



~30% of US cannabis users meet criteria for disordered use; cannabis is among the most used substances nationally



Cannabis is often inhaled, used as a sleep aid, and may inflame the upper airway — yet its effect on OSA risk is unknown



Does cannabis use disorder (CUD) increase the risk of developing OSA?



Study Population: TriNetX US Collaborative Network — 67 healthcare organizations, >115M patients (2005–2020)



84,125 adults with CUD vs an equal number of propensity-matched controls (Aim 1)



8,556 patients with OSA who later developed CUD vs matched controls (Aim 2)



29% higher risk of incident OSA at 5 years among adults with CUD (HR 1.29)



Risk was elevated across all cannabis use severity strata, without a stepwise dose–response



2× CPAP prescription at 5 years among patients with OSA who subsequently developed CUD (HR 2.00)

Introduction

- Obstructive sleep apnea (OSA) affects 9–38% of the global population and carries cardiovascular, metabolic, and psychiatric comorbidity.
- Cannabis is widely used (19% of US adults), and interest in cannabinoids as an OSA therapy has grown despite an AASM statement advising against it.
- Small trials of oral dronabinol reduced AHI, but inhaled cannabis may cause airway inflammation, and existing studies are small and conflicting.
- Prior work focuses on patients with pre-existing OSA; the impact of cannabis on OSA onset and treatment needs is unstudied.
- We examined the association between CUD and incident OSA, and CPAP utilization among OSA patients who later developed CUD.**

Methods and Materials

Design: Retrospective propensity-matched cohort study (TriNetX US Collaborative Network; queried May 2026).

Aim 1 cohorts: Adults ≥18 y with ≥5 outpatient visits (2005–2020) and cannabis-related disorder (ICD-10 F12) vs no F12. Stratified by use (F12.9), abuse (F12.1), and dependence (F12.2).

Aim 2 cohorts: Adults with index OSA diagnosis and subsequent CUD vs OSA without CUD.

Exclusions: Prior OSA, central sleep apnea, or lip/oral/pharyngeal cancer.

Matching: 1:1 greedy nearest-neighbor on age, sex, race, ethnicity, BMI, tobacco use, alcohol-related disorders, hypertension, upper respiratory disease, endocrine/metabolic disease, and healthcare utilization (SMD <0.1).

Outcomes: Incident OSA (G47.33) at 1 and 5 years (Aim 1); CPAP prescription (CPT/HCPCS) at 5 years and lifetime (Aim 2).

Analysis: Kaplan–Meier with log-rank tests and Cox proportional hazards models (HR, 95% CI); tobacco-naïve sensitivity analysis.

Results

Result #1. Adults with CUD had **higher incidence of OSA** than matched controls at 1 year (2.7% vs 2.0%; HR 1.38, 95% CI 1.30–1.47) and 5 years (8.6% vs 6.5%; HR 1.29, 1.25–1.34). The association persisted in tobacco-naïve CUD patients (5y HR 1.40, 1.28–1.53).

