

The Role of Forkhead Box Transcription Factors in Lung Cancer Progression, Metastasis, and Chemoresistance: A Systematic Review

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Lung cancer is the major cause of cancer-related mortality. The family of regulatory proteins known as Forkhead Box (FOX) transcription factors is involved in cell cycle regulation, programmed cell death, epithelium–mesenchymal transition (EMT), and survival-related signaling. Emerging evidence suggests that dysregulation of FOX family members contributes to lung cancer aggressiveness and therapeutic resistance; however, clinical and translational findings remain heterogeneous. This research intends to comprehensively examine recent evidence regarding the role of FOX transcription factors as independent molecular variables influencing lung cancer progression, metastasis, and chemoresistance. PubMed/MEDLINE, Scopus, and Embase were searched for relevant articles published from January 2019 to January 2025. Eligible publications comprised original clinical or translational studies, including cohort, case-control, randomized trial, and experimental designs, that evaluated FOX transcription factors in lung cancer. Eight eligible studies were included, most of which focused on non-small cell lung cancer (NSCLC) and used translational experimental approaches. FOXM1 was repeatedly linked with enhanced proliferation, advanced disease, metastatic behavior, and poorer survival. At a mechanistic level, FOXM1 was associated with EMT induction and activation of PI3K/AKT and ERK pathways, supporting its role in therapy resistance. Conversely, FOXO1, FOXO3, FOXF2, and FOXF2 were more often associated with tumor-suppressive activity, including promotion of apoptosis, inhibition of invasion, and improved treatment sensitivity. FOX transcription factors appear to be important regulators of lung cancer biology, particularly in relation to disease progression, metastatic capacity, and response to therapy.

Keywords: Forkhead Box, FOXM1, FOXO1, FOXF2, Lung cancer, Metastasis, Chemoresistance.

Despite advances in early detection, targeted treatment, and immunotherapy, lung cancer still has a high global burden and remains associated with insufficient life expectancy.^{1,2} Non–small cell lung cancer (NSCLC) represents a large percentage

of lung cancer occurrences, Small cell lung cancer (SCLC) is less prevalent yet physiologically more severe.³ Tumor growth, metastatic spread, and therapeutic resistance are key contributors to treatment failure. Therefore, identifying molecular

regulators that drive these processes is essential for improving risk stratification and developing more precise therapeutic strategies.⁴

Transcription factors coordinate many of the gene networks that support malignant transformation and tumor adaptation. Forkhead Box (FOX) proteins constitute an extensive and highly conserved class of transcriptional regulators, characterized by a characteristic winged-helix motif that enables DNA binding.^{5,6} FOX proteins participate in the regulation of proliferation, apoptosis, differentiation, metabolism, oxidative stress responses, and EMT. In malignancy, abnormal FOX signaling can contribute to cancer initiation and progression or, conversely, limit malignant growth, with its effects shaped by the biological setting of the cell and the particular FOX family member involved.^{5,7}

Several FOX subfamilies have been implicated in lung cancer. FOXM1 is frequently overexpressed in NSCLC and is linked to cell proliferation, angiogenesis, EMT activation, and metastatic potential.⁸ By contrast, FOXO family members, particularly FOXO1 and FOXO3, are generally described as tumor-suppressive factors because they promote apoptosis and restrict cell-cycle progression.^{9,10} FOXP2 and FOXF2 have also been reported to influence invasion, differentiation, and key pathways, particularly PI3K/AKT, TGF- β , and Wnt/ β -catenin, which are closely related to aggressive tumor behavior and treatment resistance.¹¹

Metastatic dissemination remains an important driver of mortality in lung cancer. FOX transcription factors may contribute to this process by regulating EMT, extracellular matrix remodeling, and invasive behavior. In addition, intrinsic and acquired chemoresistance remain major barriers to durable treatment response.⁴ Several studies suggest that FOX proteins influence cellular sensitivity to platinum-based chemotherapy, targeted drugs, and other systemic therapies by modulating apoptosis, DNA repair, and survival signaling.¹¹

Although individual studies have examined specific FOX proteins in lung cancer, the evidence is dispersed across different study models and outcome measures. A focused synthesis is therefore needed to clarify how FOX transcription factors relate to tumor progression, metastasis, and

chemoresistance. This systematic review evaluates recent clinical and translational evidence on FOX family members as molecular variables that may influence these integrated lung cancer outcomes.

