

Preface

The inflammatory tumor microenvironment (TME) has a multifaceted role in tumor initiation, progression, and metastasis. Whereas genetic changes are critical for the malignant transformation of epithelial cells, we now understand that components of the developing lung TME are active participants in the events precipitating lung cancer initiation and progression. Inflammation can influence the TME to orchestrate creation of a hypoxic environment, increased angiogenesis and invasion, as well as expand stem cell phenotypes.

Although the origin of the inflammatory TME is an active area of investigation, two pathways have been postulated. In the intrinsic pathway, the inflammatory microenvironment is generated by genetic alterations within premalignant or neoplastic cells that lead to increased production of inflammatory mediators. Conversely, in the extrinsic pathway, the inflammatory environment is accommodating to cancer development and progression. Thus, inflammation could be present due to an unresolved infection or chronic exposure to carcinogens.

A body of evidence exists at the preclinical, clinical, epidemiological, molecular, and pathological levels suggesting that inflammation is strongly associated with the development of lung cancer. Here, inflammation and lung cancer is addressed in the context of the molecular pathology of the disease as well as the relationship to chronic obstructive pulmonary disease. In addition, the important relationships between inflammation, epithelial mesenchymal transition (EMT), and lung cancer initiation and metastases are reviewed. Our understanding regarding inflammation-dependent regulation of angiogenesis and eicosanoid metabolism has opened new opportunities to translate findings to clinical interventions in prevention and therapy.

Finally, research in understanding the nature of inflammation and immunity in the lung cancer TME has led to ground-breaking studies applying immunotherapeutic approaches for lung cancer. The phenotype of the adaptive immune infiltrate and the diversity of cellular elements that either promote eradication of malignancy or facilitate an immunosuppressive TME favoring tumor progression are being assessed in the context of the mutational landscape of evolving and established lung cancer. These studies, reviewed here, hold promise for additional progress in controlling inflammation and leading to further improvements in immunotherapy for lung cancer.

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