Lung cancer is the leading cause of cancer-related mortality in both more and less developed countries (1). The past few years have witnessed a great change in the diagnosis and treatment of patients with advanced lung cancer. Surgery, radiofrequency ablation, radiation therapy and chemotherapy are used to be the basic treatments for NSCLC patients, but in recent years, immunotherapy and targeted therapy are increasingly important. Experts get to know the pathobiology of non-small-cell lung cancer (NSCLC) on a deeper level, which then accelerates our better understanding of certain proteins and small molecules (2).

Epidermal growth factor receptor (EGFR) has been proved to be the key molecule associates to lung cancer and it has become a significant therapeutic target for NSCLC (3). EGFR mutations predict responses to EGFR tyrosine kinase inhibitors (TKIs). In the beginning section of this new book *Targeted Therapy for Lung Cancer: Afatinib Focused*, we first introduce some topics about EGFR mutations, such as tumor heterogeneity, circulating DNA, molecular methods for somatic mutation testing, Kinase inhibitor-responsive genotypes and advances on EGFR mutation. It is well known that HER2 mutation is an oncogenic driver in lung cancer and it is responsible for 2% to 6% of lung adenocarcinomas (4). Therefore, in the second section of the book, we briefly review two papers about HER2 driven NSCLC.

It is well established that the progression-free survival (PFS) for patients receiving TKIs varies among different EGFR mutations (5). Gefitinib, erlotinib, afatinib and osimertinib are the options for treatment of patients with EGFR mutations. In the third and fourth section of the book, it gives an overview and future perspectives on the EGFR TKIs and lung cancer metastasis.

In recent years, physicians gradually recognize the role of afatinib in treating patients with metastatic NSCLC whose tumors have EGFR exon 19 deletions or exon 21 L858R substitutions as it was first approved by the Food and Drug Administration (DFA) in 2013. And now in Jan. 2018, the FDA expanded approval of afatinib (Gilotrif) to treat patients with lung cancers with EGFR L861Q, G719X, and S768I (6). Therefore, some hot and controversial topics about afatinib will be presented in the fifth section of the book.

The occurrence of intrinsic or acquired resistance may hinder the efficacy of EGFR TKIs, so the deeper understanding of mechanisms leading to inhibitor resistance will benefit the exploration of new therapeutic strategies. In the second last section of the book, it mainly focuses on the resistance mechanism of EGFR TKIs. Last, in the era of precision medicine, it is indispensable to study patients with lung cancer in a personalized way.

We hope all physicians and other interested readers will enjoy this book and find available and helpful in the daily clinical practice.

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