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March 11-12, 2019 | Amsterdam, Netherlands

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16th EuroSciCon Conference on
Immunology

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Ceren Ciraci et al., J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

DOWNREGULATION OF NLRP11 ALTERS HUMAN T CELL RESPONSES IN CO-CULTURES WITH DAUDI CELLS

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The NLR family is a relatively newly discovered family of Pattern recognition receptors whose functions have been examined almost exclusively in innate immunity. There are 22 known members of the NLR family in humans, four of which form a multiprotein complex called the inflammasome complex. Besides inflammasome complex formation, the NLR family members have been demonstrated to have associations with several diseases such as artherosclerosis, type II diabetes, obesity, Alzheimer's disease, gout and bacterial, viral and parasite infections. One of the members of the NLR family, NOD-like Receptor 11 (NLRP11) is expressed only in primates; however, its cellular functions as well as the specific stimulant(s) that activate it are largely unknown. To examine whether NLRP11 forms an inflammasome complex and whether it has regulatory roles in shaping adaptive immune responses, we investigated its potential interactors including ASC and Caspase-1 by co-IP and determined T cells responses; respectively. We also determined both extracellular and intracellular IL1β production by ELISA and western blotting, as a maker for canonical inflammasome pathway activation. High expression of NLRP11 and expression of costimulatory molecules made Daudi cells an ideal model to use in our experiments. Given that B cells are professional antigen presenting cells (APC) that interact with T cells, we co-cultured human CD4+ primary T cells with Daudi cells in vitro. 40% down regulation of NLRP11 by siRNA in co-cultures resulted in the significant reduction of Th1, Th17 responses and an increase in anti-inflammatory response whereas did not significantly affect Th2 responses when compared with control co-cultures. In brief, our studies of NLRP11 suggest a role in regulating adaptive immune responses.

Biography

Ceren Ciraci has completed her PhD from Iowa State University and Postdoctoral Studies from University of Iowa Inflammation Program. She is currently serving as a Junior Faculty at Istanbul Technical University.

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16th EuroSciCon Conference on
Immunology

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Marjan Jalali Mehr et al., J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

MOLECULAR IDENTIFICATION OF *MYCOBACTERIUM TUBERCULOSIS* COMPLEX ISOLATES IN SMEAR POSITIVE CLINICAL SAMPLES BY RD-TYPING

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Objective: Tuberculosis has long been recognized as a zoonotic disease and is one of the most important threatening diseases of death yet. Rapid identification of the species is too important for fast and correct treatment. Therefore molecular identification is suitable method for this purpose. The aim of this study was identification of ***Mycobacterium*** isolates by RD-Typing technique in tuberculosis patients in Khorasan Razavi province of Iran.

Materials & Methods: PCR 16S rRNA and IS6110 were used for one hundred isolates followed by RD-Typing method (RD1, RD4, RD9 and RD12) were done to differentiate the complex members. PCR-RFLP of oxy-R gene performed to confirm differentiation between *Mycobacterium tuberculosis* and ***Mycobacterium bovis***.

Results: PCR 16S rRNA and IS6110 amplified 543 and 245 bp which identified that all isolates are belonging to the genus ***Mycobacterium*** and *Mycobacterium tuberculosis* complex (MTBC) respectively. RD-Typing and PCR-RFLP revealed that, all isolates were detected *Mycobacterium tuberculosis* and none of them were ***Mycobacterium bovis*** or other species of the MTBC.

Conclusion: Our data suggest a low contribution of ***Mycobacterium bovis*** between human tuberculosis in the Khorasan Razavi province. This is either due to the widespread use of pasteurized milk and non-use of milk contaminated with ***Mycobacterium bovis*** or due to applying of control and eradication scheme for Bovine tuberculosis in the whole country.

Biography

Marjan Jalalimehr 40 years old is graduated from Azad University of Iran majoring in Master of Microbiology . My thesis was in the field of Molecular Identification of Mycobacterium In Department Razi Vaccine & Serum Research Institute. In the section of Tuberculin and passed an experimental period working on this . I have continued my study and work on some related techniques in the field of Microbacterium and I am still involved in this study in Razi institute.

