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REVIEW

Chronic Obstructive Pulmonary Disease and Lung Cancer: A Meta-Analysis of Risk Association

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Objective: Chronic obstructive pulmonary disease (COPD) is a heterogeneous lung condition characterized by persistent respiratory symptoms and airflow limitation, which may involve mechanisms that are also implicated in lung carcinogenesis. This meta-analysis aimed to evaluate the association between COPD and the risk of developing lung cancer.

Methods: A systematic literature search was conducted from database inception to March 20, 2022 across PubMed, Embase, Web of Science, and the Cochrane Library. Studies were independently screened by two reviewers, and relative risks (RRs) with 95% confidence intervals (CIs) were extracted and synthesized.

Results: Nine studies were included in the final meta-analysis. A significantly increased risk of lung cancer was observed among individuals with COPD, with a pooled relative risk (RR) of 3.79 and a 95% confidence interval (CI) ranging from 3.60 to 3.98. Among individuals with COPD who did not use inhaled corticosteroids (ICS), the relative risk of lung cancer was 1.26 (95% CI: 1.20–1.33). The relative risk for females was 1.02 (95% CI: 0.99–1.05), suggesting no statistically significant difference in lung cancer risk by gender. The meta-analysis identified a moderate but statistically significant association between COPD and an increased risk of lung cancer.

Conclusion: The results of this study are consistent with those of previous studies, further verifying that patients with COPD have a higher risk of lung cancer and providing stronger support for this finding. These findings underscore the need for enhanced surveillance and tailored preventive strategies among individuals diagnosed with COPD. Moving beyond traditional research paradigms to more refined, multidimensional approaches may improve the understanding of the link between COPD and lung cancer.

Keywords: COPD, inflammation, lung cancer, respiratory diseases, signaling pathways

Introduction

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable condition characterized by persistent respiratory symptoms and chronic inflammation of the airways. Lung cancer, a complex genetic disorder, arises from oncogenic mutations that progressively transform normal cells into malignant phenotypes through a combination of genetic and epigenetic alterations. COPD has been recognized as an inflammatory respiratory disease, and chronic inflammation has similarly been associated with lung carcinogenesis. The presence of COPD is frequently considered a significant risk factor for lung cancer and may contribute to its development through mutually reinforcing pathophysiological mechanisms. In certain cases, progression from COPD to lung cancer has been observed. Moreover, increased severity of COPD has been associated with higher incidence and more rapid progression of lung cancer.

Genome-wide association studies (GWAS) have identified multiple overlapping susceptibility loci for both COPD and lung cancer, suggesting shared genetic underpinnings. COPD-related lung cancer is believed to result, in part, from chronic inflammation leading to excessive proliferation of epithelial cells in the airways and alveoli. This process involves the activation of signaling pathways such as signal transducer and activator of transcription 3 (STAT3), which promote malignant

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transformation. Additionally, impairments in immune regulatory mechanisms and cellular homeostasis have been proposed to exacerbate this pathological progression.[1]

The global burden of COPD and lung cancer has evolved over decades, with substantial variations across countries due to differences in tobacco control policies (eg, smoking taxes, public smoking bans) and shifts in smoking-related behaviors.[2–4] Global Burden of Disease studies have documented rising COPD prevalence and its significant overlap with lung cancer incidence, underscoring the need for context-specific strategies to address these interconnected health challenges.[2–4]

Notably, accumulating evidence highlights that the links between COPD and lung cancer involve multiple interconnected factors, including chronic inflammation, genetic susceptibility, epigenetics, smoking, epithelial mesenchymal transformation (EMT), and oxidative stress injury.[5] Chronic inflammation, in particular, plays a central role in both diseases: in COPD, it drives airway remodeling and tissue damage, while in lung cancer, it creates a protumor microenvironment that facilitates oncogenic transformation and tumor progression.[6] Pathophysiological studies have further elaborated that common mechanisms linking COPD and lung cancer include persistent inflammation, along with genetic predisposition, epigenetic changes, telomere shortening, protease-anti-protease imbalance, mitochondrial dysfunction, premature aging, and aberrant reparative processes.[6] These overlapping pathways reinforce the hypothesis that COPD and lung cancer share a complex interplay of biological processes, with chronic inflammation serving as a key driver.

