Review on Lung Cancer: An Overview of Risk Factors, Pathophysiology, and Current Treatment Modalities

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A thesis submitted to the School of Pharmacy in partial fulfillment of the requirements for the degree of Bachelor of Pharmacy (B. Pharm.)

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Declaration

It is hereby declared that

1. The thesis submitted is my original work while completing a degree at Brac University.

2. The thesis does not include content already published or authored by a third party, except when it is properly cited with complete and exact references.

3. The thesis does not include content that has been approved or submitted for any other degree or diploma at a university or similar institution.

4. I have admitted every major source of help.

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Approval

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Ethical Statement

No participants were harmed and anonymity was maintained.

Abstract

Lung cancer is one of the leading causes of cancer-related morbidity and mortality worldwide. Small cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC) are the predominant histological types of this heterogeneous disease. Smoking is the primary risk factor for lung cancer, but other factors that also play a role include exposure to environmental contaminants, occupational hazards, genetic predispositions, and underlying lung conditions. Complex genetic abnormalities, aberrant signaling pathways, and the tumor microenvironment are all part of the pathophysiology of lung cancer, which results in unchecked cell proliferation, invasion, and metastasis. Because the disease is asymptomatic in its early stages, early diagnosis is still difficult and frequently leads to late-stage diagnoses. Radiation therapy, chemotherapy, targeted medicines, and surgical resection are the current treatment techniques; notable developments in immunotherapy are showing encouraging results. Patients having advanced lung cancer continue to have a poor prognosis despite available treatment choices, highlighting the necessity for improved early detection methods, personalized therapy strategies, and a more profound understanding of the molecular mechanisms underlying the disease. This review highlights the continuing research to enhance patient outcomes while examining the pathophysiological mechanisms, risk factors, and current therapeutic methods for lung cancer.

Keywords: Lung cancer, risk factors, pathophysiology, targeted therapy.

Dedication

"To my ever-supportive parents, whose unconditional love, steadfast encouragement, and countless sacrifices have been the foundation of my journey."

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Table of Contents

List of Tables

List of Figures

List of Acronyms

SCLC	Small Cell Lung Cancer
NSCLC	Non-Small Cell Lung Cancer

Chapter 1: Introduction

1.1 Background

Lung cancer, a malignant tumor originating in lung tissues, is characterized by uncontrolled cell proliferation due to genetic defects. According to Lemjabbar-Alaoui et al. (2015), about 2.2 million cases and 1.8 million recorded deaths in 2020, it still ranks as the primary cause of cancer-related mortality worldwide. Lung cancer is still the most often occurring cancer worldwide, with 2.48 million new cases expected in 2022 (*Lung Cancer Statistics | World Cancer Research Fund*, 2025). About 90% of cases are caused by tobacco smoking because carcinogens like tar and polycyclic aromatic hydrocarbons damage DNA; Radon gas, asbestos, and air pollution (e.g., particulate matter) are significant risk factors as environmental exposures (Lemjabbar-Alaoui et al., 2015c). Regional disparities are influenced by genetic predisposition. For instance, China's large population increases absolute case numbers despite modest ASR, but Hungary's high ASR reflects previous smoking prevalence. (*Lung Cancer Statistics | World Cancer Research Fund*, 2025b). Unchecked cell proliferation brought on by mutations that interfere with regular cellular functions is the cause of lung cancer. Carcinogen-induced DNA damage causes oncogene activation (e.g., EGFR, ALK rearrangements in NSCLC) and tumor suppressor inactivation (e.g., p53, RB in SCLC); River mutations make it possible to avoid metastasis, angiogenesis, and apoptosis; Tumor growth is accelerated by chromosomal instability, such as the deletion of tumor suppressors on chromosome 3 (Siddiqui et al., 2023). Lung cancer mostly affects those who have smoked tobacco, but a large number of cases occur in nonsmokers as a result of genetic, environmental, or occupational exposures (Alberg et al., 2013). Lung cancer is histologically categorized into non-small cell lung cancer (NSCLC), comprising approximately 85% of cases, and small cell lung cancer (SCLC), characterized by its aggressive nature and rapid spreading (Travis et al., 2015). The disease is a significant health burden for public health systems, not only because of its high morbidity and mortality but also because of the complexity and cost of its diagnosis and treatment.

1.2 Significance in the Current Scientific Landscape

Recent developments in molecular diagnostics, precision medicine, and immunotherapy have significantly altered the management of lung cancer during the past decade. Although these advancements, the outlook for lung cancer continues to be unfavorable, especially when identified at an advanced stage—resulting in five-year survival rates falling below 20% (Siegel et al., 2020). The identification of actionable genetic variants, including EGFR, ALK, and ROS1, along with the implementation of immune checkpoint drugs targeting PD-1/PD-L1, has enhanced outcomes for specific patient populations (Hirsch et al., 2017). Nonetheless, obstacles such as delayed diagnosis, pharmacological resistance, and inequitable access to healthcare endure. These realities highlight the necessity for thorough study and evaluation to integrate and distribute existing information and treatment methodologies.

