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Review Article

Marijuana (Cannabis) Smoking Increases the Risk of Lung Cancer: Evidence from a Systematic Review and Meta-Analysis

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Abstract

Introduction: Marijuana (cannabis) use has become increasingly prevalent worldwide, raising concerns about its potential long-term health effects, particularly on the respiratory system. While tobacco smoking is a well-established risk factor for lung cancer, the association between cannabis use and lung cancer risk remains unclear and controversial. This systematic review and meta-analysis of existing observational studies aimed to evaluate the potential relationship between marijuana use and the risk of developing lung cancer.

Methods: A systematic search was conducted across PubMed, Web of Science, and Scopus to identify cohort and case-control studies up to September 2025. Studies were selected, relevant data were extracted, and statistically analyzed using effect sizes (ES) and 95% confidence intervals (CI). All analyses were conducted using STATA software.

Findings: This meta-analysis included six observational studies, comprising 16 effect sizes, consisting of two cohort studies and four case-control studies. The pooled analysis of the cohort studies (7 effect sizes) indicated a statistically significant association between marijuana (cannabis) smoking and increased lung cancer risk (ES: 2.06; 95% CI: [1.04–4.07]; $P=0.038$; $I^2=75\%$). In contrast, the meta-analysis of the case-control studies (9 effect sizes) showed a non-significant increase in risk (ES: 1.16; 95% CI: [0.71–1.88]; $P=0.542$; $I^2=82\%$).

Conclusion: This meta-analysis indicates that marijuana smoking may increase lung cancer risk, with cohort studies showing a significant twofold association. Although case-control studies were not statistically significant, the upward trend highlights the importance of considering cannabis use in lung cancer risk assessments and patient counseling.

Keywords: Marijuana, Cannabis, Lung cancer, Smoking, Meta-analysis

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Introduction

Marijuana (Cannabis) is the most commonly used illicit psychoactive substance worldwide, with its legal status undergoing significant changes in recent years. An increasing number of countries have legalized or decriminalized cannabis use for medical and recreational purposes, reflecting shifting societal attitudes and regulatory policies.[1] As the prevalence of cannabis use rises,[2] there is growing concern about its long-term health effects, particularly its potential role in the development of malignancies.[3] One area of emerging interest is the possible association between cannabis smoking and the risk of lung cancer.

Lung cancer remains the leading cause of cancer-related mortality globally, with substantial variation in incidence and death rates across regions. Smoking is the primary risk factor for lung cancer, with both active and passive exposure contributing to disease risk.[4,5] Given the well-documented carcinogenic effects of tobacco smoke,[5] concerns have been raised about the health implications of inhaling combusted cannabis plant material,[3] which contains many of the same harmful constituents, including polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds, and other toxic combustion products.[6,7] Additionally, cannabis users often inhale more deeply and hold smoke in their lungs longer than tobacco

users, potentially increasing pulmonary exposure to toxic substances. Despite these similarities, some researchers have suggested that cannabis may exhibit anti-neoplastic properties due to the presence of cannabinoids such as Δ9-tetrahydrocannabinol (THC) and cannabidiol (CBD), which have demonstrated anti-proliferative effects in in vitro studies.[8-10] This biological complexity has contributed to ongoing debate regarding the carcinogenic potential of cannabis.

While the carcinogenic effects of tobacco are well-established, the role of marijuana in the development of lung cancer remains unclear and continues to be debated. Epidemiological studies investigating this association have yielded inconsistent results. Some studies have reported an increased risk associated with long-term or heavy marijuana use,[11,12] whereas others have found no significant association after adjusting for confounding factors.[13,14] These discrepancies may be attributable to variations in usage patterns, such as inhalation depth, frequency of use, and concurrent tobacco use, as well as to methodological limitations, including small sample sizes, reliance on self-reported data, and inadequate control for key confounders, particularly tobacco use. Given these complexities, a comprehensive synthesis of the existing literature is essential to generate more reliable and informative conclusions regarding the potential relationship between marijuana use and lung cancer.

