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Abstracts

15th International Conference on

Immunology

July 05-07, 2018 Vienna, Austria

EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Yong-Suk Jang et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

C5A RECEPTOR SIGNALLING IN PEYER'S PATCH DENDRITIC CELLS ENHANCES THE ANTIGEN-SPECIFIC CD8+ T CELL RESPONSE IN DENGUE MUCOSAL VACCINE MODEL

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Chemoattractant complement 5a receptor (C5aR) is associated with mucosal immunity. Although C5aR is expressed in several cell types including macrophages, monocytes, and neutrophils, its expression is controversial in mucosal dendritic cell (DC) subsets. In this study, we found that $CD11c^{+}CD11b^{-}CD8^{-}$ Peyer's patch (PP) DCs located in subepithelial dome expressed the C5aR where stimulation of C5aR with its cognate ligand, C5a, or a specific peptide (Co1) effectively induced antigen-specific IFN-γ^{+} Th1 cells through the induction of pro-inflammatory cytokines. Based on the previous observation that Co1 peptide has an M cell-targeting ability and its moiety is homologous to C5aR agonist, EP67, which is capable of inducing $CD8^{+}$ cells, we assumed that oral administration of Co1 peptide-conjugated antigen may induce the antigen-specific Th1 and/or CD8 response through both M cell antigen-targeting and the induction of pro-inflammatory cytokines via C5aR signalling in systemic and mucosa immune compartments. To this end, a model antigen, partial-nonstructural 3 (NS3) protein of dengue virus serotype 2 (DENV-2) was conjugated with Co1 peptide (p-NS3-Co1) and M cell-targeting of the antigen and co-localization with C5aR on M cells were confirmed. As we assumed, oral prime and boost immunization with p-NS3-Co1 effectively induced the NS3-specific IFN-γ^{+} effector $CD8^{+}$ T cells. In addition, challenge with DENV-2 at 4 weeks post immunization with p-NS3-Co1 induced not only the functional restimulation of memory effector $CD8^{+}$ T cells but also proliferation of $CD107a^{+}$ cytolytic effector $CD8^{+}$ T cells in mucosal and systemic compartments. Collectively, we concluded that C5aR plays a role as mucosal immune modulator in PPs and Co1 peptide ligand-mediated C5aR activation contributes to develop the $CD8^{+}$ T cell immune response induction.

Biography

Yong-Suk Jang has completed his PhD from Northwestern University, Chicago, IL, USA in 1993 and Postdoctoral study at Cancer Research Institute of Seoul National University Medical School, Seoul Korea. He is a Professor and Chairman in the Department of Molecular Biology, College of Natural Science, Chonbuk National University, Republic of Korea. He has published more than 150 papers in reputed journals including the *Journal of Immunology, Biomaterials, European Journal of Immunology,* and *Scientific Reports*. He will serve as a President of the Korean Association of Immunologists for 2019. His current research is mainly on Mucosal Immune Regulation and Mucosal Vaccine Development.

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Ying-Chyi Song et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

THE EFFICACY AND MECHANISM OF CHINESE HERBAL MEDICINE ON THE INDUCTION OF SPECIFIC ANTI-TUMOR RESPONSE

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Virus infections may account for the development of several cancers, such as HPV16/18 are known to cause around 70% of cervical cancer cases. Until now, several immunotherapeutic approaches for virus-induced cancer are under development. Peptide-based vaccines have several advantages over conventional whole-protein vaccines in terms of purity, lot-to-lot consistency, production costs, and the high antigenic specificity. However, the use of peptide antigens in vaccine development has been hampered by problems, such as weak immunogenicity coupled with a paucity of potent adjuvants. Specific T cell immune response is crucial for anti-tumor immunity. Manipulating specific T cell immunity by Chinese herbal medicine (CHM) is a promising field to explore. Therefore, we aimed to investigate whether CHM can modulate specific T cell responses to apply for the development of cancer vaccine. We have identified a CHM extract that could increase expression of maturation cytokines and activation markers of murine bone marrow-derived dendritic cells (BM-DCs). Furthermore, in a murine TC-1 tumor-bearing model, we found that the CHM extract could act as an adjuvant to induce cellular immune responses and anti-tumor effect in peptide vaccine strategy. We suggested that immune-stimulator CHM combined with cancer vaccine endows them with increased immunologic activity, which may be used to bypass the requirement for the conditional adjuvant. Further delineation of the mechanism may provide new clues for vaccination strategy.

Biography

Ying-Chyi Song has completed her PhD from National Yang Ming University and Postdoctoral studies from National Health Research Institutes (NHRI) in Taiwan. She is an Assistant Professor in China Medical University, Taiwan. His current research interests are development of cancer vaccines and immune adjuvants. She has published more than 15 papers in reputed journals.

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Ju-Pi Li et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

DYSREGULATED IMMUNE SYSTEM IN MDA5-POSITIVE DERMATOMYOSITIS PATIENTS

Shih-Hsin Chang, Ju-Pi Li and Joung-Liang Lan
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Dermatomyositis, a subgroup of idiopathic inflammatory myopathy, is characterized by inflammation of muscles and skin. In Asian cohorts, the presence of anti-melanoma differentiation-associated gene-5 (MDA5) autoantibody in dermatomyositis patients is often associated with rapidly progressive interstitial lung disease (RP-ILD). Here we examined the immune system of MDA5-positive dermatomyositis patients (n=9) and compared with healthy controls (n=10). The percentages of Th17 and Treg cells in the peripheral bloods from MDA5-positive dermatomyositis patients were higher than those from healthy controls. Furthermore, circulating proinflammatory levels of IFNγ, IL-17, TNFα, IL-6, CCL18, and IP-10 from MDA5-positive dermatomyositis patients were significant increase compared with healthy controls. These data suggest that MDA5-positive dermatomyositis patients had the dysregulated immune system and the exacerbated inflammation response. In addition, plasma surfactant protein D and Krebs von den Lungen-6 levels in MDA5-positive dermatomyositis patients were markedly higher than those in healthy controls, supporting that the presence of anti-MDA5 autoantibody in dermatomyositis patients usually had lung fibrosis phenomena.

Biography

Ju-Pi Li has completed her PhD from National Tsing Hua University and Postdoctoral studies from Immunology Research Center, National Health Research Institutes in Taiwan. She is an Assistant Research Fellow of China Medical University Hospital. She has published about 20 papers in reputed journals.

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Hsin-Wei Chen et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

A NOVEL ANTIGEN DELIVERY SYSTEM VIA FCγ RECEPTORS INDUCES ROBUST IMMUNE RESPONSES

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Antigen-presenting cells are important for the induction of immune responses. Dendritic cells (DCs) are most effective antigen-presenting cells. They patrol in the peripheral and continuously sample their surrounding environment with various receptors. Fcγ receptors (FcγRs) express on dendritic cells. They bind to the constant fragment of IgG and are crucial to mediate internalization of antigen-antibody complexes (immune complexes, ICs). Internalization of IC facilitates antigen uptake and presentation by DCs. In this context, targeting of antigen to DCs via FcγRs potentially constitutes an effective strategy for modulation of antigen-specific immune responses. Formyl peptide receptor-like 1inhibitory protein (FLIPr), secreted by *Staphylococcus aureus*, is a potent FcγR antagonist and bind to various FcγRs. In this study, we developed a novel antigen delivery system by fusion antigen with FLIPr. Ovalbumin (OVA) was used as a model antigen. Our results show that OVA-FLIPr fusion protein (OVA-FLIPr) possesses FcγR binding ability. Immunization of mice with OVA-FLIPr but not OVA can induce both OVA-specific CD4 and CD8 T cell responses without exogenous adjuvant formulation. In addition, we demonstrate that OVA-specific cytotoxicity is elicited and mediate antitumor response in mice immunized with OVA-FLIPr. These results indicate that FLIPr is a potential vector to deliver antigen to DCs via FcγRs which induce robust immune responses without exogenous adjuvant formulation.

Biography

Hsin-Wei Chen obtained his PhD in the field of Agricultural Chemistry from National Taiwan University, Postdoctoral training was in the immunology field at Academia Sinica and National Health Research Institutes. He is an Associate Investigator of National Health Research Institutes. He has published more than 50 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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Al-Nesf M et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

POLLEN ATLAS TO INVESTIGATE THE LOCAL FLORA IN QATAR

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All Palynological applications and researches related to pollen require the definition of pollens and establishment of an illustrated Pollen Atlas. Pollen Atlas is a reference resource that records the specimens of many species collected from local flora. It is used by researchers involved in agriculture, forensic, allergy, phenology and biological specialties. Furthermore, the atlas will help plant taxonomist to solve the problems of distinguishing between the morphologically similar plants and will improve our understanding of the evolution of Arabian plant communities and environments. We initiated a pioneer project on Aerobiological studies in Qatar and Sharjah: toward the establishment of a network for pollen analysis and allergenicity; collaborative work between Hamad Medical Corporation (Qatar), and University of Malaga (Spain) teams working on the local flora of Qatar. The Project aims to develop innovative solutions for the sustainable management for the endemic flora in the Arabian Peninsula, through the preparation of an illustrated Atlas with the most common pollen types in the atmosphere of Qatar (Doha and Al-Khor). Pollen obtained from flowering samples of native, ornamental, crop and horticultural plants were collected from different parts in Qatar from January to September 2017. A total of 277 species were collected. Reference slides of pollens grains were prepared using each of blooming plants. Pollen grains were mounted in glycerine jelly solution. Pollen grains are represented by family or genus level. For each species, a high-quality image was taken for the pollens collected from the flowering plants with the magnification of 400 X. Differences in species, abundance and distribution, were observed. This tool is a rapid method to identify and determine pollen season overlap where species and genera cannot be distinguished by microscopy. This is the first step, but we expect to expand the process for most of the flora of Qatar.

Biography

Maryam Ali Al-Nesf Al-Mansouri has completed her MD from Sultan Qaboos University, Sultanate of Oman in 1999 and Arab Board of Medical Specialization in 2005 in Medicine. She finished speciality training in Pulmonary Medicine and in the Allergy and Clinical Immunology fellowships from 2005-2009. She is the Head of Allergy and Immunology Section, Hamad Medical Corporation, Qatar since 2016. Currently, she is advancing her academic career by studying the degree of Cellular and Molecular Medicine (MSc) (R) in the Faculty of Biomedical Science at the University of Bristol, UK. She has published more than six papers in reputed journals and multiple abstracts.