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Dounya Bounid et al., J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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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Immunology

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Shih-Hsin Chang et al., J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

DYSPHAGIA IN IDIOPATHIC INFLAMMATORY MYOPATHY PATIENTS WITH ANTI-FHL1 AUTOANTIBODY

Shih-Hsin Chang, 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 dierentiation, 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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Martin Ungerer, J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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

16th EuroSciCon Conference on
Immunology

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Kimihiko Okazaki, J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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 eliminations of the causes of these diseases are 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 has completed his Graduation from Kyoto University, Faculty of Medicine in 1959. He was engaged in medical chemical research from Apr' 1960 to Jul' 1981. He has started working in Internal Medicine in July, 1981. He has started running a private medical clinic in Sep' 1989. His main achievements are as follows: discovery of a novel in coenzyme of thiamine pyrophosphokinase Baker's yeast; identification of initiator of rat liver regeneration as biliverdin and discovery of a novel and complete cure-method for allergic and autoimmune diseases.

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Immunology

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Shaymaa M. Mohammed, J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

LYSYL OXIDASE, HYPOXIA INDUCIBLE FACTOR –1 ALPHA AND INDUCIBLE NITRIC OXIDE SYNTHASE AS POSSIBLE BIOLOGICAL MARKERS IN BLADDER CANCER

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Bladder Cancer (BC) is the commonest malignancy of the urinary tract ; it is a heterogeneous disease, with 70% of patients presenting with superficial tumors, that tend to recur and are generally not life threatening, About 30% of BC cases presenting as muscle-invasive disease associated with a high risk of death from distant metastasis. Several molecules and pathways have been identified to be linked to the pathogenesis of bladder cancer; while major efforts was directed to characterize molecular alterations to improve disease prognostication, only a few biomarkers of potential clinical relevance have been identified. The hallmark of cancer comprise of six biological capabilities acquired during the multistep development of human tumors, forming an organizing principle for rationalizing the complexities and diversity of neoplastic disease. They include sustaining proliferative signaling, evading growth suppressor, enabling replicative immortality and resisting cell death. In the past decades, majority efforts of cancer research have focused on the functional consequences of oncogene and tumor suppressor gene mutations. However tumors are complex tissues composed of multiple distinct cell types and the extracellular matrix (ECM), these participate in heterotypic interactions with one another in the genesis of tumors by influenceing the tumor microenvironment . Features of the tumor microenvironment that are significantly different from normal tissue are the reduction in oxygen pressure (PO2) which is indicated by hypoxia and pH reduction (acidosis) . Two important means by which cancer cells adapt to their microenvironment, by reprogramming cellular glucose / energy metabolism to use pathways that generate ATP in the absence of oxygen, and by stimulating angiogenesis to increase oxygen delivery. The microenvironment of solid tumors, is exposed to low oxygen tension (hypoxia), a key regulator of the cellular oxygen - signaling pathway is the Hypoxia Inducible Factor-1α (HIF-1α) , a transcription factor that facilitate adaptation to oxygen deprivation by regulating the expression of genes that control cell metabolism, angiogenesis, cell proliferation and apoptosis. On the other hand, lysyl oxidase enzyme (LOX), an important modulator of extracellular matrix, is one critical HIF-1α targets having an important role in the tumor development and progression. Furthermore, hypoxia associated with the maintained inflammatory state, resulted in activation of inducible nitric oxide synthase (iNOS), resulting in nitric oxide (NO) release, a further interesting affecter in the tumor microenvironment. Hypoxia-inducible factors (HIFs) are important angiogenic molecules, as they control a cell's response to a hypoxic stress. Over expression of hypoxia inducible factor -1 alpha (HIF-1α) has been demonstrated in many types of cancer with poor prognosis; its prognostic value is even more significant when combined with p53.