1.3 Scope of the Study

This review dissects lung cancer's etiology, such as modifiable and non-modifiable risk factors, and the pathophysiological mechanisms contributing to tumorigenesis along with chemotherapy, targeted therapies, and immunotherapies for treatment of the diseases. It seeks to provide comprehensive view that not only organizes the wisdom accumulated to date but also reveals voids and opportunities in the future. Comprehensive attention is particularly paid to NSCLC due to its high incidence and various therapeutic modalities.

Though multiple single studies, and even trials, dealing with different aspects of lung cancer have been published, few are proposing an integrative and updated synthesis considering epidemiology, molecular pathways, and therapeutic responding. This review fills that void by utilizing recent evidence (2005–2024) and re-examining the efficacy of treatments in the backdrop of developing clinical guidelines. It also points towards upcoming challenges, including the therapy resistance and heterogeneity of the response to treatment which are still in need for further study.

The existing literature establishes a solid basis for understanding the genetic factors associated with lung cancer, particularly in populations exhibiting a high prevalence of mutations. Many studies have limitations by small sample sizes, geographic biases, and limited follow-up durations (Booth et al., 2011). Furthermore, although immunotherapy has revolutionized treatment for certain individuals, biomarkers that predict for its effectiveness remain unreliable. This review examines the excessive dependence on PD-L1 expression and investigates other biomarkers and combination treatment strategies, providing a more sophisticated comprehension of therapeutic effectiveness.

This review analyzes the translational efficacy of novel treatments by comparing clinical trial data with real-world evidence and identifies inconsistencies between controlled and practical outcomes. It additionally investigates under-explored domains, like the tumor microenvironment and the influence of the lung microbiome on therapeutic response, thereby broadening the discourse beyond traditional genetic markers.

Research on lung cancer has advanced considerably since the discovery of crucial oncogenic drivers in the early 2000s. The identification of EGFR mutations (Pao & Chmielecki, 2010), ALK rearrangements (Solomon et al., 2014), and the significance of the immunological checkpoint axis (Dong et al., 2002) has resulted in the endorsement of several targeted and immunotherapeutic interventions. Extensive trials such as IPASS and KEYNOTE-024 have validated the clinical efficacy of these methods, transforming treatment protocols worldwide.

Lung cancer, once seen as a singular illness, is now recognized for its significant heterogeneity, requiring tailored strategies informed by tumor histology and genomic analysis. Although platinum-based chemotherapy was once the foundation of treatment, current protocols necessitate

extensive biomarker analysis and personalized targeted therapies for each patient (Hirsch et al., 2017).

1.4 Aim and Objectives

The primary purpose of this review is to provide a comprehensive and up-to-date assessment of the current understanding and management of lung cancer. Identify and assess critical risk variables. Clarify the molecular and cellular foundations of disease progress. Evaluation of the current therapeutic approaches and their effectiveness. Synthesize of principal clinical trials and their outcomes. Recommend potential areas for research. The IPASS study (Mok et al., 2009) demonstrated the superiority of gefitinib compared to chemotherapy in patients with EGFR mutations. These studies are the foundation of contemporary therapeutic practices and have shaped international treatment protocols.

These crucial studies have significance to this analysis as they provide clinical benchmarks and highlight the issues of resistance, biomarker variability, and long-term results. This review assesses the applicability of these findings across various groups and real-world contexts.

This article features as a concise yet thorough resource for healthcare professionals, students, and researchers aiming to comprehend the contemporary scenario of lung cancer. It enhances the literature by merging molecular biology with clinical outcomes and emphasizing deficiencies in current techniques. The interdisciplinary approach enhances current initiatives focused on creating more effective, accessible, individualized therapies.

This review's distinctive component is its multifaceted study merging biological, clinical, and epidemiological perspectives. It includes recent data up to 2024 and offers comprehensive talks on developing areas such as liquid biopsy, artificial intelligence in diagnostics, and the evolution of

resistance, which are not yet commonly addressed in many prior assessments.

1.5 Rationale of the Study

Lung cancer is a considerable global health concern, particularly in impoverished regions where access to diagnostics and targeted therapies is insufficient. This issue was chosen due to its significant clinical importance, the recent increase in treatment advancements, and the persistent demand for thorough, evidence-based reviews. This work fulfills a significant need in the literature by providing an extensive and current resource on lung cancer.