Pharmacologic interventions in COPD primarily target airway smooth muscle contraction, inflammation, mucus hypersecretion, respiratory tract infections, and alpha-1 antitrypsin deficiency. Anti-inflammatory therapies include the use of glucocorticoids, phosphodiesterase inhibitors, and long-term macrolide antibiotics.[7] The potential role of inhaled corticosteroids (ICS) in reducing lung cancer incidence among individuals with COPD has been a topic of ongoing investigation. ICS has been proposed as potential chemopreventive agents, with some studies reporting a reduced incidence of lung cancer among individuals with COPD receiving ICS therapy.[8] In addition to reducing the frequency of acute exacerbations, ICS has been shown to improve health-related quality of life. As nonspecific anti-inflammatory drugs, ICS reduce airway inflammation, which may mitigate the persistent low-grade inflammation believed to contribute significantly to lung cancer development.[9] Given this potential, further investigation into the effect of ICS on lung cancer risk among individuals with COPD is warranted.

Reports on sex-based differences in the prevalence of COPD have yielded inconsistent findings. Historically, both COPD and lung cancer have been reported more frequently in males, a pattern commonly attributed to higher rates of tobacco use and greater occupational exposure to respiratory irritants.[10] However, recent trends indicate an increase in smoking prevalence among females. Emerging evidence suggests that females may exhibit greater susceptibility to the detrimental effects of various risk factors, including tobacco smoke, exposure to cooking oil fumes, and a positive family history. Consequently, an increased risk of developing both COPD and lung cancer has been observed among females in certain populations. Current research gaps include limited global data on COPD-lung cancer association variability across regions/populations; conflicting evidence on ICS effects and unclear dose-response relationships; and undetermined gender-specific mechanisms with small-sample data. This meta-analysis integrates multi-regional data to quantify associations and clarify ICS/gender impacts, addressing these gaps.

Methods

Eligibility Criteria

Studies were considered eligible if they met the following criteria: (1) cohort studies, case-control studies, or randomized clinical trials that evaluated the use of any ICS, including beclometasone, budesonide, triamcinolone, fluticasone, ciclesonide, mometasone, or flunisolide; (2) study populations comprising individuals diagnosed with COPD; (3) inclusion of an intervention involving ICS, placebo, or a clearly defined comparator; and (4) availability of data on lung cancer incidence, including studies in which no lung cancer events were reported during the follow-up period. Exclusion criteria included in vitro or animal studies, review articles, incomplete manuscripts, conference abstracts, and duplicate publications.

Search Strategy

A systematic literature search was conducted across PubMed, Embase, Web of Science, and the Cochrane Library from database inception to March 20, 2022. The search utilized individual and combined terms, including "COPD", "chronic obstructive pulmonary disease", "lung cancer", "ICS", and "inhaled corticosteroids". Additionally, a manual search of reference lists from the included studies was performed to identify any relevant publications not captured in the database search.

Study Selection

Titles and abstracts suggesting a potential association between COPD and lung cancer risk were independently screened by two reviewers. Full-text articles of potentially eligible studies were retrieved and assessed independently to determine eligibility based on the predefined inclusion and exclusion criteria. Discrepancies between reviewers were resolved through discussion, with a third reviewer consulted to reach a consensus when necessary.

Data Extraction and Outcome Measures

Data were extracted using a standardized form designed to capture general study characteristics, including the first author's name, country of origin, study period, year of publication, source and number of individuals with COPD, and gender distribution. The risk of bias within individual studies was assessed using the Cochrane Risk of Bias Assessment Tool, which evaluated domains such as random sequence generation, completeness of outcome data, and selective reporting. Particular attention was given to studies identified as having a high risk of bias, and potentially biased data were critically examined during extraction. Sensitivity analyses were subsequently conducted to assess the influence of these studies on the overall pooled estimates.