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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* which was 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 were estimated by immunohistochemistry assay. Our results showed that there was streptococcal isolates from placenta 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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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

INVESTIGATION OF THE EFFECTS OF NOBILETIN THROUGH TOLL-LIKE RECEPTOR-9 SIGNALLING PATHWAY IN PROSTATE CANCER

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In this study, we investigated the effect of Nobiletin (NOB) on Toll-like receptor-9 (TLR9) signaling pathway and to highlight the potential for developing a treatment for this pathway that plays an important role in prostate cancer. We investigated the effects of NOB on TLR9 in LNCaP, PC-3 as prostate cancer cells and HUVEC as a control cell. Oligodinucleotide (ODN) was used for TLR9 stimulation. Cell viability was analyzed with the WST-1 assay. TLR9 gene expression was examined by Quantitative Reverse Transcription Polymerase Chain Reaction (qPCR). Cytokines (INF-α and INF-β) were analyzed with Enzyme-Linked Immunosorbent Assay (ELISA). Gelatinase activity and protein expression were examined by zymography and western blotting, respectively. Inhibitory concentrations (IC50) of NOB were found 20 μM for LNCaP and 40 μM for PC-3 and HUVEC. It was observed that NOB increased TLR4 gene expression in PC-3 but decreased in LNCaP and HUVEC. NOB reduced the amount of INF-α and INF-β in PC-3. It was found that NOB reduced TLR9 protein levels in PC-3 and increased IRF-7 protein levels in PC-3 and LNCaP. Gelatinase activity of MMP-9 and MMP-2 was found low in PC-3 although there was high MMP-2 activity in LNCaP and MMP-9 activity was not observed in HUVEC. In conclusion, the effect of NOB is AR-dependent and shows a reducing effect on TLR9 signalling pathway. NOB may be effective on prostate cancer via TLRs and also TLR9-mediated signalling pathway with great potential may be important for new therapeutic approaches in prostate cancers.

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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

THE EFFECT OF NEEM LEAF GLYCOPROTEIN ON HUMAN GLIOBLASTOMA CELL

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Glioblastoma (GBM) is an aggressive brain tumour and the treatment options are limited. Despite advancement in surgical and adjuvant radiation therapy and chemotherapy strategies, malignant gliomas continue to be associated with poor prognosis. Immunotherapeutic use seems to be an effective remedy for cancer. Immunotherapeutic compounds harness the power of the hosts' immune system. *Azadirachta indica*, also known as neem, is commonly found in many semi-tropical and tropical countries including India, Pakistan and Bangladesh. The components extracted from neem plant have been traditionally used as the curative of cancer. Neem leaf glycoprotein has been seen to have a broad-range of inhibition activity on different cancer in culture and is currently being used in different cancer treatments as an immunotherapeutic agent. Uncontrolled cell growth and proliferation are one of the fundamental hallmarks in this disease and play important role in the development of tumour and metastasis. Extracts of neem suppress the proliferation and growth of tumour cells through disruption of cell cycle progression. But it is not clear that how neem leaf glycoprotein inhibits the action of glioblastoma. In this experiment, we are using several experimental approaches to identify that NLGP is an essential component for anti tumoral action. We are testing neem leaf glycoprotein, the most abundant plant-derived, on the U251, U87 and SF126 glioblastoma cell lines. The treatment of glioblastoma cells with neem leaf glycoprotein is expected to have an apoptosis as well as inhibitiory activity. Our expected test results may improve the overall effectiveness in the treatment of glioblastoma in cancer patients. In this study, we are trying to find that NLGP has inhibitory activity on glioblastoma cell proliferation and survival. We are also trying to find that NLGP induce apoptosis of glioblastoma cell. Glioblastoma multiforme (GBM) cell lines are treated with freshly prepared NLGP from mature Azadirachta indica.MTT assay is being done to find out the cell metabolic activity. Cell culture is being analysed for cell apoptosis. Glioblastoma cells are being grown in petri dishes and are being treated with NLGP. Cell cycle and Western blot analysis is being done to find out how after NLGP treatment cellular content of proteins that regulate cell progression through the cell cycle works. Finally PCR is being done. NLGP, as an immunotherapeutic agent, has shown promising result in cancers. If our experiment meets the expected result then NLGP can be used as an immunotherapeutic agent for brain tumours and can bring a revolutionary change in medical field.