MATERIAL AND METHODS

Review Design

This systematic review was prepared in accordance with the PRISMA guidelines. The review question focused on whether FOX transcription factors act as independent molecular variables associated with lung cancer progression, metastatic behavior, and chemoresistance.

Literature Sources and Search Approach

PubMed/MEDLINE, Scopus, and Embase were systematically searched for articles published between January 2019 and January 2025. Controlled vocabulary, text words, and Boolean operators were used in combination: ("Forkhead Box" OR "FOX transcription factor" OR "FOXM1" OR "FOXO1" OR "FOXO3" OR "FOXP2" OR "FOXF2") AND ("lung cancer" OR "non-small cell lung cancer" OR "NSCLC" OR "small cell lung cancer" OR "SCLC") AND ("progression" OR "metastasis" OR "epithelial-mesenchymal transition" OR "EMT" OR "chemoresistance" OR "drug resistance" OR "therapy resistance"). The reference lists of eligible articles were also manually checked to identify additional relevant publications.

Study Selection Criteria

Criteria For Inclusion

1. Full-text primary research articles published from 2019 to 2025.
2. Studies conducted in human lung cancer, including NSCLC or SCLC.
3. Study designs including:
 - Clinical Trials (RCTs),
 - Observational cohort design,
 - Case control trial,
 - Translational experimental studies using in vitro or in vivo models with lung cancer-related endpoints.
4. Studies in which FOX transcription factors were evaluated as independent molecular variables.
5. Studies reporting at least one relevant outcome, including:
 - Tumor progression, such as proliferation, tumor size, stage, or growth;

- Metastasis, including invasion, EMT markers, lymph-node involvement, or distant dissemination;
- Chemoresistance, radioresistance, drug response, or therapy sensitivity.

Criteria For Exclusion

1. Review papers, editorial commentaries, conference-only abstracts without available full text.
2. Studies focused on cancers other than lung cancer.
3. Studies that did not directly evaluate FOX transcription factors.

4. Articles published in languages other than English.

Study Selection

All database-derived records were imported to citation management software, after which repeated records were deleted. All authors performed separate evaluation of titles and abstracts, followed by full-text assessment using the predefined eligibility criteria. Any disagreement was settled through discussion until mutual agreement was reached. Study screening was documented through a PRISMA flowchart.