Given the global rise in marijuana use and ongoing debate over its health effects,[2] a systematic evaluation of existing evidence is urgently needed. This study addresses the lack of a comprehensive meta-analysis by reviewing observational studies on the association between marijuana use and lung cancer risk. The goal is to clarify this potential relationship and inform public health policy, clinical practice, and future research.

Methods

Study Protocol and Design

This study was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline.[15] The study was designed based on the PICOS (Population, Intervention, Comparison, Outcomes, and Study) framework,[16] including:

- **P** (Population): Adolescent and adult males and females (aged 13 years or older)
- **I** (Intervention/Exposure): Marijuana (Cannabis) smoking
- **C** (Comparison): Non-smoking individuals who reported no use of any smoking substances (e.g., cigarettes, hookah, marijuana)
- **O** (Outcomes): Risk of developing any type of lung cancer, including both small-cell and non-small-cell lung cancers
- **S** (Study Design): Cohort and case-control studies

Search Strategy

A comprehensive literature search was conducted in PubMed/MEDLINE, the ISI Web of Science, and Scopus from their inception through September 2025. The search strategy incorporated both MeSH (Medical Subject Headings) and non-MeSH terms, along with relevant synonyms for "marijuana (cannabis)" and "lung cancer," based on titles and abstracts. Specific keywords and detailed search strategies for each database are provided in Supplementary Tables 1 and 2. No language or publication date restrictions were applied to maximize the capture of relevant studies. Manual searches were performed using Google Scholar and the reference lists of all included studies to identify gray literature and locate additional reviews and eligible studies.

Eligibility Criteria

Studies that met the following criteria were included: (1) observational in design (including case-control and cohort studies), (2) published in English as original articles, (3) involved adult participants (≥18 years), regardless of gender, geographic location, or ethnicity, and (4) provided data on the association between marijuana (cannabis) use and the risk of lung cancer. Conference abstracts, case reports, case series, review articles, commentaries, editorials, in vitro or animal studies, and studies that lacked data on cancer outcomes or did not primarily assess marijuana exposure were excluded from the study.

Data Extraction

All retrieved records were imported into EndNote software (version 21; Clarivate, Philadelphia, PA, USA) for citation management and duplicate removal. Title and abstract screening were conducted independently by two reviewers, with discrepancies resolved by consensus in consultation with a third reviewer.

Two independent reviewers conducted a comprehensive screening and data extraction process. They meticulously cross-verified the extracted data to ensure accuracy. Any discrepancies or inconsistencies were resolved through discussion. Key data extracted included: First author's name, publication year, country, study design, cohort duration, follow-up period, sample size, participant age, outcome measures, adjusted covariates, confounding variables, and effect estimates with corresponding 95% confidence intervals (CI).

Quality Assessment

Two independent reviewers (A.J.N. and P.I.) evaluated the quality of the included studies using the Newcastle-Ottawa Scale (NOS) tailored for observational research. Any disagreements or inconsistencies were addressed through discussion or resolved with input from the project supervisor (M.K.). The NOS assesses studies based on three core domains: Selection of participants, comparability of study groups, and outcome assessment. Based on their total scores, studies were categorized as high, fair, or low quality.[17]

Statistical Analysis

In analyzing studies, relative risk (RR) with 95%

confidence intervals (CI) served as the effect size metric, while hazard ratios (HR) from certain studies were treated as equivalent to RR

values.[18] The effect size for each study was determined by comparing the highest and lowest marijuana smoking rates with those of non-smokers. Fixed-effects models were employed to examine potential associations, with heterogeneity assessed using Q and I^2 statistics, where an I^2 over 50% indicated high heterogeneity.[19] Publication bias was evaluated by funnel plot visualization and Egger's and Begg's tests,[20,21] and if bias was detected, the trim-and-fill method was used for adjustment.[22] Sensitivity analysis involved removing one study at a time to assess the impact of each study on the overall results. All analyses were conducted using STATA software (version 17.0). Associations with P-values below 0.05 were considered significant.

