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METHODS AND COMPOSITIONS FOR PREVENTING ANTI-GAL PRODUCTION IN XENOGRAFT RECIPIENTS

Technology Reference
R046

Keywords
Research Tool

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Field
Microbiology and Immunology

Patent Status
[US Patent 6,613,330](#)

Cross Reference
This technology is related to discovery R044

A shortage of allografts has prompted researchers to search for an alternative source of organs and tissue from other mammalian species. This invention provides a toxin complex and methods for preventing immune rejection of xenografts.

AREAS OF APPLICATION

- Advance studies in using organ and tissue xenotransplantation
- Animal model for immune suppression to treat autoimmune diseases

ADVANTAGES

- Cytotoxic agent can be any agent, that when internalized by the cell, causes cell death
- Carrier can be any macromolecule capable of being modified to display the ligand and be linked to the cytotoxin
- Provides a method for the destruction of a selected population of target cells by administering an amount of the toxin complex
- Provides a method for the inhibition of immune mediated xenograft rejection in an animal or human subject
- Provides a method for the inhibition of autoimmune disease in an animal or human subject

THE TECHNOLOGY

Cell toxins are coupled to carriers that display ligands which allow the complex to be recognized by antigen binding sites (i.e., specific B cell and T cell receptors) on the lymphocytes as the target cells responsible for the specific immune response. The toxin complexes interact with the specific receptors (antigen binding sites) on lymphocytes, and are internalized by these target cells. Once internalized, the toxin complex dissociates to release the cell toxin, which destroys the target cell. In one embodiment, the target cells are B lymphocytes and T lymphocytes, the antigen binding sites are anti-Gal B cell receptors and T cell receptors which interact with α -gal epitopes displayed on an α 1-acid glycoprotein carrier. In other embodiments, the toxin complex is directed by the corresponding ligands to target cells responsible for autoimmune diseases in which the specificity of the autoantibodies is defined.

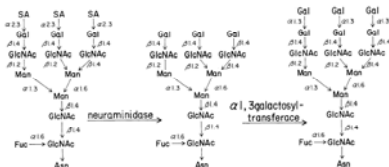


FIG. 1

Rush Medical Center University
www.rush.edu/research
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