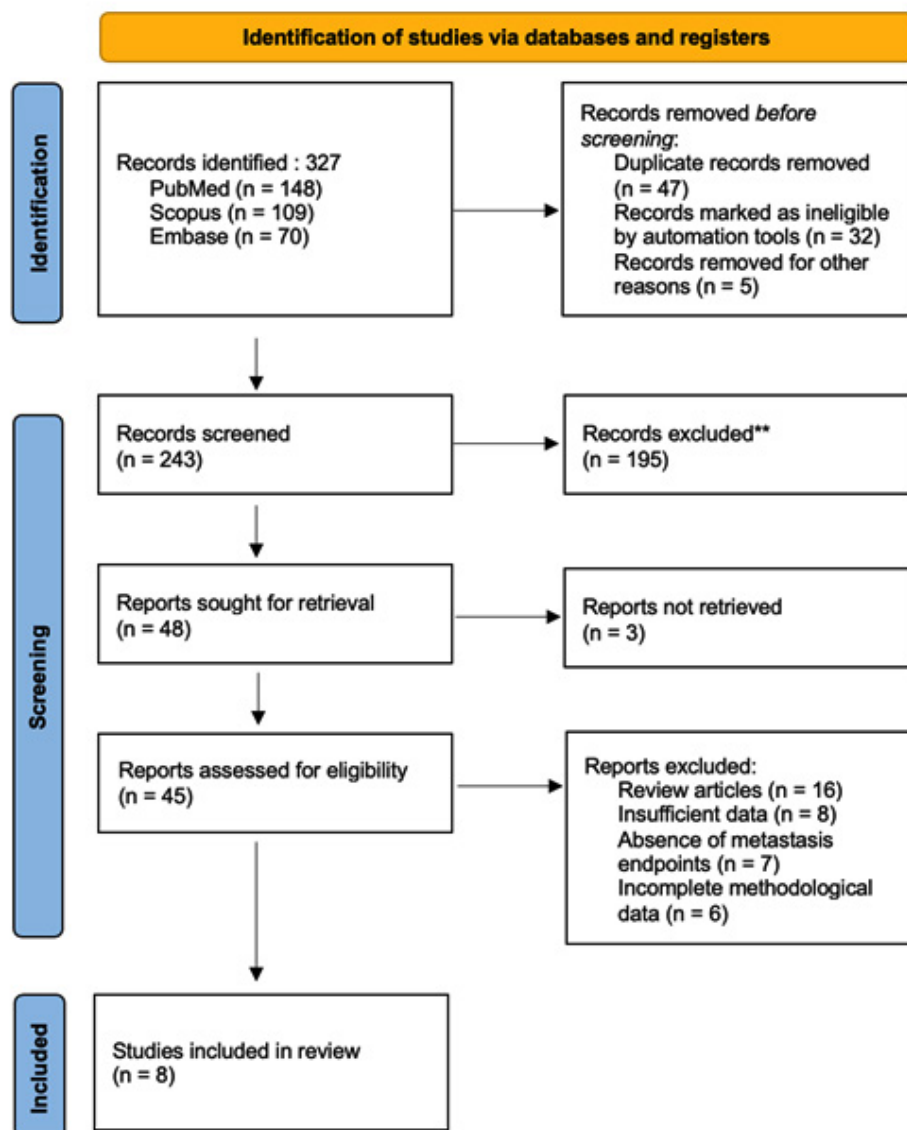


Fig. 1. PRISM Flowchart

Synthesis of Findings

Because the included studies varied in design, biological model, and outcome reporting, the evidence was summarized using a qualitative narrative approach. Results were organized by FOX subfamily and then mapped to the main outcomes of interest: progression, metastasis, and chemoresistance. In the original studies, statistical measures were reported qualitatively.

RESULTS

Study Selection

The first database search yielded 327 records (PubMed = 148, Scopus = 109, Embase = 70). After excluding 84 duplicate records, 243 titles and abstracts were assessed. Among them, 198 records were omitted because they were unrelated to lung cancer, had non-original study designs, or lacked FOX transcription factor assessment. Forty-five complete articles were evaluated against the selection criteria. Thirty-three were eliminated due to: review articles ($n = 16$), insufficient lung cancer-specific data ($n = 8$), absence of progression/metastasis/chemoresistance endpoints ($n = 7$), or incomplete methodological reporting ($n = 6$). A total of 8 studies fulfilled the selection requirements and were analyzed qualitatively. A meta-analysis was not conducted due to variations in study design and measured outcomes.

Characteristics of Included Studies

The eligible evidence consisted entirely of translational experimental studies that employed cell-based and/or animal models and, in some studies, were validated with clinical specimens. No randomized clinical trial directly evaluating FOX modulation as a lung cancer intervention was found within the search period.

Across studies with clinical samples, the pooled sample size exceeded 534 patients, with NSCLC as the predominant cancer type. FOX expression was commonly measured using immunohistochemistry, quantitative PCR, or Western blotting.