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EuroSciCon

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Immunology

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Jongseon Choe et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

OPPOSING ROLE OF ERK AND P38 IN THE POSITIVE FEEDBACK PRODUCTION OF PROSTAGLANDINS BY HUMAN FOLLICULAR DENDRITIC CELL LIKE CELLS

Jongseon Choe and Bongjae Kwon
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Prostaglandins (PGs) are recognized as important immune regulators. Using human follicular dendritic cell (FDC) like cells, we have investigated the immunoregulatory role of PGs and their production mechanisms. We have recently reported on the positive feedback effect of PGs, and the present study was aimed at determining the role of ERK and p38 MAP kinases in PG-induced cyclooxygenase-2 (COX-2) expression by immunoblotting. COX-2 is the key enzyme responsible for PG production in FDC-like cells, which produce PGE2, PGI2, and PGF2α. An ERK inhibitor inhibited PGF2α from inducing COX-2 whereas a p38 inhibitor prevented PGE2- and PGI2-stimulated COX-2 induction. In line with these results, PGE2 and PGI2 treatment resulted in up-regulation of p38 phosphorylation while PGF2α stimulation led to phosphorylation of ERK. We are currently confirming these results with RNA interference technology. These findings suggest that ERK and p38 play differential roles in COX-2 expression in FDC-like cells and shed a therapeutic potential in the treatment of immune inflammatory disorders.

Biography

Jongseon Choe had completed Ph.D. degree in immunology at Seoul National University, Korea in 1995 and worked at Ochsner Medical Institute (New Orleans, USA) as a research fellow. His published papers include "In this issue" article in Journal of Immunology (180:1390-1397) and "Highlighted article" in International Immunopharmacology (12:635-642). As a faculty member of Kangwon National University, he is currently interested in the production mechanisms and inherent roles of eicosanoids in the germinal center of peripheral lymphoid tissue.

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Thamer A Hamdan et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

AUTOANTIBODIES ARE INDUCED DURING CHRONIC VIRAL INFECTION AND ACCOUNT TO CD8+ T CELL EXHAUSTION

Thamer A Hamdan, Vikas Duhan and Karl S Lang
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CD8+ T cell exhaustion is a hallmark of chronic viral infection. Mechanisms underlying CD8+ T cell exhaustion, is still moot. Upregulation of inhibitory receptors (i.e. PD-1, CTLA-4, Tim3) is the major explanation how CD8+ T cells are dampened during chronic viral infection. However, PD-1 is up-regulated on basically all activated CD8+ T cells and *in vitro* data showed limited inhibition of proliferation in the presence of its ligand, PD-L1, so that it remains elusive whether other mechanisms contribute to PD-1 dependent failure of CD8+ T cell function. Commensurately, protective immune responses against viral infection are generally accompanied with production of autoantibodies that might jeopardize the host. In the current study, we propose that induction of autoantibodies during viral infections might attack CD8+ T cells, through binding to the cytotoxic T-cells and deplete them by NK cell mediated cytotoxicity. The scope of our proposed study is to delineate the underlying mechanism of T cells attack/exhaustion via NK cell mediated cytotoxicity and to find out if the depletion of NK cells, B cells or lack of Fc-receptor signaling blunt CD8+ T cell deletion, culminating in robust CD8+ T cell response and effective control of viral infection.

Biography

Thamer A Hamdan has completed his Bachelor's degree in Medical Laboratory Sciences from Jordan University of Science and Technology in 2005. In 2007, he affiliated the same institute to pursue his Master's degree major in Clinical Microbiology and Immunology and obtained the degree in 2010. From 2006-2011, he has worked as Medical Laboratory Technologist at King Abdulla University Hospital, Jordan. Later, he has worked as a Lecturer in Faculty of Applied medical Sciences in University of Tabuk, Kingdom of Saudi Arabia, from 2011 till 2016. Since October 2016, he has commenced his PhD studies at the Institute of Immunology, Medical Faculty, University of Duisburg-Essen, Germany. He has PhD scholarship from DAAD (Deutscher Akademischer Austauschdienst) (German Academic Exchange Service).

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Cheng-Chi Chan et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

FETAL OVALBUMIN EXPOSURE RESULTING IN MURINE HYPER-IMMUNE RESPONSIVENESS

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Allergen exposure during prenatal or postnatal period may alter immune programming and affect the fate of infant's allergic disease. However, the relationship and mechanism between prenatal allergen exposure and the development of allergic disease is still unclear. We aimed to investigate whether prenatal allergen exposure induces immune tolerance or sensitization to allergen. The peritoneal cavity of each FVB/N fetus was directly injected with different doses of adjuvant-free ovalbumin (OVA) or normal saline (NS) on day 14 of gestation. Eight weeks after the birth, i*n utero* NS- and OVA-injected mice were challenged by inhaling OVA aerosols. I*n utero* OVA-injected adult mice with OVA challenge manifested significant induction of airway hyperresponsiveness, lung eosinophilia, serum levels of OVA-specific antibodies and Th2 cytokines of OVA-stimulated splenocytes. These mice also developed serious anaphylactic reactions following intraperitoneal injection of OVA. To further understand the mechanisms of OVA-induced hyper-immune responsiveness, we analyzed the OVA-specific immunity and gene expression of lungs and spleens in prenatal OVA-exposed neonates. I*n utero* OVA-injected neonates already had dominant OVA-specific humoral and cell-mediated immunity. Cytokine expression pattern in the lungs of i*n utero* OVA-injected neonates evidently favoured Th2-biased immune responses. Furthermore, splenocytes of i*n utero* OVA-injected neonates expressed higher RNA levels of Notch ligands (Jagged1 and Jagged2). Inhibition of notch signalling by γ-secretase inhibitor significantly reduced OVA-induced Th2 cytokine production and proliferative responses *in vitro*. The results suggested that intervention of allergen exposure or notch signalling during pregnancy may be beneficial for modulating the development of allergic asthma.

Biography

Cheng-Chi Chan has accomplished his PhD degree in 2016 from Graduate Institute of Biomedical Sciences, Chang Gung University. Postdoctoral training is being performed in the Department of Microbiology and Immunology, Chang Gung University. His studies focus on the mechanisms among prenatal or postnatal allergen exposure and allergic asthma development. He has a good training in the field of Immunology and Molecular Biology and a great skill in the asthmatic animal model and related experiments. Simultaneously, He is familiar with the operation of intra-utero injection and the investigation of the development and differentiation of various immune cells. The related experimental results had been published in reputed journals.

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EuroSciCon

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Immunology

July 05-07, 2018
Vienna, Austria

You Jin Hwang et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

MOLECULAR IDENTIFICATION OF ESBL-PRODUCING GENE BLATEM IN MRSA AND MEATS ISOLATED BACTERIA

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Staphylococcus aureus **is one of the most pathogenic organisms which cause infections in both healthcare and community settings. The proportion of methicillin-resistant S. *aureus* (MRSA) in healthcare-associated (HA) isolates was very high in South Korea. In this study, antimicrobial resistance profiles of *S. aureus* and meats isolated bacteria were determined by disc diffusion method. PCR was applied for detecting the presence of antibiotic resistance genes and blaTEM types. In total of 18 *S. aureus* and 36 meats isolated bacterias. All strains carried blaTEM gene which encodes an enzyme belongs to extended-spectrum β-lactamases (ESBLs), conferring significant penicillin resistance. MRSA strains which carried type II SCC*mec* element presenting characteristic of multidrug resistance (MDR), these strains harbored resistance genes ant(4')-Ia and *ermA*, which showed positive correlation with kanamycin and erythromycin resistance, respectively. Genes *aac(6')-aph(2")* and *tetM* were also found in these strains which phenotypically associated with gentamicin and tetracycline resistance. In addition, type IVA was the most prevalent SCC*mec* element in our study, which carried a variant class B *mec* complex. The J3 region of type IVA element may integrate a copy of plasmid pUB110, harboring *ant(4')-Ia* which phenotypically associated with kanamycin resistance. The molecular characteristics of MRSA strains in our study are unique compared with isolates which have spread internationally. This is the report to show the presence of blaTEM in meats isolated bacterias.**

Biography

Hwang has completed his PhD from Inha University. He is the director of RIS, a Bio Health solution. He has published more than 50 papers in reputed journals.

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Daeyoung Kim et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

SHEEPHEAD MUSHROOM (*GRIFOLA FRONDOSA*) ALLEVIATES OBESITY AND HYPERGLYCEMIA IN DB/DB MICE

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The sheephead mushroom (*Grifola frondosa*, GF) that contains several physiologically active compounds, of which polysaccharides, specifically β-glucans. Its efficacy as to possess various promising bioactivities, mainly including anti-tumor and anti-oxidation, immunomodulation, anti-hyperglycemia. This study aimed to investigate the possible protective or ameliorative effect of GF on obesity and anti-hyperglycemia in diabetic mice. Forty male and female genetically diabetic mice (BKS.Cg-+Leprdb/+Leprdb/OlaHsd) were used and divided into five groups, 5 animals each; at the dose of 0, 75, 150, and 300 mg/kg BW GF extract mixed with fed and insulin treat group. After eight weeks of feeding, serum biochemistry, histopathological, and immunohistochemical definding were performed. In contrast to the control group, treat groups caused significant decreased in absolute and relative weights of major organs, levels of insulin as well as leptin and triglyceride in plasma demonstrated a change similar to blood glucose with feeding of sheephead mushroom. These results suggest that the long term *G. frondosa* consumption alleviates the obesity and hyperglycemia in diabetic mice and to provide biological activities of G. frondosa polysaccharides to support their further therapeutic potentials.

Biography

D. Kim graduated from the college of veterinary medicine and earned his doctorate degree at Seoul National University, South Korea in a research paper on the production of transgenic pigs by somatic cell nuclear transfer in 2003. The same year, he joined the Department of Life Sciences at Gacheon University and became a professor. He served as a school affairs committee member, head of the student ministry, and vice chairman of the committee on animal ethics, and lectures on developmental biology are being conducted. Major areas of interest are diabetes, sarcopenia, pulmonary fibrosis and tendinopathy animal model.

