INNOVATION PARTNERSHIPS
UNIVERSITY OF MICHIGAN

Novel Cancer Immunotherapy targeting CD6 and/or CD6 Ligands

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OVERVIEW

A novel monoclonal antibody that affects the binding of CD318 to CD6 to enhance cancer cell death

- An approach that is similar to, but distinct from, existing checkpoint inhibitor treatments
- Suppresses autoimmunity and may therefore serve as a safer anti-cancer therapy

BACKGROUND

CD6 is a transmembrane glycoprotein that is almost exclusively expressed by lymphocytes, including most mature T cells and about half of natural killer cells. CD6 functions to promote T-cell activation and proliferation, and it is also involved in the pathogenesis of autoimmune diseases including multiple sclerosis, inflammatory bowel disease, psoriasis, and rheumatoid arthritis. CD318 is a ligand of CD6, and their interactions correlate with autoimmune diseases that affect the central nervous system and synovial lining of joints. Additionally, CD318 is known to be expressed by a variety of tissue cell types including many cancers. The expression of CD318 on cancer cells is distinct from other pathways which have been manipulated to derive checkpoint inhibitor therapies, a group of treatments that have revolutionized cancer immune therapy. While cancers respond with varying efficacy to checkpoint inhibition, many patients suffer autoimmune-related adverse events from these treatments. As such, additional targets are needed on cancer cells and lymphocytes that enhance immune cell elimination of tumors without engendering autoimmune toxicities through induction of lymphocyte self-reactivity.

INNOVATION

Researchers at the University of Michigan have described the use of a monoclonal antibody named UMCD6 that blocks the binding of CD318 to CD6 and enhances killing of cancer cells by lymphocytes. The scientists discovered that the use of antibody UMCD6 to CD318 augments the death of cell lines derived from breast, lung, and prostate cancers through direct effects on both CD8+ T-cells and natural killer (NK) cells. The cancer cell death fostered by this approach created a more robust effect than those from monoclonal antibody checkpoint inhibitors. UMCD6 also augmented in vivo killing by human peripheral blood lymphocytes of a human breast cancer line xenotransplanted into immunodeficient mice. This approach is analogous to the mechanism of existing cancer cell checkpoint inhibitor treatments, though these CD6/CD6 interactions potently suppress autoimmunity in several experimental systems and may therefore offer the potential for cancer treatments with a more favorable side effect profile. The safety of anti-CD6 monoclonal antibodies has been confirmed through their use in psoriasis treatments. Therefore, a CD6/CD6 ligand-directed approach to cancer treatment may be used

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independently or in combination with other checkpoint inhibitors to prevent or reverse autoimmune effects of those agents.