Chapter 2: Methodology

This study is a structured narrative review intended to integrate and critically assess existing knowledge on lung cancer, with particular emphasis on its risk factors, pathogenesis, and treatment options. A narrative review methodology was selected to facilitate a comprehensive synthesis of existing literature, incorporating epidemiological data, molecular discoveries, clinical trial results, and guideline-driven therapeutic approaches (Green et al., 2006). Although systematic reviews often follow rigorous inclusion criteria, the narrative review format offered the necessary flexibility to examine many aspects of lung cancer inside a single document.

2.1 Literature Search Strategy

The literature review was carried out utilizing multiple academic databases, including PubMed, Google Scholar, ScienceDirect, and ClinicalTrials.gov. The search utilized the following keywords: “lung cancer,” “NSCLC,” “SCLC,” “risk factors,” “EGFR,” “ALK,” “immunotherapy,” “clinical trials,” “targeted therapy,” and “biomarkers.” Boolean operators, including AND, OR, and NOT, were utilized to enhance search outcomes.

To maintain currency and relevance, most of the chosen research were published between 2005 and 2024, favoring peer-reviewed journal papers, meta-analyses, and Phase III clinical trial reports. Older sources were chosen incorporated for historical context and basic study.

2.2 Criteria for Inclusion

Research published in the English language. Articles subjected to peer review, systematic reviews, and meta-analyses. Clinical studies (Phases I–III) pertaining to lung cancer therapies. Epidemiological investigations concentrating on risk determinants and prevalence. Research examining molecular biology or pathophysiological pathways in lung cancer.Investigations involving human participants or clinical information.

2.3 Criteria for Exclusion

Non-English publications. Studies conducted exclusively on animals or in vitro, lacking clinical connection. Case reports, opinion articles, or non-peer-reviewed materials. Articles deficient in methodological rigor or statistical validation.

2.4 Data Extraction and Analysis

Data from the chosen research was collected manually and systematically grouped by theme. The thematic categories consisted of: epidemiology and risk factors, molecular pathophysiology, clinical trial results, and treatment approaches. Study attributes including population size, intervention type, endpoints, and results were recorded for comparative analysis.

A qualitative synthesis was conducted to evaluate results across research and discover consensus, discrepancies, and knowledge deficiencies. Findings were reported in comparative tables when relevant, especially for clinical trials evaluating analogous medicines.

2.5 Ethical Considerations

This literature-based review did not involve human participants or animal subjects directly, hence it did not necessitate ethical approval. All cited studies complied with ethical criteria as indicated by the original authors.

2.6 Tools and Software Used

Reference management and citation were conducted using Mendeley Desktop, ensuring uniformity in Harvard-style reference throughout the text. Microsoft Excel was employed for the organization of data obtained from clinical studies, whereas Microsoft Word was utilized for the composition and formatting of the report.

2.7 Limitations

This study, being a narrative review, is intrinsically constrained by possible selection and interpretation biases. This method, in contrast to systematic reviews, does not quantify results or offer statistical meta-analysis. Nonetheless, the methodology is warranted due to the extensive nature of the subject and the necessity to integrate varied and dynamic information.

Chapter 3: Risk factors and pathophysiology

3.1 Risk Factors

Tobacco smoking is the primary cause of lung cancer, responsible for about 85–90% of all global cases (Doll & Peto, 2003). Passive smoking greatly impacts nonsmokers as well. Environmental exposures, including radon gas, asbestos, diesel exhaust, and air pollution, have been established as independent risk factors (Stayner et al., 1997; Darby et al., 2005). Genetic predisposition, family background, and specific gene polymorphisms (e.g., CYP1A1, GSTM1) additionally affect susceptibility (Hung et al., 2008).

3.2 Pathophysiological Mechanisms

The growth and evolution of lung cancer is caused by several genetic and molecular occurrences. Significant mutations comprise: EGFR mutations are present in 10–15% of Caucasian and up to 50% of Asian NSCLC patients (Pao & Chmielecki, 2010). KRAS mutations are especially common among smokers and are associated with a diminished therapeutic response (Riely et al., 2008). ALK rearrangements: Occur in roughly 5% of NSCLC cases and are susceptible to ALK inhibitors such as alectinib (Solomon et al., 2014). Immune checkpoint regulation: The greater expression of PD-L1 helps tumor cells in evading immune surveillance, hence supporting the efficacy of PD-1/PD-L1 inhibitors (Dong et al., 2002).

