BOOK OF ABSTRACTS

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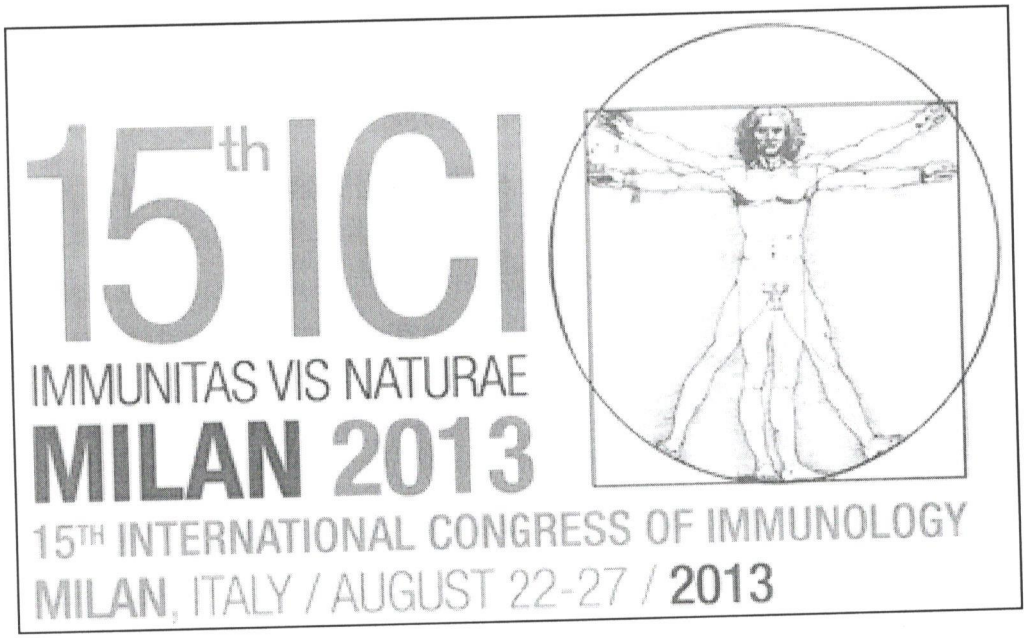

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Galectin-3 (Gal3), a β-galactoside-binding lectin, has been reported to regulate various immune functions. Furthermore, reports indicate that Gal3 expression is greatly induced by HIV-1 infection in T cells. However, the role of Gal3 in HIV-1 infection remains unclear. Lipid raft integrity plays a critical role in virological synapse formation during HIV infection, and previously we have demonstrated that endogenous Gal3 is accumulated in lipid rafts of dendritic cell surface membrane. Therefore, we hypothesized that galectin-3 may play a role in viral cell-to-cell transmission during virological synapse formation. Here, we show that Gal-3 was co-localized with Gag and Env in lipid raft at cell-to-cell junction of HIV-1-infected Magi5 (HeLa cells expressing CD4, CXCR4 and CCR5) and Hut78 $CD4^+$ T cells. In addition, results from conventional cell-to-cell transmission assay indicate that HIV-1 transmission efficacy is significantly attenuated in Gal3 knockdown Magi5 and Hut78 T cells ($p<0.05$). On the other hand, HIV-1 transmission efficacy is significantly increased by overexpression of Gal3 in Jurkat T cells ($p<0.05$). Moreover, Gal3-promoted HIV-1 cell-to-cell transmission efficacy is positively correlated with the expression level of Gal3 in the effector cells ($p<0.05$). Time-lapse confocal microscope indicates colocalization of Gal3-EGFP and HIV Gag-iCherry at virological synapses between effector and target cells. In addition, both Gal3-EGFP and Gag-iCherry were cotransmitted from the effector cells to the target cells via virological synapses. These results were confirmed by using human primary $CD4^+$ T cells. Altogether, our studies demonstrated an active role of Gal3 in HIV-1 cell-to-cell transmission and revealed a potential target for HIV-1 therapy.

