



**Kokand University
Andijon filiali**

**O'ZBEKISTON RESPUBLIKASI OLIY TA'LIM, FAN VA INNOVATSIYALAR VAZIRLIGI
QO'QON UNIVERSITETI ANDIJON FILIALI**

“FARMASEVTIKA, BIOTEXNOLOGIYA VA BIOTIBBIYOTDA ZAMONAVIY TADQIQOTLAR VA ILMIY YANGILIKLAR”

MAVZUSIDAGI XALQARO ILMIY-AMALIY

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**EGFR-ASSOCIATED MOLECULAR MARKERS OF LUNG CANCER IN
UZBEKISTAN**Mirakbarova Zebinisa^{1,2,3}, Turdikulova Shahlo⁴

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[*Corresponding author: E-mail: zebyniso@gmail.com]**Abstract**

Epidermal growth factor receptor tyrosine kinase inhibitors (EGFR-TKIs), including gefitinib and erlotinib, are reversible competitive inhibitors of the tyrosine kinase domain of EGFR, binding to its adenosine-5'-triphosphate (ATP)-binding site¹. The presence of somatic activating mutations in the EGFR gene, an increased gene copy number, and specific clinical and morphological tumour characteristics correlate with the high efficacy of these inhibitors, leading to significant tumour regression and improved clinical outcomes in patients with non-small-cell lung cancer (NSCLC)².

Lung cancer remains one of the leading causes of cancer-related mortality worldwide, including in Uzbekistan. The epidermal growth factor receptor (EGFR) is one of the most frequently mutated driver genes in lung cancer. However, unlike lung adenocarcinoma, EGFR mutations are relatively rare in squamous cell lung carcinoma (SCC), with a reported prevalence ranging from 3% to 18%³. The objective of this study was to investigate the prevalence of activating EGFR mutations (L858R mutation and E746_A750 deletion) in patients with SCC.

The study included formalin-fixed paraffin-embedded (FFPE) tumour tissue samples from 39 patients with SCC and 79 healthy individuals as a control group. DNA was extracted from these samples using nucleosorption with prior paraffin removal, followed by polymerase chain reaction (PCR) amplification to detect the L858R mutation and E746_A750 deletion in the EGFR gene. Genotyping analysis revealed the following results: in the case group, the genotype distribution was TT - 15 (38.5%), TG - 6 (15.4%), GG - 18 (46.1%), whereas in the control group, all individuals exhibited the TT genotype - 79 (100%) ($p < 0.0001$). The frequency of the E746_A750 deletion in the case group was 26 (66.6%), while the wild-type EGFR genotype was present in 13 (33.3%). In contrast, no EGFR deletions were detected in the control group ($p < 0.0001$).

Further studies on the role of EGFR activating mutations in different populations, including European and Asian cohorts, have demonstrated their impact on survival and response to targeted therapies⁴⁻⁶. In particular, EGFR mutations, especially exon 19 deletions and the L858R mutation in exon 21, have been associated with high response rates to targeted treatments⁷. Additionally, patients with EGFR L858R mutations who received anti-angiogenic therapy as part of their treatment regimen exhibited a significantly prolonged overall survival (OS) compared to those who had never received an anti-angiogenic agent⁸.

Analysis of this polymorphism provides an opportunity for the primary prevention of NSCLC and the personalization of treatment in this patient group. It facilitates early diagnosis, individualized therapy, and improvements in overall survival (OS) and progression-free survival (PFS). The findings of this study further confirm the critical role of EGFR mutations in SCC pathogenesis and their potential as prognostic biomarkers, which could aid in the development of more effective therapeutic strategies. However, large-scale studies in this cohort are necessary to further elucidate the underlying mechanisms and therapeutic potential of these mutations.

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