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Chemokine Receptor Antagonist for Cancer Treatment

Stanford researchers have found that a chemokine receptor antagonist can reduce immunosuppression in the tumor microenvironment and thereby delay tumor progression.

Immunotherapy is a cancer treatment approach that modulates the patient's immune system to target and eliminate tumor cells. While it has greatly improved the outlook for patients with liquid tumor cancer, its effectiveness against solid tumors, which account for most cancers, has been relatively low. This is because solid tumor cells release chemokines to their microenvironment to attract certain cells that exert immunosuppressive effects. Accumulation of these cells leads to immunosuppression, tumor cell proliferation, angiogenesis, and metastasis – all of which could interfere with cancer treatment. Therefore, removing these cells from the microenvironment could help improve patient prognosis.

The Stanford researchers discovered an antagonist that acts on chemokine receptor found on these immunosuppressing cells and showed that such antagonist can delay tumor progression by interfering with recruitment of these immunosuppressing cells. Administration of the antagonist could be a promising treatment option for cancer patients, especially those with immunotherapy-resistant solid tumor, either as a stand-alone therapy or in conjunction with other neoadjuvant therapies. It has shown synergistic effects when combined with immunotherapy and chemotherapy.

Stage of Development

The inventors are conducting *in vivo* experiments to study the therapeutic efficacy in mice with different cancer types.

Applications

- Cancer treatment
    - Stand-alone treatment to delay tumor progression
    - In combination with other neoadjuvant therapies (ex. Immunotherapy, chemotherapy)
- Inflammatory and autoimmune disease treatment
    - Peritonitis
    - Multiple sclerosis
    - Rheumatoid arthritis
    - Autoimmune hepatitis

Advantages

- Effective therapy for several kinds of tumors that have insignificant response to immunotherapy.
- Specific to certain type of cells, not interfering with effector neutrophils and monocytes.
- Synergistic effects with the existing neoadjuvant therapies like immunotherapy and chemotherapy.

Publications

- Banuelos A, Zhang A, Berouti H, Baez M, Y?lmaz L, Georgeos N, Marjon KD, Miyanishi M, Weissman IL (2024). CXCR2 inhibition in G-MDSCs enhances CD47 blockade for melanoma tumor cell clearance. Proc Natl Acad Sci U S A. 2024 Jan 30;121(5):e2318534121.

Patents

- Published Application: WO2024010842

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