Results

Study Selection

The study selection process for the systematic review, as depicted in the PRISMA flow chart (Figure 1), began with the identification of 1,911 records from three databases: PubMed (n = 306), Scopus (n = 960), and Web of Science (n = 645). After removing 618 duplicate records, 1,293 records remained for initial screening based on titles and abstracts. Of these, 1,282 were excluded because they were review articles, non-human studies, or irrelevant to the research question. The full texts of the remaining 11 articles were then assessed for eligibility, and 5 studies were excluded for failing to meet the inclusion criteria. Ultimately, six studies were included in the final systematic review and meta-analysis.[11-14,23,24]

Study Characteristics

The basic characteristics of the studies included in the meta-analysis are summarized in Table 1. These encompass both cohort and case-control designs conducted across multiple countries. In total, six observational studies comprising 16 effect sizes were included: Two cohort studies [13,23] and four case-control studies.[11,12,14,24]

The cohort studies, conducted in the USA[23] and Sweden,[13] focused on younger populations aged 15–49 and 18–20 years, respectively. Both studies included male and female participants, with large sample sizes ranging from 49,321 to 64,855 individuals. They reported risk ratios (RRs) or hazard ratios (HRs) with effect sizes ranging from 0.66 to 10.3, and adjusted for confounders such as age, race, education, tobacco use, and alcohol consumption.

The case-control studies[11,12,14,24] were carried out in the USA, Tunisia, Morocco, Algeria, and New Zealand, involving participants of both sexes aged 18–65 years or older. Sample sizes varied considerably, and reported odds ratios (OR) ranged from 0.56 to 4.1. These studies adjusted for factors including age, sex, race, occupational exposures, tobacco use, and other lifestyle factors.

Quality Assessment

The quality assessment of the included studies using the NOS revealed that both cohort studies demonstrated high methodological quality, with Callaghan et al. (2013)

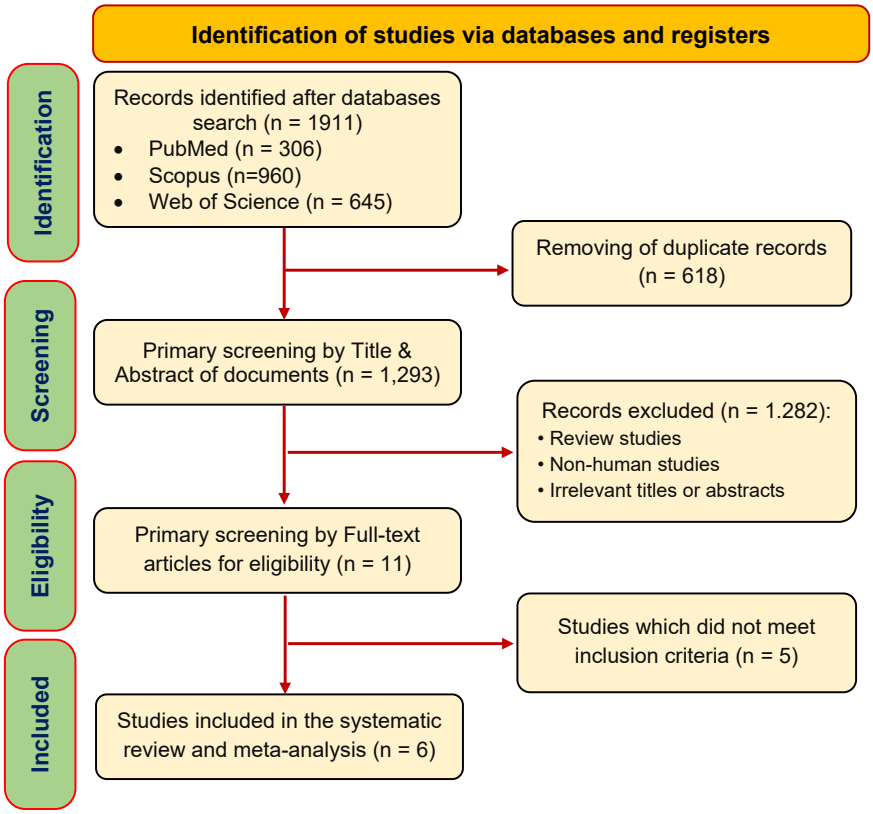

Figure 1. PRISMA Flow chart of the study selection process for inclusion studies in the systematic review