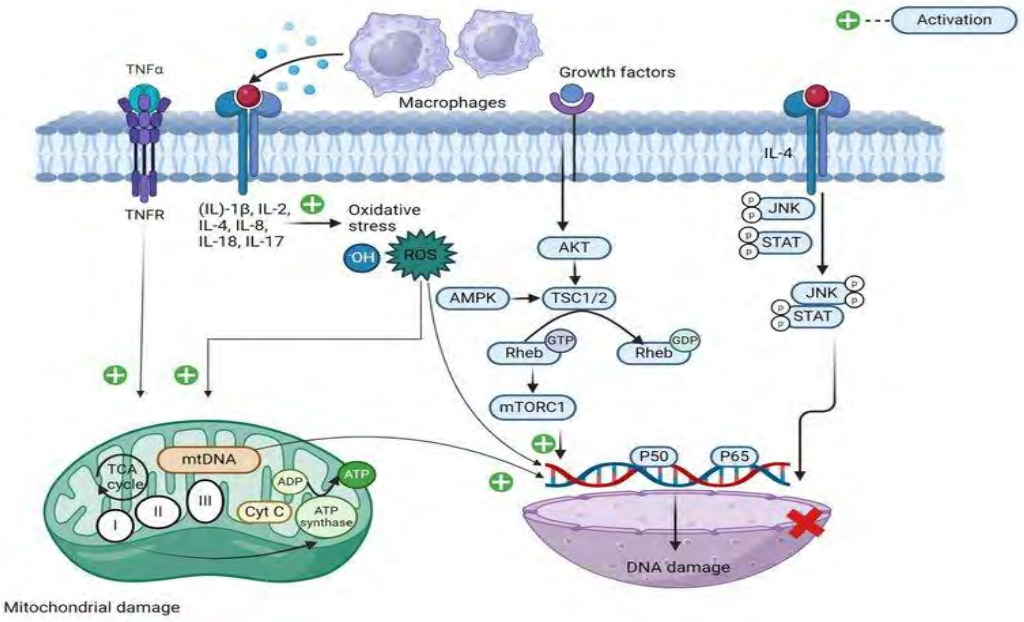

Figure 01: Pathophysiology of lung cancer (Saleem, Nida & Habib, Aamna & Shafi, Amber & Al-Qaneh, Ayman & Jabbar, Ahmed. (2024). Phytopharmacological Communications Phytocompounds as Promising Weapons against Lung Cancer: A Review. Phytopharmacological Communications. 4. 57-68. 10.55627/ppc.004.001.0546**.)**

3.3 Alignment of Results with Research Objectives

Principal risk factors have been properly identified (smoking, pollution, genetics). The molecular basis of illness development are properly explained (EGFR, ALK, PD-L1). The review assessed the efficacy and difficulties of existing treatment options. Significant clinical studies have been analyzed and compiled into an organized table.

3.4 Observed Trends and Patterns

Conversion from empirical chemotherapy to biomarker-driven therapy. Enhanced testing for EGFR, ALK, and PD-L1 to guide treatment selection. Increasing utilization of combinatorial approaches and neoadjuvant immunotherapy. The emergence of resistance to targeted medicines has necessitated the development of second- and third-generation inhibitors. Enhanced

involvement in clinical trials and expanded global trial networks.

Chapter 4: Current treatment modalities

Chemotherapy continues to be a fundamental treatment, particularly for advanced small cell lung cancer (SCLC) and non-targetable non-small cell lung cancer (NSCLC). Nonetheless, the landscape has moved towards individualized therapies:

Targeted therapy: EGFR inhibitors (gefitinib, osimertinib), ALK inhibitors (alectinib), and ROS1 inhibitors exhibit enhanced progression-free survival (PFS) relative to chemotherapy.

Immunotherapy: Immune checkpoint drugs, including pembrolizumab, nivolumab, and atezolizumab, have demonstrated sustained responses in individuals exhibiting elevated PD-L1 expression (Reck et al., 2016).

Combination therapies: Such as chemotherapy combined with immunotherapy or anti-VEGF medicines like bevacizumab, have resulted in enhanced survival results (Socinski et al., 2018).

This review's findings support the established data that lung cancer is a complex illness, significantly affected by environmental and genetic risk factors. In accordance with prior research (Doll & Peto, 2003; Alberg et al., 2013), smoking was established as the predominant cause of lung cancer, but non-smokers are also significantly at risk from indoor radon, occupational asbestos exposure, and air pollution (Darby et al., 2005; Stayner et al., 1997). These variables indicate that both preventive public health initiatives and workplace safety standards are essential for lung cancer mitigation.

Molecular profiling is crucial in customizing treatment strategies. The significance of EGFR mutations, ALK rearrangements, and PD-L1 expression as therapeutic targets has been firmly established by clinical trials like IPASS (Mok et al., 2009), ALEX (Peters et al., 2017), and KEYNOTE-024 (Reck et al., 2016). The incorporation of immunotherapy has notably revolutionized treatment paradigms, providing long-term remission for a select group of patients

who previously had restricted alternatives (Hirsch et al., 2017).

