

# Preface

Non-small cell lung cancer (NSCLC) tumors with specific mutations in the epidermal growth factor receptor (EGFR) tyrosine kinase have been defined as ‘oncogene addicted’ to indicate their dependence on EGFR and their high susceptibility to the inhibitory effects induced by EGFR tyrosine kinase inhibitors (EGFR-TKIs; eg, gefitinib, erlotinib, afatinib). The most common *EGFR* mutations include a deletion in exon 19 (del E746\_A750) and a point substitution in exon 21 (L858R). During the last few years, eight phase III randomized studies comparing an EGFR-TKI versus platinum-based chemotherapy demonstrated that gefitinib, erlotinib, or afatinib are superior to chemotherapy in terms of response rate, progression-free survival, quality of life, and toxicity profile only in patients harboring activating *EGFR* mutations. Moreover, these mutations are also prognostic of a relatively indolent course of disease, regardless of treatment, as compared with classical NSCLC. Although the vast majority of patients harboring *EGFR* mutations respond to EGFR-TKI treatment, no patient achieves a definitive cure and inevitably acquired resistance occurs. The aim of the present handbook is to summarize the role of *EGFR* mutations in NSCLC and to describe the strategies for treating patients.

Guide to Targeted Therapies: EGFR mutations in NSCLC

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