Primary outcomes included the incidence of lung cancer among individuals with COPD, the effect of ICS on lung cancer development, and the gender-specific differences in lung cancer risk.

Data Analysis

A random-effects model was applied to account for potential heterogeneity among the included studies and evaluate the impact of various factors on lung cancer risk. Statistical heterogeneity was assessed using the I^2 statistic. High heterogeneity was defined as $I^2 \geq 75\%$ in conjunction with a p-value ≤ 0.05. A p-value ≤ 0.05 was considered indicative of statistical significance. All statistical analyses were performed using STATA software.

Results

Literature Search Results

The comprehensive search of four databases yielded a total number of potentially relevant studies. Following the screening of titles and abstracts, 68 studies were selected for full-text review. Of these, 9 studies fulfilled the predefined eligibility criteria and were included in the final analysis. The study selection process is illustrated in Figure 1, presented in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow diagram.[11–19]

Study Characteristics

Of the nine studies included in the analysis, one was the Bergen COPD cohort study, two were conducted in China, one was a Korean cohort study, three were based on U.S cohorts, and two were derived from the UK General Practice Research Database. Detailed characteristics of the included studies are summarized in Table 1.

Meta-Analysis

The findings of the systematic review and meta-analysis are summarized as follows: Figure 2 presents the pooled risk of lung cancer among individuals diagnosed with COPD ($P = 0.000$); Figure 3 illustrates the association between ICS use

