AJPMHS GLOBAL RESEARCH NETWORK

**AMERICAN Journal of Pediatric Medicine and Health Sciences**
*Volume 01, Issue 10, 2023 ISSN (E): 2993-2149*

# Pathomorphology of Lung Cancer Development

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**Abstract:** Lung cancer is one of the common malignant tumors that threaten human life with serious incidence and high mortality. According to the histopathological characteristics, lung cancer is mainly divided into non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC). NSCLC accounts for about 80–85% of lung cancers. In fact, lung cancer metastasis is a major cause of treatment failure in clinical patients. The underlying reason is that the mechanisms of lung cancer metastasis are still not fully understood. The metastasis of lung cancer cells is controlled by many factors, including the interaction of various components in the lung cancer microenvironment, epithelial-mesenchymal transition (EMT) transformation, and metastasis of cancer cells through blood vessels and lymphatics. The molecular relationships are even more intricate. Further study on the mechanisms of lung cancer metastasis and in search of effective therapeutic targets can bring more reference directions for clinical drug research and development. This paper focuses on the factors affecting lung cancer metastasis and connects with related molecular mechanisms of the lung cancer metastasis and mechanisms of lung cancer to specific organs, which mainly reviews the latest research progress of NSCLC metastasis. Besides, in this paper, experimental models of lung cancer and metastasis, mechanisms in SCLC transfer and the challenges about clinical management of lung cancer are also discussed. The review is intended to provide reference value for the future research in this field and promising treatment clues for clinical patients.

**Keywords:** lung cancer, pathomorphology, development, factors, mechanism.

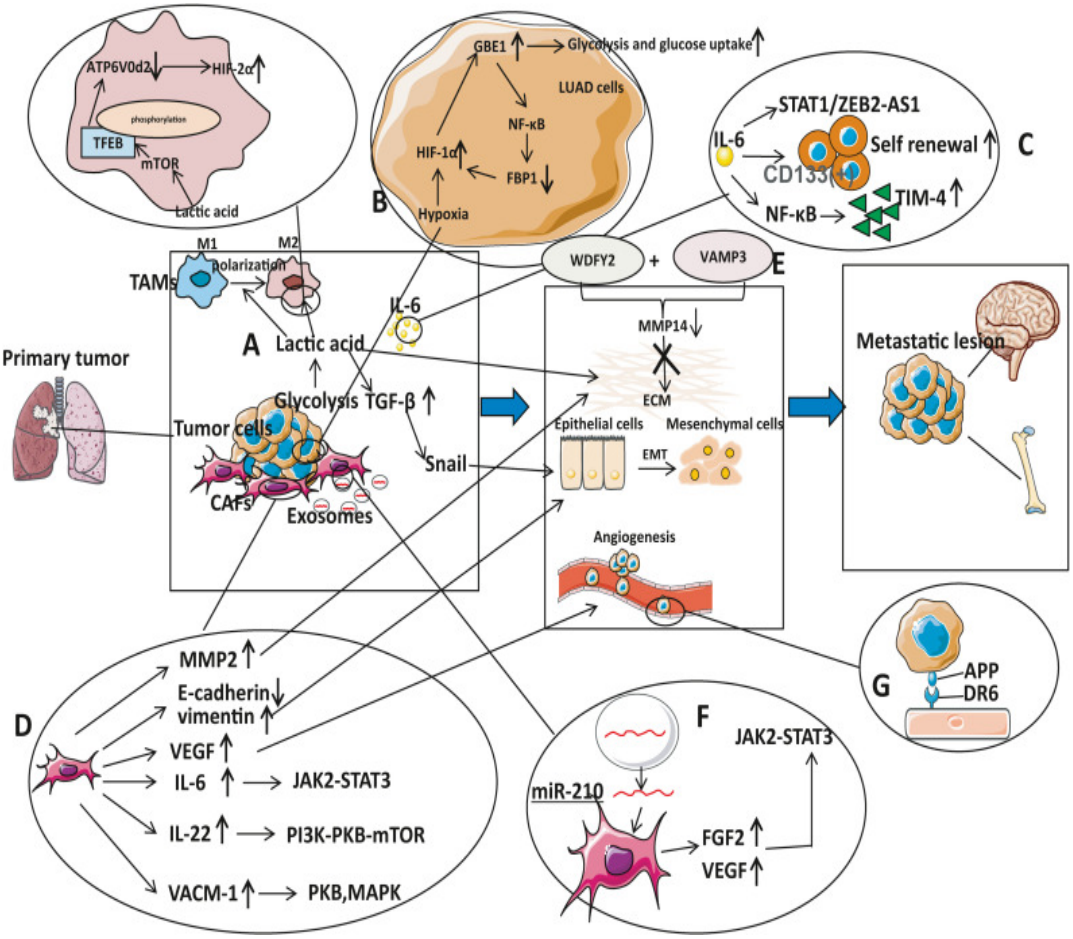
Lung cancer metastasis is a process in which the primary malignant tumor in the lung leaves the primary site and grows at a distance through a variety of ways. The most common sites of metastasis are brain, bone, lymph nodes, and liver [1], [2]. Lung cancer metastasis is an extremely complex process including the involvement of lung cancer microenvironment and lung cancer stem cells (LCSCs) [3], [4], [5] as well as various mechanisms such as EMT formation and angiogenesis and lymphangiogenesis [6], [7], [8]. These influencing factors are related to many non-coding RNAs (ncRNAs) [9], [10], related factors of lung cancer metastasis and multiple signaling pathways. The purpose of the study on the mechanisms of lung cancer metastasis is to find more efficient schemes for the control of lung cancer metastasis and to provide guidance for clinical treatment. Here we mainly review recent advances of NSCLC metastasis, focusing on the influencing factors of lung cancer metastasis, the relevant molecular mechanisms and the mechanisms of lung cancer metastasis to specific organs. We also briefly explain the establishment of lung cancer metastasis models, the mechanisms of SCLC metastasis and the correlational studies in the clinical treatment of lung cancer, hoping to be able to offer references for future studies on lung cancer metastasis.

