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# Targeted therapies in non-small cell lung cancer: A paradigm shift in oncology.

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## Introduction

Non-small cell lung cancer (NSCLC) accounts for approximately 85% of all lung cancers. The prognosis for advanced NSCLC was once grim, with limited options beyond cytotoxic chemotherapy. However, the discovery of driver mutations and molecular alterations has ushered in an era of precision medicine, transforming NSCLC treatment. Targeted therapies, designed to interrupt specific signaling pathways that promote cancer cell proliferation and survival, have changed the standard of care and significantly improved patient outcomes [1].

The identification of oncogenic driver mutations such as EGFR, ALK, ROS1, BRAF, KRAS, and MET has redefined how NSCLC is diagnosed and treated. These mutations are often mutually exclusive and are found predominantly in non-smokers or light smokers with adenocarcinoma histology. Advances in next-generation sequencing (NGS) and liquid biopsy technologies have made it feasible to identify these mutations early and accurately, allowing clinicians to select the most appropriate targeted therapy for each patient [2].

Mutations in the epidermal growth factor receptor (EGFR) gene are among the most common actionable mutations in NSCLC, especially in Asian populations. First-generation EGFR TKIs like erlotinib and gefitinib provided initial clinical benefit. However, resistance inevitably developed, primarily due to the T790M mutation. This led to the development of osimertinib, a third-generation TKI that effectively targets both sensitizing EGFR mutations and T790M. Osimertinib is now considered the first-line therapy for EGFR-mutated NSCLC due to its superior efficacy and central nervous system penetration [3].

Anaplastic lymphoma kinase (ALK) rearrangements, found in about 5% of NSCLC cases, are effectively targeted by ALK inhibitors such as crizotinib, alectinib, brigatinib, and lorlatinib. These agents have significantly prolonged progression-free survival compared to chemotherapy. Similarly, ROS1 rearrangements, which share similarities with ALK mutations, respond well to crizotinib and newer agents like entrectinib. Both ALK and ROS1 testing are now standard in the molecular workup of NSCLC [4].

Beyond EGFR and ALK, newer targets continue to emerge. BRAF V600E mutations are treated with a combination of BRAF and MEK inhibitors. MET exon 14 skipping mutations, identified in about 3-4% of NSCLC patients, can now be treated with MET inhibitors like capmatinib and tepotinib. The RET and NTRK gene fusions are rare but actionable, with therapies such as selpercatinib, pralsetinib, and larotrectinib demonstrating efficacy [5].

## Conclusion

Targeted therapies have revolutionized the treatment landscape of non-small cell lung cancer, marking a true paradigm shift in oncology. By focusing on the molecular drivers of disease, these therapies offer a personalized approach that significantly improves survival and quality of life. Continued investment in molecular diagnostics, drug development, and resistance research will be crucial to extending these benefits to a broader patient population and ultimately transforming lung cancer into a manageable chronic disease.

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