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Lung cancer is the leading cause of deaths in worldwide. e high mortality associated with this disease is due primarily to the fact that most of the lung cancers are diagnosed advanced stages when lung carcinoma (NSCLC) types squamous cell carcinoma (30%), adenocarcinoma (including the non-invasive type of bronchial-alveolar carcinoma, BAC; 45%), and large-cell carcinoma (9%) [5,6]. Advances in molecular technologies are providing insight metastasis. Molecular advances have provided unique opportunities for rational targeted therapies for lung cancer that have led to an emerging and exciting new area of therapy, which takes advantage of cancer-speci c molecular defects that render the cancer cells to respond speci c agents.

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In this setting, the analysis of molecular abnormalities of lung cancers is becoming increasingly important and represents an interesting challenge for adequate integration of routine pathological and molecular examination for the diagnosis, classi cation, and choice of therapy options [8]. Although many molecular abnormalities have been described in clinically evident lung cancers, relatively little is known about the molecular events preceding the development of lung carcinomas and the underlying genetic basis of lung carcinogenesis. Several studies have provided information regarding the molecular characterization of the pre-neoplastic changes involved in the pathogenesis of lung cancer in last decade, especially squamous cell carcinoma and adenocarcinoma. Many molecular changes have been detected in histologically normal respiratory mucosa of smokers.

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e genetic abnormalities of lung adenocarcinomas include point mutations of dominant oncogenes, K-rash, BRAF, and EGFR, and tumour-suppressor genes TP53 and p16Ink4 [2, 46 ± 49]. In lung cancer, activating K-transmutations preferentially target adeno-carcinoma histology (20 ± 30%). Most K-transmutations lung cancer are G->T transversions and they a ect exons 12 (~90% of mutations). ese types of K-transmutation have been associated with tobacco related cancer. Activation of BRAF gene mutations, a Raf serine-threonine kinase pathway component, has also been detected in lung adeno-carcinoma cell lines (11%) and primary tumors (3%). Recently, a body of evidence has indicated that EGFR mutations a ecting the tyrosine kinase domain of the gene (exons 18 ± 21) are present in approximately 20 ± 55% of adenocarcinomas that they are almost entirely absent in other types of lung cancer. EGFR mutations are somatic in origin, and they occur more frequently in adenocarcinomas in patients who have never smoked (51 ± 68%), women (42 ± 62%), and patients from countries in East Asia (30 ± 50%) [48,51 ± 54]. In addition, although infrequent t (3%), HER-2/Neogene mutations have been detected predominantly in lung adenocarcinoma histology and patients with an East Asian ethnic background. e remarkable similarities of mutations in EGFR and HER2/aneugens involving adenocarcinoma histology type, mutation type, gene location (tyrosine kinase do-main),

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