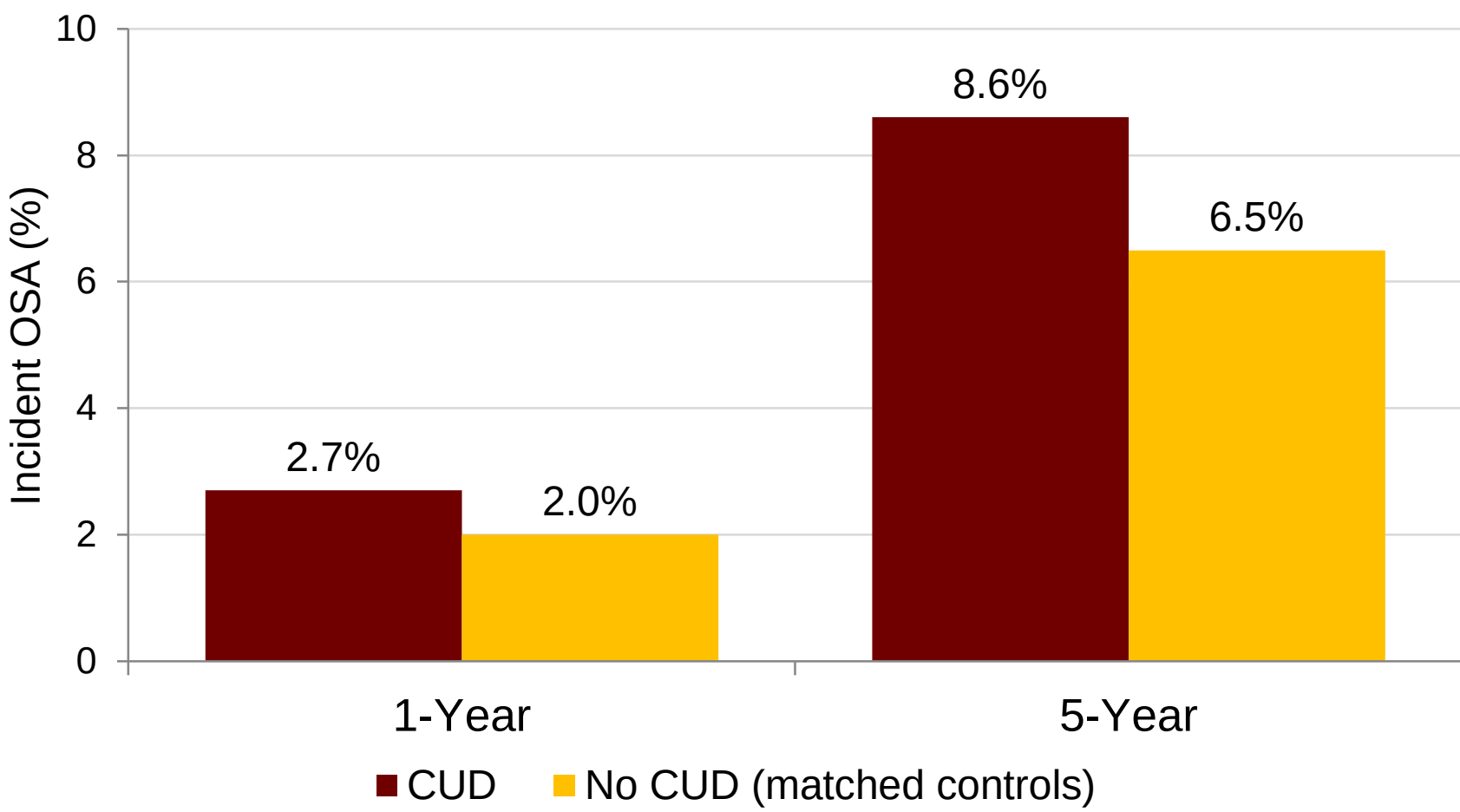


Figure 1. Incidence of OSA at 1 and 5 years after index visit in propensity-matched CUD and control cohorts (log-rank $p<0.001$ for both).

Result #2. At 5 years, **every severity stratum** carried increased OSA risk - cannabis use (HR 1.39), abuse (1.22), and dependence (1.23). Effect sizes were **largest in the lowest-severity stratum** and did not increase stepwise with severity.