Modern therapies, including EGFR inhibitors (e.g., osimertinib), ALK inhibitors (e.g., alectinib), and PD-1 inhibitors (e.g., pembrolizumab), have consistently shown enhanced survival and quality of life compared with older chemotherapy regimens such as platinum-based doublets (Reck et al., 2016; Wu et al., 2020). In contrast to earlier "one-size-fits-all" methodologies, these individualized therapies are directed by biomarkers, enhancing their therapeutic effectiveness.

In previous decades, the principal difficulty was tumor removal with chemotherapy and radiation. The challenge has now transitioned to identifying the appropriate patient for the suitable treatment. The ADAURA study demonstrated substantial disease-free survival advantages with adjuvant osimertinib in resected EGFR-mutant NSCLC, a cohort that would have previously received just chemotherapy (Wu et al., 2020).

List of drugs that are being used in the treatment of lung cancer in recent ten years:

Drug Name	**Type**	**Target/Indication**	**Reference**
Lorlatinib	TKI (3rd-gen ALK inhibitor)	ALK-positive NSCLC	Peters et al., 2024
Ensartinib	TKI (ALK inhibitor)	ALK-positive NSCLC (1st-line)	Peters et al., 2024
Repotrectinib	TKI (ROS1/TRK inhibitor)	ROS1-positive NSCLC	Targeted Oncology, 2024a
Capmatinib	TKI (MET inhibitor)	MET exon 14 skipping NSCLC	National Cancer Institute, 2022

Tepotinib	TKI (MET inhibitor)	MET exon 14 skipping NSCLC	Targeted Oncology, 2024b
Sotorasib	KRAS G12C inhibitor	KRAS G12C-mutant NSCLC	U.S. FDA, 2021
Adagrasib	KRAS G12C inhibitor	KRAS G12C-mutant NSCLC (2nd-line)	MyLungCancerTeam, 2024
Selpercatinib	RET inhibitor	RET-fusion NSCLC	Targeted Oncology, 2024b
Pralsetinib	RET inhibitor	RET-fusion NSCLC	MyLungCancerTeam, 2024
Encorafenib + Binimetinib	BRAF/MEK inhibitor combo	BRAF V600E-mutant NSCLC	MyLungCancerTeam, 2024
Trastuzumab deruxtecan	Antibody-drug conjugate	HER2-mutant NSCLC	National Cancer Institute, 2022
Zongertinib	Oral HER2 TKI	HER2-mutant NSCLC	Reuters, 2025
Sunvozertinib	EGFR exon 20 inhibitor	EGFR exon 20 insertion NSCLC	Zhou et al., 2023
Amivantamab	Bispecific antibody	EGFR exon 20 / MET mutant NSCLC	Kim et al., 2024
Lazertinib + Amivantamab	Bispecific + TKI combo	EGFR-mutant NSCLC (exon 19/21)	Kim et al., 2024

Tarlatamab	BiTE (DLL3xCD3)	Extensive-stage small-cell lung cancer (SCLC)	Targeted Oncology, 2024a