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Andrzej K. Siwicki et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

THE IMPACT OF BIOMATERIAL'S COMPONENTS ON THE CELLULAR AND HUMORAL IMMUNITY

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Biomaterials are commonly characterized as materials used to construct artificial organs, rehabilitation devices, or implants to replace natural body tissues. In general, materials fall into the three categories: metals, ceramics and polymers. The use of biomaterials in clinical practices depends on solving the following problems: 1) toxicity, 2) biological compatibility (biocompatibility), 3) mechanical properties. Biocompatibility has been defined as the ability of a material to perform with an appropriate host response in a specific application. The corrosion of metal is the main problem in construction of implants. Metals commonly used for implants are: cobalt-chromium alloys, stainless steel, titanium alloys, gold, platinum, silver-tin-copper alloys. These elements could be accumulated at the highest doses in the adjacent tissues and bone marrow and could circulate in bloodstream and penetrate to other organs in the body. These released metal ions may cause type IV inflammatory and hypersensitivity reactions, and alternations in bone modeling that lead to aseptic loosening and implant failure. The ions of metals released from the surface of the implant are absorbed by present macrophages which are involved in many of the processes associated with phagocytose orthopaedic biomaterials particles and release the pro-inflammatory mediators as interleukin-1 (IL-1), interleukin-6 (IL-6), tumor necrosis factor (TNF-α) and prostaglandin. The pro-inflammatory cytokines such as IL-1 α and β stimulate resorption of bone and then they act synergistically to the tumor necrosis factor TNF-α. Moreover, macrophages release matrix metalloproteinases (MMPs), and chemokines. Another investigation has shown that Cr and Co ions inhibitis osteoblasts, osteoclasts and T and B cell proliferation.

Biography

Professor Andrzej K. Siwicki, VMD, Ph.D., DSc is a head of Department Microbiology and Clinical Immunology, Faculty of Veterinary Medicine, University of Warmia and Mazury and Department of Pathology and Immunology IFI in Olsztyn, Poland. He is author of over 500 original papers in reputed journals and about 200 scientific communications (index-h 32, index of citation: 4502). His fields of interest are modulation of defence mechanisms and protection against diseases by natural and synthetic products in animals, influence of pollutants on the cell-mediated immunity and restoration of immunity after suppression induced by xenobiotics. Developed a new possibility in the comparative immunotoxicology for control of effect of xenobiotics and pharmaceutical products on the defence mechanisms and protection against diseases. He attended scientific missions in USA, Japan, France and Israel, was a co-ordinator of USDA and FAO projects and he is professor conferring of 14 PhDs and 4 DSc.v.

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THE TOXICOLOGY OF NANOPARTICLES, MICROELEMENTS AND MACROELEMENTS IN CELLULAR AND HUMORAL IMMUNITY

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The immune response is the process of recognition of potentially harmful agents by specialized cells of the immune system. It is expressed as cellular and humoral immunity. Micro-, macro-, and nanoelements can give adverse effects in immunity. The nanoelements (e.g. Silver, TiO_2, ZnO) modulated immune responses via TLR signaling pathways. AgNP, ZnONP, PtNP, TiO_2NP, AuNP enhanced proinflammatory cytokine expression: IL-6-, IL-1, TNF-α, IFN-γ and T cell activation. TiO_2 nanoparticles dose-dependently increased histamine secretion and increased cytosolic Ca^{2+} concentration in mast cells. TiO_2 nanoparticles provoke inflammatory cytokines and increase dendritic cell maturation, expression of costimulatory molecules, and prime naive T cell activation and proliferation. The micro and macroelements on the one hand, they can be expected to increase the production of reactive oxygen species (ROS). They can initiate lipid peroxidation and cellular damage. Immune cells are particularly sensitive to oxidative stress, because their membranes contain high concentrations of polyunsaturated fatty acids which are very susceptible to peroxidation and, when stimulated, they produce large amounts of ROS. On the other hand, trace elements are involved in the antioxidant system and the deficiency of any of them may depress immunity. Uncontrolled oxidation reactions may impair the animal's immune status. Moreover, immune cells and their mechanisms of phagocytic activities are affected by microelements deficiencies. It has been proved that selenium supplementation improves neutrophil's phagocytic capacity. However, low copper status reduces neutrophil phagocytic capacity. Moreover, it has been shown that selenium deficiency affects blood levels of IgG, IgM and IgA as well as T cell function. A number of nutrients (eg Zinc, Selenium, and Chromium) have the ability to modulate immune response through the production of antibodies or cytokines. Moreover the elements are required for immune cells proliferation or activation (e.g. Iron).

Biography

Professor Andrzej K. Siwicki, VMD, Ph.D., DSc is a head of Department Microbiology and Clinical Immunology, Faculty of Veterinary Medicine, University of Warmia and Mazury and Department of Pathology and Immunology IFI in Olsztyn, Poland. He is author of over 500 original papers in reputed journals and about 200 scientific communications (index-h 32, index of citation: 4502). His fields of interest are modulation of defence mechanisms and protection against diseases by natural and synthetic products in animals, influence of pollutants on the cell-mediated immunity and restoration of immunity after suppression induced by xenobiotics. Developed a new possibility in the comparative immunotoxicology for control of effect of xenobiotics and pharmaceutical products on the defence mechanisms and protection against diseases. He attended scientific missions in USA, Japan, France and Israel, was a co-ordinator of USDA and FAO projects and he is professor conferring of 14 PhDs and 4 DSc.v.

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DOI: 10.21767/2471-304X-C1-003

REGULATION OF CHROMIUM (III) AND NICKEL (II) OF RNA VIRUS REPLICATION

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The transition elements: chromium and nickel are essential micronutrients for human, animals and plants. Micronutrients in human and animal organisms play crucial role in prevention and treatment of various diseases. They also play an important role in the optimization of physical and mental functions. Inside the cells, chromium (III) may interact with microfilaments, mitochondria, lysosomes and nucleus. Cr (III) compounds can bind directly to DNA *in vitro,* forming Cr-DNA adducts and DNA-DNA crosslinks. Nickel can induce genotoxic effects, such as DNA strand breaks, sister chromatin exchanges and DNA-protein crosslinks. Moreover, nickel can generate ROS production, which interacts with nucleic acids or proteins. The aim of this study was to examine the effect of treating of chromium (III) and nickel (II) and their combinations on Bovine Viral Diarrhoea Virus (BVDV) replication. The BVDV has been a good model virus for investigating HCV, which is a member of genus *Hepacivirus*, which belongs to the same family. The antiviral efficacies of chromium (III) and nickel (II) on BVDV were evaluated using Real Time PCR method. Moreover, the cytotoxicity of these microelements was examined using the MTT reduction assay. The IC_{50} (50% inhibitory concentration) for the chromium chloride was 1400 µM for BT cells. The IC_{50} for the nickel chloride was more than 1200 µM for BT cells. The concentration-dependent antiviral activity of chromium chloride and nickel chloride against BVDV was observed. In cultures simultaneously treated with 1) 200 µM of $CrCl_3$ and 1000 µM of $NiCl_2$, 2) 1000 µM of $CrCl_3$ and 200 µM of $NiCl_2$, a decrease in number of RNA copies was observed compared with control cells and cells incubated with chromium(III) and iron(III) used separately. The synergistic antiviral effects were observed for chromium (III) and nickel (III) against BVDV.

Biography

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Alencar L et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

THE ROLE OF WESTERN DIET ON LOW-GRADE INFLAMMATION

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In the last decade, a food pattern known as Western diet, heavily based on ultra-processed foods with high levels of fats and refined sugar, thus very palatable and easy to adhere, has been presenting an exponential growth. However, a number of negative effects to those who adopt said diet have been identified, such as excess of energy, lack of fibres, phenolic compounds and micronutrients, apart from the high presence of xenobiotics, compounds that may initiate or worsen a process of low-grade inflammation. Evidence shows that this type of diet promotes endotoxin translocation to the bloodstream, stimulating innate immune cells and leading to a transient postprandial inflammatory response. Binding of LPS-protein complexes to the toll-like receptor 4 (TLR4) activates cellular nuclear factor kappa B (NF-κB) signalling pathway which in turn leads to production of diverse proinflammatory cytokines and chemokines (IL-1β and TNF). Moreover, a low fiber diet and the lack of phenolic compounds may affect the gut microbiota, leading off dysbiosis and a reduction of probiotic strains capable of converting phenolic compounds, such as *Lactobacillus plantarum* and L. *brevis*. These strains contain an enzyme called phenolic acid decarboxylase (e.g. caffeic acid to 4-vinyl catechol), responsible for converting phenolic compounds into metabolites which, in turn, can activates nuclear factor 2-related factor 2 (NRF2), responsible for the synthesis of phase 2 enzymes who have the ability to neutralising reactive oxygen species (ROS). In addition, once micronutrients are essential co-factors in the synthesis of enzymes that play an important role on the conversion of hydrophobic xenobiotic into hydrophilic xenobiotic (e.g. selenoproteins), a lack of those essential elements can impair physiologic detoxifying pathways. Therefore, aiming to reduce the risks that a low-grade inflammation can present, it is recommended a fiber rich diet, based on whole foods, with a variety of vegetables, fruits, roots and minimally-processed foods.

Biography

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Anna Przybyła et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

CLONALLY EXPANDED, STEM CELL-LIKE MELANOMA-ANTIGEN SPECIFIC CD8 MEMORY CELLS CAN BE DETECTED IN HEALTHY HUMANS

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We used four-color ImmunoSpot® assays, in conjunction with peptide pools that cover the sequence of tyrosinase (Tyr), MAGE-3, Melan/MART-1, gp100, and NY-ESO-1 to charact erize the melanoma antigen (MA)-specific CD8 cell repertoire in PBMC of 40 healthy human donors (HD). Tyr triggered IFN-γ-secreting CD8 cells in 33% HD within 24h of antigen stimulation ex vivo. MAGE-3, Melan/MART-1, and gp100 also induced recall responses in 10%, 5%, and 5% of HD, respectively. At this time point, these CD8 cells did not yet produce GzB. However, they engaged in GzB production 72h after antigen stimulation. By this 72h time point ex vivo, 58% of the HD responded to at least one, and typically several, of the MA. A closer characterization of the Tyr-specific CD8 cell repertoire showed it to be of low affinity, and to entail primarily the stem cell-like subpopulation.

