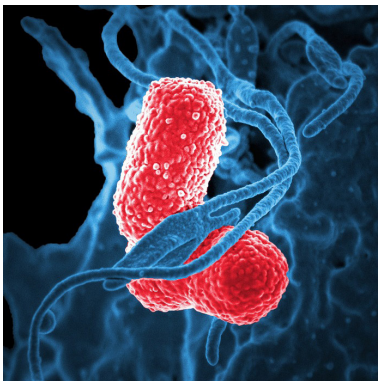
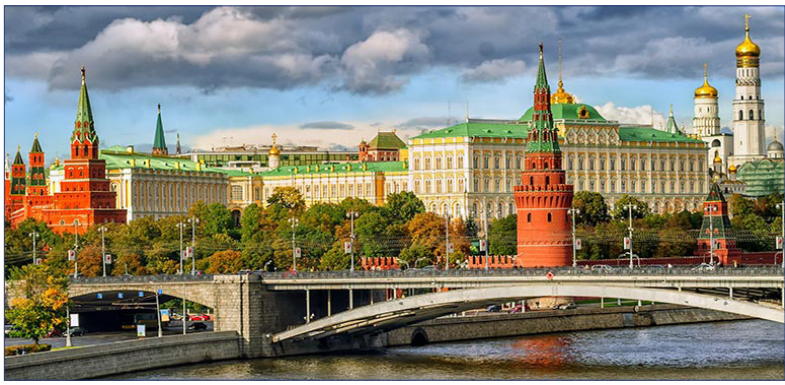
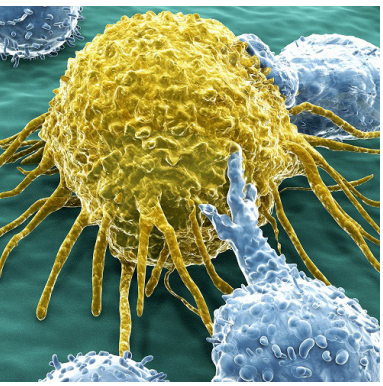
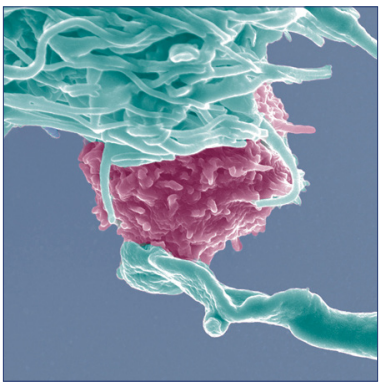
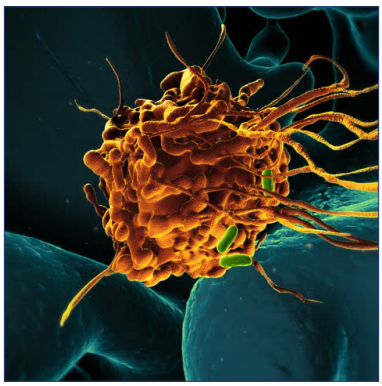
Poster Presentation

Immunology Congress 2018

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Outbreak of Yellow Fever in Brazil: Clinical considerations, diagnosis and its implications

Patricia Fabrini, Marco Fabrini Araujo and **Lorran Fabrini Araujo**
Santhè Clinic, Brazil

Yellow fever (YF), a mosquito-borne viral hemorrhagic fever, is one of the most lethal viral diseases.YF first registers in Brazil dated in 1685. The disease was erradicated in 1942. Virus transmission is highest during the rainy season (January-March) in Brazil , when the number of insects that transmict the disease is high.It is characterized by a rapid evolution and hepatitis, renal failure, hemorrhage, shock, and death; the mortality rate is 20%-50%.The diagnosis is based on viral Antigens detection or when antibodies titles are higher than 4X. To prevent the disease, prophylactic measures and vaccination must be highlighted.The vacination can be iniciated by nine months of age. Last studies have shown that the immunization in adults can provide permanent immunization.To control the epidemics, precaution against exposure to vector mosquitoes,as a continuous ritual and the proper use of 17D vaccine to prevent infection in travelers must be highlighted.Including the verification of vaccination card in the airports, while checking passports must restrict YF in the world .In Brazil, vacination and continued prophylatic measures can erradicated, in the a near future, this disease again.

Speaker Biography

Patrícia Fabrini helds a infusional center in Belo Horizonte , Minas Gerais, Brazil. She also works as a dermatologist in Santa Casa , a great hospital in the city.She is nowadays responsible for the infusion of many of the patients diagnosed with immunomediated diseases, like Psoriaisis, Arthritis, Crohn's disease and Ankyloosing Spondyhlitis where patients with psoriasis and arthritis receive immunobiological treatment.

