

ImmunoTools IT-Box-Cy55M-Award 2013



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Role of Notch receptor signaling in CD4⁺ T helper 17 cell differentiation

CD4⁺ T helper cells (T_H) are involved in host defense to pathogens and in the maintenance of immune homeostasis. Upon antigen-specific recognition, naive CD4⁺ T cells receive signals to differentiate into several functionally distinct subsets of CD4⁺ T_H cells characterized by the secretion of a specific pattern of effector cytokines. Inside these different effector CD4⁺ T helper subset, T_H17 cells have been described on the basis of their secretion of specific cytokines (IL-17A, IL-17F) and their dependence of specific transcription factors (RORγt, RORα). Among the different factors contributing to the differentiation of naive CD4⁺ T cells towards a given T_H cell subset, Notch receptor signaling has been reported to influence either the differentiation and/or the function of CD4⁺ T_H cells. However, the mechanisms involved in this control are not defined. To investigate the mechanisms of Notch signaling in the control of T_H17 cell function, mice carrying a T cell specific deletion of both Notch1 and Notch2 (N1N2^{ΔCD4⁺Cre}) were infected with different infection models that preferentially induce a T_H17 cell response *in vivo*. For this project, we are currently defining an *in vitro* T_H17 cell differentiation model that is highly dependent on the presence of different murine cytokines. The important plasticity inside the T_H17 cell subset was reported to be correlated to the cytokine environment and to be critical in inflammation process. We want therefore investigate the involvement of Notch receptor in either the T_H17 cell differentiation and also in the T_H17 cell plasticity *in vitro* by using the different variety of cytokines that are present in IT-Box-Cy55M from **ImmunoTools**.

ImmunoTools IT-Box-Cy55M for **Manuel Coutaz**

includes 55 recombinant mouse cytokines

rm EGF, rm Eotaxin / CCL11, rm FGF-a / FGF-1, rm FGF-b / FGF-2, rm FGF-8, rm Flt3L / CD135, rm G-CSF, rm GM-CSF, rm GRO-a / CXCL1, rm GRO-b / CXCL2, rm IFNγ, rm IL-1α, rm IL-1β, rm IL-2, rm IL-3, rm IL-4, rm IL-5, rm IL-6, rm IL-7, rm IL-9, rm IL-10, rm IL-11, rm IL-13, rm IL-15, rm IL-16, rm IL-17A, rm IL-17C, rm IL-17F, rm IL-19, rm IL-20, rm IL-21, rm IL-22, rm IL-25 / IL-17E, rm IL-27, rm IL-31, rm IL-33, rm IP-10 / CXCL10, rm LIF, rm MCP1 / CCL2, rm M-CSF, rm MIP-1α / CCL3, rm MIP-1β / CCL4, rm MIP3α / CCL20, rm MIP3β / CCL19, rm NGF-beta, rm PDGF-AA, rm PDGF-BB, rm RANTES / CCL5, rm sCD40L / CD154, rm SCF, rm SDF-1α / CXCL12a, rm SDF-1β / CXCL12b, rm TNFα, rm TPO, rm VEGF

[DETAILS](#)