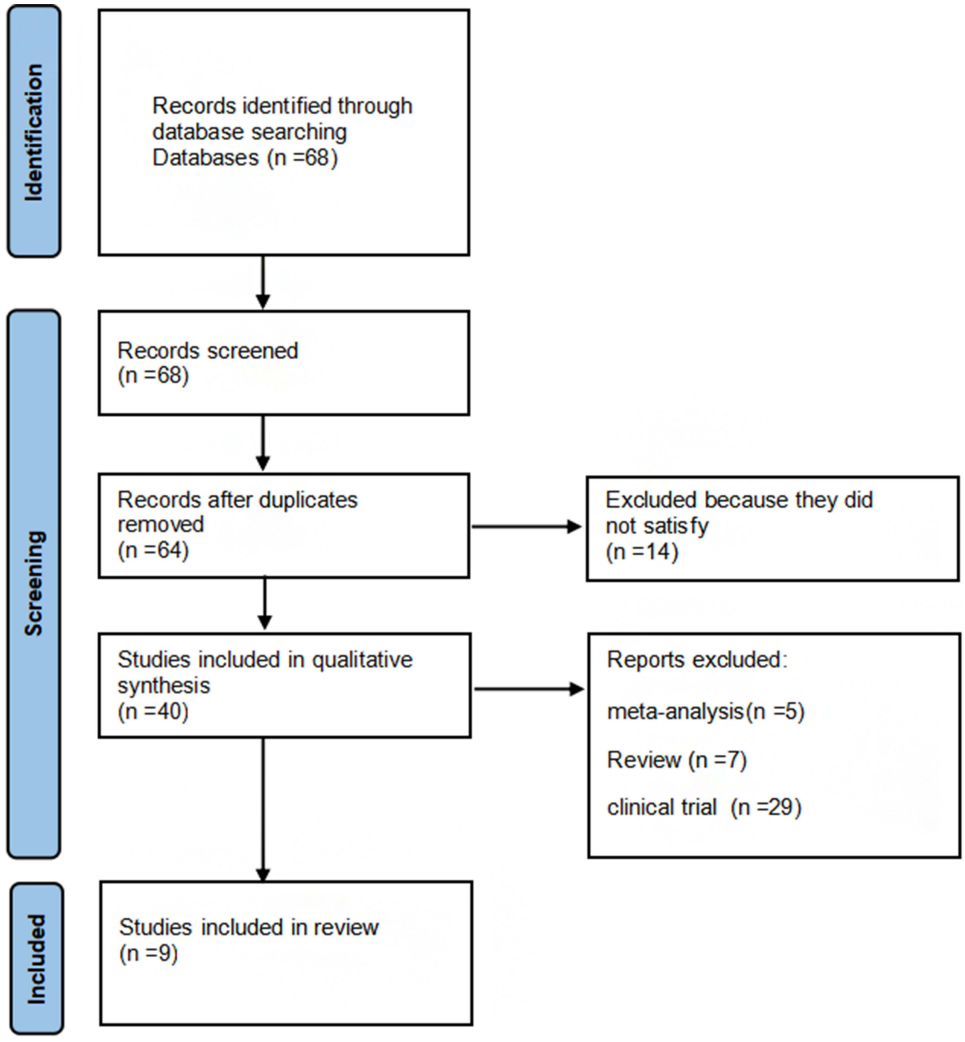

Figure 1 PRISMA flow diagram depicting the study selection process.

and lung cancer risk among individuals with COPD receiving ICS therapy ($p = 0.000$); and Figure 4 depicts the influence of gender on lung cancer risk in this population ($p = 0.720$).

A significantly increased risk of lung cancer was observed among individuals with COPD, with a pooled relative risk (RR) of 3.79 and a 95% confidence interval (CI) ranging from 3.60 to 3.98. Among individuals with COPD who did not

Table 1 Demographic and Study Characteristics of Included Studies

Study	Region	Year	Case COPD+LA/ COPD-LA	Controls COPD+LA/ COPD-LA	Case Women/ Men	Controls Women/Men	Case ICS/Non-ICS	Controls ICS/Non-ICS
DAVID M[5]	Southeastern Minnesota	1986	9/104	0/113	93/20	93/20	NA	NA
Gunnar R[6]	Bergen	2019	28/0	405/276	NA	NA	NA	NA
Helen A[7]	UK	2013	2757/9131	2286/35319	4863/15639	7025/21966	NA	NA
Yu Min Lee[8]	South Korea	2018	78/187	62/998	61/237	204/823	162/103	671/389
Samy Suissa[9]	Canada	2019	NA	NA	NA	NA	2304/818	507/447
Shih-Feng Liu[10]	China	2017	NA	NA	NA	NA	1290/12396	22/283
Tanyalak Parimon[11]	Washington	2006	NA	NA	NA	NA	517/9957	21/402
Lei Yang[12]	China	2015	NA	NA	158/184	345/439	NA	NA
Victor A. Kiri[13]	UK	2009	NA	NA	46/522	81/948	1095/375	81/46

