Cannabis Use as Risk or Protection for Type 2 Diabetes: A Longitudinal Study of 18 000 Swedish Men and Women

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Abstract

Whether or not cannabis use may increase or decrease the danger of type 2 diabetes isn't clear. We analyzed the association between cannabis and subsequent type 2 diabetes and if a possible positive or reverse association persisted after controlling for potential confounders. Methods. during this population-based cohort study, 17,967 Swedish men and ladies (aged 18-84 years), who answered an in depth questionnaire in 2002 (including questions on cannabis use), were followed up for brand new cases of type 2 diabetes (n = 608) by questionnaire (in 2010) and in health registers during 2003-2011. Odds ratios (ORs) with 95% CIs were estimated in an exceedingly multiple logistic multivariate analysis. Potential confounders included age, sex, BMI, physical inactivity, smoking, alcohol use, and occupational position. Results. The crude association showed that cannabis users had a reduced risk of type 2 diabetes OR = 0.68 (95% CIs: 0.47-0.99). However, this inverse association attenuated to OR = 0.94 (95% CIs: 0.63-1.39) after adjusting for age. Conclusions. the current study suggests that there's no association between cannabis use and subsequent type 2 diabetes after controlling for age. to form more robust conclusions prospective studies, with longer periods of follow-up and more detailed information about cannabis use, are needed.

Keywords: : Cannabis study, Diabetes, cutaneous xanthoma ,Primary Healthcare, Pancreatic cells.

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Introduction

Annabis is that the most ordinarily used narcotic globally Diabetes features a worldwide prevalence of 8.8%, and by 2045 quite 600 million people are estimated to be living with diabetes As more countries legalize the sale and consumption of cannabis, the amount of users is constant to rise Given the increasing prevalence of both medicinal and recreational cannabis, it's increasingly important to grasp its impact on public health. Although several observational studies have reported that cannabis use had favorable metabolic associations including a lower prevalence of diabetes and lower glucose levels evidence that cannabis is causally linked to the event of type 2 diabetes is insufficient.

This literature is proscribed by a preponderance of cross-sectional study designs Available prospective observational studies could also be subject to social desirability and recall bias, and lack of valid cumulative exposure assessment Notably, cannabis users also engage in other behaviors that are related to poor outcome. specifically, confounding bias (e.g., by tobacco smoking) may result in spurious associations that preclude conclusions about causality.

As cannabis smoking together with tobacco is that the predominant method of use thanks to positivity violations (i.e., few cannabis users refrain from tobacco smoking), traditional confounding adjustment (e.g, regression, weighting, matching) is infeasible in observational studies. However, establishing causality is vital, as this is often essential for recommending public policies and clinical interventions. during this study, we use Mendelian randomization (MR) to look at whether cannabis use may cause the event of type 2 diabetes.

MR makes use of genetic instrumental variables to represent the exposure of interest and infers a relationship between exposure and outcome . MR isn't tormented by reverse causation, as genetic variants are fixed at conception. MR is additionally less liable to environmental confounding compared with conventional observational studies because genetic instruments are assumed to affect the result only via the exposure and to be independent of confounders. Using genetic instrumental variables for lifetime cannabis use from GWAS of quite 180,000 individuals and 74,000 cases of diabetes, we examined the connection between lifetime cannabis use and sort 2 diabetes. We additionally used cannabis use disorder as an exposure variable that reflects heavy cannabis use. This study provides no evidence for a task of cannabis use within the development of type 2 diabetes.

Several cross-sectional studies have suggested that cannabis has beneficial metabolic effects (3; 5; 7; 28-30) but prospective observational studies haven't supported inverse associations with type 2 diabetes Cannabis intake stimulates appetite and increases the utilization of low nutritional value carbohydrates and it promotes adipogenesis, which is anticipated to extend insulin resistance ; Consequently, the arteria Risk Development in Young Adults (CARDIA) cohort found an increased risk of pre-diabetes among current (OR: 1.65, 95% CI: 1.15-2.38) and lifelong cannabis users (OR: 1.49, 95% CI: 1.06-2.11), but no increased risk for manifest diabetes. Another prospective study of over 17,000 Swedish men and girls found no association between lifetime cannabis use and diabetes risk . alittle randomized double-blind trial showed that in patients with type 2 diabetes, tetrahydrocannabivarin (a CB1 receptor antagonist at low dose) decreased fasting plasma glucose levels and improved pancreatic β-cell function. Likewise, in another small randomized trial among healthy cannabis users , cannabis use lowered blood insulin, glucagon-like peptide 1, and ghrelin levels.

We combined nationwide Danish registers to identify 21 066 cases with schizophrenia and 176 935 sex-and-age-matched controls. Two models were analyzed for the associations between CUD and digestive disorders in time-varying Cox regressions: one adjusted for sex, year of birth, and calendar year; and one further adjusted for alcohol and other substance use disorders and parental education.

Results. CUD was associated with a decreased risk of developing disorders of gut–brain interaction (e.g. irritable bowel syndrome, dyspepsia, etc.) among cases with schizophrenia (HR = 0.84, 95% CI 0.74–0.94, p = 0.003). CUD was associated with decreased risk of inflammatory bowel disease (HR = 0.70, 95% CI 0.49–0.99, p = 0.045) in the basically adjusted model, dropping just below statistical significance in the fully adjusted model (HR = 0.71, 95% CI 0.48–1.03, p = 0.07). CUD displayed a tendency toward a decreased risk of serious disorders of the digestive organs among cases with schizophrenia (HR = 0.89, 95% CI 0.77–1.02, p = 0.09) in the fully adjusted model. No associations were observed among controls. In people with schizophrenia, but not in controls, CUD is associated with decreased risk of disorders of gut–brain interaction and inflammatory bowel disease, and possibly other serious disorders of the digestive organs. Our findings could lead to new targets for treatment and prevention of disorders of the digestive organs.

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