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High CD8 cell percentage and HCV control in HIV-1 controllers and HTLV-2 coinfected patients

Alejandro Vallejo
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Natural control of HIV-1 infection occurs in less than 1% of patients, maintaining very low plasma HIV-1 RNA loads or even below the limit of detection, and usually with no clinical signs of disease progression for many years without any antiretroviral treatment.

HTLV-2/HIV-1 co-infection is found with relatively high frequency among injection drug users in North America and Western Europe. These patients have been reported to have lower levels of plasma HIV-1 RNA loads before antiretroviral treatment, and slower decrease of CD4 T cell counts. These two groups of patients show an immune capacity that enables a certain control of viral infections, dramatic control of HIV-1 replication in the case of controllers. The aim of this study was to compare viral and immunologic parameters between HIV-1 controllers (N=75), HTLV-2/HIV-1 chronic progressors (N=57), and HIV-1 chronic progressors (N=182).

Speaker Biography

Alejandro Vallejo is a biologist and completed his PhD at Complutense University, Madrid, Spain. One of his fields of research is the study of immune parameters of HTLV infections among HIV-1 infected patients. He moved to the Laboratory of Molecular Virology, CBER, Food and Drug Administration, Bethesda, MD, USA, as a Post-Doctoral Fellow (1995-2000) and developed several works on molecular epidemiology of HTLV and HIV, and viral tropism. Then he joined the Immunovirology Laboratory at the Virgen del Rocío University Hospital, Seville, Spain, as an independent researcher (2000-2008). He focused his research on immune recovery of HIV-1-infected patients. Then he moved to Ramon y Cajal University Hospital in Madrid to follow his research on HTLV-1/2 immunopathology and continuing the research on HTLV-1/2 infections (2008) running the Laboratory of Molecular Virology within the Infectious Diseases Department.

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Strategies to avoid lack of response of Immunobiological drugs delivered subcutaneously

Patricia Fabrini, Marco Araujo, Luísa Coutinho Teixeira and **Marina Rodrigues Costa Lages**
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The present work aims to highlight different ways to prevent diminished or lack of response of immunobiological drugs delivered subcutaneously, therefore reducing the burden of treatment with immunobiological drugs for both patients and healthcare system. Etanercept is an immunobiological drug with anti-TNF action, applied subcutaneously, which is indicated in cases of psoriasis refractory to other treatments. The present study shows a possible lack of response to the treatment after consecutive applications in the same anatomical site.

Speaker Biography

Patrícia Fabrini helds an infusional center in Belo Horizonte, Minas Gerais, Brazil. She also works as a dermatologist in Santa Casa, a great hospital in the city.She is nowadays responsible for the infusion of many of the patients diagnosed with immunomediated diseases, like Psoriaisis, Arthritis, Crohn's disease and Ankyloosing Spondyhlitis where patients with psoriasis and arthritis receive immunobiological treatment.

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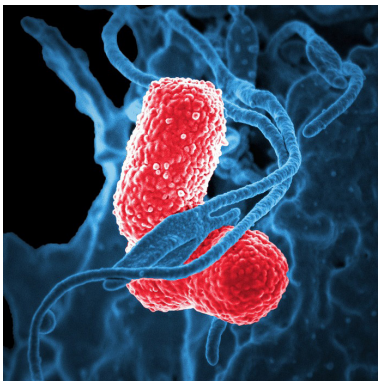
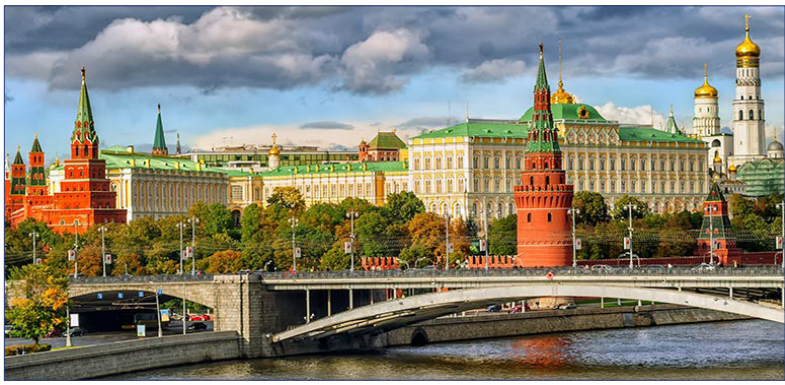
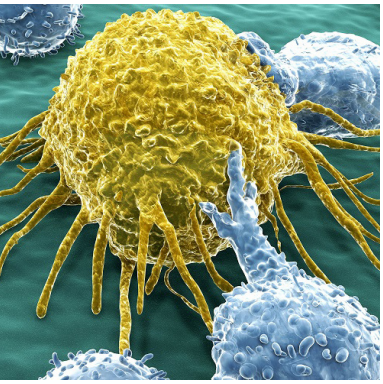
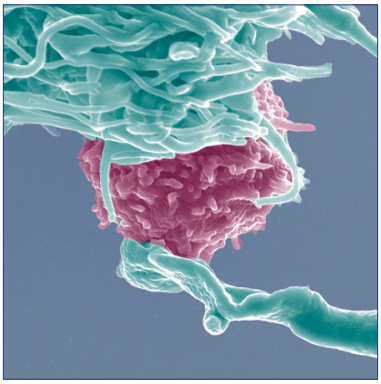
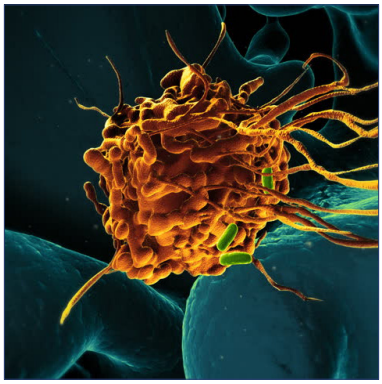

