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Harnessing immune regulation for treatment of human diseases : CD4+CD25+ regulatory T cells & antibody glycosylation

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PROPOSITIONS

Harnessing Immune Regulation for Treatment of Human Diseases

CD4⁺CD25⁺ regulatory T cells & antibody glycosylation

1. FOXP3 is transiently expressed in human activated non-regulatory T cells; therefore, expression of endogenous FOXP3, in humans, is not sufficient to induce regulatory T cell activity or to identify Treg cells (this thesis).
2. The absence of cell surface TNF α expression can be used to identify and isolate a subset of CD4⁺CD25⁺⁺ T cells with stronger *in vitro* suppressive capability (this thesis).
3. The combination of TGF- β and all-*trans* retinoic acid not only consistently converts human naive T cells into Treg cells, but also enhances the suppressive ability of isolated CD4⁺CD25⁺⁺ T cells during *in vitro* expansion (this thesis).
4. IL-4 signaling plays a dominant role in preventing TGF- β -induced Treg conversion both in mice and humans (this thesis).
5. Microenvironmental factors present during the activation and differentiation of B cells modulate the Fc-linked glycosylation of secreted IgG1 (this thesis).
6. Next to the regulation of the amount and type of antibodies, modulation of the carbohydrate structures of IgG may represent an additional and independent checkpoint to control the strength of humoral immune responses (this thesis).
7. Treg lineage may be determined by a higher-order regulatory process that ensures stable and high levels of Foxp3 expression (Hori S, *Eur. J. Immunol.* 2008; 38: 928-930).
8. Regulatory T-cell-based immunotherapy should not be envisaged as an all-or-nothing approach to re-establish immunological tolerance on its own (Roncarolo MG et al, *Nat. Rev. Immunol.* 2007; 7: 585-598).
9. Foxp3 expression is dynamically regulated in normal physiological conditions, and its instability can lead to the generation of pathogenic effector-memory T cells that promote autoimmunity (Zhou X et al, *Nat. Immunol.* 2009; 9: 1000-1007).
10. The distribution of the glycoform populations is well controlled and, in a given state, is not random but reproducible; therefore, disease-associated alternations provide diagnostic biomarkers and contribute to disease pathogenesis (Arnold JN et al, *Annu. Rev. Immunol.* 2007; 25: 21-50).
11. Rejection of your manuscript by a journal does not necessarily mean that your findings are not important.