coronary heart disease. Clin Cardiol 2012;35:329–335. [PubMed: 22278660]
27. Lazarte J, Hegele RA. Cannabis effects on lipoproteins. Curr Opin Lipidol 2019;30:140–146. [PubMed: 30649023]
28. Steffens S, Veillard NR, Arnaud C, Pelli G, Burger F, Staub C, Karsak M, Zimmer A, Frossard JL, Mach F. Low dose oral cannabinoid therapy reduces progression of atherosclerosis in mice. Nature 2005;434:782–786. [PubMed: 15815632]
29. Falkstedt D, Wolff V, Allebeck P, Hemmingsson T, Danielsson AK. Cannabis, Tobacco, Alcohol use, and the risk of early stroke: a population-based cohort study of 45 000 swedish men. Stroke 2017;48:265–270. [PubMed: 28028147]
30. ElSohly MA, Mehmedic Z, Foster S, Gon C, Chandra S, Church JC. Changes in cannabis potency over the last 2 decades (1995–2014): analysis of current data in the united states. Biol Psychiatry 2016;79:613–619. [PubMed: 26903403]

Table 1:

Population Characteristics

Characteristics	Cannabis User Status	
	n=2586 (36.1%)	n=4573 (63.9%)
	Never	Ever
Age (years)	37.8 ± 12.5	37.8 ± 12.3
Men	1070 (41.4%)	2411 (52.7%) *
White	1493 (57.7%)	2907 (63.6%) *
Non-white	1093 (42.3%)	1666 (36.4%) *
Hypertension	941 (36.3%)	1693 (37.0%)
Obesity	1135 (43.9%)	1802 (39.4%) *
Diabetes mellitus	290 (11.2%)	359 (7.9%) *
Total Cholesterol (mg/dL)	187 ± 40.3	188 ± 42.6
LDL (mg/dL)	112 ± 34.8	111 ± 34.9
HDL (mg/dL)	53.4 ± 15.7	53.4 ± 16.0
Current Tobacco Smoker	275 (10.6%)	1585 (34.7%) *
ASCVD Risk Score (%)	3.0 ± 4.1	3.6 ± 4.6 *

* $p < 0.05$ compared with never users

Obesity = body mass index ≥ 30 kg/m^2

LDL = low-density lipoprotein

HDL = high-density lipoprotein

ASCVD = atherosclerotic cardiovascular disease

Table 2:

Association of Cannabis Use and ASCVD Risk Category

Cannabis Use	Reference Level	ASCVD Risk Category		
		Borderline	Intermediate	High
		OR (95% CI)	OR (95% CI)	OR (95% CI)
Ever User	Never User	1.51 (1.23–1.84, p<0.001)	1.49 (1.26–1.78, p<0.001)	1.60 (1.04–2.45, p=0.03)
Current User	Never User	1.32 (1.01–1.72, p=0.04)	1.37 (1.09–1.73, p=0.007)	1.41 (0.80–2.51, p=0.24)
Every month for ≥ 1 year	Never User	1.73 (1.38–2.16, p<0.001)	1.96 (1.62–2.37, p<0.001)	1.87 (1.16–3.00, p=0.01)
≥ 2 uses per month	Never User	1.78 (1.43–2.23, p<0.001)	1.90 (1.56–2.30, p<0.001)	1.79 (1.10–2.92, p=0.02)
≥ 1 use per day	Never User	1.72 (1.38–2.16, p<0.001)	1.94 (1.60–2.35, p<0.001)	1.87 (1.16–3.01, p<0.001)

OR (95% CI) = Odds Ratio (95% Confidence Interval); ASCVD = atherosclerotic cardiovascular disease;

Borderline = ASCVD score 5.0–7.4%; Intermediate = ASCVD score 7.5%−19.9%; High = ASCVD score ≥20%

Table 3:

Association of Ever Cannabis Use and ASCVD Risk Category Among Sub-groups

Sub-group		ASCVD Risk Category[†]			
		Borderline	**Intermediate**	**High**	
		Odds Ratio *(95% CI)*	**Odds Ratio *(95% CI)***	**Odds Ratio *(95% CI)***	***Interaction p-value*** [*]
Race	*Non-white*	1.23 (0.91–1.66, p=0.18)	1.12 (0.89–1.42, p=0.33)	1.59 (0.91–2.78, p=0.10)	0.17
	White	1.71 (1.31–2.24, p<0.001)	2.08 (1.59–2.72, p<0.001)	1.62 (0.84–3.14, p=0.15)	
Gender	*Men*	1.26 (0.97–1.63, p=0.08)	1.37 (1.09–1.71, p=0.006)	1.24 (0.72–2.15, p=0.44)	0.38
	Women	1.53 (1.11–2.10, p=0.009)	1.17 (0.88–1.56, p=0.29)	1.79 (0.91–3.51, p=0.09)	
Hypertension	*Present*	1.47 (1.14–1.90, p=0.003)	1.28 (1.04–1.57, p=0.02)	1.60 (0.99–2.59, p=0.05)	0.16
	Absent	1.58 (1.13–2.21, p=0.007)	2.89 (1.87–4.47, p<0.001)	1.56 (0.61–3.98, p=0.36)	
Obesity	*Present*	1.26 (0.96–1.66, p=0.09)	1.43 (1.13–1.82, p=0.003)	1.42 (0.85–2.37, p=0.18)	0.76
	Absent	1.82 (1.35–2.44, p<0.001)	1.55 (1.20–2.01, p<0.001)	2.02 (0.92–4.43, p=0.08)	
Hyperlipidemia	*Present*	1.55 (1.20–1.99, p<0.00l)	1.39 (1.11–1.72, p=0.003)	1.38 (0.87–2.20, p=0.17)	0.19
	Absent	1.39 (0.99–1.94, p=0.05)	1.61 (1.19–2.18, p=0.002)	3.32 (0.97–11.33, p=0.06)	

ASCVD = atherosclerotic cardiovascular disease; 95% CI = 95% Confidence Interval; Borderline = ASCVD score 5.0–7.4%;

Intermediate = ASCVD score 7.5%−19.9%; High = ASCVD score ≥20%

[†]Association of ever cannabis use and ASCVD risk category

[*]Interaction p-value calculated from linear regression model

Obesity = body mass index ≥30 kg/m^2

Hyperlipidemia = Total cholesterol ≥200 mg/dL, serum triglycerides ≥150 mg/dL, low-density lipoprotein ≥190 mg/dL, or use of lipid-lowering medications

Table 4:

ASCVD Risk Score and Cannabis Consumption

Cannabis Use	n	ASCVD Risk Score	p-value*
Never User	2586	2.95 ± 4.09	--
Ever User	4573	3.58 ± 4.63	<0.001
Current User	1443	3.29 ± 4.45	0.02
Every month for ≥ 1 year	2362	3.92 ± 4.89	<0.001
≥ 2 uses per month	2219	3.87 ± 4.79	<0.001
≥ 1 use per day	2351	3.91 ± 4.88	<0.001

ASCVD = atherosclerotic cardiovascular disease

*Compared to never users

Table 5:

Cannabis Consumption and Hyperlipidemia

Cannabis Use	n	Hyperlipidemia	p-value*
Never User	2586	1097 (42.4%)	--
Ever User	4573	1966 (43.0%)	0.65
Current User	1443	498 (34.5%)	<0.001
Every month for ≥ 1 year	2362	978 (41.4%)	0.47
≥ 2 uses per month	2219	918 (41.4%)	0.46
≥ 1 use per day	2351	974 (41.4%)	0.48

Hyperlipidemia = Total cholesterol ≥200 mg/dL, serum triglycerides ≥150 mg/dL, low-density lipoprotein ≥190 mg/dL, or use of lipid-lowering medications

*Compared to never users