IL-15 and STAT3 Inhibition for Bone and Soft Tissue Sarcoma Cancer Therapy

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ABSTRACT

Researchers at the University of California, Davis have developed a combinatorial cancer treatment that enhances IL-15 mediated immune activation while inhibiting STAT3 to suppress myeloid-derived immune suppression in bone and soft tissue sarcomas.

FULL DESCRIPTION

This technology involves a novel therapeutic approach combining IL-15 pathway activation with STAT3 inhibition to treat bone and soft tissue sarcomas. IL-15 typically stimulates cytotoxic lymphocytes to promote tumor killing; however, it paradoxically activates STAT3 signaling in myeloid-derived suppressor cells (MDSCs), enhancing their immunosuppressive functions and limiting IL-15's anticancer efficacy. By selectively inhibiting STAT3, this method uncouples IL-15's beneficial immune effects from its undesirable pro-tumor effects, restoring robust antitumor immunity. The approach employs IL-15 activating agents such as recombinant proteins or superagonists alongside STAT3 inhibitors including small molecules or oligonucleotides, optionally combined with NF-κB inhibitors to further suppress MDSCs.

APPLICATIONS

- Therapeutic treatment of bone sarcomas and soft tissue sarcomas in humans.
- Development of novel immunotherapy drugs combining IL-15 agonists and STAT3 inhibitors.
- Adjunct therapy alongside chemotherapy, surgery, or other cancer treatments.
- Veterinary cancer immunotherapy applications, especially for canine osteosarcoma.
- Pharmaceutical development of combination biologics and targeted small molecule inhibitors for immuno-oncology.

FEATURES/BENEFITS

- Activates NK cells and CD8+ T cells to strengthen antitumor immunity via IL-15.
- Inhibits STAT3 signaling in MDSCs to reduce IL-15–associated immunosuppression.
- Improves treatment responses in bone and soft tissue sarcomas by limiting immune escape in the tumor microenvironment.
- Enables flexible development and delivery by supporting multiple IL-15 agonist types and STAT3-inhibitor formats (e.g., small molecules, peptides, siRNAs).
- Supports combination regimens by pairing with NF-κB inhibitors to further suppress MDSC accumulation and function.
- Prevents IL-15 from driving pro-tumor MDSC activation, thereby avoiding paradoxical pro-tumor effects. Overcomes STAT3-mediated myeloid suppression to reduce immunotherapy

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OTHER INFORMATION

KEYWORDS

arginase-1, cancer immunotherapy, cd8+ t cells, il-15 receptor alpha, immunosuppression, myeloid-derived suppressor cells, natural killer cells, stat3 inhibition, tumor microenvironment

CATEGORIZED AS

- **Medical**
  - Disease: Cancer
  - Therapeutics

RELATED CASES

resistance.

▶ Targets tumor-promoting immune cells to relieve suppression without impairing cytotoxic lymphocytes.

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PATENT STATUS

Patent Pending

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