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Prognostic Value of Mutations in Key Cancer Driver Genes in Advanced Lung Adenocarcinoma

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Introduction

Lung adenocarcinoma, the most common histological subtype of Non-Small Cell Lung Cancer (NSCLC), remains a leading cause of cancer-related mortality worldwide, particularly in its advanced stages. Despite progress in targeted therapies and immunotherapy, prognosis for patients with advanced lung adenocarcinoma varies widely and remains poor overall. A major contributor to this variability is the genomic heterogeneity of the disease, which is driven by mutations in a number of key cancer driver genes [1]. These genetic alterations influence tumor behavior, treatment response, and clinical outcomes. Understanding the prognostic value of specific driver gene mutations can refine risk stratification, inform treatment decisions, and potentially uncover new therapeutic opportunities [2].

Description

In this study, we examine the association between mutations in key cancer driver genes and clinical outcomes in a large cohort of patients with advanced lung adenocarcinoma. Using publicly available datasets, including The Cancer Genome Atlas (TCGA) and clinical-genomic data from institutional cohorts and commercial platforms, we assess the mutational status of frequently altered genes such as TP53, KRAS, EGFR, STK11, KEAP1, and ALK. These genes were selected based on their known involvement in lung adenocarcinoma pathogenesis and therapeutic relevance. We analyze Overall Survival (OS), Progression-Free Survival (PFS), and response to standard therapies in relation to mutation status, while adjusting for key clinical variables such as age, smoking history, stage, and treatment type [3,4].

The analysis reveals distinct prognostic patterns associated with specific gene mutations. TP53 and STK11 mutations are significantly associated with worse overall survival, suggesting a more aggressive disease phenotype. In contrast, patients with EGFR mutations tend to exhibit improved survival, particularly when treated with Tyrosine Kinase Inhibitors (TKIs), reflecting the therapeutic sensitivity conferred by these alterations. KRAS mutations, especially in combination with co-mutations in STK11 or KEAP1, are linked to poorer outcomes and reduced response to immunotherapy, underscoring the need for combination treatment strategies in these subgroups. Furthermore, gene mutation combinations appear to be more predictive of prognosis than single-gene alterations alone, highlighting the value of integrated genomic profiling [5].

Conclusion

In conclusion, this study demonstrates that mutations in key cancer driver genes carry significant prognostic implications for patients with advanced lung adenocarcinoma. The findings support the incorporation of comprehensive genomic profiling into routine clinical assessment to better stratify patients and guide personalized treatment strategies. By identifying mutation-specific patterns of survival and therapeutic response, this research contributes to a more nuanced understanding of lung adenocarcinoma biology and paves the way for precision oncology approaches that consider not just the presence of driver mutations, but their broader clinical context and combinatorial effects.

Acknowledgment

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Conflict of Interest

None.

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