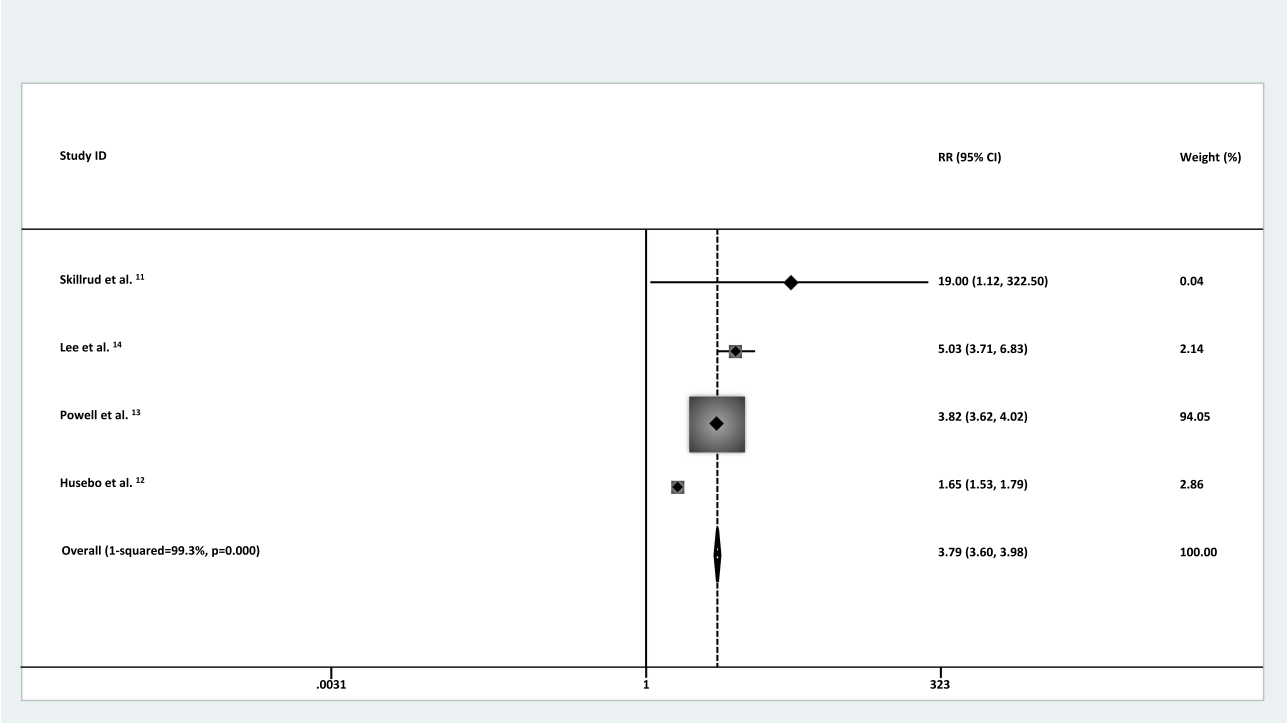

Figure 2 Forest plot presenting the pooled relative risk of lung cancer among individuals with COPD.