Table 1. Basic characteristics of the cohort and case-control studies included in the systematic review and meta-analysis

Study	Country	Study design	Age (y)	Sex	Total Sample Size	Duration	Effect Size (95%CI)	Adjustment
Sidney et al. (1997) (a) [23]	USA	Cohort	15-49	Male	64855	NR	RR: 10.3 (2.4 – 43.7)	Age, race, education, alcohol consumption
Sidney et al. (1997) (b) [23]	USA	Cohort	15-49	Female	64855	NR	RR: 9.8 (3.6 – 26.5)	
Callaghan et al. (2013) (a) [13]	Sweden	Cohort	18-20	Male	49321	Once time	HR: 1.52 (0.77 – 3.01)	
Callaghan et al. (2013) (b) [13]	Sweden	Cohort	18-20	Male	49321	2–4 times	HR: 0.66 (0.27 – 1.62)	
Callaghan et al. (2013) (c) [13]	Sweden	Cohort	18-20	Male	49321	5–10 times	HR: 0.68 (0.21 – 2.16)	Tobacco smoking, alcohol consumption
Callaghan et al. (2013) (d) [13]	Sweden	Cohort	18-20	Male	49321	11–50 times	HR: 1.68 (0.77 – 3.66)	
Callaghan et al. (2013) (e) [13]	Sweden	Cohort	18-20	Male	49321	>50 times	HR: 2.12 (1.08 – 4.14)	
Hashibe et al. (2006) (a) [24]	USA	Case-control	18-65	Both	478	>0 to<1 joint-years	OR: 0.63 (0.46 - 0.87)	
Hashibe et al. (2006) (b) [24]	USA	Case-control	18-65	Both	189	1 to<10 joint-years	OR: 0.71 (0.46 – 1.1)	
Hashibe et al. (2006) (c) [24]	USA	Case-control	18-65	Both	89	10 to<30 joint-years	OR: 0.56 (0.31 – 1.0)	Age, sex, race, education, drinking years, tobacco use, pack-years
Hashibe et al. (2006) (d) [24]	USA	Case-control	18-65	Both	43	30 to<60 joint-years	OR: 0.82 (0.38 – 1.7)	
Hashibe et al. (2006) (e) [24]	USA	Case-control	18-65	Both	68	≥60 joint-years	OR: 0.62 (0.32 – 1.2)	
Voirin et al. (2006) [12]	Tunisia	Case-control	Mean: 57	Male	337	NR	OR: 4.1 (1.9 – 9.0)	Age, occupational exposure, duration of tobacco smoking, cannabis use
Berthiller et al. (2008) (a) [11]	Morocco	Case-control	Case: 59.6±11.1, Control: 59.2±10.9	Male	353	NR	OR: 3.4 (1.8 – 6.6)	age
Berthiller et al. (2008) (b) [11]	Algeria	Case-control	Case: 64.9±11.1, Control: 63.8±11.5	Male	507	NR	OR: 2.9 (1.1 – 7.7)	
Aldington et al. (2008) [14]	New Zealand	Case-control	≤55	Both	60	NR	RR: 1.2 (0.5 – 2.6)	Age, sex, ethnicity, family history, pack-years

Footnote: (**NR**: Non-reported, **RR**: Risk ratio, **OR**: Odds ratio, **HR**: Hazard ratio, **BMI**: Body mass index, **PA**: Physical activity).