Lung cancer microenvironment is composed of lung cancer cells, stromal cells and extracellular matrix (ECM) which is suitable for the growth and development of tumor cells. Among them, stromal cells include fibroblasts, immune cells, and some vascular endothelial cells. In 1889,

Stephen Paget proposed the "seed-soil" theory on the mechanism of tumor metastasis. This revealed the correlation between tumor cells and tumor microenvironment (TME), which laid a foundation for future studies. The coordination between tumor cells and the tumor microenvironment is the foundation for the metastasis of tumor cells to specific organs and the formation of metastases. In 2018, Lambrechts et al. [11] revealed the highly complex nature of the lung cancer microenvironment. Through in-depth studies, they identified 52 stromal cell subtypes to produce the most complete lung cancer cell map. This could be useful for scientists to study these cell types in the future. At the same time, they found a positive correlation between tumor aggressiveness and the number of stromal cells in the microenvironment. These stromal cell subtypes and their related marker genes may be used as biomarkers to evaluate the prognosis and therapeutic efficacy of lung cancer patients. Deeper understanding of the microenvironment of lung cancer will certainly provide a different perspective for the anti-lung cancer therapy targeting the microenvironment of lung cancer, which will be helpful to the research and development of anti-cancer drugs and drug strategies.

The TME can promote the proliferation and invasion of tumor cells. Acidity, hypoxia and inflammation are the three main characteristics of TME [12], [13], [14], [15]. It has been proved that these characteristics of TME can help tumor invasion and metastasis through different mechanisms. Tumor cells can also undergo glycolysis under aerobic conditions to produce large amounts of lactic acid, making the TME acidic. This abnormal form of metabolism is called the Warburg effect [16]. However, lactic acid is no longer a wasted product of tumor metabolism. Studies have shown that lactic acid is an important source of nutrients in the tricarboxylic acid cycle in cancer cells. And it plays a promoting role in the growth, proliferation and metastasis of lung cancer cells [17], [18].

Li et al. [19] found lactic acid can cause ECM remodeling and increase the level of transforming growth factor-β1 (TGF-β1), which is tantamount to induce the expression of snail and EMT process (Fig. 1A and Table 1). Liu et al. [20] revealed the role of lactic acid in M2-like transformation of tumor-associated macrophage (TAM) phenotypes. Lactic acid from glycolysis activates mammalian target of rapamycin (mTOR) and leads to transcription factor EB (TFEB) phosphorylation [21], [22], which reduces the expression of ATPase H+ transporting V0 subunit d2 (ATP6V0d2), the target gene of TFEB. Their study further showed that ATP6V0d2 results in hypoxia inducible factor-2α (HIF-2α) degradation, so as to play the function of inhibiting tumor metastasis (Fig. 1A). The down-regulation of ATP6V0D2 stabilizes HIF-2α and increases the expression of its downstream target gene vascular endothelial growth factor (*VEGF*) and M2-related genes. What's more, a study has found that lactic acid produced in metabolism, as a key regulatory factor, participates in the modification of histone lysine lactic acid and promotes the expression of related genes in M2 macrophages [23] One of the breakthroughs made in this study is the discovery of a new epigenetic regulation mode of histone lactic acid modification, which provides a new way for the study of metabolite lactic acid in tumors.




Studies have shown that cancer cells exposed to oxygen deprivation in the body are more aggressive [24]. Liu et al. [25] discovered that hypoxia can induce increased expression of snail family transcriptional repressors 1 and 2, which promoting the transcription activity of β-catenin and causing EMT transformation . Mo et al. [26] investigated the effect of exosomes on tumor metastasis in different oxygen environments. It was found that exosomes released by NSCLC cells under hypoxia could contribute to metastasis of lung cancer cells. Li et al. [27] found the related mechanism of anoxic glycolysis triggered by hypoxia. They showed that HIF-1α gives rise to the increase of glycogen branching enzyme 1 (GBE1) in lung adenocarcinoma (LUAD) cells, which suppresses fructose bisphosphate 1 (FBP1) expression through the nuclear factor-κ-gene binding (NF-κB) signaling pathway. The reduction of FBP1 pushes for the increase in HIF-1α, which constitutes a positive feedback that promotes anaerobic glycolysis and glucose uptake (Fig. 1B).

Chronic inflammation can also be beneficial to the invasion of cancer cells. Li et al. [28] found that interleukin-1-β (IL-1-β) was widely present in lung cancer microenvironment as a strong pro-inflammatory cytokine (Table 1). It advances the EMT phenotype. Chen et al. [9] showed the mechanism of IL-6 in the invasion and metastasis of lung cancer (Fig. 1C and Table 1). IL-6 increases the level of signal transducer and activator of transcription 1 (STAT1) and strengthens metastasis. They also concluded the connection between long non-coding RNA (lncRNA) zinc finger E-box binding homeobox 2 antisense RNA1 (ZEB2-AS1) and STAT1 in lung cancer metastasis by chromatin immunoprecipitation assay. In brief, IL-6 activates STAT1/ZEB2-AS1 to affect metastasis

Acidities, hypoxia, and inflammation are closely linked and interactional. Hypoxia and inflammatory mediators cause glycolysis pathways to occur and enhance the content of lactic acid in TME. Lactic acid can inhibit inflammation through a variety of mechanisms and play an immunosuppressive role [30]. They form a complex network through various mechanisms to regulate the progression of lung cancer metastasis.

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