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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

HOW BACTERIAL LIPOPROTEINS INFLUENCE OUR IMMUNE SYSTEM

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Bacterial lipoproteins (Lpp) belong to the class of lipid-anchored proteins that are in Gram-negative bacteria anchored both in the cytoplasmic and the outer membrane and in Gram-positive bacteria only in the cytoplasmic membrane. In contrast to Gram-negative bacteria, lipoprotein maturation and processing is not vital in Gram-positive bacteria, however, they play an important physiological role, in nutrient and ion acquisition allowing particularly pathogenic species to better survive in the host. In Gram-positive bacteria Lpp represent the major protein group of the surfacome. They also represent important MAMPs (microbe-associated molecular patterns) by alerting our immune system via interaction with TLR2 (Toll-like receptor 2). More recently it has been shown that the lipid structure of Lpp has a profound influence on the intensity of our immune response. In commensal staphylococcal species Lpp carry a long-chain N-acyl group, while non-commensal species carry only N-acetylated lipid moiety. While the non-commensal species and their isolated Lpp induce a fulminant immune reaction the commensal species rather lulls our immune system. These findings confirm our hypothesis that successful pathogenic bacteria but also harmless commensal bacteria can only survive in the host when they manage to escape or evade the immune defense system. There are two main strategies of bacteria to circumvent the immune system. One is directed against phagocytes including inhibition of chemotaxis or phagocytosis or colonization of phagocytes. The other strategy is directed against the innate and adaptive immunity such as avoiding to evoke an immune response.

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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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DOI: 10.21767/2471-304X-C1-009

BRINGING TO WIDESPREAD PUBLIC USE LATEST TECHNOLOGIES AND INNOVATIONS IN VACCINES AND IMMUNIZATION: A POINT OF VIEW FROM A DEVELOPING COUNTRY, MOZAMBIQUE

Helder Fernando Brígido MARTINS
Public Health Planning and Management

Despite the generalized consensus that most effective technologies are useless if not widely used, between the discovery of the various effective vaccines and their widespread use it has always elapsed a long time. In the past, among others, two main factors have justified this long gap of time: widespread use of vaccines implies a complex managerial and logistic delivery system; and the introduction of new vaccines imply costs. Money is not always available, particularly in developing countries. In 1974, the EPI programme approved by WHO and recommended to be implemented all over the world, was a gigantic step forward to create the managerial and logistic system to deliver vaccines. At same time, a new era of vaccine promotion started, to raise conscience of vaccines as one of the most cost/effective measures in public health. However, even after the EPI was widely adopted all over the world, there has been always a considerable period, between the discovery of new vaccines and their introduction in EPI national programmes. In recent years, a lot of progress had been made in vaccine research and development and a great number of new vaccines were approved for public use and some are still on the pipeline. However, the time between the approval of a vaccine for public use and its effective widespread use is still much more than the desirable and there still are a lot of underutilized vaccines. To complicate the situation, in the last 10 to 20 years, an unexplainable anti-vaccine lobby has been very active, involving many Medical Doctors, to discourage the general public to use vaccines. On the other side, the paradigm of the original EPI programme was based on vaccines for children and women in the fertile age. This was understandable, because, at the time, the important task was to address priority problems. In the last 20 to 30 years, the conscience of the health professionals has moved to realize that there are also very useful and effective vaccines for teenagers, elderly people and adults that should not remain underutilized. Consequently, the EPI paradigm has changed, to include a much bigger number of vaccines. With well-established EPI Programmes in almost all countries of the world, the managerial and logistic delivery system problems are solved (or at least, they are not very constringent any more) and the financial constraints to bring to widespread public use an increasing number of vaccines became the main issue, but not the only one. Therefore, nowadays, the great challenge is how to reduce the time gap between the approval of a vaccine for public use and its effective widespread use. In this paper, the author uses his wide technical and managerial skills and experience to present suggestion on how to minimize this problem.