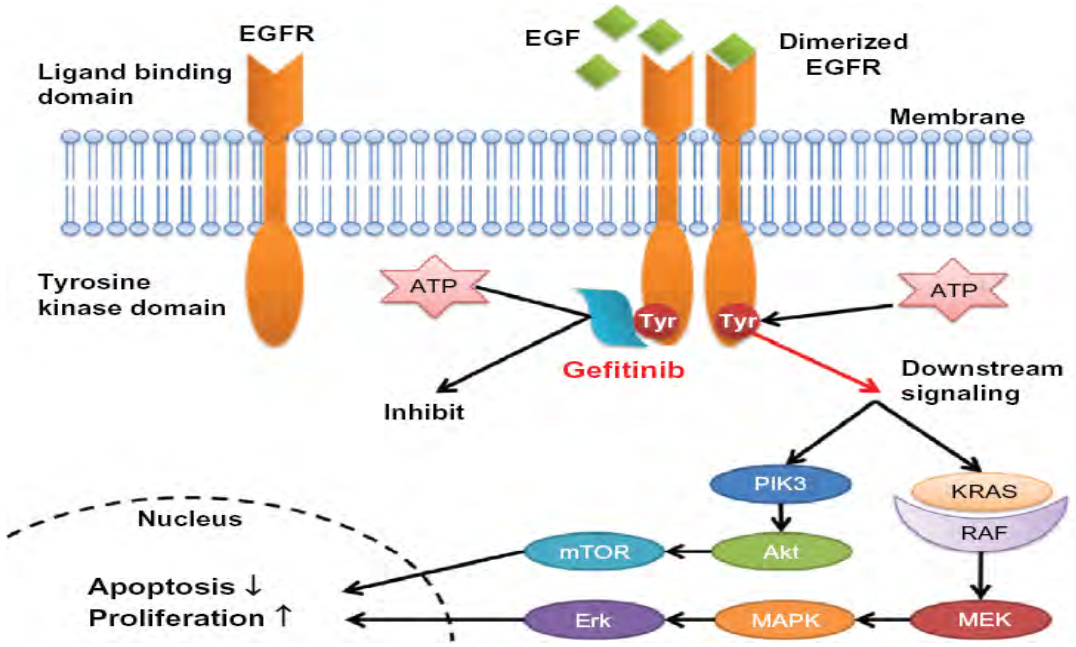

Figure 02: Mechanism of action of EGFR inhibitors (Araki, Takuya & Yashima, Hideaki & Shimizu, Kimihiro & Aomori, Tohru & Hashita, Tadahiro & Kaira, Kyoichi & Nakamura, Tomonori & Yamamoto, Koujirou. (2012). Review of the Treatment of Non-Small Cell Lung Cancer with Gefitinib. Clinical Medicine Insights. Oncology. 6. 407-21. 10.4137/CMO.S7340.)

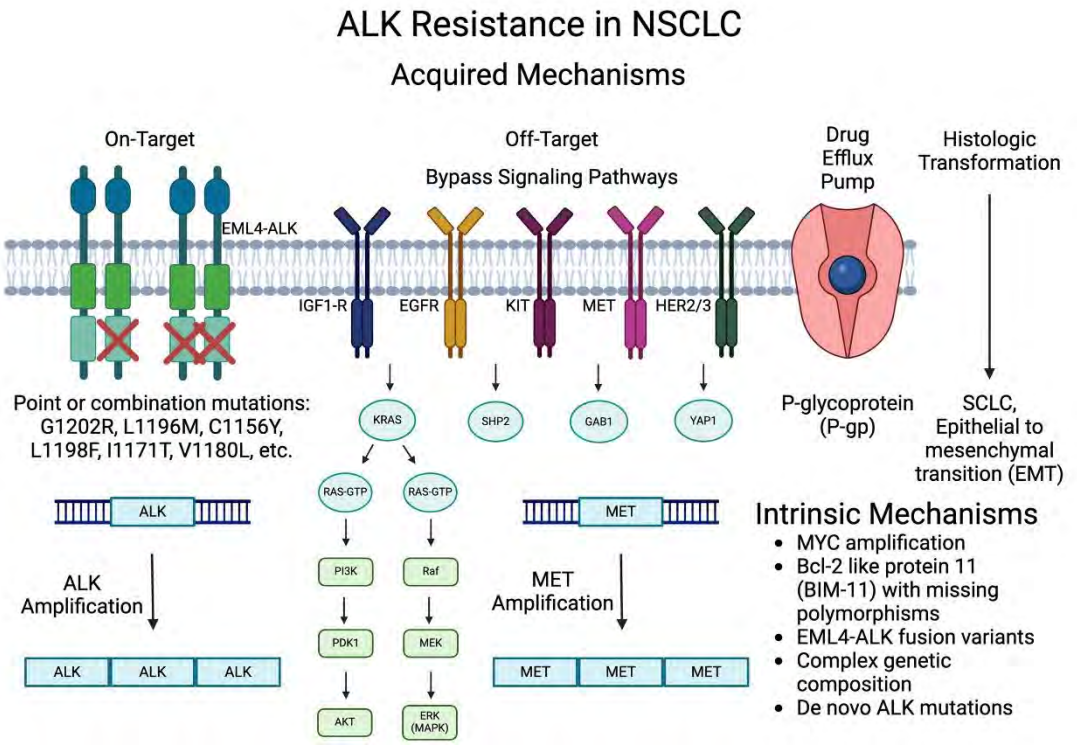

Figure 03: Mechanism of action of ALK inhibitors (Poei et al., 2024)