Biography

Anna Przybyla has completed her PhD from Poznan University of Medical Science and Postdoctoral fellowship from Cellular Technology Limited (CTL) in Cleveland, USA. She is an Adjunct in the Department of Cancer Immunology at the Poznan University of Medical Sciences where she is also involved in teaching college students. Her research field are melanoma genetics and immunology, immunomonitoring and genetically modified melanoma vaccine. She has published 10 papers in medical and scientific journals.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Liangping Li et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

ANTITUMOUR T CELLS GENERATED FROM EMBRYONIC STEM CELLS MODIFIED BY TUMOUR ANTIGEN SPECIFIC TCR

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TCR-T cell therapy is a promising immunotherapy for cancer patients. A large number of tumour-reactive TCR genes are cloned and need the preclinical experiments. Therefore, it is very important to establish an experimental animal model to quickly and effectively evaluate the function of TCR. The development of stem cell technology provides a new way to solve these technological bottlenecks. Embryonic stem cells (ES) / inducible pluripotent stem cells (iPS) can grow infinitely *in vitro,* are easy to culture, and maintain normal cell karyotype and have the potential to differentiate into various normal cells, including T cells. Therefore, we propose to combine TCR and ES technology to produce single specific T cells using TCR modified ES cells. In order to mimic human HLA-TCR recognition system in mouse T cells, we use human and mouse hybrid molecular system of MHC and TCR. The MAGEA1-specific TCR1367 contained human V (D) J and mouse C gene fragment was sub cloned into lentiviral vector for the transduction of mouse ES cells, and then OP9-DL1-HHD cell line was generated with HHD retroviral vector which express the chimeric MHC molecule HHD to create human HLA-TCR antigen recognition system in mouse T cells, TCR1367-ES, OP9 and OP9-DL1-HHD cells were subcutaneously co-injected into NOD/SCID or NSG mice to generate ES cell-derived teratoma. 8 weeks later, mature T cells which have single antigen specificity for anti-tumour antigen could be detected in peripheral blood and spleen. After isolating these T cells, we found that they could specifically recognize MAGEA1 epitopes, and kill MAGEA1+ cells. Using this simple and low cost TCR-ES cell differentiation technique, NOD/SCID or NSG mice can be used as a biological generator to produce antitumor T cells, which can be used to test the functions of various human TCR genes in preclinical animal experiments.

Biography

Liangping Li has completed his PhD from Humboldt-Universität zu Berlin and Postdoctoral studies from Max-Delbrück-Centrum für Molekulare Medizin, Berlin, Germany. He is the director of Institute of Clinical Oncology, Jinan University, First Affiliated Hospital, Guangzhou, China. He has published several important papers in reputed journals such as Nature Medicine, Nature Protocol, Blood, Cancer Res. et al. and has been serving as an Editorial Board Member of several journals.

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Immunology 2018

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Immunology

July 05-07, 2018 Vienna, Austria

EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Dounya Bounid et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

CLINICAL SIGNIFICANCE OF ANTITHYROID ANTIBODIES

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The thyroid injury is the most frequent organ specific autoimmune disease. The thyroid gland is the target of two main autoimmune pathologies; Grave disease (GD) and Hashimoto's thyroiditis (HT). The autoimmune thyroiditis (AIT) shares a common immunological marker, which is the presence of circulating antithyroid antibodies (ATA). The type of these ATA and their targets define the specificity of each disease. The objective of our study was to determine the clinical significance of the ATA; thyroid pyroxidase (TPOAb), thyroid globuline (TGAb) and the TSH-receptor antibodies (TRAb), in thyroidal and non-thyroidal pathologies. We conducted a cross-sectional and retrospective study on patients having positive antithyroid antibodies enrolled at the laboratory of immunology of the University Hospital of Marrakesh during the period from January 2014 to January 2016. The mean age of our patients was 38±16 years with a sex-ratio M/F of 0.57. The ATA were associated in 70.9% of cases to hypothyroidism, in 22.15% to hyperthyroidism and in 7 % of cases to euthyroidism. The hypothyroidism was noted in 83.2% of TPO Ab positive cases, the hyperthyroidism in 87.2% of TRAb positive cases and the euthyroidism in 6.4 % of TPOAb positive ones. Thyroiditis were represented essentially by the HT, noticed in 110 patients (69.6%) and the GD in 37 (23.4%). ATA were associated to non-thyroidal autoimmune diseases in 29.7% of cases specially represented by type 1 diabetes, sjogren syndrome, celiac disease, lupus and the PBC, associated to TPOAb in (10.4%), (4.8%) ,(4 %), (3.2 %) and (3.2%). The autoimmune polyendocrinopathy was associated with TRAb in 7.7 % and TPOAb in 1.6% of cases. These ATA were also associated to non-thyroidal and non-autoimmune pathologies such as type 2 diabetes and hypertension which were especially associated to the TPOAb in 5.6 % and 4 % respectively followed by Turner's syndrome and Crohn's disease who were especially associated to TGAb in 6.25 % for each one. The results objectified in our series suit generally to various series of literature. These results underline the importance of ATA in clinical practice especially in thyroidal and/or non-autoimmune pathologies and required a finical interpretation to establish exactly their real clinical significance and to help for better medical care of patients.

Biography

Dounya Bounid (MD) has completed his Medical Studies from School of Medicine, Cadi Ayyad University (Marrakech, Morocco) and actually Resident in Medical Biology (second year) in the same university.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Ju-Pi Li et al., Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

DYSPHAGIA IN IDIOPATHIC INFLAMMATORY MYOPATHY PATIENTS WITH ANTI-FHL1 AUTOANTIBODY

Ju-Pi Li and Joung-Liang Lan

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FHL1 (four and a half LIM domains protein 1), is a cysteine-rich double Zinc-finger structure protein, highly expresses in skeletal and cardiac muscles. FHL1 is shown to involve in muscle growth, myoblast di erentiation, sarcomere formation and structural maintenance. The gene and protein of FHL1 is associated with several diseases, including Emery–Dreifuss muscular dystrophy, reducing body myopathy, X-linked myopathy characterized by postural muscle atrophy, and scapuloperoneal myopathy. Recent study further shows that the anti-FHL1 autoantibody has a potential pathogenic role in idiopathic inflammatory myopathies (IIMs) patients. Thus, our study aims to examine whether the anti-FHL1 autoantibody is associated with IIMs patients in Taiwan. Anti-FHL1 autoantibodies in plasmas from IIM patients are compared with healthy controls, as well as disease controls from SLE patients via ELISAs and immunoblot analyses. We found that the anti-FHL1 autoantibody is shown to be a novel and muscle-specific autoantibody in Taiwan IIMs patients. It may coexist with other myositis-specific autoantibody. IIM Patients with anti-FHL1 autoantibody have higher disease severity, especially in dysphagia and muscle weakness.

Biography

Ju-Pi Li has completed her PhD from National Tsing Hua University and Postdoctoral studies from Immunology Research Center, National Health Research Institutes in Taiwan. She is an Assistant Research Fellow of China Medical University Hospital. She has published about 20 papers in reputed journals.

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15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Martin Ungerer, Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

INDUCTION OF TOLERANCE FOR ANTIGEN-SPECIFIC THERAPY OF GRAVES´ DISEASE AND ORBITOPATHY

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Graves' disease is an autoimmune disorder, which is characterized by stimulatory antibodies targeting the human thyrotropin receptor (TSHR), resulting in hyperthyroidism and multiple organ damage. The disease can be modelled in mice using adenoviral immunizations with the extracellular A subunit of the TSHR, which induces a long-term stable disease state. TSHR binding cAMP-stimulatory antibodies, thyroid enlargement, elevated serum thyroxin levels, tachycardia, cardiac hypertrophy and orbitopathy are observed in these Ad-TSHR-immunized mice. T cell epitope-derived linear peptides have been identified using immunized HLA-DR3 transgenic mice, which may induce tolerance towards TSHR at the group of David Wraith, Birmingham, UK. A combination of such peptides have being investigated in a first clinical phase I trial with encouraging results in patients with Graves´ disease at Apitope Inc. Alternatively, cyclic peptides derived from the interaction site of the TSHR A domain with stimulatory anti-TSHR antibodies were injected intravenously in monthly intervals into mice modelling Graves´ disease. These administrations of cyclic peptides were each timed two weeks after the respective Ad-TSHR immunizations, and re-established tolerance towards the antigen, improving symptoms of Graves´ disease within 3 – 4 months after starting these therapies. In immunologically naïve mice, administration of the cyclic peptides did not induce any immune response.

Biography

Medical school at the University of Munich, Germany, and at the Universities of Marseille and Nice, France post-doc in the lab of Prof. Martin Lohse and Prof. Ernst Winnacker, Gene Center, Max-Planck-Institute for Biochemistry, Martinsried, Germany Resident/Senior House Officer at the1st Medical Clinic Rechts der Isar and German Heart Center, TUM University of Munich, training in internal medicine and cardiology Phd promotion ("Habilitation") at TUM University of Munich. Founder and member of the company management of ProCorde GmbH, Corimmun GmbH and advanceCOR GmbH - biotech companies which established novel technologies and drugs for the treatment of thrombosis, cardiac and immunological diseases approved professor of internal medicine and cardiology at the University of Würzburg member of the „Translational Research Group" steering committee of the German Centre for Cardiovascular Research, Berlin.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Kimihiko Okazaki, Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

AN EASY WAY TO ELIMINATE CAUSES OF COLLAGEN AND ALLERGIC DISEASES

Kimihiko Okazaki

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According to the traditional concept of the contemporary immunology, neither autoimmune diseases nor allergic diseases can be cured completely. Nevertheless, a fortunate coincidence led the author to discover a novel concept that eliminating the causes of these diseases is possible. In other words, combinations of pathogenic antibodies with responsible cells, namely, cytolytic T lymphocytes in cases of autoimmune diseases and mast cells in cases of allergic diseases, can be decomposed by replacing the pathogenic antibodies with non-specific antibodies. In more detail, intradermal injections with a non-specific antigen preparation induce productions of non-specific antibodies in the body of the patient. Repetitions of the injections bring about an accumulation of them. Accumulated non-specific antibodies will occupy most of the receptors on the surface of responsible cells. When the accumulation reaches the sufficient level, virtually no pathogenic antibodies would remain on the receptors. That is, no causes of the diseases remain.

Biography

Kimihiko Okazaki Born in 1933, in Osaka. Graduated from Kyoto University in 1959. Engaged in Medical Chemical research work from April, 1960 to July, 1981, his Main achievements of my work are (1)Discovery of a novel coenzyme of thiamine pyrophosphokinase in Baker's yeast, i.e. mho-inositol 1-pyrophosphate. and (2)Identification of the initiator of rat liver regeneration as biliverdin. Each work was published in Biochemical and Biophysical Research Communications, in 1975 and 1978, respectively. Engaged in clinical internal medical work since July, 1981. Started running a private clinic in September, 1989.