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EuroSciCon

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

APPLICATION OF THE PRINCIPAL COMPONENTS ANALYSIS FOR THE ASSESSMENT OF THE RELIEF RATE AFTER THE INITIATION OF CHEMOTHERAPY IN CAMEROONIAN KAPOSI SARCOMA'S PATIENTS

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Adriblastin-Bleomycin-Vincristin (ABV) chemotherapy has improved the survival of Kaposi Sarcoma (KS) infected people. In human herpes virus-8 (HHV-8, the causative agent of KS) endemic areas, the immune system is in the constant rate of activation, leading to a chronic immune activation against the virus. This chronic activation can leads to immune cells progressive depletion as T-lymphocytes and/or to inflammatory reaction and represent a major concern in patient care. However, many markers can measure those dysfunctions and they are often used without accounting for their possible interdependency. In the order to better understand the impact of this chemotherapy on the dynamics of immune activation and associated inflammation markers in Kaposi Sarcoma patients, the use of PCA for such task could be beneficial. In this view, four markers such as CD4 T-lymphocytes, IgG, Il-6 and Il-10 from the analysis of blood samples, collected between 1st Jul' 2014 and 31st Dec' 2015, on a total of 52 SK patients at the Yaoundé General Hospital, were analyzed. KS advanced stages and progressive response variables were associated to abnormal biomarkers levels post ABV therapy. At the end of this work, the ABV chemotherapy impacts on CD4 evolution and the persistence of biomarkers abnormal levels post treatment were observed. These results suggested that chemotherapy was not efficient to rise up immune activation and inflammation biomarkers to normal level.

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16th EuroSciCon Conference on
Immunology

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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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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Immunology

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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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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Immunology

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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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

16th EuroSciCon Conference on
Immunology

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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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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16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

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

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

MAST CELLS' RESPONSES TO ALLERGEN CHALLENGE AND THEIR MODULATION

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The quest for novel therapeutic interventions becomes important due to increase in allergic and inflammatory disorders worldwide including developing countries like India. Allergic reactions predominantly are mediated by activation and degranulation of mast cells through cross-linking of their high affinity surface receptors FcεRI on binding of IgE and allergen. Mast cell activation leads to release of early phase pre-stored mediators and late phase newly synthesized cytokines. We explored if mast cells can be trained to become tolerant of allergen. RBL-2H3 mast cell line and primary bone marrow mast cells were used to study mast cell secondary responses to allergens and their modulation. For this study, cells were first sensitized with DNP-BSA specific IgE and treated with different combinations of DNP-BSA to mimic allergen challenge *in vitro*. Cells were further sensitized and treated with IgE and DNP-BSA for a secondary challenge. β-hexosaminidase release and cytokine expression was analysed after each allergen challenge. Surface receptor expression was also analysed. The β-hexosaminidase release was significantly decreased after secondary challenge. Secondary challenge also resulted in reduced pro-inflammatory cytokine expression at mRNA level. Further, use of inhibitors revealed that various signalling pathways and histone modifications are involved in such modulations. Our study revealed the mast cell responses can be modulated in vitro after a primary challenge, to become tolerant or less responsive to the next allergen challenge.

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EuroSciCon

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
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J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

LNCRNA TMEVPG1 EXPRESSION AND IFN γ CORRELATES WITH HIGHER DISEASE SEVERITY AND OCCLUSION RATE ACCORDING TO GENSINI SCORE IN PATIENTS WITH CORONARY ARTERY DISEASE

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Background: In this study, we investigated the associations of circulating long non-coding RNA (lncRNA) Tmevpg1, transcription factor T-bet and IFN-γ cytokine relative expressions with severity and occlusion rate of coronary artery disease (CAD) patients.

