

Drug Resistant Immunotherapy for the Treatment of Cancer

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Technology description

Emory University is looking for companies interested in commercializing a new therapy for treating cancer which can be applied in concert with traditional chemotherapies. This combination approach could be used to successfully and rapidly treat a broad range of tumors, especially those that typically confer a poor prognosis upon a patient.

Technical Summary

Chemotherapy agents are among the most used and successful cancer treatments but, are not uniformly effective, and can cause unwanted and harmful side effects. Many of these effects stem from the drug's inability to differentiate the body's own rapidly proliferating cells from tumor cells. For example, chemotherapy agents are highly toxic to lymphocytes (immune cells), the very cells that could aid in finding and destroying tumor cells. Emory University researchers recently have discovered a method of protecting lymphocytes from the cytotoxic effects of chemotherapeutic agents. Because the molecular mechanisms of drug-induced toxicities are understood for most cancer therapeutics, it is feasible to genetically engineer selected cells to withstand these cytotoxic drug treatments using gene therapy techniques. These researchers have successfully generated lymphocytes that are resistant to a number of chemotoxic therapeutics, and when transplanted in animal models facilitate anti-tumor responses in combination with chemotherapeutics. Proof of principle has been demonstrated in leukemia cell lines, and mouse sarcoma models, with mice treated displaying a rapid and prolonged reduction in tumor size compared to either treatment alone.

Application area

A novel method of genetic engineering that increases effectiveness of current chemotherapeutic- and immune-based cancer therapies.

Advantages

Ability to engineer chemotherapeutic-resistant immune cells.

Ability to overcome cytotoxic side effects of chemotherapeutics.

Combined application of chemotoxic and immune treatments significantly enhances tumor cell death.

Institution

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