Fred Hutch
Cancer Center

Designing universal donor B cells that will respond to antigen independently of T cell boost

Business Opportunity

Exclusive license
Non-exclusive license
Sponsored research

Technology Type

Hybridoma/Antibody
Immuno Oncology
Therapeutic
Vaccine

State of Development

Preclinical in vitro

Patent Information

WO/2022/170271 A2, patent pending

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Brief Description

Methods to link CD40 signaling to antigen binding independently of CD40L binding in order to potentiate B-cell responses.

Technology Overview

Vaccines increase immunity against infections by stimulating B cells to produce antibodies against the infectious agent. In addition to infections, antibodies are also useful as treatment for different autoimmune conditions, and cancer. To mount a potent B cell response, interaction between CD40 (expressed on the surface of B cells) and CD40 ligand (CD40L; expressed on the surface of CD4+ T cells) is typically required for expansion, survival and differentiation of the B cells. B cell dependence on T cell interaction via MHC presentation also limits development of universal donor B cells for broader therapeutic applicability. To overcome this, Fred Hutchinson Cancer Center researchers have developed a method to genetically modify B cells to express fusion proteins that link extracellular antigen binding domains to CD40 signaling domain. This allows CD40 signaling activation upon antigen binding, independently of interaction with CD40L on T cells. Two approaches to link CD40 signaling to antigen binding have been developed: (a) Linking CD40 signaling to B cell receptor (BCR) signaling and (b) Linking CD40 signaling domain to engineered extracellular antigen binding domains

Applications

- Enhancing responses of B cells engineered to combat common respiratory viruses (e.g., RSV, HMPV, influenza, etc.)
- Enhancing responses of B cells engineered to combat other viral (e.g., HIV, CMV, EBV, hepatitis C, herpes simplex, etc.) or bacterial (e.g., Bordetella, clostridium difficile, etc.) infections
- Potentiating therapeutic response of B cells producing antibodies for autoimmune diseases and cancer

Advantages

- Improves response activity of B cells independent of CD40L interaction and thus leads to a greater response to a vaccine or treatment
- Obviates the need for MHC II interaction between T cell and B cell allowing for ‘universal’ donor B cell therapeutics