[13] scoring 9 out of 9 and Sidney et al. (1997) [23] scoring 8 out of 9, indicating robust selection, comparability, and outcome assessment. Among the case-control studies, Aldington et al. (2008) [14] and Hashibe et al. (2006) [24] also achieved high scores of 9, reflecting strong design and comprehensive adjustment for confounders. In contrast, Voirin et al. (2006) [12] and Berthiller et al. (2008) [11] received lower scores of 7 due to limited control for additional confounders and less clarity in exposure assessment. Overall, the majority of studies included in the meta-analysis were of high or fair quality, supporting the reliability of the pooled findings (Table 2).

Meta-Analysis of Cohort Studies

The pooled analysis of the two cohort studies encompassing seven effect sizes [13,23] indicated a statistically significant positive association between marijuana smoking compared to non-smokers and increased risk of lung cancer (ES = 2.06; 95% CI: [1.04–4.07]; $P = 0.038$; $I^2 = 75\%$). Heterogeneity among the cohort effect sizes was high ($I^2 = 75.5\%$, p-heterogeneity < 0.001) (Figure 2, Table 2).

Meta-Analysis of Case-Control Studies

The meta-analysis of the nine effect sizes from 4 case–control studies [11,12,14,24] showed a positive association between marijuana smoking compared to non-smokers and increased risk of lung cancer, although it was not statistically significant (ES = 1.16; 95% CI: [0.71–1.88]; $P = 0.542$). Notably, there was a high heterogeneity among these studies ($I^2 = 82\%$, p-heterogeneity < 0.001) (Figure 3, Table 2).

Sensitivity Analysis

The sensitivity analysis for cohort studies highlights the influence of specific studies on the overall effect estimate. Excluding individual studies such as Sidney et al. (1997) [23] and various ES from Callaghan et al. (2013) [13] resulted in ES ranging from 1.57 to 2.21, with wide 95% CIs that all included the null value. This suggests that no single study unduly influenced the overall pooled estimate and that the association between marijuana use and lung cancer risk remained relatively consistent, although imprecise. Notably, no sensitivity analyses were reported for the case-control studies (Table 3).

Publication Bias

The assessment of publication bias using funnel plots and statistical tests revealed differing results between cohort and case-control studies. For cohort studies, the funnel plot appeared symmetrical, and no significant publication bias was detected, as confirmed by Egger's test ($P=0.518$) and Begg's test ($P=1.000$) (Figure 4, Table 4). In contrast, the funnel plot for case-control studies showed asymmetry, suggesting potential publication bias, which was supported by Begg's test ($P=0.048$). However, Egger's test did not reach statistical significance ($P=0.066$), indicating borderline evidence for bias in these studies (Figure 5, Table 4).

Discussion

Given the global rise in marijuana use and ongoing debate over its health effects,[2] a systematic evaluation of existing evidence is urgently needed. The present systematic review and meta-analysis aimed to clarify the potential relationship between marijuana (cannabis) smoking and

Table 2. Quality assessment of the cohort studies according to the Newcastle-Ottawa Scale (NOS) criteria.

Study	Study designs	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Score
Sidney et al. (1997) [24]	Cohort	★	★	★	★	★★	★	★	☆	-	8
Callaghan et al. (2013) [14]	Cohort	★	★	★	★	★★	★	★	★	-	9
Hashibe et al. (2006) [25]	Case-control	★	★	★	★	★★	★	★	★	☆	9
Voirin et al. (2006) [13]	Case-control	★	★	★	★	★☆	☆	★	★	☆	7
Berthiller et al. (2008) [12]	Case-control	★	★	★	★	★☆	☆	★	★	☆	7
Aldington et al. (2008) [15]	Case-control	★	★	★	★	★★	★	★	★	★	9