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Immunology

July 05-07, 2018 Vienna, Austria

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15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

ROLE OF INFLAMMATORY CYTOKINES AND IMMUNE REACTIVE MOLECULES IN PATHOGENESIS OF STREPTOCOCCUS AGALACTIAE IN ABORTED WOMEN

Ali Anok Njum
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S. *agalactiae* has been appearing as a vital human pathogen and a gradually important cause of aggressive infections in immunocompromised adults and older. The aim of the study was to find the effect of inflammatory cytokines (interleukin 2 and 8) and immune reactive molecules (CD79 and CD54 molecules) on pathogenesis of *S. agalactiae* that isolated from aborted women. A total of 100 aborted women aged between (16 - 42) years, were involved in this study. Placentas specimens were cultured to isolate the *Streptococcus agalactiae*, the level of cytokine in the serum was measured by commercial ELISA tests, while CD molecules was estimated by immunohistochemistry assay. Our results showed that there was streptococcal isolates from placental specimens, specific isolation and identification were done for *S. agalactiae*. Significant difference could be found in serum levels of inflammatory cytokines (P≤ 0.05) between these two investigated groups (infected and uninfected with *S. agalactiae*) in addition to high expression for CD79 and CD54 in infected women as compare with non *S. agalactiae* infected women.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

DIFFERENTIAL EXPRESSION OF TRANSLOCATOR PROTEIN (TSPO) IN MULTIPLE SCLEROSIS REFLECTS ACTIVATED MICROGLIA AND ASTROCYTES

Sandra Amor[1,2], Erik Nutma[1], Jodie Stephenson[1,2], Rianne P G Gorter[1], Paul van der Valk[1], Paul M Matthews[3] and David R Owen[4]

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Microglia are the resident immune cells of the central nervous system. A frequently used marker for *in vivo* activated microglia is the 18kDa translocator protein (TSPO). TSPO is widely used as a Positron Emission Tomography (PET) imaging target to visualize injured brain areas and microglial activation. TSPO, formerly known as the peripheral benzodiazepine receptor, is a cholesterol binding protein localized to the outer mitochondrial membrane. Despite the proposed roles of TSPO, little is known about the cells expressing TSPO, namely the microglia and astrocytes, in neuroinflammatory and neurodegenerative disorders. However, localisation of TSPO in relation to lesions and cell types is unknown. Therefore, we performed immunohistochemistry on brain tissue containing different white and grey matter. Multiple sclerosis (MS) lesions (n = 56) were compared with brain tissues from healthy controls (n = 10) to determine expression of TSPO. Low levels of TSPO expression were found in the white matter of healthy controls (16.9±2.4%) and normal appearing white matter (NAWM) (22.9±16.4%) in MS cases. Conversely, significantly increased levels of TSPO expression were observed in microglia of active white matter lesions (12.9±7.5%) and the rim of chronic active lesions (15.1±8.5%) in MS cases compared to controls (1.2±0.6%) and NAWM (0.7±0.8%). Furthermore, TSPO was also found to be expressed in astrocytes at the centre of active lesions. These findings show the localisation of TSPO in MS lesions in the brain and highlight that PET imaging using TSPO ligands in people with MS not only reflects microglia and macrophage activity *in vivo* but also the response of astrocytes, as well as cells of the adaptive immune response.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

CLONALLY EXPANDED, STEM CELL-LIKE MELANOMA-ANTIGEN SPECIFIC CD8 MEMORY CELLS CAN BE DETECTED IN HEALTHY HUMANS

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We used four-color ImmunoSpot® assays, in conjunction with peptide pools that cover the sequence of tyrosinase (Tyr), MAGE-3, Melan/MART-1, gp100, and NY-ESO-1 to charact erize the melanoma antigen (MA)-specific CD8 cell repertoire in PBMC of 40 healthy human donors (HD). Tyr triggered IFN-γ-secreting CD8 cells in 33% HD within 24h of antigen stimulation *ex vivo*. MAGE-3, Melan/MART-1, and gp100 also induced recall responses in 10%, 5%, and 5% of HD, respectively. At this time point, these CD8 cells did not yet produce GzB. However, they engaged in GzB production 72h after antigen stimulation. By this 72h time point ex vivo, 58% of the HD responded to at least one, and typically several, of the MA. A closer characterization of the Tyr-specific CD8 cell repertoire showed it to be of low affinity, and to entail primarily the stem cell-like subpopulation.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

FACING THE CHALLENGE OF DEVELOPING ULTRA-SENSITIVE MOLECULAR ASSAYS FOR HIV VACCINE AND CURE-RELATED RESEARCH

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Current internationally-approved HIV viral load assays detect down to 20 copies of HIV RNA in plasma samples but are not suitable for detecting ultra-low DNA and RNA within host cellular compartments. Even when it is undetectable by commercial assays, HIV persists within patient plasma, CD4 T cells and other cellular compartments. International human subject research regulations now stipulate that all research subjects who test positive for HIV must be placed on highly active anti-retroviral treatment (HAART) immediately. This makes the continued monitoring of their HIV infection more challenging. As we continue to bank and study these samples, there will be an increasing need for assays that can reliably detect ultra-low quantities of HIV DNA or RNA from limited amounts of archival serum, plasma or cellular material. There will be an increasing need for lower cost assays for monitoring ultra-low viral levels of the virus, in resource-limited settings, where such studies are being conducted. Ultra-sensitive laboratory developed PCR-based assays (LDAs) will thus play an increasingly critical role in human subject-based HIV research and vaccine efforts. This presentation outlines the presenters experience in developing ultra-sensitive HIV molecular assays. The presenter provides an overview of the field in general, including current approaches in the study and elimination of the HIV viral reservoir. The presenter discusses current challenges and provides an opportunity for the audience on approaches that may be used to address these issues.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

IMMUNOMODULATORY EFFECT OF LOW MOLECULAR WEIGHT GARLIC PROTEINS IN CROSSTALK BETWEEN PERIPHERAL BLOOD MONONUCLEAR CELLS AND COLON CANCER CELLS

Elahe Safari, Mona Amani and Samaneh Khorami
Iran University of Medical Sciences, Iran

Cancer is one of the non-infectious diseases with high mortality and colorectal cancer is the third common cancer worldwide. Herbal medicine can use as a supplement in cancer. Among the various type of herbal medicine, garlic has different medicinal properties and biological effects. Garlic active ingredients can improve the immune system to defend against different microorganism and also cancer. In this study, the immunomodulatory effect of low molecular proteins of garlic was evaluated in the co-culture of peripheral blood mononuclear cell (PBMCs) and colorectal cancer cell lines SW48 and SW837. After extraction from garlic cloves, protein fractions were purified by G-75 gel filtration chromatography and confirmed by SDS-PAGE. To define the protein identity, MALDI-TOF spectrometry was done. In the cell culture phase, PBMCs and cell lines alone and in co-culture were treated with desired protein and PBMCs proliferation was assayed by CFSE. Also, cell culture supernatants were collected to evaluate the secretion of mediators by ELISA test. Finally, the rate of T regulatory and MDSC in co-culture medium was measured by flow cytometry. The result shows that purified protein fraction was a lectin binding protein with 11-16 kDa molecular weight. In proliferation assay, these proteins were able to stimulate PBMCs alone and in co-culture with tumour cell lines ($p<0.05$). In cytokine assay, PBMC treatment with protein fraction caused reduction in TGF-β and Galectin-3 secretion; in opposite IL-6 and IFN- secretion level was upregulated and it has no significant effect on IL-10 secretion in comparison with a negative control($p<0.05$). Also, the result shows that this garlic fraction could decrease T regulatory induction in the co-culture milieu ($p<0.05$). By stimulating PBMCs proliferation, inhibiting suppressor cell induction and upregulating inflammatory cytokine and reversely reducing inhibitory mediators; low molecular weight garlic proteins may use as an immunomodulatory supplement in cancer treatment. Also, the *in vivo* study should be done.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

THYMIC STROMAL LYMPHOPOIETIN PROMOTES INTERPLAY BETWEEN TUMOUR CELLS AND MYELOID CELLS TO REGULATE BREAST TUMOUR PROGRESSION

Emma L Kuan and Steven F Ziegler
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The cytokine thymic stromal lymphopoietin (TSLP) has been implicated in controlling various human cancer developments through regulating type-2 inflammation via T cells. However, it is still largely unknown the role of TSLP in other cell types within tumours since TSLP receptor (TSLPR) is widely expressed on many cell types, including hematopoietic cells and epithelial cells under inflammatory conditions. We found that both human and mouse breast tumour cells express functional TSLPR and TSLP signalling is important to maintain their survival through inducing Bcl-2 *in vitro*. By using murine metastatic breast tumour models we found the most important TSLP source for maintaining breast tumour cell survival *in vivo* is not derived from tumour cells but rather from myeloid cells. Furthermore, tumour cell-derived IL-1a is important to increase TSLP expression in myeloid cells. Blocking TSLP systemically after tumour development has reduced primary tumour size in a spontaneous mouse breast cancer model. Constitutive expression of TSLP in lungs of mice that mimicked human asthma enhanced tumour cell survival in lungs that further led to more lung metastases and TSLP blockage only in lungs reduced tumour metastases. Besides tumour cells, we discovered that TSLP signalling in Ly6Chi monocytes is also crucial for promoting tumour progression by regulating monocyte suppressor functions and their ability to differentiate into tumour associated macrophages. Our work is the first to show a tumour-myeloid cell axis, mediated through IL-1a and TSLP, to promote tumour cell survival. We also provided another novel mechanism of the requirement of TSLP signalling in regulating the pro-tumour functions in Ly6Chi monocytes. These studies define a novel TSLP-mediated crosstalk between tumour-infiltrating myeloid cells and tumour cells and provide an effective therapeutic intervention in metastatic breast cancer.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

COMBINATORY EFFECTS OF BOVINE HSP70 INDUCER AND SIRNAS ON EXPRESSION PROFILE OF PATHOGEN RECOGNITION RECEPTORS

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Despite its chaperone and stress tolerance actions, heat shock protein-70 (HSP70) is known to modulate immune status of an individual. The aim of the present study was to explore the modulatory effects of an HSP70 inducer Geranyl geranyl acetone (GGA) on the in-vitro expression profile of bovine major pattern recognition receptors (PRRs) viz. Toll-like receptors-2/4 (TLR2/4) and NOD-like receptors-1/2 (NOD1/2). The expression levels in NOD1/2 and TLR2/4 in GGA induced groups were significantly (P<0.01) upregulated than the non-induced groups. However, among all the PRRs, the highest level of expression was observed in TLR4, followed by NOD2, TLR2, and NOD1. Further, we observed that after treatment with HSP70 specific siRNAs on GGA induced cells, significantly (P<0.01) down regulates all the four receptors. Future understanding the basic molecular mechanisms of interaction between the PRRs and HSP70 will make it potential to realistically modulate immune responses towards immunity or thermal tolerance in cattle during summer stress.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