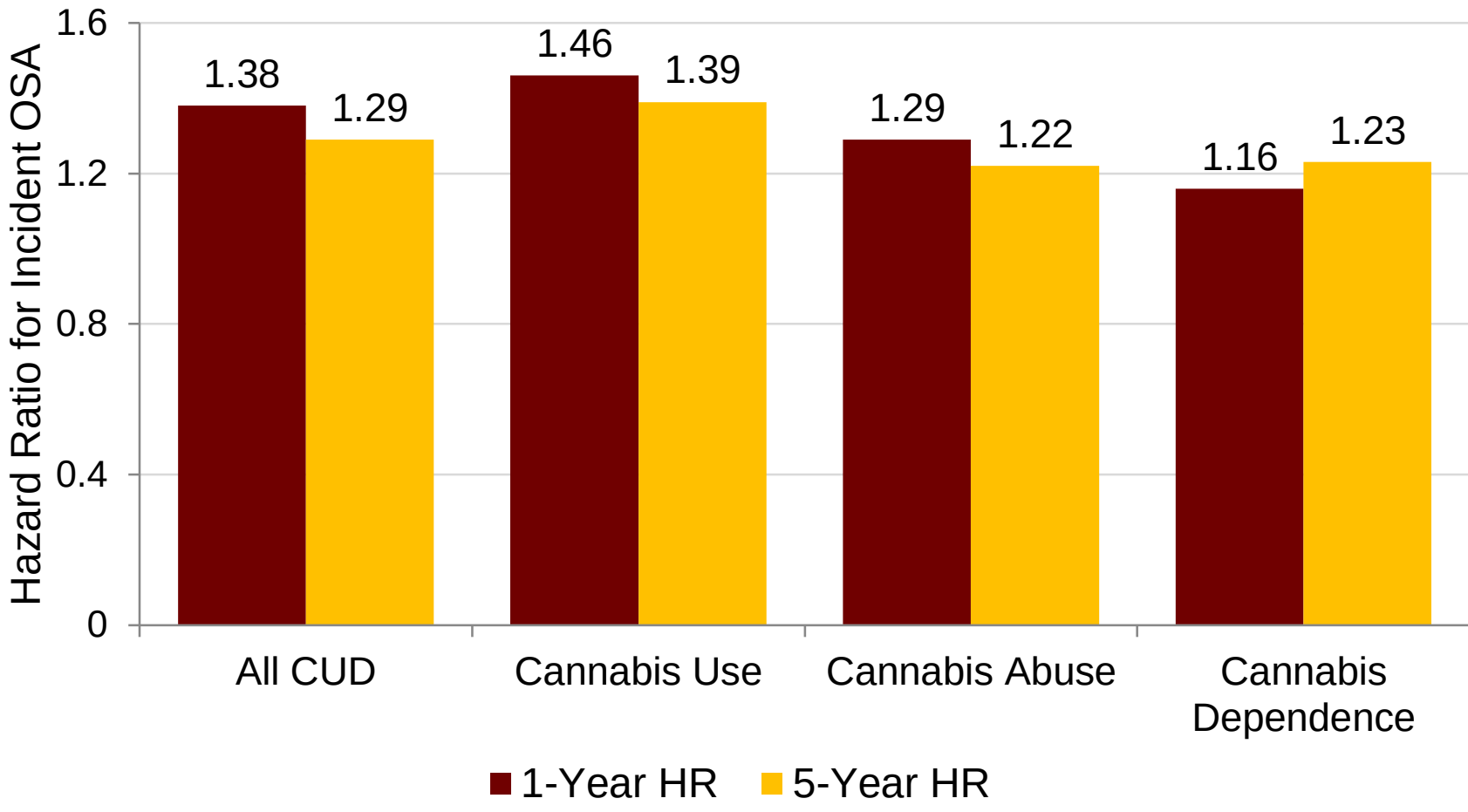


Figure 2. Hazard ratios for incident OSA by cannabis-related disorder severity stratum. All $p<0.001$ except dependence at 1 year ($p=0.25$).

Result #3. Among patients with pre-existing OSA, those who subsequently developed CUD were **twice as likely to be prescribed CPAP** at 5 years (17.7% vs 9.0%; HR 2.00, 1.83–2.19) and over lifetime follow-up (21.9% vs 12.9%; HR 1.80, 1.67–1.94).

