Animal Model

CCR7 -/- mice: a model for studies on early immune reponses towards alloantigens

Reference Number 03-00244

Abstract

Challenge

Genetically modified animals are essential research tools in modern molecular biology. The chemokine receptor 7 (CCR7) is described as an important regulator of lymphocyte trafficking to and within secondary lymphoid organs, and plays a significant role in organization of the primary immune response, like e.g. in the development of transplant rejection.

Technology

The technology provides mice deficient in CCR7. The complex phenotype of these knock out mice is reflected by the lack of fast primary B and T cell responses. Following application of T-cell-dependent antigens, CCR7 mutant mice fail to produce specific antibodies of any IgG isotype within the first 10 days, indicating the control of a productive allospecific primary immune response by CCR7. Furthermore, in CCR7-/- mice a profound reduction in the cytotoxic T cell (CTL) response towards alloantigens can be observed, providing evidence for a defective T cell priming. The CCR7 deficiency prevents the appropriate presentation of allogeneic peptides in draining lymph nodes and results in a marked loss of allogeneic T cell priming. Overall CCR7-/- mice offer a valuable tool for studies on early immune responses and thus provide insight into diseases and clinical syndromes like e.g. allograft acceptance and rejection.

Commercial Opportunity

Breeding pairs are available under a Tangible Property License Agreement.

Patent Situation

No patent application was filed.

Further Reading

- Forster et al., 1999, Cell, 99(1):23-33
- Höpken et al., 2004, Eur. J. Immunol., 34:461-470

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