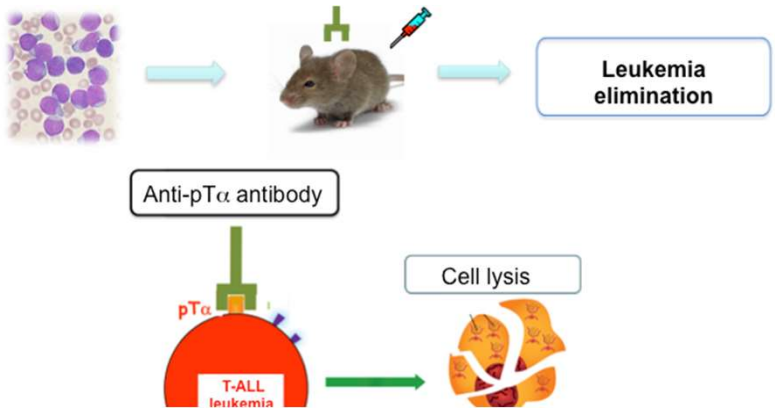
Technology Offer CSIC/EG/116

Monoclonal antibody for treatment of T-cell acute lymphoblastic leukemia (T-ALL)



A new immunotherapy strategy based on the administration of a monoclonal antibody specific for pre-TCR has been developed and validated in a preclinical human T-ALL xenotransplantation model in mice.

Intellectual Property

Patent filed in Europe and United States of America

Stage of development

Preclinical in vivo

Intended Collaboration

Licensing and/or co-development

Contact

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Market need

T-cell acute lymphoblastic leukemia (T-ALL) is an aggressive tumor, mainly pediatric, that appears due to the oncogenic transformation of T-lymphoid progenitors during their development in the thymus. Although intensive chemotherapy treatments have notably increased the life expectancy of patients in recent years, the frequency of refractory cases is high, and these patients have a poor prognosis, which demands the availability of new therapies directed against the cells Leukemia Initiating Cells (LIC) responsible for relapses.

CSIC solution

Our researchers have shown that the pre-TCR receptor is expressed on the surface of leukemic cells during all stages of disease progression in a model of human T-ALL generation in mice, as well as in LIC cells from T-ALL patients. It is also demonstrated that pre-TCR is a therapeutic target for the identification, screening or design of compounds, molecules, drugs, etc., useful for the diagnosis, treatment and / or prevention of T-ALL leukemia. The efficacy of a proprietary anti-pre-TCR monoclonal antibody for the treatment of this disease has been validated in a preclinical xenotransplantation model of human T-ALL.

Competitive advantages

- It can be used in the diagnosis, treatment and / or prevention of relapses of leukemia, preferably relapses of T-cell acute lymphoblastic leukemia (T-ALL), more preferably relapses of pre-TCR + T-ALL.
- Validated in a preclinical xenotransplantation model of human T-ALL.
- Validated in primary T-ALL cells from patients.