NOS criteria for case-control studies: Q1: Is the case definition adequate?, Q2: Representativeness of the cases
Q3: Selection of controls, Q4: Definition of controls, Q5: Comparability of cases and controls based on the design or analysis, controlled for confounders, Q6: Study controls for any additional factor, Q7: Ascertainment of exposure, Q8: Same method of ascertainment for cases and controls, Q9: Non-response rate.
NOS criteria for cohort studies: Q1: Representativeness of the exposed cohort, Q2: Selection of the non-exposed cohort, Q3: Ascertainment of exposure, Q4: Demonstration that the outcome of interest was not present at the start of the study, Q5: Comparability of cohorts based on the design or analysis, Q6: Assessment of outcome, Q7: Was the follow-up long enough for outcomes to occur (>5 years), Q8: Adequacy of follow-up of cohorts (loss-to-follow-up<20%).
★=Criterion fully met, ☆=Criterion partially met or unclear. ★★=Two stars for comparability when controlled for two major confounders, "-"=Not applicable.

Table 3. Meta-analysis findings of the association between Marijuana (Cannabis) use and the risk of lung cancer.

Study designs	N. of Effect Sizes	Pooled ES (95% CI)	*P*-value	I^2 (%)	P-heterogeneity
Cohort studies	7	2.06 (1.04, 4.07)	**0.038***	75.5%	$P<0.001$
Case-control studies	9	1.16 (0.71, 1.88)	0.542	82.8%	$P<0.001$

Footnote: Aesthetics (*) indicates statistical significance ($P<0.05$).

Table 4. Publication bias and Sensitivity analysis for the association between Marijuana (Cannabis) use and the risk of lung cancer.

Study designs	Publication bias		Sensitivity analysis
	Egger's test	Begg's test	
Case-Control	$P=0.066$	$P=0.048$	None
Cohort	$P=0.518$	$P=1.000$	Sidney et al. (1997) (a) (ES: 1.71; 95% CI: [0.87, 3.33]) Sidney et al. (1997) (b) (ES: 1.57; 95% CI: [0.88, 2.79]) Callaghan et al. (2013) (a) (ES: 2.21; 95% CI: [0.95, 5.13]) Callaghan et al. (2013) (d) (ES: 2.16; 95% CI: [0.94, 4.94]) Callaghan et al. (2013) (e) (ES: 2.07; 95% CI: [0.88, 4.86])

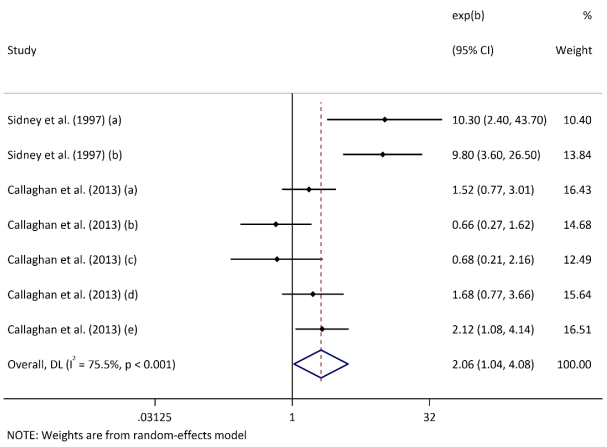

Figure 2. Forest plot detailing effect sizes (ES) and 95% confidence intervals (CIs) for the association between Marijuana (Cannabis) use and the risk of lung cancer in cohort studies

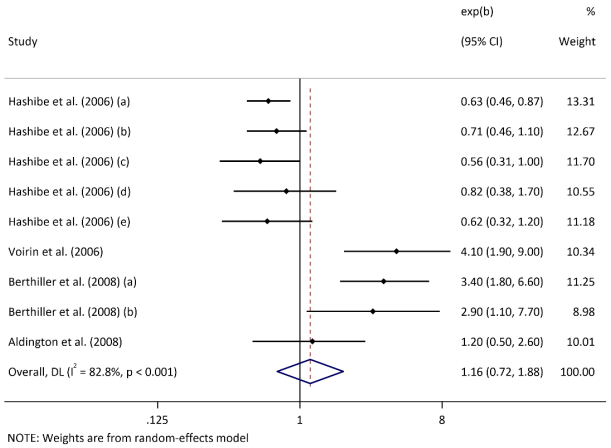

Figure 3. Forest plot detailing effect sizes (ES) and 95% confidence intervals (CIs) for the association between Marijuana (Cannabis) use and the risk of lung cancer in case-control studies

