

Unraveling the enigma: Fibroblast activation protein - A Key player in tumor microenvironment.

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Introduction

The field of cancer research has seen immense advancements in recent years, leading to the discovery of novel targets for therapies. One such fascinating protein that has garnered attention is the Fibroblast Activation Protein (FAP). Initially known for its role in tissue remodeling and wound healing, FAP's presence in the tumor microenvironment has emerged as a potential therapeutic target. This article aims to shed light on the structure, function, and significance of Fibroblast Activation Protein in cancer biology and its potential implications for targeted cancer therapies [1].

FAP, also known as Sepase or FAP-alpha, is a cell surface-bound serine protease belonging to the prolyl peptidase family. It is primarily expressed on the surface of activated fibroblasts, which are essential components of the stroma or supportive tissue surrounding cancer cells. While FAP is absent or minimally expressed in healthy adult tissues, its expression is upregulated in various cancers, including breast, pancreatic, colorectal, and lung cancers. In normal physiological conditions, FAP plays a crucial role in tissue repair, embryogenesis, and wound healing. It is responsible for the degradation of extracellular matrix components like collagen and fibronectin, facilitating tissue remodeling and wound contraction. However, in the tumor microenvironment, FAP's overexpression leads to profound changes that support tumor growth, progression, and metastasis. FAP-expressing fibroblasts in the tumor stroma promote extensive remodeling of the extracellular matrix, which creates a favorable environment for cancer cell invasion and migration. Immune Evasion: FAP can suppress the immune response by degrading cytokines and chemokines that recruit immune cells to the tumor site, leading to reduced antitumor immunity [2].

FAP promotes the formation of new blood vessels (angiogenesis) by processing factors that stimulate blood vessel growth, facilitating the supply of nutrients and oxygen to the growing tumor. FAP's enzymatic activity contributes to the activation of growth factors and cytokines that drive cancer

cell proliferation, invasion, and metastasis. The discovery of FAP's aberrant expression and prominent role in the tumor microenvironment has sparked interest in its potential as a therapeutic target [3].

Monoclonal antibodies and adoptive T-cell therapies directed against FAP have shown promise in preclinical studies. These therapies aim to selectively target FAP-expressing cells and modulate the tumor microenvironment. Researchers are developing drug conjugates that specifically deliver cytotoxic agents to FAP-expressing cells, thereby increasing the specificity and efficacy of cancer treatments. Efforts are underway to identify small molecule inhibitors that can selectively block FAP's enzymatic activity, hindering its pro-tumorigenic functions [4,5]

Conclusion

Fibroblast Activation Protein has emerged as a compelling target in cancer research due to its selective expression in the tumor microenvironment. Understanding the biology of FAP and its interactions with cancer cells and the immune system offers new opportunities to design innovative and effective therapies against cancer. As research progresses, FAP-targeted approaches may become an integral part of precision medicine, leading to improved outcomes for patients battling cancer.

References

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