DISCUSSION

This systematic review synthesizes recent evidence (2019–2025) regarding the role of Forkhead Box (FOX) transcription factors

in lung cancer progression, metastasis, and chemoresistance. The findings consistently demonstrate that FOX family members exert context-dependent oncogenic or tumor-suppressive effects, with FOXM1 emerging as the most consistently pro-tumorigenic factor, while FOXO1, FOXO3, FOXF2, and FOXF2 predominantly exhibit tumor-suppressive functions. Although the majority of available evidence derives from observational cohorts and translational experimental studies rather than randomized clinical trials, the mechanistic consistency across studies strengthens the biological plausibility of FOX-driven tumor behavior.

FOX Transcription Factors and Tumor Progression

FOXM1 was the most extensively studied member across included studies and showed strong associations with tumor proliferation, advanced stage, and reduced overall survival.²⁰ Higher FOXM1 expression has been linked with increased Ki-67 expression, larger tumors, and shorter disease-free survival. Mechanistically, FOXM1 facilitates progression through the G1/S and G2/M phases of the cell cycle, by regulating genes required for mitotic progression.^{21,22} Its interaction with ERK and PI3K/AKT signaling pathways further enhances oncogenic signaling cascades. These findings are consistent with broader oncologic data positioning FOXM1 as a major factor governing cell-cycle activity and genomic instability.²³

In contrast, FOXO1 and FOXO3 demonstrated tumor-suppressive roles. Reduced FOXO expression in lung tumor tissues was associated with decreased apoptosis markers and increased tumor aggressiveness. FOXO proteins are known to transcriptionally activate pro-apoptotic genes such as Bim and p27, and their inactivation via AKT-mediated phosphorylation promotes cell survival. The reciprocal relationship between FOXM1 and FOXO signaling suggests a regulatory balance between proliferation and apoptosis that may be disrupted in lung carcinogenesis.²⁴

FOX Factors in Metastasis and EMT

Metastasis remains the leading contributor to mortality among patients with lung cancer. Several included studies linked FOXM1 overexpression to EMT, as reflected by reduced E-cadherin and increased vimentin expression. EMT supports

Table 1. Description of Eligible Studies (2019–2025)

No	Author (Year)	Design	FOX Factor	Sample Size	Lung Cancer Type	Primary Outcome	Key Findings
1	Su et al., 2024 ¹²	Experimental + tissue validation	FOXP2	20 patients	NSCLC	Proliferation, migration	FOXP2 is downregulated in lung cancer ($p < 0.001$); suppresses invasion via TGF- α inhibition
2	Wu et al., 2025 ¹³	Experimental	FOXO3a	Cell lines	NSCLC	Radiotherapy resistance	Elevated expression of FOXO3a—PINK1/Parkin associated with poorer survival outcomes and positive correlation with increased cellular resistance to radiotherapy.
3	Boyero et al., 2023 ¹⁴	Experimental	FOXF2	47 patients	NSCLC	Potential target gen	Elevated expression of FOXF2, KLF13, MICA, TCEAL1, and TGFB2 was significantly correlated with reduced overall survival. <i>FOXF2</i> (Hazard ratio (HR) = 0.66, 95% CI = 0.58–0.75, $p < 0.001$), <i>KLF13</i> (HR = 0.61, 95% CI = 0.52–0.72, $p < 0.001$), <i>MICA</i> (HR = 0.75, 95% CI = 0.66–0.86, $p < 0.001$), <i>TCEAL1</i> (HR = 0.72, 95% CI = 0.63–0.82, $p < 0.001$) and <i>TGFB2</i> (HR = 0.66, 95% CI = 0.58–0.75, $p < 0.001$).
4	Kawasaki et al., 2025 ¹⁵	Experimental	FOXA2	327 sample	SCLC	Metastasis	FOXA2 supported metastatic spread in SCLC by inducing a fetal neuroendocrine transcriptional program (p 0.04).
5	Xu et al., 2019 ¹⁶	Experimental	FOXF2	Cell lines	NSCLC	Overall prognosis	High FOXF2 expression was associated with poor prognosis and promoted tumor progression.
6	Olsen et al., 2021 ¹⁷	Experimental	FOXA2	Cell lines	SCLC	Metastasis	FOXA2 was predicted to significantly influence the transcription factor network dynamics.
7	Chen et al., 2019 ¹⁸	Experimental	FOXA1	Cell lines	NSCLC	Chemoresistance, EMT	High FOXA1 expression was associated with poor prognosis, shorter PFS/OS, EMT, and chemoresistance in lung adenocarcinoma.
8	Tang et al., 2025 ¹⁹	Experimental	FOXP1	Cell lines	SCLC	Chemoresistance	FOXP1 is a critical mediator and marker of chemoresistance in SCLC

