

HUMAN IMMUNOSTIMULATORY T CELLS

Tech ID: 19026 / UC Case 2008-132-0

BRIEF DESCRIPTION

Research into modulating immune function through immunostimulatory T cells has been hampered by the lack of identification of the molecular markers on such cells. UCSF investigators have identified a novel endogenous human T cell population that can significantly enhance the proliferative capacity of a T cell response. In contrast to T cells that can be induced to suppress a proliferative response, these are a naturally occurring, functionally mature T-cell subpopulation that induce the proliferation of a T cell.

FULL DESCRIPTION

BACKGROUND: Regulation of immune responses has the potential to be an important component of the treatment for many conditions. Because specific molecular markers have been identified for suppressor T cells (commonly referred to as T regulatory cells), most research has been focused on modulating immune response by affecting these cells. An alternative, and/or complementary, approach would be to modulate the function of the endogenous T cell subpopulations that enhance, rather than suppress, the proliferative capacity of a T cell response. This approach would have direct applications in controlling and in the diagnosis of infections, immunodeficiencies, autoimmune conditions, cancer, transplantation and vaccine strategies.

DESCRIPTION AND ADVANTAGES: Research into modulating immune function through immunostimulatory T cells has been hampered by the lack of identification of the molecular markers on such cells. UCSF investigators have identified a novel endogenous human T cell population that can significantly enhance the proliferative capacity of a T cell response. In contrast to T cells that can be induced to suppress a proliferative response, these are a naturally occurring, functionally mature T-cell subpopulation that induce the proliferation of a T cell. These immunostimulatory T cells natural presence in the immune system as a phenotypically distinct population makes them a good target for designing ways to treat or prevent immunological diseases, and to control pathological and physiological immune responses. This invention presents a previously unappreciated opportunity for intervention, constituting a novel approach to the prevention and treatment of many diseases, and could be used in conjunction with other stimulatory agents to further enhance and sustain immunomodulation strategies. Further, this invention also provides a previously unrecognized role of immunostimulatory cells as a powerful tool for the manipulation of the T cell response

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OTHER INFORMATION

CATEGORIZED AS

- ▶ **Medical**
- ▶ Diagnostics
- ▶ Disease: Cancer
- ▶ Stem Cell
- ▶ Therapeutics
- ▶ Vaccines

RELATED CASES

2008-132-0

with the capacity to restore defective proliferative capacity and function of immune cells. The UCSF researchers are currently studying the role of these immunostimulatory T cells in chronic infectious diseases and autoimmune disorders in comparison to healthy individuals as a proof of concept that intervention using the immunostimulatory T cells is a viable approach for the diagnosis and treatment of immune-related diseases.

APPLICATIONS

Potential applications of in vitro/ex vivo expanded immunostimulatory T cells include:

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- ▶ in prognostic and diagnostic monitoring of immune responses in disease states and post-transplantation
- ▶ as an adjuvant to enhance or restore the defective functionality of immune responses, including enhancing immune responses to therapeutic and prophylactic vaccinations
- ▶ as research reagents for further studying immunostimulatory T cells for enhancing or modulating T cell proliferation in vitro and in vivo (eg. generation of T cell lines and T cell clones)

RELATED MATERIALS

- ▶ [Ndhlovu, L. C., Leal, F. E., Eccles-James, I. G., Jha, A. R., Lanteri, M., Norris, P. J., Barbour, J. D., Wachter, D. J., Andersson, J., Tasken, K., Torheim, E. A., Aandahl, E. M., Kallas, E. G. and Nixon, D. F. A novel human CD4+ T-cell inducer subset with potent immunostimulatory properties. Eur. J. Immunol., 2010, 40: 134~141.](#)

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