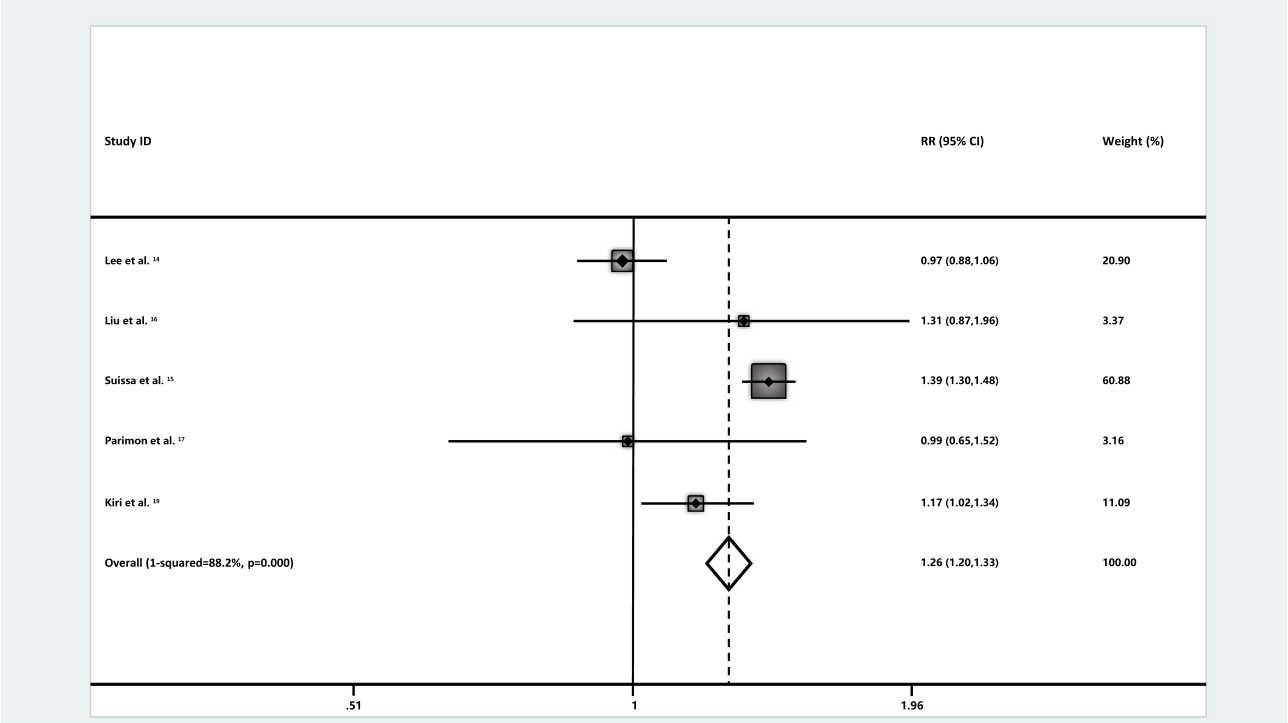

Figure 3 Forest plot presenting the pooled relative risk of lung cancer associated with ICS use among individuals with COPD.

https://doi.org/10.2147/COPD.S539213

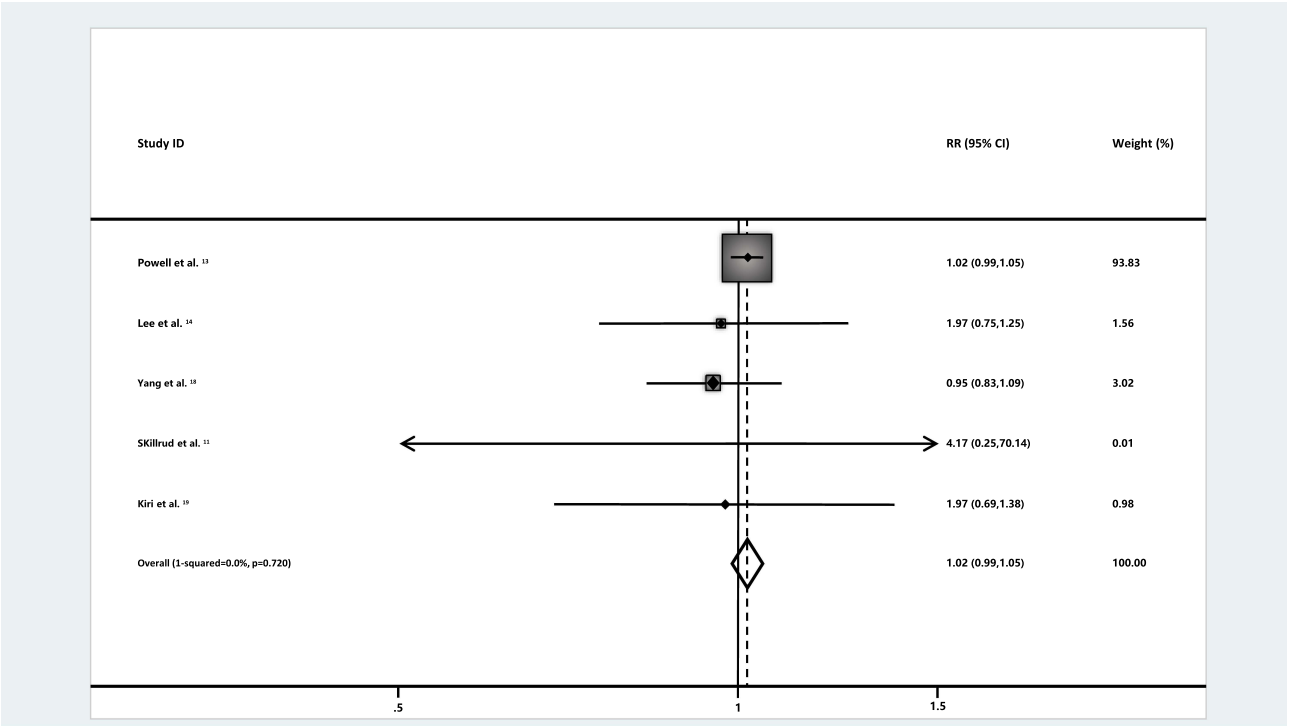

Figure 4 Forest plot presenting the relative risk of lung cancer among females with COPD.

use ICS, the relative risk of lung cancer was 1.26 (95% CI: 1.20–1.33). The relative risk for females was 1.02 (95% CI: 0.99–1.05), suggesting no statistically significant difference in lung cancer risk by gender.