SCORPION VENOM-INDUCED NEUROINFLAMMATORY RESPONSE: IMPACT ON THE SEVERITY OF ENVENOMATION

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The central nervous system (CNS) was originally ruled out as a site of action for scorpion venom, however, neurological manifestations like convulsion are commonly observed after scorpion stings, especially in young children. Scorpion venom components stimulate the neuro-endocrino-immunological axes inducing the activation of an inflammatory response. In this study, we have tested the neuroinflammatory response after an injection of *Androctonus australis hector* (Aah) venom to 7, 21 postnatal days (pnd) and adult mice by subcutaneous route. Our results showed that Aah venom stimulation lead to a stronger neuroinflammatory response in immature mice, characterized by an important leukocyte activation and migration from the circulation to the cerebral tissue. Oxidative stress markers nitric oxide (NO) and malondialdehyde (MDA) were significantly higher in cerebral tissue of 7 and 21 pnd when compared to adult mice. An increase in reduced glutathione (GSH) and catalase levels after 1 and 3 h post envenomation was observed in adult and 21 pnd mice in comparison to the control groups. A significant decrease of antioxidant markers was observed in new borne mice. One hour after envenomation, the immature mice (7 and 21 pnd) revealed alterations in cerebral tissue characterized mainly by hemorrhage and diffuse edema that were more severe than those observed in adult mice. The results show that Aah venom is able to act on the CNS inducing alterations that could be involved in scorpion envenomation severity and high mortality especially in children. More studies in this field are necessary to develop new therapeutic approach taking into consideration the effect of the scorpion venom on the central nervous system.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

SEROPREVALENCE OF IMMUNOGLOBULING AND OF IMMUNOGLOBULINM ANTI-TOXOPLASMA GONDII ANTIBODIES IN HUMAN IMMUNODEFICIENCY VIRUS INFECTION/ACQUIRED IMMUNODEFICIENCY SYNDROME PATIENTS AT TIKUR ANBESSA SPECIALIZED HOSPITAL, ADDIS ABABA, ETHIOPIA

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Background: In Ethiopia, only a few studies on the seroprevalence of toxoplasmosis have been carried out among HIV/AIDS patients. The objective of this study was to determine the seroprevalence of toxoplasmosis among HIV/AIDS patients at Tikur Anbessa Specialized Hospital, Ethiopia and to determine risk factors associated with seroprevalence.

Materials & Methods: Blood samples were collected from randomly selected 150 HIV-positive patients, IgM and IgG anti-toxoplasma antibodies were quantified by using Enzyme immunoassay technique (Human-ELISA, Germany). Ethical approval for the study was obtained from the Institutional Review Board of the Faculty of Medicine, Addis Ababa University. Questionnaire was administered to assess the risk factors associated with the prevalence of toxoplasmosis in HIV patients.

Results: Of the 150 patients, 108 (72%) were females and 42 (28%) were males. The mean (sd) age was 38.4 (9.5). Based on IgG anti-Toxoplasma antibodies status, the seroprevalence of toxoplasmosis in HIV-positive patients was 94%. No IgM antibody was detected. Consumption of raw vegetables and not having primary information about toxoplasmosis were significant association with the presence of anti-toxoplasma antibody ($p<0.05$). Inexact logistic regression analysis of consumption of raw vegetable (adjusted OR=7.49, 95% CI 1.29-58.93) was significant risk factor for toxoplasmosis and having information about toxoplasmosis (adjusted OR=0.083, 95% CI, .011-.499) had significant protective effect. The mean (sd) CD4 count was 341.1(173.6) cells/µl. The association between the presence of anti-toxoplasma antibody and CD4+ T lymphocyte cells count was not statistically significant.

Conclusions & Recommendations: The findings showed that there is a high prevalence of chronic toxoplasmosis in HIV/AIDS patients and the risk factors were consumption of raw vegetable and lack of information about toxoplasmosis. Therefore, routine screening for Toxoplasma should be undertaken for all HIV-infected patients. Moreover, creating awareness about toxoplasmosis and its risk factors should be prioritized

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

THE ROLE OF CD44 AND ITS LIGAND GALECTIN-8 IN THE INDUCTION OF APOPTOSIS IN THE ARTHRITIC JOINTS: ROUTES TO NEW THERAPIES FOR AUTOIMMUNE AND CANCER DISEASES

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Autoimmune inflammatory and cancer diseases are intractable disorders, involving pathological activities mediating tissue destruction. In Europe, and other parts of the world, these diseases cause immense human suffering and inflict on society an annual economic burden of hundreds of billions of Euros, associated with direct and indirect medical costs resulting from lost work time, disability payments and premature death. Traditional treatments and drugs for various autoimmune and cancer diseases either produce undesirable side effects or provide a relief of symptoms or delay in deterioration rather than a cure. Most, if not all, have the deleterious effect of destroying normal cells as well as the cells involved in pathological activities. Results of efforts made to develop improved treatments for autoimmune and cancer diseases using disease specific cell surface chemical entities have been disappointing until now. Rheumatoid arthritis (RA) is a common chronic inflammatory arthropathy, leading to joint destruction and disability as a consequence of the chronic inflammatory processes. The etiology is unknown and the pathogenesis of this disorder is not well understood, yet the molecular events leading to tissue inflammation resulting with cartilage and bone destruction are now better defined. As a result there is a better chance now than ever before to develop new therapeutic modalities; an example of such an effort will be presented here. We have found new variant of human galectin-8 protein expressed in the joints of arthritic patients. This protein induces apoptosis in treated cells and has approximately 80% therapeutic effect in CIA mouse model

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

WHAT DO LEBANESE WOMEN KNOW ABOUT CERVICAL CANCER AND HUMANPAPILLOMAVIRUS? A REPORT ON AWARENESS LEVELS IN URBAN COMMUNITIES

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Objectives: To evaluate the knowledge of Lebanese women about cervical cancer (CC) and human papillomavirus (HPV) infection. To measure the uptake of the cervical cancer screening test (Pap smear), the uptake of HPV vaccination and to determine the influencing factors.

Methods: 444 women with no medical background filled out a 32 item questionnaire. Collected data was analyzed in SPSS® v. 21.0.

Results: 45.7% aged 18 to 25 with high education qualifications (73.9%) and employed in a field not related to health (84.9%). They did not visit a general physician (64%) or a gynecologist (64.6%) regularly. 85.6% were aware of CC with a median CC symptom knowledge score of 3.00±2.13. HPV infection involvement in the pathogenesis of CC was identified in 53.9% of cases. 35.6% of women were aware of HPV infection. The median HPV general knowledge score was 5.39±2.38 and the median HPV vaccination score was 6.00±2.41. 37.6% of participants had been screened by Pap smear for CC at least once in their lives whereas 9% did not know what a Pap smear was. Screening was significantly associated with CC awareness and regular visits to physicians. Only 11.7% of participants aged 18 to 35 were vaccinated against HPV. Vaccination uptake was significantly associated with CC awareness, religion, field of work and studies, and regular visits to gynecologists.

Conclusion: Lebanese women residing in the urban communities are not well informed about CC and HPV. Screening by Pap smear and HPV vaccination uptakes are non-satisfactory.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

VACCINATION OF HEPATITIS B IN HEALTHY ADULTS: EFFECT OF SEX AND AGE, TWO PROTOCOLS OF REVACCINATION IN NON-RESPONDERS

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Objective: To assess, in non vaccinated healthy adults, the response to the standard protocol SP of vaccination for hepatitis B (0, 1, 6 months), and the response to two boosters protocols in non-responders (NR).

Methods: 192 adults, mean age of 44.3 receive (Engerix® B 20 µg/1 ml) by the SP. The non-responders (anti-Hbs antibodies <10UI/l) are divided into two groups. The first group (P1) receives a single booster (R1) 4 months after the SP, and those who remained non-responders receive a double booster (R1b) 2 years after the SP. The second group (P2) receives a unique double booster (R2), 2 years after the SP.

Results: The rate of response after the SP is 75.5 %(145/192). 28 NR in (P1) have a response rate of 32.14 %(9/28). The 19 NR in (R1b) have a response of 36.84 %(7/19). 19 NR (P2) have a response of 63.16 %(12/19). 9.9 %(19/192) of individuals don't respond to any booster. The rate of response decreases with the age(5% every year and 41% every 10 years) For the same age, men are 2 fold more responders then women.

Conclusion: A unique double booster done 2 years after the SP gives a better response then a single booster done after 4 months, and a similar response to a single booster done after 4 months followed by a double booster done after 2 years. The female gender and the age are 2 factors that decrease the response to the vaccination

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

EFFECTS OF HELMINTH ERADICATION ON THE IMMUNE SYSTEM

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Helminth infection has a profound effect on the immune system. However, the precise natures of the immune changes that are elicited by human helminth infection have not been sufficiently characterized. Furthermore, the reversibility of these changes after treatment has not been documented sufficiently. We observed immune profiles of Ethiopian immigrants to Israel cohort. A longitudinal follow up study involving different group of subjects were conducted. Each group had a baseline data for series of peripheral blood tests, including: IgE and Eosinophil levels, T-cell populations, T-cell receptors phenotypes, and cytokines measurement. These tests were all repeated at one year interval. Results were compared between newly-arrived-Ethiopian-Israelis (NEW-Eth-Il), Long-stayed-Ethiopian-Israelis (OLD-Eth-Il), and Non-immigrant- Israeli controls (NON-Imm-Il). Out of the 184 individuals 111 were NEW-Eth-Il, who had a high prevalence of helminth infection, the immunological changes were elivated IgE levels and eosinophil counts, decreased CD4/CD8 ratio, increased proportion of HLA-DR+CD3+, HLA-DR+CD4+ and HLA-DR+CD8+ cells, decreased proportion of CD45RA+CD4+ (naive) and CD28+CD8+ cells, increased proportion CD45RO+CD4+ (memory) cells, and increased secretion of IL-4 and IL-5 (Th2 type cytokines). In the OLD-Eth-Il, who was all negative for helminth infection, we did not observe these immune changes and did not differ markedly from that of the NON-Imm-Il controls. Significant normalizations in the above-mentioned variables were observed in 33 NEW-Eth-Il who have received successful treatment but not in those who missed the treatment. These findings demonstrate that helminth infection is associated with profound immune changes, and can be normalized within a short time after helminth eradication