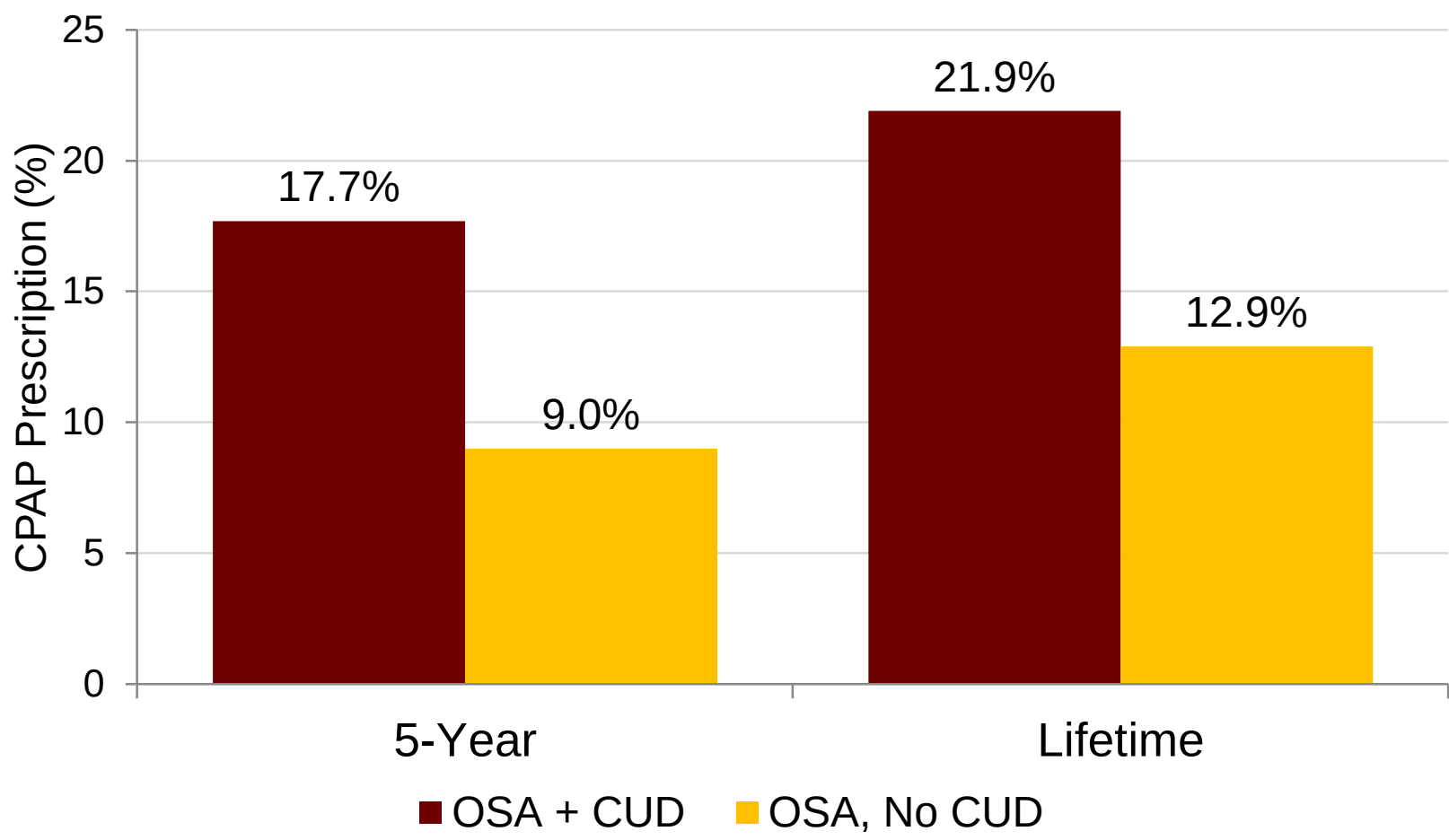


Figure 3. CPAP prescription among propensity-matched patients with pre-existing OSA, with vs without subsequent CUD.

Table 1. Incident OSA (Aim 1) and CPAP prescription (Aim 2) in propensity-matched cohorts.

Cohort / Outcome	Exposed	Control	HR (95% CI)
Aim 1: Incident OSA, 5-year (n per arm)			
All CUD (84,125)	8.6%	6.5%	1.29 (1.25–1.34)
Cannabis use (32,480)	9.7%	6.8%	1.39 (1.32–1.47)
Cannabis abuse (24,275)	7.7%	6.1%	1.22 (1.14–1.31)
Cannabis dependence (6,387)	7.3%	5.7%	1.23 (1.08–1.41)
Tobacco-naïve CUD (20,779)	6.0%	4.2%	1.40 (1.28–1.53)
Aim 2: CPAP prescription in pre-existing OSA (8,556 per arm)			
s5-year follow-up	17.7%	9.0%	2.00 (1.83–2.19)
Lifetime follow-up	21.9%	12.9%	1.80 (1.67–1.94)
Tobacco-naïve CUD, 5-year (1,687)	11.8%	5.9%	2.00 (1.57–2.55)

All log-rank $p<0.001$. HR from Cox proportional hazards models.

Conclusions

- CUD was independently associated with increased incident OSA at 1 and 5 years — across all severity strata and in tobacco-naïve patients.**
- In pre-existing OSA, subsequent CUD doubled the likelihood of CPAP prescription.**
- No dose–response was seen; co-exposures and detection bias may contribute.
- A detailed substance use history is warranted in OSA evaluation and treatment.**

References

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Oluwatobiloba (Tobi) Ayo-Ajibola, MD
Otolaryngology—Head & Neck Surgery, UC Davis
ayoajibo@usc.edu