Publication Bias

Sensitivity analyses demonstrated that the exclusion of any single study did not substantially alter the overall findings. Given the limited number of included studies, the Harbord test was used to assess the presence of publication bias. The results of the test indicated minimal to no evidence of publication bias, suggesting that its impact on the overall conclusions was negligible. Detailed results of the sensitivity and publication bias analyses are presented in Tables 2–4.

Table 2 Publication Bias Assessment for Studies Examining Lung Cancer Risk Among Individuals with COPD

Z/sqrt(V)	Coefficient	Sde.err.	t	P>| t |	[95% conf.interval]	
sqrt(V)	1.844449	0.0765633	24.09	0.002	1.515024	2.173874
bias	0.9049238	1.119511	0.81	0.504	−3.911944	5.721792

Notes: Number of studies=4; Root MSE=1.727; Test of NO: no small-study effects. P=0.504.

Table 3 Publication Bias Assessment for Studies Evaluating the Association Between ICS Use and Lung Cancer Risk in Individuals with COPD

Z/sqrt(V)	Coefficient	Sde.err.	t	P>| t |	[95% conf.interval]	
sqrt(V)	1.353089	0.4061561	3.33	0.045	0.0605188	2.645659
bias	−6.032416	3.003479	−2.01	0.138	−15.59083	3.525995

Notes: Number of studies=5; Root MSE=2.749; Test of NO: no small-study effects. P=0.138.

Table 4 Publication Bias Assessment for Studies Investigating Lung Cancer Risk Among Females with COPD

Z/sqrt(V)	Coefficient	Sde.err.	t	P>| t |	[95% conf.interval]	
sqrt(V)	0.0235292	0.0262439	0.90	0.436	0.0599907	0.1070492
bias	0.0170722	0.05647894	0.03	0.978	−1.78034	1.814484

Notes: Number of studies=5; Root MSE=0.9894 Test of NO: no small-study effects. P=0.978.

Discussion

The findings of this meta-analysis indicated a significantly elevated risk of lung cancer among individuals with COPD. The use of ICS was associated with a slightly increased risk in lung cancer development, and male individuals exhibited a higher risk compared to females. These conclusions were drawn from a synthesis of evidence from both observational and interventional studies, including cohort studies, case-control studies, and randomized controlled trials.

Given the elevated risk of lung cancer in individuals with COPD, there is growing advocacy for the implementation of targeted lung cancer screening among this population. Early detection strategies may contribute to a reduction in lung cancer–related mortality.[5] This meta-analysis focused on studies investigating the subsequent development of lung cancer in individuals with COPD and presents findings with important implications for real-world application.

Although the precise mechanisms underlying the association between COPD and lung cancer remain incompletely understood, emerging evidence suggests that immune dysfunction may play a key role in the pathogenesis of both conditions.[20] These diseases appear to share overlapping pathological features, including genetic susceptibility, altered epithelial and endothelial cell plasticity, dysregulated inflammatory responses—characterized by excessive extracellular matrix deposition—angiogenesis, and increased vulnerability to DNA damage and cellular mutations.[21,22]

From a preventive standpoint, a clearer understanding of the association between COPD and lung cancer may support the delivery of more individualized and evidence-based guidance for lung cancer prevention among individuals diagnosed with COPD. In clinical practice, heightened vigilance regarding the increased risk of lung cancer in this population is warranted. Incorporating regular lung cancer screening into routine follow-up care may offer clinical benefit, particularly for individuals with established COPD. For instance, tailoring the frequency of low-dose CT screening based on clinical risk profiles could facilitate earlier detection, thereby improving both survival outcomes and quality of life.