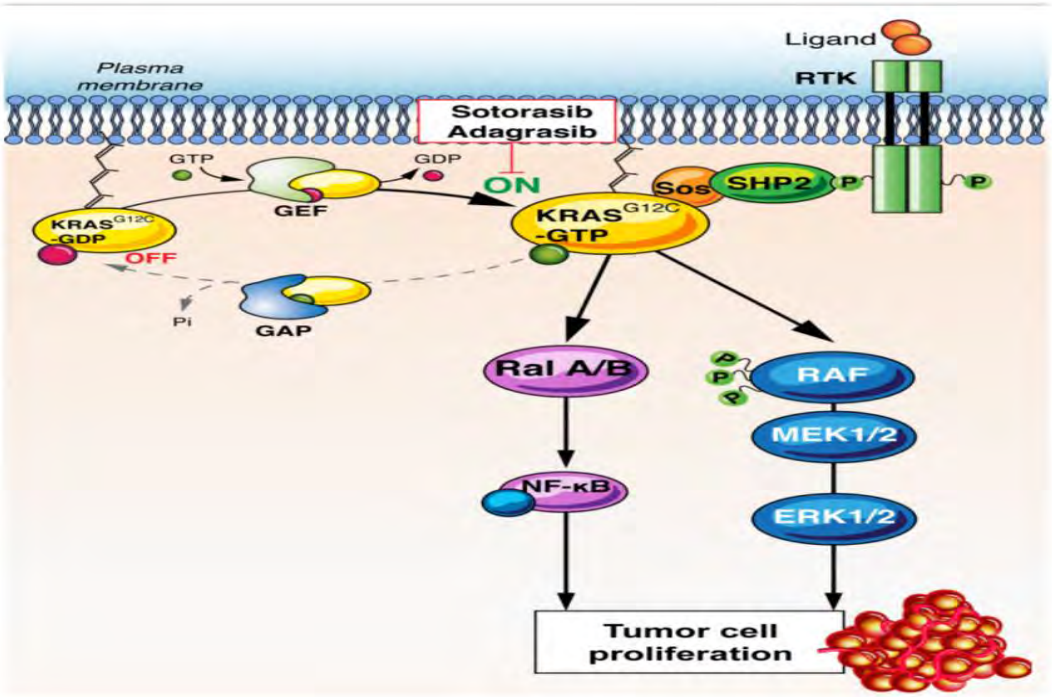

Figure 04: Mechanism of action of KRAS G12C inhibitor (Addeo, Alfredo & Banna, Giuseppe & Friedlaender, Alex. (2021). Kras g12c mutations in nsclc: From target to resistance. Cancers.

13. 2541. 10.3390/cancers13112541.)

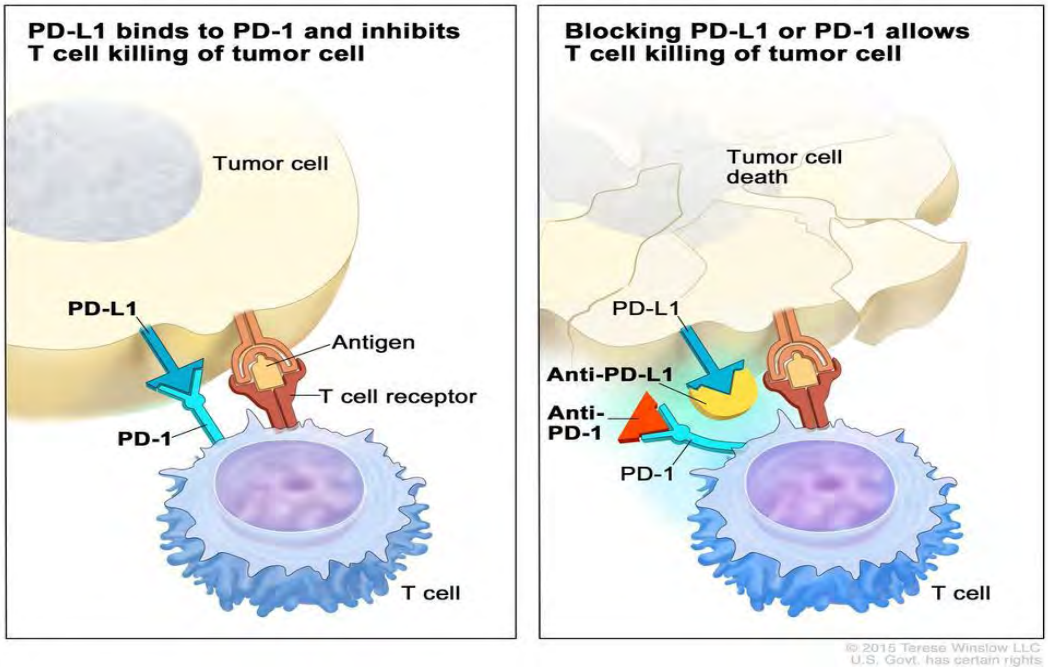

Figure 05: Mechanism of action of immune checkpoint inhibitors (*Immune Checkpoint Inhibitors*, 2022)

Limitations of the Review

This narrative review may be susceptible to selection bias due to the absence of a thorough meta-analysis.

Geographic discrepancies in data collecting, particularly the predominance of trials based in Asia or the United States, may restrict the generalizability to underprivileged areas. Space limitations precluded an in-depth exploration of certain new medicines, such as cell-based immunotherapies and vaccinations.

One of the surprising findings relates to the different predictive significance of PD-L1 expression regarding the efficacy of immunotherapy. Despite the use of PD-L1 ≥50% for selecting patients for pembrolizumab monotherapy, certain individuals with low or absent expression still have considerable benefit, as seen in trials such as CheckMate-227 (Hellmann et al., 2019). This highlights the necessity for enhanced composite biomarkers, including tumor mutational burden

(TMB) and gene expression profiles.