Methods: Sixty-four patients suspected of CAD who underwent coronary angiography were consecutively enrolled in this study and divided into four group CAD 2, 3, 4 patients (N = 48) and CAD1 or controls (N=16) according to coronary angiographic results and Gensini Score. Blood samples of all participants were collected. Quantitative polymerase chain reaction (qPCR) was used to measure lncRNA Tmevpg1, transcription factor T-bet and IFNG cytokine expressions in whole blood samples. Serum interferon gamma (IFN-γ) was evaluated using enzyme-linked immunosorbent assay (ELISA). Gensini Score was used to assess the disease intensify and occlusion rate of CAD patients.

Results: LncRNA Tmevpg1 relative expression in CAD patients was upregulated compared with that in controls (P<0.003). LncRNA Tmevpg1 relative expression was remarkably associated with Gensini Score (P<0.01). Additionally, IFNG expression and IFN-γ serum levels were significantly increased (P<0.001), while, T-bet expression was not significantly increased. (P=0.06).

Conclusion: lncRNA Tmevpg1 and INF-γ expression correlated with higher disease severity, elevated inflammation and occlusion rate in CAD patientS.

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EuroSciCon

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

DISTURBED GENE EXPRESSION OF TLR NEGATIVE REGULATORS IN XLA PATIENTS

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X-linked Agammaglobulinemia (XLA) is a prototype primary antibody deficiency which is the most common form of primary immunodeficiency diseases. Mutated BTK in these patients affect many immune cells, immunologic responses and molecular interactions. TLRs, in a close interaction with BTK, reported being defected in different subcellular populations of PBMCs of XLA patients. In this concern, we aimed to assess LPS and CpG-A stimulatory action on TLR4 and TLR9 by measuring the activation of some TLR negative regulatory molecules' transcription and cytokine production. Higher transcripts of SOCS1 and RNF216 were found in unstimulated PBMCs of patients. Despite this, interesting patterns of TLR-induced transcription were observed: upregulation of IRAKM and SOCS1 in healthy subjects but downregulation in XLA, lack of RNF216 induction in healthy subjects while downregulation in patients and similar TNFAIP3 downregulation in both XLA and healthy subjects. Further, a lower amount of TNF-α was also produced by XLA patients PBMCs after LPS stimulation by disturbed cytokine production and dysregulated transcription of selected downstream signalling molecules. Our results strengthen the potential TLRs defect pointing out TLR involvement in the pathogenesis of different complications of XLA patients and also the scale of this defectiveness form TLRs expression to downstream signalling and cytokine production.

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EuroSciCon

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

MOLECULAR REGULATION OF INFLAMMATORY SIGNALING IN CHRONIC RHEUMATIC DISEASES

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Systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) are chronic autoimmune inflammatory diseases that predominantly effect women. RA primarily affects the synovial joints where infiltrating immune cells release inflammatory cytokines such as tumour necrosis factor (TNF) and interleukin-1 (IL-1). This leads to the destruction of cartilage and bone, whilst the synovial membrane becomes thickened due to cell infiltration and proliferation. The disease is both rapid and progressive, causing irreversible damage to the joints. SLE is a more systemic autoimmune disease with damage occurring to many tissues throughout the body. Symptoms can include arthritis, nephritis, neurological disorders and dermatological problems, with patients experiencing varying combinations of symptoms leading to a range of disease severity. Overall, SLE presents as one of the most clinically diverse autoimmune diseases, making diagnosis and treatment highly challenging. In both diseases, a family of innate immune receptors the toll-like receptors (TLRs) have been proposed to contribute to the pathogenesis. TLRs recognise both pathogens and damage associated molecular patterns resulting in inflammatory cytokine production. Using primary human tissue and blood samples from rheumatology patients our research has demonstrated potential roles for individual TLRs. Most recently, our work has focused on the molecular regulation of the downstream TLR signalling pathways in patient monocytes. This has led to the identification of several negative regulators of signalling that are altered in either RA or SLE compared to healthy volunteers. Changes to the function of these regulators have the potential to perpetuate inflammatory cytokine production in these patients. Understanding which receptors and signalling molecules are contributing to the production of excessive inflammation in these diseases is of great importance, as this knowledge will provide targets for the development of future therapies.