W1.12.06
Galectin 3 affects DCs:NKT cell interaction in the development of αGalCer-induced hepatitis

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We used α-galactosylceramide (αGalCer) induced liver injury, well established murine models of NKT cell mediated hepatitis, to study the role of Galectin 3 (Gal-3) in acute liver injury.
We tested susceptibility to αGalCer-induced hepatitis in galectin-3-deficient (Gal-3KO) mice and wild-type (WT) C57BL/6 mice.
One microgram/mouse of αGalCer i.v. significantly enhanced expression of Gal-3 on dendritic cells (DCs) and NKT cells of WT mice. Gal-3KO mice were less sensitive to αGalCer-induced hepatitis as evaluated by liver enzyme test, histology, cytokine production and intracellular staining of immune cells in the liver. The level of IL-10 in the sera and percentage of IL-10-producing NKT cells were significantly higher in αGalCer-treated Gal-3KO mice. Percentage of liver infiltrating CD11c (+) DCs, CXCR3(+) DCs, CD1d(+) DCs and TNFα-, IFNγ-, and IL-12-producing DCs was significantly lower in αGalCer-treated Gal-3KO mice. In vitro, αGalCer-loaded DCs, isolated from livers of untreated Gal-3KO mice, produced significantly higher amounts of IL-10 and significantly lower amounts of IFNγ compared to DCs from WT mice confirming that. αGalCer-induced production of IL-10 and IFNγ by DCs is Gal-3 dependent. Liver DCs (5x105/mouse) of untreated WT mice, but not from Gal-3KO mice, transferred in Gal-3KO recipient significantly enhanced αGalCer-induced hepatitis. Pretreatment of WT mice with a selective inhibitor of Gal-3 led to the attenuation of αGalCer-induced liver injury and provided similar effect as Gal-3 deletion.
Gal-3 plays an important pro-inflammatory role in αGalCer-induced hepatitis by affecting DCs:NKT cell interaction in the liver.
Supported by grants 175069, 175103 from the Serbian Ministry of Education and Science.

W1.13 Fc and Fc-like receptors

IL1.13.01
Inside-out and outside-in Fc receptors: impact on antibody therapy of cancer

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Fc receptors are an important bridge between the cellular and humoral branches of the immune system. Residing on monocytes, granulocytes, platelets, B lymphocytes, NK cells and dendritic cells, triggering of Fc receptors by complexed antibodies can lead to e.g. phagocytosis, respiratory burst, cytokine production and antigen presentation. Over the past decades, Fc receptors got more attention because of their important role in antibody therapy. Immunotherapy using monoclonal antibodies (mAb) has emerged as an excellent therapeutic strategy for cancer, immune deficiencies, immune regulation and autoimmunity. The first FDA-approved antibody for human neoplasia was Rituximab, targeting B-cell CD20 for treatment of non-Hodgkin's lymphoma and immune diseases.
An important mechanism of action of rituximab is antibody-dependent cellular cytotoxicity (ADCC), depending on Fc receptor bearing effector cells, such as NK cells and macrophages. In vitro also complement dependent cytotoxicity (CDC) and apoptosis induction are described as important mechanisms. The Immunotherapy group investigates the importance of these distinct effectormechanisms in vivo, including the possibility of apoptosis induction by crosslinking of Fc-receptors. For the latter purpose an transgenic mouse was developed, with normal expression of Fc receptors, but without the signaling of these receptors.(de Haij et al, Cancer Research 2010). Also the effectiveness of IgA as a novel isotype in antibody therapy is being explored in vivo (EMBO MM, in press). Knowledge of the biology of both IgG and IgA receptors is essential for these investigations.

IL1.13.02
Fc gamma Receptors: a 50 year anniversary

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Fcgamma R are an heterogeneous family of molecules of the Ig superfamily with effector and immunoregulatory functions. The Fcgamma RI bind IgG with high affinity whereas FcgRII, FcgRIII and FcgRIV (in the mouse) bind IgG immune complexes. They comprise several activating receptors characterized by an Immunoreceptor Tyrosine Activation Motif (ITAM) in their intracytoplasmic region or in their associated chains, and a unique single chain inhibitory receptor, FcgRIIB, with an Immunoreceptor Tyrosine Inhibition Motif (ITIM) in the intracytoplasmic domain.
Fcgamma R are expressed by most if not all cells of the immune system. The innate cell subpopulations co-express activating and inhibitory Fcgamma R, whereas B cells selectively express inhibitory FcgRIIB and NK cells mostly FcgRIII. Expression of FcgRII can be found on T lymphocytes as well. The ectopic expression of FcgRIIB can be found on some cancer cells such as in human metastatic melanoma.
I will present the FcgR family members and their functions to introduce the workshop.

W1.13.01
Role of IgG, IgG receptors and neutrophils in anaphylactic reactions

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Allergic reactions are generally considered to be triggered by the activation of mast cells or basophils, when IgE antibodies bound to their receptors (FcεRI) get crosslinked by specific antigens. Challenging this dogma, we could recently demonstrate that a murine model of allergic shock (anaphylaxis) depended primarily on IgG, IgG receptors and the activation of neutrophils (JCI 2011). Anaphylaxis was abolished in mice lacking all activating IgG and IgE receptors (FcRγ-/- mice). Interestingly, transfer of human neutrophils into

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