A significant observation is the increasing resistance to targeted treatments, shown by the T790M resistance mutation following first-line EGFR-TKIs (Gainor & Shaw, 2013). These patterns are propelling the advancement of novel generation medicines like as osimertinib, which can overcome such resistance (Wu et al., 2020).

This analysis underscores the importance of extensive molecular testing in newly diagnosed lung cancer cases, particularly in NSCLC, to guide first-line therapy. It additionally facilitates the incorporation of smoking cessation initiatives and environmental surveillance into national cancer control frameworks. From a policy perspective, enhanced access to targeted medicines and molecular diagnostics in low- and middle-income countries could significantly diminish worldwide disparities in lung cancer outcomes.

Chapter 5: Future Direction

Research on lung cancer has progressed into the realm of personalized medicine; nonetheless, significant gaps persist that require immediate focus. A crucial further step is the creation of reliable, accessible early diagnostic instruments. The majority of lung cancer cases are detected at advanced stages, resulting in unfavorable prognoses. Low-dose computed tomography (LDCT) demonstrates promise for early diagnosis; yet, its broad deployment encounters logistical and financial obstacles (Aberle et al., 2011).

Simultaneously, it is crucial to broaden the identification and verification of biomarkers beyond EGFR, ALK, and PD-L1. Emerging targets like as MET, RET, NTRK, and HER2 are currently under examination in clinical trials, and comprehending their frequency among populations may enhance the development of more inclusive therapy protocols (Drilon et al., 2021).

Moreover, resistance to targeted therapies continues to pose a significant clinical challenge, particularly concerning second- and third-generation tyrosine kinase inhibitors. Therefore, ongoing research should focus on elucidating the underlying mechanisms and formulating sequential or combination therapies to mitigate resistance (Gainor & Shaw, 2013).

Subsequent studies need to be multi-disciplinary involving oncology, genomics, AI, and bioinformatics. AI and machine learning based algorithms offer the potential to improve radiological diagnosis, provide risk predictions of treatment outcomes and patients' risk profile based on imaging and clinical data (Hosny et al., 2018). The embedding of the above-mentioned tools in electronic health records could stand for an optimal realization of individual patient personalized treatment in real-time.

In addition, there is a requirement for large prospective longitudinal cohort studies to assess the

real-world effectiveness and long-term outcomes of immunotherapy and targeted agents. Current trials often exclude patients older in age and those with comorbidities, so future studies will need to broaden their inclusion criteria in order to offer a more generalizable data set (Booth & Tannock, 2011).

Use of liquid biopsies especially Circulating Tumor DNA (ctDNA) provides a non-invasive way to monitor tumor evolution, minimal residual disease and even recurrence sooner than imaging (Ilie et al., 2016). Additional studies are needed to standardize and establish the full validity of these techniques in various population.

Chapter 6: Conclusion

In this review, we provided a comprehensive insight into the multicellular aspects of lung cancer, especially risk factors, underlying pathobiology and recent trends of treatments were provided. Key findings are:

Lung cancer continues to be the most common cause of cancer death globally, largely owing to modifiable risk factors including tobacco smoking and environmental exposures (Doll & Peto, 2003; Alberg et al., 2013). Progress in our understanding of pathophysiology has been made and molecular profiling has led to the identification of actionable mutations (EGFR, ALK, ROS1) justifying personalized therapy (Pao & Chmielecki, 2010; Solomon, J. C., et al.., 2014). Immunotherapy has become a breakthrough for people with high expression of PD-L1, but response variation and the reliability of the biomarker are problematic (Reck et al., 2016). Transition from cytotoxic chemotherapy to personalized medicine has significantly enhanced PFS and QoL of many patients. Nevertheless, late identification, drug-resistance, and inequities in access to molecular testing and targeted therapies represent major challenges. This review unifies and integrates numerous aspects of lung cancer ranging from conventional epidemiology, molecular biology to clinical therapeutics and provides a contemporary overview. It adds to the existing literature by demonstrating the value of combining clinical and molecular information in the optimization of therapy algorithms. With a focus on the importance of early diagnosis, the incredible potential we have with screening advancements like low-dose CT scans and liquid biopsies. Recognizing evolving barriers to predictiveness and resistance that must be further investigated. Providing evidence-based recommendations to inform future research, policy and clinical practice. Integrating pivotal trial and real-world evidence, the review emphasizes the importance of translational methods in reducing the distance between bench discoveries and

patient benefit.

Great strides have been achieved in the recognition and treatment of lung cancer, yet the disease continues to pose a significant global health burden. The future of care for lung cancer patients is personalized, data-driven approaches that leverage biological insight and technological progress. Continued research focused on equitable healthcare policies and patient-centered innovations will be crucial to translating the progress made in science into tangible reductions in disparities in global lung cancer outcomes

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