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EuroSciCon

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

DIRECT EVIDENCE OF VIRAL INFECTION AND MITOCHONDRIAL ALTERATIONS IN THE BRAIN OF FETUSES AT HIGH RISK FOR SCHIZOPHRENIA

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There is increasing evidences that favor the prenatal beginning of schizophrenia. These evidences point toward intra-uterine environmental factors that act specifically during the second pregnancy trimester causing a direct damage to the brain of the fetus. The current available technology doesn't allow observing what is happening at cellular level since the human brain is not exposed to a direct analysis in that stage of the life in subjects at high risk of developing schizophrenia.

Methods: **In 1977, we began a direct electron microscopic research of the brain of fetuses at high risk from schizophrenic mothers in order to find differences at cellular level in relation to controls.**

Results: **In these studies, we have observed the presence of complete and incomplete viral particles within the nuclei of neurons, that reacted in positive form with antibodies to herpes simplex hominis type I [HSV1] virus, and mitochondria alterations.**

Conclusion: **The importance of these findings can have practical applications in the prevention of the illness keeping in mind its direct relation to the aetiology and physiopathology of schizophrenia. A study of the gametes or the amniotic fluid cells in women at risk of having a schizophrenic offspring is considered. Of being observed the same alterations that those observed previously in the cells of the brain of the studied foetuses, it would intend to these women in risk of having a schizophrenia descendant. Previous information of the results, the voluntary medical interruption of the pregnancy or an early anti HSV1 viral treatment as preventive measure of the later development of the illness.**

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EuroSciCon

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

BIFUNCTIONAL PEPTIDE INHIBITORS FOR CONTROLLING AUTOIMMUNE DISEASES

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Bifunctional peptide inhibitors (BPI) are conjugates between antigenic peptides and cell adhesion peptides or protein. The hypothesis is that BPI molecules simultaneously bind to MHC-II and ICAM-1 on antigen-presenting cells (APC) to inhibit the formation of the immuno¬logical synapse at the interface between T cells and APC followed. Therefore, this inhibition induces selective alteration of T-cell differentiation from inflammatory to regulatory responses. Our results showed that BPI molecules suppressed experimental autoimmune encephalomyelitis (EAE) disease in mice significantly better than antigenic peptide (i.e., PLP peptide) or PBS. BPI molecules have been shown to suppress rheumatoid arthritis in collagen-induced arthritis mice significantly better than antigenic peptide (i.e., Collagen-II peptide) or PBS. In EAE mice, BPI molecules suppressed the production of inflammatory cytokines and induced the production of regulatory and/or suppressor cytokines. We have also shown that BPI molecules suppressed EAE in antigen specific manner. Currently, we are working on understanding the mechanisms of action of BPI molecules in suppressing autoimmune diseases in animal models.

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EuroSciCon

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

THE HUMAN TOPONOME PROJECT: TRANSLATING THE SPATIAL PROTEIN NETWORK CODE INTO EFFICIENT THERAPIES

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Imaging cycler® technology (IC®M) is presented as key technology: for the spatial resolution of large protein networks at the target sites of disease with a discriminatory power for an unlimited number or proteins at a time (dimension unlimited imaging); for the in situ detection of thousands of distinct multi protein complexes; for the construction of machines able to decode the mechanism of cell invasion into organs, such as the invasion of autoimmune cells and cancer cells; for the application of this technology for the efficient finding of therapies selectively blocking these invasions. The example of amyotrophic lateral sclerosis (ALS) is presented showing that ALS cells were seen by IC® for the first time in the blood, the mechanism of CNS invasion and pathogenic neuronal axotomy of these cells was completely decoded by IC®, and these ALS cells were efficiently depleted in blood of patients. This ALS example can be translated for other diseases based on cell invasion. The IC® detection of somatotopic coding in the innate immune system is key.