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

IMMUNE-RELATED ADVERSE EVENTS WITH IMMUNE CHECKPOINT BLOCKADE THERAPIES

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Cancer immunotherapy is coming of age; it has prompted a paradigm shift in oncology, in which therapeutic agents are used to target immune cells rather than cancer cells. The first generation of new immunotherapies corresponds to antagonistic antibodies that block specific immune checkpoint molecules cytotoxic T-lymphocyte-associated antigen 4 (CTLA-4), programmed cell death protein (PD-1) and its ligand PD-L1. Targeting these checkpoints in patients living with cancer had led to long-lasting tumour responses. By unbalancing the immune system, these new immunotherapies also generate dysimmune toxicities, called immune-related adverse events (IRAEs) that mainly involve the gut, skin, endocrine glands, liver, and lung but can potentially affect any tissue. In view of their undisputed clinical efficacy, anti-CTLA-4 and anti-PD-1 antibodies are entering in the routine oncological practice, and the number of patients exposed to these drugs will increase dramatically in the near future. Although steroids can be used to treat these IRAEs, the associated immunosuppression may compromise the antitumour response. Oncologists must be ready to detect and manage these new types of adverse events. This topic will focus on the mechanisms of IRAE generation, putative relationship between dysimmune toxicity and antitumour efficacy, immune-haematological and emergent IRAEs, and basis for management guidelines.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

MOLECULAR ASPECTS INVOLVED IN THE MODULATION OF THE INFLAMMATORY RESPONSE BY INSULIN

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Insulin is a key regulator of the glucose metabolism and has an important anabolic function throughout the body. Insulin controls glucose uptake by many different cells and can modulate various processes where there is need for energy, such as mitogenesis, gene transcription and autophagy. Under certain conditions, for example, diabetes mellitus, the homeostasis of many tissues and organs are affected, leading to an increased mortality due to an enhanced susceptibility to infections. This vulnerability to infection may be partially explained by an inefficient inflammatory response. Several studies in animal models and patients have demonstrated that diabetic individuals have shown ineffective inflammatory response. This deficiency is reflected by a decrease in chemotaxis and neutrophils recruitment, altered production of inflammatory mediators such as cytokines and chemokines, changes in expression of adhesion molecules, the latter two on both: protein synthesis and gene expression. In addition, macrophages from diabetic animals showed decreased phagocytic and microbicidal activities. In most of the parameters studied on this animal model, once the insulin therapy is introduced, these parameters can be reverted. To explore the susceptibility to infections in diabetic patients, the role of insulin in natural immunity against pathogens and inhibiting/reduction of deleterious effects of inflammation, is the nature of my line of research.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

MIF PROTEINS AS PROTOTYPICAL INNATE CHEMOKINES IN INFLAMMATORY AND CARDIOVASCULAR DISEASE

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Inflammatory processes such as those promoting atherosclerotic lesion formations are pivotally driven by components of the innate and adaptive immune axis. Chemokines and their receptors are particularly prominent part of the innate immune arm. While the role of classical chemokines, i.e., belonging to the CC or CXC families is increasingly well understood, an emerging family of chemokine like inflammatory mediators termed innate chemokines, CLF chemokines or micro-chemokines, which additionally structurally and functionally overlaps with the mediator class of alarmins, has been identified, but it yet has to be comprehensively characterized regarding its molecular mechanism and role in disease. For example, innate chemokines modulate inflammatory reactions in the atherogenic arterial wall and numerous other inflamed tissues, but the precise receptor signaling mechanisms are still only poorly understood. What is known is that many innate chemokines share functional homology with classical chemokines and signal through classical chemokine receptors, whereas they do not exhibit conserved structural features such as N-terminal tandem cysteine residues or the chemokine fold. Thus, important receptor binding motifs yet have to be characterized. This lecture will give an overview of the mechanisms underlying molecular hijacking of classical chemokine receptors by innate chemokines, featuring their pathophysiological role. Examples will encompass high mobility group binding protein-1 (HMGB1), macrophage migration inhibitory factor (MIF), MIF-2/D-D (D dopachrome tautumerase) T and certain β-defensins. Receptor usage, binding domains, signalling, innate immune cell regulation and involvement in various inflammatory conditions, including atherosclerosis will be discussed. The lecture will outline strategies to target such mediators in disease either in conjunction or explicit exclusion of the co-targeting of classical chemokines. Finally, a cross kingdom analysis will be shared offering more general understanding of some of these mediators.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

EARLY IDENTIFICATION FOR AUTOIMMUNE TYPE 1 DIABETES

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With a solid establishment of disease biomarkers, the consideration of clinical autoimmune type 1 diabetes (T1D) is now pushed back to the presence of islet autoimmunity or insulitis with islet autoantibodies (iAbs) in peripheral blood before clinical overt T1D appears. It is accepted that the classification of T1D is divided into 3 disease stages: Stage 1 is the presence of two or more iAbs with a normal glucose metabolism, Stage 2 is the presence of two or more iAbs with an impaired Oral Glucose Tolerance Test (OGTT) and Stage 3 is overt diabetes. The biomarker for islet autoimmunity is well known to be started with single iAb (usually IAA or GADA) at early stage and further progressed to two or more iAbs. But unfortunately single iAb identified by the current standard radio-binding assay (RBA) is not able to be adopted as an official biomarker for disease classification since it is problematic for disease specificity and the rate of progression of diabetes in children with persistent single iAb is only 15% in 15 years across populations. We have recently developed, validated, and patented a new generation of iAb assays using the technology of electrochemiluminescence (ECL). In addition to higher sensitivity and earlier identification of iAbs among longitudinally followed young children from birth, the ECL assay has been demonstrated in multiple clinical trials to be more disease specific. It is able to discriminate the high risk, high affinity iAbs from the low risk; low affinity iAbs generated in the RBA that usually appear among subjects with a single iAb. The ECL assay can substantially refine the selection of single iAb positive individuals at high risk and could be used as a reliable early biomarker for T1D islet autoimmunity.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

BUTYRYLCHOLINESTERASE AND ACETYLCHOLINESTERASE POLYMORPHISMS AND SERUM CHOLINERGIC AND INFLAMMATORY PROFILES IN MULTIPLE SCLEROSIS PATIENTS

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Multiple sclerosis (MS) is an autoimmune disease, having not fully understood aetiology, both genetic and environmental factors contribute to the pathogenesis of the disease. The cholinergic system has been indicated as a mediator of neuro-immune interactions, as well as an internal regulator of immune responses. The aim of the present research was to assess the associations between butyryl cholinesterase (BChE) and acetyl cholinesterase (AChE) genetic variations, serum cholinergic and inflammatory profiles in 102 relapsing remitting (RR) MS patients and 117 healthy controls. Results showed that in patients and controls, the reduction of BChE enzymatic activity in subjects carry the BChE polymorphic allele. Serum levels of BChE were higher in RR-MS patients compared to HD subjects, resulting in reduced amounts of circulating ACh. An increased frequency of the BChE K-allele in MS patients as compared to controls was found. The BChE-K-allele seems a promising marker to assess the role of non-neuronal cholinergic system in regulating peripheral inflammation via ACh regulation. This study shed light on the role of the non-neuronal cholinergic system in immune cells to better understand MS aetiology and progression. The cross-talk between the periphery and the CNS could have a new undescribed crucial role for MS, regarded as a systemic disease.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

MODULATORY EFFECTS OF VIP (VASOACTIVE INTESTINAL PEPTIDE) IN THE BONE HEALING PROCESS

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Host inflammatory immune response has been described as an essential step of bone healing process. In this context, immunoregulatory molecules that consequently modulate inflammatory cell migration, and the subsequent bone repair, have been considered as potential targets for improving bone repair. This study aim evaluates the experimental role of the immunoregulatory molecule VIP in the process of alveolar bone healing in C57Bl/6 (WT) mice. Experimental groups (N=5/time point) comprised animals submitted to right upper incisor extraction, maintained how control or treated with VIP (Sigma Aldrich-0.05 mg/kg, IP, 24/24h) or VIP-Antagonist (VIP-Antagonist-GRF1-29-Sigma Aldrich-0.05 mg/kg, IP, 24/24h) starting one day before surgery; assessed by microtomographic-µCT and histomorphometric analysis, in the periods 0h,3,7 and 14d, for quantification of tissue repair indicators and cell migration to the repair site. The results of µCT did not show significant difference between groups in relation to hyperdense regions. The histomorphometric analysis demonstrated a greater area of newly formed bone tissue and a higher number of osteoblasts in the VIP-antagonist group when compared to the control and VIP groups (7d). Referent to osteoclasts density, the groups VIP and VIP-Antagonist presents a major density in relationship to the control groups (14d), which suggests a greater bone remodelling in these groups. Connective tissue formation was also analysed, with the density similar of collagen fibers and blood vessels between groups. Density of fibroblasts, the group treated with VIP-antagonist show a significant diminution compared to the control group (7d). Density of inflammatory infiltrate was also observed, and the group treated with VIP-antagonist showed a higher density in relation to the VIP and control groups. Density of blood clot post-extraction, the group control presents a major density when compared with the VIP-antagonist. These results suggest that inhibition of VIP modifies the course of post-exodontic alveolar bone repair; and further analyses are underway to determine the immunoregulatory mechanisms involved in this modulation.

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

EVALUATION OF IL-21 GENE EXPRESSION IN CELIAC PATIENTS COMPARE TO CONTROL

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Objective: Considering the role of adaptive immunity in the progression and pathogenesis of celiac disease, we investigated the role of Th17 by examining the gene expression of one of its related cytokines, IL-21, in duodenal biopsy of celiac patients in comparison to healthy controls.

Methods: In this study, duodenal biopsy were collected from 60 celiac disease patients under gluten-free (between 6 months and 2 years) and 60 healthy subjects as control group. RNA was extracted from tissue according to the protocol of the commercial kits, cDNA was synthesis, primer pairs designed and then IL-21 gene expression was run by using Real-time PCR technique.

Result: Out of 60 CD patients, 17.6% were female and 12.6% were male with mean age of 38.85 and in control group 55% were female and 45% were male with an average age of 35.60. The most common GI symptoms were bloating (17.6%) and diarrhoea (15.1%), and non-GI symptoms, fatigue (21.6%), weight loss (14.6%) and anaemia (16.1%). Most of the patients were Marsh III (54.2%). The result of this study was shown that IL-21 in gluten free diet (GFD) patients was expressed relatively more than healthy controls, but this difference was not statistically significant (P<0.2).