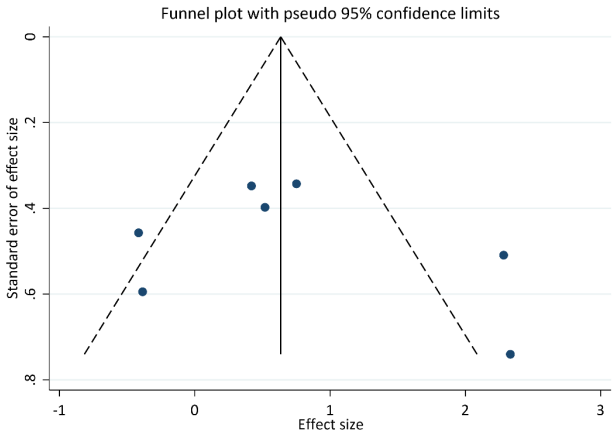

Figure 4. Funnel plot of publication bias demonstrating effect sizes (ES) and 95% confidence intervals (CIs) for the association between Marijuana (Cannabis) use and the risk of lung cancer in cohort studies

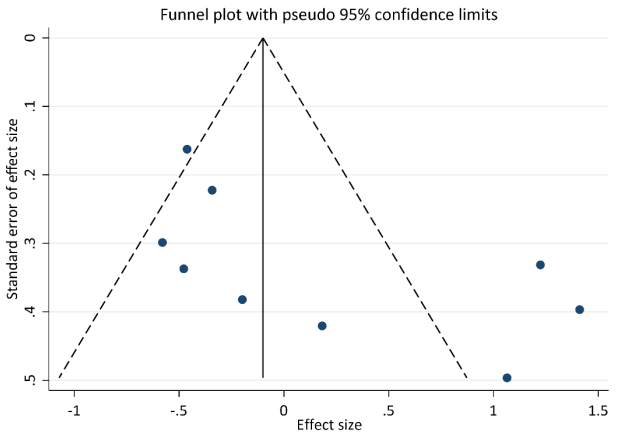

Figure 5. Funnel plot of publication bias demonstrating effect sizes (ES) and 95% confidence intervals (CIs) for the association between Marijuana (Cannabis) use and the risk of lung cancer in case-control studies

the risk of lung cancer. The pooled analysis of cohort studies demonstrated a statistically significant increase in lung cancer risk among marijuana smokers, aligning with concerns that cannabis smoke, like tobacco smoke, may possess carcinogenic properties. In contrast, the analysis of case-control studies revealed a non-significant association, suggesting that heterogeneity in study designs, sample populations, and exposure assessments may influence the observed relationships.

These findings are consistent with some previous studies that have proposed a potential relationship between heavy or long-term marijuana use and respiratory malignancies. For example, Aldington et al. (2008) [14] reported that each joint-year of cannabis smoking was associated with an increased risk of lung cancer after adjusting for tobacco use. However, other studies, such as those by Hashibe et al. (2006) [24], did not observe a clear association, particularly among light or moderate users. This inconsistency may reflect limitations in sample sizes, underreporting of marijuana use, or confounding by concurrent tobacco smoking.

A meta-analysis in 2019 evaluated the association between marijuana use and risk of cancer and found no significant association between ever using marijuana and the risk of head and neck or oral cancers. However, long-term marijuana use (over 10 years) was associated with an increased risk of testicular germ cell tumors (TGCT), particularly nonseminoma TGCT. Evidence for other cancers, including lung cancer, was inconclusive due to low exposure levels, poor exposure assessment, and limited data on marijuana-only users.[25]