From a therapeutic standpoint, the management of individuals with coexisting COPD and lung cancer requires a comprehensive and integrative approach. Treatment strategies should be carefully tailored to account for the presence and severity of both conditions. Evidence from Parimon et al indicated a dose-response relationship, wherein the use of higher doses of ICS (≥1200 μg/day) was associated with a 61% reduction in lung cancer risk among individuals with COPD.[23] Due to frequently compromised pulmonary function in this population, oncologic interventions—whether surgical, radiological, or chemotherapeutic—should be planned following thorough assessment of pulmonary reserve to mitigate the risk of exacerbating underlying COPD. Such individualized approaches may contribute to more balanced and effective treatment outcomes.

Although COPD status, ICS exposure, and gender have been associated with variations in lung cancer risk, the biological mechanisms underlying these associations remain insufficiently understood. Further research is warranted to elucidate the pathophysiological pathways linking these factors to inform the development of targeted prevention and treatment strategies.

Several limitations should be considered when interpreting the findings of this meta-analysis. First, the relatively small number of included studies may have introduced potential bias and contributed to observed heterogeneity, arising from both clinical and methodological variations. Multiple assessment strategies were applied to evaluate heterogeneity, and a random-effects model was used to account for its presence in the pooled analysis. Nonetheless, heterogeneity remained a complicating factor in interpreting the results. Additionally, potential flaws in data interpretation and methodological limitations, as noted, may affect the reliability of conclusions, and the paucity of literature further renders the findings less robust. Caution is therefore warranted when generalizing these findings, and the influence of heterogeneity should be thoroughly considered. Future investigations are encouraged to adopt more rigorous study designs and to reduce heterogeneity, thereby improving the validity

and generalizability of meta-analytic conclusions. At the same time, the clinical relevance of understanding the relationship between COPD and lung cancer should be emphasized, with particular attention to the role of potential confounding factors in optimizing clinical decision-making and patient management. Second, the analysis was limited by the lack of detailed information regarding patterns of ICS use and occupational exposures, both of which represent important variables that may influence lung cancer risk. Moreover, the study did not sufficiently refine the research question by stratifying analyses by country/region or time period (eg, by decade), which may have obscured variations in COPD-lung cancer associations across different populations or over time. Third, due to limitations in the available data, it was not feasible to assess the impact of smoking status on lung cancer risk among individuals with COPD, despite its recognized significance as a major confounding factor. The literature included in this meta-analysis was restricted to studies published before March 20, 2022, introducing a degree of temporal lag that may limit the reflection of recent epidemiological changes. Furthermore, the paucity of background citations and related work in the introduction may have constrained the contextualization of this study within the broader field, particularly regarding evolving factors such as shifts in smoking prevalence and attitudes.

Conclusion

This meta-analysis identified a significant association between COPD and an elevated risk of developing lung cancer. The use of ICS among individuals with COPD was associated with a reduced risk of lung cancer, suggesting a potential protective effect. No statistically significant association was observed between the female sex and the risk of lung cancer in the context of COPD.

These findings offer clinically relevant insights for guiding lung cancer prevention strategies in individuals diagnosed with COPD. The results support a transition from conventional research paradigms toward more precision-based and comprehensive approaches in investigating the relationship between COPD and lung cancer. This analysis provides a scientific foundation for the formulation of evidence-based interventions, with the potential to improve patient outcomes and reduce disease burden.

This study contributed meaningfully to the advancement of medical research and the refinement of clinical practice. Future investigations may be enhanced by the integration of multi-omics technologies, such as genomics, transcriptomics, and proteomics, to enable a more comprehensive elucidation of the molecular mechanisms involved in the progression from COPD to lung cancer. Analysis of cross-omics relationships may facilitate the identification of critical molecular biomarkers, which could serve as novel targets for early diagnosis and precision therapy.

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Disclosure

The authors have no conflicts of interest in this work.

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