



FRONTIERS IN IMMUNOLOGY RESEARCH NETWORK

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FRONTIERS IN IMMUNOLOGY RESEARCH 2011 INTERNATIONAL CONFERENCE

< <http://www.firnweb.com/FIRNCall2011.pdf> >

SPLIT, CROATIA (July 1-4, 2011), Le Meridien Lav Hotel

<<http://www.lemeridienlavsplit.com/>>

SUBJECT CATEGORIES (scroll down)

SCHEDULE OF MEETINGS & ACTIVITIES (scroll down)

INSTRUCTIONS TO PROGRAM PARTICIPANTS (scroll down)

PROGRAM & OBSERVERS (scroll down)

SUBJECT CATEGORIES

- | | |
|---|--|
| <ul style="list-style-type: none">• Allergy and immunopathology• Antigen presentation• Autoimmunity• B cells• Cellular immunology• Cytokines, chemokines, inflammation• Dendritic cells• Diabetes• Imaging at the molecular, cellular or whole animal level• Immune responses to infectious diseases• Immunogenetics and gene discovery• Immunotherapy• Innate immunity• In silico modelling• Knockout and knock-in studies• Lymphocyte activation and homing• Lymphocyte differentiation and migration | <ul style="list-style-type: none">• Mechanisms of cell death• Molecular immunology• Mucosal immunology SIG• NK cells• NKT and T regulatory cells• Plasmon resonance• Protein crystallography• Science communication - BD award session• Signal transduction• Stem cells• T cell biology• The immune response to viral infections• The MHC and its interactions• Tolerance• Transplantation• Tumour immunology• Vaccine development• Veterinary immunology SIG |
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Friday, July 1, 2011 [continued]

Friday, July 1, 2011, 1:30 PM – 2:30 PM
Session [3], Room: Lobby by Conference Registration Desk

POSTERS

**Melanoma cell derived exosomes transform macrophage
and dendritic cell functions *in vitro***

Annamaria Marton, Csaba Vizler, Robert Katona, Viktoria Temesfoi,
Erno Duda, Janos Minarovits, Zsuzsa Szathmary, Zsolt Szegletes, Laszlo Siklos,
Erzsebet Kusz, O.M. Zack Howard, **Krisztina Buzas**

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The response of dendritic cells to nanobiomaterials

Miodrag Čolić^{1,2}, Sergej Tomić¹, Tanja Džopalić^{1,2}, Saša Vasilijić¹,
Petar Milosavljević¹ and **Rebeka Rudolf**^{3,4}

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TCR Peptide Therapy of EAE Requires Foxp3⁺ Treg-cells and Inhibits Heterogeneous T-Effector Cells

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Mouse models of experimental autoimmune encephalomyelitis (EAE) that mimic certain aspects of the human disease multiple sclerosis (MS) have been utilized to determine if the therapeutic effects induced by vaccination with T-cell receptor (TCR) peptides requires TCR-specific Foxp3⁺ regulatory T-cells (Treg). T-cell recognition of self TCR sequences represents an effective autoregulatory mechanism for limiting inflammation mediated by T-effector cells. In previous studies using a humanized MBP-specific TCR transgenic mouse strain, administration of TCR CDR3 peptides derived from the pathogenic TCR could effectively prevent and treat EAE. Moreover, TCR peptide treatment resulted in suppressed proliferation and IL-2 production in response to MBP peptide, and induced expansion of Foxp3⁺ Treg cells. These findings are consistent with our previous studies in MS subjects showing that a TCR CDR2 peptide vaccine was able to increase Foxp3⁺ Treg-cell expression levels. The TCR BV8S2⁺ CDR2 peptide successfully treated C57BL/6 mice after EAE onset. However, TCR CDR2 peptide treatment was not effective in EAE mice when the Foxp3⁺ Treg population was depleted. In the CNS, EAE mice treated with TCR CDR2 peptide demonstrated a reduction of T cells producing IL-17 and IFN- γ . TCR CDR2 peptide therapy requires induction of Foxp3⁺ Treg cells and is effective in regulating heterogeneous, encephalitogenic T cell populations in CNS and might be an effective therapy for MS.

Melanoma cell derived exosomes transform macrophage and dendritic cell functions *in vitro*

Annamaria Marton, Csaba Vizler, Robert Katona, Viktoria Temesfoi, Erno Duda, Janos Minarovits, Zsuzsa Szathmary, Zsolt Szegetes, Laszlo Siklos, Erzsebet Kusz, O.M. Zack Howard, **Krisztina Buzas**

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Previous studies showed that tumor cells release microvesicles (exosomes) with a size of 20-100 nm that contain molecular markers characteristic of their plasma membranes. Because exosomes carry surface proteins associated with antigen presentation and apoptosis, they may affect anti-tumoral immune responses. However, the immunomodulatory functions of tumor cell derived exosomes are poorly understood. Both activatory and inhibitory effects were reported depending on the exosome phenotype and tumor stage. In the present study we demonstrate that B16F1 melanoma cell derived exosomes (mcd-exosomes) affect immune cell functions *in vitro*. We found that bone marrow derived dendritic cells exposed to mcd-exosomes enhanced CD4⁺ T cell proliferation in an allogeneic system. In addition we detected that mature macrophages stimulated with mcd-exosomes exhibited an increased level of NF- κ B activity, suggesting that mcd-exosomes activate the immune system. However, exosome-treated macrophages secreted lower amounts of inflammatory cytokines and chemokines thought to mediate anti-cancer immune responses. This study illustrates that soluble and cellular responses to exosomes can lead to opposing immune status. Our findings suggest that the tumor derived exosomes could contribute to tumor escape using different pathways.