The potential biological mechanism connecting marijuana smoking to lung cancer involves the inhalation of carcinogenic compounds similar to those found in tobacco smoke. Cannabis contains over 400 chemical constituents, including more than 60 cannabinoids.[26] Its smoke carries polycyclic aromatic hydrocarbons (PAHs), volatile aldehydes, and other toxic compounds such as benzopyrene, benzanthracene, ammonia, and hydrocyanic acid—substances also present in tobacco smoke, excluding nicotine.[27-29] Unlike tobacco users, marijuana smokers often inhale more deeply, take larger puffs, and hold their breath up to four times longer, increasing the deposition of harmful particles in lung tissue. Additionally, marijuana is commonly smoked without filters, further enhancing exposure. Chronic use has been associated with airway inflammation, epithelial cell damage, and histological changes that may contribute to carcinogenesis. Despite differences in active ingredients, D9-tetrahydrocannabinol (THC) in cannabis versus nicotine in tobacco, the combustion process produces a similar array of toxic byproducts, suggesting that marijuana smoking may pose comparable risks to lung health.[28-30]

A major strength of this study lies in its comprehensive synthesis of existing epidemiological data, utilizing a systematic approach and meta-analytic techniques, which enhances the reliability of the pooled estimates. The inclusion of both cohort and case-control studies enabled the evaluation of the association across different study designs, providing a broader perspective on the evidence. However, several limitations should be acknowledged. First, the number of high-quality longitudinal studies remains limited, and many studies included in the meta-analysis had small sample sizes or relied on self-reported cannabis use, which is subject to recall bias and misclassification. Additionally, confounding factors, particularly concurrent tobacco use, are difficult to fully control for and may obscure the independent effect of marijuana. Variations in cannabis potency, frequency of use, and methods of inhalation further complicate exposure assessment. Finally, many existing studies were conducted before widespread changes in cannabis legality and consumption patterns occurred, which may limit the applicability of their findings to current populations.

Future research should focus on elucidating dose-response relationships, distinguishing the independent effects of marijuana from those of tobacco, and investigating the long-term impact of varying patterns of use. Well-designed prospective cohort studies with accurate exposure assessment and control for confounding

variables are essential. Additionally, examining potential synergistic effects in individuals who use both marijuana and tobacco will be critical to understanding combined risks and informing public health strategies.

Conclusion

This systematic review and meta-analysis highlight a potential association between marijuana smoking and an increased risk of lung cancer, particularly as evidenced by cohort studies showing a statistically significant relationship. These findings support growing concerns about the carcinogenic potential of cannabis smoke, which shares many harmful constituents with tobacco smoke. However, the non-significant results from case-control studies suggest variability in the existing literature, likely due to differences in study design, population characteristics, and methods of exposure assessment. Given the global rise in cannabis use, these results underscore the need for more rigorous, large-scale prospective studies to clarify this association. Until stronger evidence becomes available, public health messaging and clinical guidance should adopt a cautious approach to marijuana use, especially in populations at risk for respiratory disease.

Authors' Contribution

Conceptualization: Mehdi Karimi, Kimia Kazemi, Omid Asbaghi
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Project Administration: Mehdi Karimi
Resources: Mehdi Karimi, Kimia Kazemi, Mohammad Hossein Pourhanifeh, Parsa Irajian, Omid Asbaghi
Software: Mehdi Karimi, Omid Asbaghi
Supervision: Mehdi Karimi
Validation: Mehdi Karimi, Kimia Kazemi, Mohammad Hossein Pourhanifeh, Parsa Irajian, Omid Asbaghi
Visualization: Mehdi Karimi, Omid Asbaghi
Writing–Original Draft: Mehdi Karimi, Kimia Kazemi, Mohammad Hossein Pourhanifeh, Parsa Irajian, Omid Asbaghi

Competing Interests

The authors declare no conflict of interest.

Data Availability

All data used in this meta-analysis were extracted from published studies. The datasets supporting the findings of this study are available from the original sources, which are cited in the manuscript. Additional information can be provided by the corresponding author upon reasonable request.

Ethical Approval

Not applicable.

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Supplementary Files

Specific keywords and detailed search strategies for each database are provided in Supplementary Tables 1 and 2.

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