Conclusions: IL-21 plays an important role in the onset of tissue damage in celiac disease, and in this study as patients were on the gluten free diet; its expression is slightly higher than the healthy group. This gene may consider as a biomarker that can be used to follow the histological improvement

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

PERIPHERAL BLOOD MONOCYTES WITH AN ANTI-INFLAMMATORY PHENOTYPE DISPLAY LIMITED PHAGOCYTOSIS AND OXIDATIVE BURST IN VISCERAL LEISHMANIASIS PATIENTS

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Background: Monocytes are important effector cells during *Leishmania* infection and changes in their functions may impact development of immunity. However, functional characteristics of monocytes in visceral leishmaniasis (VL) patients remains poorly understood.

Methods: Peripheral blood monocytes from VL patients and healthy endemic controls (ECs) from Muzaffarpur, Bihar, India were isolated and compared *ex-vivo* using cell-culture techniques, flow cytometry and RT-qPCR.

Results: A blood monocyte population with a gene signature comprising upregulated expression of *TGM2, CTLRs, VDR, PKM, SOCS1, CAMP1*, accompanied by downregulated expression of *NOS2* and *HIF1A* was observed in VL patients, compared to ECs. VL patient monocytes also had impaired expression of chemokine receptors, adhesion molecules and decreased frequencies of IL-1β- and IL-6-producing cells. Importantly, monocytes from VL patients had a marked reduction in their capacity for phagocytosis of amastigotes, as well as expression of p47phox, p67phox and ROS-production.

Conclusions: VL monocytes express anti-inflammatory molecules and lack a classically activated phenotype. They have reduced expression of molecules related to activation and anti-parasitic effector functions, indicating that monocytes are skewed towards anti-inflammatory phenotype. These findings provide insights into functional status of monocytes during VL and advise that therapeutic manipulation of this important cell population may result in favourable patient outcomes

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
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Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

IGE+ AND REGULATORY B CELLS IN ALLERGIC ASTHMATIC SUBJECT FOLLOWING ALLERGEN INHALATION

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Global prevalence of allergic diseases has been on the rise for the last 30 years. In Canada, this upward trend in allergic diseases has resulted in over 3 million Canadians being affected by allergic asthma. Asthmatic airway inflammation is initiated by the release of inflammatory mediators (histamine) released by granulocytic cells (mast cells and basophils). However, immunoglobulin E (IgE) antibody is also necessary for the initiation of the allergic cascade, and IgE is produced and released exclusively by memory B cells and plasma cells. Acute allergen exposure has also been shown to increase IgE levels in the airways of patients diagnosed with allergic asthma; however, more studies are needed to better understand local airway inflammation. Although regulatory B cells (Bregs) have been shown to modulate IgE-mediated inflammatory processes in allergic asthma pathogenesis, particularly in mouse models of allergic airway disease, the levels and function of these IgE+ B cells and Bregs remain to be elucidated in human models of asthma. Thus, the overall objective for this research was to investigate the frequency of IgE+ B cells and Bregs in allergic asthma, and the kinetics of these cells after allergen exposure. Our research shows that allergic asthmatics have elevated levels of IgE+ B cells in the airways, that can be further increased after allergen exposure. Therefore, local B cell production of IgE in the lungs may be an important source of IgE for initiation of acute inflammatory responses in allergic airways. Additionally, we showed that Bregs were lower in the blood of allergic asthmatics compared to controls, highlighting a possible dysregulation of this regulatory cell type in allergic asthmatics, which may contribute to disease pathology. Furthermore, after whole lung allergen challenge Bregs decreased in the bone marrow with a co-incident increase in the blood and sputum of allergic asthmatics. This pattern reflects potential trafficking of these cells from bone marrow to the airways after exposure to allergic stimuli. However, further functional studies are warranted. Taken together, the findings of this research highlight the local compartmental changes in IgE+ B cells and Bregs following allergen challenge of allergic airways. Better understanding the temporal and compartmental shifts in B cell subpopulations, particularly IgE+ B cells and Bregs, may aid in future development of therapeutics

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EuroSciCon

15th International Conference on

Immunology

July 05-07, 2018
Vienna, Austria

Insights Allergy Asthma Bronchitis 2018, Volume: 4
DOI: 10.21767/2471-304X-C1-003

ACE GENE INSERTION/DELETION POLYMORPHISM IN PAKISTANI RHEUMATOID ARTHRITIS PATIENTS

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Recent research explained that there may be various causes of rheumatoid arthritis i.e. an autoimmune disorder. Different genes including *HLA, TRF* (receptor family) may be involved in the occurrence of rheumatoid arthritis. ACE gene polymorphisms are also considered to be associated with rheumatoid arthritis. The main focus of this study was to check the association of ACE gene I/D polymorphism with rheumatoid arthritis. The general population of Pakistan was selected. Total numbers of the blood samples were 80, out of which 40 were of rheumatoid arthritis patients and 40 were of healthy controls. Body weight, blood glucose levels, rheumatoid factor and gender were the parameters noted in the performa. The mean age of male patients was 51.833±0.17 and that of female patients were 54.90±1.65. The angiotensin enzymes cleave the proteins and regulate fluids and salts balance in the body. The blood samples were collected and stored at 4∏C for further purposes. Serum samples were also collected for screening of the blood samples. Other immunoassays used were agglutination assays including C-reactive protein levels in blood and rheumatoid factor of the patients. Immunofluorescence of the rheumatoid positive samples was also done. The mostly observed patterns for anti-nuclear antibodies were homogenous and speckled. Blood glucose levels of the patients were recorded. In the study, the ACE gene was selected for the study of ACE gene insertion or deletion polymorphism. Nested PCR cycle was run for the specified sequence portion of ACE gene intron 16. All the controls showed 100% DD genotype. The 22.5% subject samples showed the ID genotype and 77.5% showed the DD genotype. No subject either control or patient showed II genotype. As a result, the null hypothesis was rejected and p value was less than 0.05. A significant association was found between rheumatoid arthritis and ACE gene I/D polymorphism

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DOI: 10.21767/2471-304X-C1-003

IMMONO-MODULATORY PROTOCOL FOR HAND AND ARM TRANSPLANTATION

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Transplant paradigm has shifted from immuno-suppression to immuno-modulation, which aims to balance the host and graft responses against each other and achieve co-existence without high-dose, multi-drug immunosuppression. Following the new paradigm, our team developed an experimental protocol first in a rat then swine hind-limb transplant model, utilizing donor bone marrow cells infusion into transplant recipients as a way to facilitate clonal exhaustion and deletion mechanisms and thus modulate the host immune responses towards graft acceptance. We showed that, following an induction protocol of lymphoid depletion and donor bone marrow infusion, the immunosuppression requirement for limb transplant in our swine model was reduced to a single agent of Tacrolimus. Furthermore, the dosage of Tacrolimus may be weaned over time to very low trough levels without jeopardizing the limb allograft. We also showed in the laboratory the feasibility of local, targeted treatment of allograft rejection by topical application of steroid or Tacrolimus to the skin component, thus obviating the need for increased systemic immunosuppression for treatment of mild to moderate rejection episodes of limb allografts. These experimental findings provided the foundation for a clinical immuno-modulatory protocol for VCA that permits allograft survival with minimum, single-agent (monotherapy) immunosuppression. In our clinical protocol, Campath1-H, an anti-CD52 monoclonal antibody, was used for lymphoid depletion just prior to transplant. Donor marrow was obtained by removing the lower thoracic and lumbar vertebral bodies (with donor family permission), from which marrow cells were extracted in a GMP laboratory. A high dose of unmodified donor marrow cells, numbering over a billion, were then prepared into a suspension and infused intravenously into the hand allograft recipient 2 weeks after transplant. All patients were maintained on Tacrolimus monotherapy with its blood trough levels reduced from 12-15 ng/ml initially after transplant to 4-6 ng/ml typically after one to two years. Our hand transplant recipients have not experienced any long term metabolic, infectious, or neoplastic complications. Our team has performed transplantation of 11 hands/arms in 7 patients since 2009, including 4 bilateral transplants, at Johns Hopkins and University of Pittsburgh. Surgical complications were readily treated without long-term consequences, including bleeding, delayed wound healing, bony nonunion and deep venous thrombosis. Each recipient has been maintained on our immuno-modulatory protocol of low-dose Tacrolimus monotherapy without apparent adverse effects of immunosuppression. Good to excellent functions were achieved in all but one recipient, restoring functional independence and personal autonomy and allowing returning to work or school. The functional recovery in the three patients with trans-humeral transplantation has been notable in the restoration of wrist and digital flexion and extension from re-innervation of donor forearm musculature. In one transplant recipient whose biceps and triceps were replaced with those from the donor, near-normal range of active elbow motion was obtained. For the majority of our recipients, therefore, hand transplantation transformed their lives as they became independent and productive

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SYNERGISTIC ACTIVATION OF ANTI-TUMOR IMMUNITY BY AN ARMED ONCOLYTIC VIRUS VG161

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Oncolytic viruses (OVs) are among the most powerful approaches in cancer immunotherapy. OVs not only cause cancer cell lysis but more importantly, their infection in tumors induces anti-tumor immune response from the host, resulting in lasting anti-tumor immunity. It has been recognized that anti-tumor immune response requires multiple immune regulatory factors that act synergistically and tumor microenvironment is critical for tumor to grow. Herpes simplex virus type-1 (HSV-1) has been approved by FDA as an oncolytic viral drug to treat melanoma. One advantage of HSV-1 is its large genomic capacity for carrying multiple exogenous genes. A HSV-1 oncolytic viral vector (VG161) was constructed to simultaneously express IL12, IL15 with its receptor alpha unit and a PDL-1 blocking peptide. Anti-tumor activity of VG161 was tested in both immune competent mice (CT26 and A20 tumor models) and nude mice for human tumor models (LNCaP and U87). Since CT26 and A20 are poorly permissive for HSV-1 replication, the mouse tumor models were able to demonstrate the anti-tumor immune response induced by VG161 while oncolytic activity of VG161 was demonstrated in LNCaP and U87 models since the immune system is compromised in those models. VG161 completely inhibited tumors in all the models tested and the animals survived tumor-free for many months till sacrificed. VG161 induced tumor oncolysis in both LNCaP and U87 tumors. In the CT26 model, animals were protected from the second challenging with CT26 cells following previous virally induced tumor regression. Furthermore, in a A20 double tumor model, intratumoral injection into the tumor on one side caused tumor regression on both sides. Transcriptom analysis showed significant change in tumor microenvironment. Finally, tumor specific memory T-cells were evident in the treated animals. The anti-tumor immune response by VG161 was significantly stronger than similar viruses that did not express any immune stimulating gene or only express GM-CSF. These results showed that intratumorally expressed multiple immune regulatory factors by an oncolytic virus may significantly change the tumor immune microenvironment to enhance efficacy of the oncolytic virus

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Immunology 2018