

Technology Offer

GPCR83 as a molecular switch for the induction of regulatory T-cells

Reference Number: TO 02-00208



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Life-Science Foundation
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Challenge

G-protein coupled receptors (GPCR) play a central role in signal transduction and as such they are meanwhile validated targets for drug development. GPCRs are associated with a wide range of therapeutic categories including cancer and autoimmune diseases. A GPCR of particular interest is GPCR83 that is upregulated in regulatory T-cells (Treg) indicating that this receptor is involved in immune response. Tregs are a specialized subpopulation of T-



Dendritic cell with T-cells (read)
source: HZI/ Manfred Rohde

cells critical for maintaining the immune system homeostasis and tolerance to self by actively suppressing the activation of the immune system. Thus Tregs have a therapeutic potential for instance to treat autoimmune diseases or to facilitate transplantation tolerance. Therefore, the induction or expansion of Tregs for the treatment of undesired immunoreactions is a central aspect in this therapeutic field. Due to various disadvantages of *in vitro* induction of Tregs there is a need for compounds which could effectively be used for the *in vivo* Treg induction whereby knowledge relative to the specific antigens would not be required.

Technology

The invention relates to methods for identifying compounds interacting with GPCR83, and compounds acting as immunomodulators. GPCR83 plays a crucial role in the generation of Tregs, and activation or inactivation of GPCR83 is an essential step for the induction or suppression of the development of Tregs. By using GPCR83 as a target an undesired generation of Tregs could be avoided. Thus the invention provides methods of treatment of humans suffering from undesired immunoreactions.

Commercial Opportunity

In-licensing of IP for identification and development of a drug for the treatment of diseases due to or accompanied by undesired immunoreactions. Cooperation for further development is also possible.

Patent Situation

A US patent application filed in 2007 is pending. An international application will follow.

Further Reading

GPR83-overexpression in naïve CD4+CD25- T cells leads to the induction of Foxp3+ regulatory T cells in vivo. W. Hansen et al. (2006). Journal of Immunology, Vol. 177, pp. 209-215.

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