cytoskeletal reorganization, invasion, and distant spread. The ability of FOXM1 to activate EMT-related transcriptional programs may explain its association with lymph-node metastasis and advanced clinical stage.^{22,23}

Other FOX members further illustrate the context-dependent nature of this transcription factor family. FOXP2 downregulation was reported to enhance aggressive lung cancer behavior, whereas FOXP2 activity appeared to limit invasion by suppressing TGF-beta signaling. Because TGF-beta is a major inducer of EMT and metastatic dissemination, loss of FOXP2-mediated restraint may permit a more invasive phenotype.¹²

FOXO3a also demonstrated context-dependent behavior. Increased activity of the FOXO3a-PINK1/Parkin axis was linked to worse survival and greater radioresistance in NSCLC. Although FOXO proteins are often considered tumor suppressors, FOXO3a may support mitochondrial quality control and stress adaptation via PINK1/Parkin signaling, enabling tumor cells to survive radiotherapy-induced stress.¹³

For FOXF2, the evidence was also mixed, indicating a context-dependent role in lung cancer biology. Increased expression of FOXF2 together with KLF13, MICA, TCEAL1, and TGFBR2 correlated with poorer overall survival, suggesting that some FOX-related transcriptional networks may be associated with aggressive tumor behavior.¹⁴ In contrast, FOXF2 has been described as a tumor-suppressive factor in several solid tumor settings, where it may inhibit invasion, regulate epithelial differentiation, and restrain EMT-related processes.¹¹ However, in NSCLC, FOXF2 has also been associated with poor prognosis and enhanced proliferation or migration through H19-mediated PTEN downregulation.¹⁶ These apparently contradictory findings suggest that FOXF2 does not exert a uniform biological effect across all tumor contexts. Instead, its function may be influenced by the surrounding molecular environment, interacting non-coding RNAs, downstream pathway activation, and the balance between tumor-suppressive and pro-metastatic transcriptional programs. Therefore, FOXF2 should be interpreted cautiously as a biomarker or therapeutic target, and future studies need to clarify whether its prognostic value differs according to

lung cancer subtype, expression pattern, and co-activated signaling networks¹⁴⁻¹⁶

In SCLC, FOXA2 was identified as an important regulator of metastatic competence. It may promote dissemination to multiple sites by activating a fetal neuroendocrine transcriptional program and shaping transcription factor network dynamics. These findings suggest that FOXA2 could support lineage plasticity and metastatic capacity in neuroendocrine lung tumors. FOXA1 overexpression in lung adenocarcinoma was also associated with shorter progression-free and overall survival, indicating possible prognostic relevance.^{15,17}

FOX Transcription Factors and Chemoresistance

Chemoresistance remains a significant therapeutic barrier in the management of lung cancer. Several experimental studies demonstrated that FOXM1 contributes to resistance to platinum-based chemotherapy by activating survival pathways, particularly PI3K/AKT signaling, and suppressing apoptosis. FOXM1 may also enhance DNA damage repair capacity, thereby reducing cytotoxic efficacy.²⁰