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EuroSciCon

16th EuroSciCon Conference on
Immunology

March 11-12, 2019
Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

HOUTTUYNIA CORDATA SHOWS POTENT ANTI-HIV-1 INFECTION IN VITRO

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[5]Princess of Naradhiwas University, Thailand

Objective: HIV-1/AIDS therapy remains concerned problems due to side effects of highly active antiretroviral therapy. Thus, seeking of alternative strategies for HIV-1 inhibition is still needed. *Houttuynia cordata* Thunb (*H. cordata*) is a plant that possesses several antimicrobial activities. This study aimed to investigate the cytotoxicity of *H. cordata* extract on CD4+ lymphocytes; the anti-HIV-1 activity of the extract on $CD4^+$ lymphocytes and the effects of the extract on anti-HIV-1 enzymatic activities.

Methods: Cytotoxicity of the extract was determined using MTT assay. To investigate anti-HIV-1 activity of *H. cordata*, C8166 cells were treated with the extract either before or after being exposed to HIV-1NPO3 and incubated for 72 h. The amounts of p24 antigen were detected by ELISA. Anti-HIV-1 integrase and protease activities of both the water and ethanolic extracts of *H. cordata* were determined using enzymatic assay.

Results: No cytotoxicity of *H. cordata* extract up to 400 µg/ml was observed. The extract showed potent anti-HIV-1 activity with 69% inhibition in pre-treated C8166 cells. However, HIV-1 replication was not significantly suppressed when the cells firstly exposed to the virus and then treated with the extract. *H. cordata* water extract possessed inhibitory effect against HIV-1 integrase while the ethanolic extract showed weakly inhibitory activity. Neither the water nor ethanolic extracts had anti-HIV-1 protease activity.

Conclusion: This study demonstrated that *H. cordata* can inhibit HIV-1 at the early stages of infection without cytotoxicity. These findings may lead to the development of a new effective anti-HIV-1 agent that is effective at the early stages of the infection.

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EuroSciCon

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Immunology

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Amsterdam, Netherlands

J Clin Immunol Allergy 2019, Volume:5
DOI: 10.21767/2471-304X-C1-009

DEVELOPMENT OF A NOVEL ADENOVIRUS-BASED CANCER IMMUNOTHERAPY THAT TARGETS THE SLAMF7 PATHWAY AND SUPPRESSES ESTABLISHED TUMORS

Yasser Ali Aldhamen, Yuliya Pepelyayeva, Patrick O Connell, Sean Thomas Hyslop, Maja Kuranz Blake, Cristiane Pereira Hicks, Sarah Godbehere Roosa and Andrea Amalfitano

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Objective: HIV-1/AIDS therapy remains concerned problems due to side effects of highly active antiretroviral therapy. Thus, seeking of alternative strategies for HIV-1 inhibition is still needed. Houttuynia cordata Thunb (H. cordata) is a plant that possesses several antimicrobial activities. This study aimed to investigate the cytotoxicity of H. cordata extract on CD4+ lymphocytes; the anti-HIV-1 activity of the extract on $CD4^+$ lymphocytes and the effects of the extract on anti-HIV-1 enzymatic activities.

Methods: Cytotoxicity of the extract was determined using MTT assay. To investigate anti-HIV-1 activity of H. cordata, C8166 cells were treated with the extract either before or after being exposed to HIV-1NPO3 and incubated for 72 h. The amounts of p24 antigen were detected by ELISA. Anti-HIV-1 integrase and protease activities of both the water and ethanolic extracts of H. cordata were determined using enzymatic assay.

Results: No cytotoxicity of H. cordata extract up to 400 µg/ml was observed. The extract showed potent anti-HIV-1 activity with 69% inhibition in pre-treated C8166 cells. However, HIV-1 replication was not significantly suppressed when the cells firstly exposed to the virus and then treated with the extract. H. cordata water extract possessed inhibitory effect against HIV-1 integrase while the ethanolic extract showed weakly inhibitory activity. Neither the water nor ethanolic extracts had anti-HIV-1 protease activity.

Conclusion: This study demonstrated that H. cordata can inhibit HIV-1 at the early stages of infection without cytotoxicity. These findings may lead to the development of a new effective anti-HIV-1 agent that is effective at the early stages of the infection.

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