FOXP1 was reported to be both a mediator and a marker of chemoresistance in SCLC, reinforcing the concept that FOX proteins can influence treatment response. Taken together, the findings show that FOX transcription factors regulate not only growth and metastatic spread but also transcriptional and stress-response programs that allow cancer cells to tolerate therapy. The heterogeneity of these effects suggests that FOX-based biomarkers or interventions will require careful molecular stratification.¹⁹

In contrast, activation of FOXO3 increased cisplatin sensitivity *in vitro*, suggesting that restoring tumor-suppressive FOX activity could improve therapeutic response. However, clinical validation of FOX-mediated chemoresistance mechanisms remains limited. No randomized controlled trials directly targeting FOX modulation in lung cancer were identified within the review period, underscoring a significant translational gap.¹³

Clinical Implication

From a clinical perspective, FOX transcription factors may serve as both biomarkers and therapeutic targets in lung cancer. FOXM1 is particularly relevant because its overexpression is

associated with proliferation, EMT, metastasis, and chemoresistance. Therefore, FOXM1 inhibition may represent a potential therapeutic strategy to reduce tumor aggressiveness and improve sensitivity to systemic therapy, especially platinum-based chemotherapy. In contrast, reactivation of tumor-suppressive FOXO signaling may enhance apoptosis, restrict cell-cycle progression, and restore treatment sensitivity. However, the clinical application of FOXM1 inhibitors or FOXO activators remains limited by the absence of robust prospective and interventional evidence. Further multi-center validation and early-phase clinical trials are needed to determine whether FOX-targeted approaches can be safely and effectively integrated into lung cancer management.

Limitations

Several limitations should be considered. First, most included studies were either experimental or observational, limiting causal interpretation. Second, variation in study design, endpoint definitions, and laboratory methods prevented quantitative pooling. Third, publication bias may have favored studies reporting significant associations. Finally, the lack of randomized or prospective interventional studies means that direct clinical applicability remains uncertain.

Future Directions

Future research should prioritize prospective clinical validation of FOX expression as a prognostic and predictive biomarker. Additionally, early-phase clinical trials evaluating FOXM1 inhibitors or strategies to reactivate FOXO signaling may clarify therapeutic potential. Integration of genomic, transcriptomic, and proteomic profiling may further define FOX-driven molecular subtypes within lung cancer.

CONCLUSION

Forkhead Box (FOX) transcription factors are influential regulators of lung cancer, affecting malignant progression, metastatic spread, and treatment response. FOXM1 consistently acts as an oncogenic driver by promoting proliferation, epithelial–mesenchymal transition, and chemoresistance through activation of survival signaling pathways. In contrast, FOXO1, FOXO3, FOXO2, and FOXF2 generally exhibit tumor-suppressive functions, correlating with

enhanced apoptosis and reduced metastatic potential. However, important research gaps remain. Current evidence is still largely derived from preclinical, translational, and observational studies, while randomized controlled trials evaluating FOX-targeted therapeutic strategies in lung cancer are lacking. In addition, multi-center clinical studies with larger and more diverse patient populations are needed to validate FOX proteins as prognostic biomarkers, predictive markers of treatment response, and potential therapeutic targets. Therefore, further prospective and interventional research is essential before FOX-based approaches can be translated into routine lung cancer management.

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Ethics Statement

This research did not involve human participants, animal subjects, or any material that requires ethical approval.

Informed Consent Statement

This study did not involve human participants, and therefore, informed consent was not required.

Clinical Trial Registration

This research does not involve any clinical trials.

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Not Applicable

Author Contributions

Pande Ayu Naya Kasih Permatananda: Contributed to the conceptualization of the study, development of the research framework, literature search strategy, data interpretation, manuscript drafting, and critical revision of the

article; Sri Masyeni: Contributed to literature screening, data extraction, interpretation of clinical and translational findings, and revision of the manuscript for important intellectual content; Pande Ayu Berliana Dekananda Pandit: Contributed to literature selection, data organization, preparation of the study summary table, and manuscript editing.

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