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A key to the backdoor into the castle: The clinical ramifications of immunoediting driven by antigenic competition

Michael G Hanna
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Over the last decade the field of cancer biology has gained considerable data on genomic heterogeneity. This situation creates challenges and possibly opportunities for cancer treatment. The evolution of the tumor at all stages also requires the growing malignancy to confront and avoid the immune system. What we describe here is the interaction of two immune phenomena that work together to change the characteristics of the tumor, i.e., antigenic competition and immune editing. These two systems are mutually functional, and their interaction is capable of altering the characteristics of the tumor for protection and survival in an immune competent host as well as restricting the diversity of the tumor clones. Therefore, the final outcome of these interactions can also become the key to the backdoor into the castle. Through an additional immune manipulation, autologous tumor cell immunization, we can achieve prevention of disease recurrence after surgical resection and by analyzing induced human monoclonal antibodies to the neoantigens, gain in site into the restriction of diversity of the mutant clones. These findings may also open the door for a pathway to immune prevention of cancer.

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The monogenic disorder called alpha-tryptasemia fosters our understanding of the biologic and pathobiologic roles of human α/β-tryptases

Lawrence B Schwartz
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α/β-Tryptases are preferentially expressed by mast cells in humans, where they serve as biomarkers for systemic anaphylaxis, systemic mastocytosis and mast cell cytoreductive therapies. Their biologic or pathobiologic clinically-relevant activities are less well understood. Recently, a monogenic autosomal-dominant condition called alpha-tryptasemia has been described due to a copy number variation in the gene encoding alpha-tryptase, TPSAB1. Affected patients present with multi-organ system defects, including autonomic dysfunction, joint hyper-extensibility, gastrointestinal symptoms and vibratory urticaria along with elevated baseline tryptase levels, but with a normal bone marrow biopsy and no c-kit mutation. As α-tryptase tetramers are ineffective as proteases due to a G245D change in which the aspartic acid side chain clogs the substrate binding pocket, essentially all tryptase proteolytic activity resides with β-tryptase, primarily via β-tryptase tetramers, because monomers, when active, unlike tetramers, are rapidly inactivated by biologic protease inhibitors. So how might alpha-tryptasemia provide new insights in the function of these tryptases? New research aiming to better understand the relationship between tryptases and the clinical phenotype of alpha-tryptasemia patients and how this might lead to better therapeutic options for such patients will be discussed.

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Anti-inflammatory activity and modulate oxidative stress of Bucida buceras in Lipopolysaccharide-Stimulated RAW 264.7 macrophages and Carrageenan-Induced acute paw edema in rats

Simon Bernard Iloki Assanga
Rubio Pharma y Asociados, Mexico

As oxi.dative stress is an important mediator that provoke or sustain inflammatory processes, we evaluated here the effects of Bucida buceras on inflammatory response in lipopolysaccharide (LPS)-stimulated RAW 264.7 cells and anti-inflammatory effect and redox biomarkers in carrageenan-induced paw edema in rats. In a continuous effort to find more potent, non-toxic natural product inhibitors that suppress inflammation, the present study was carried out to analyzed the influence of aqueous extract on NO, TNF-α, IL-6 and IL-1β in LPS-induced murine macrophages and paw thickness, NO, CPR, organoperoxide, oxidation protein and reducing power antioxidant in paw edema in rats. Treatment with Bucida buceras aqueous extract inhibited not only the protein (albumin) denaturation but also, in LPS-induced inflammatory response, including increased secretion of proinflammatory cytokines (IL-6 and IL-1β) and NO were inhibited by aqueous extract in a concentration-dependent manner. Furthermore, B. buceras suppressed significantly edema in a dose-dependent fashion in inflamed rat paws; decrease the C-reactive protein, lipid peroxidation levels (OT) and oxidation protein product and exerted strong reducing antioxidant power.

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Antiphospholipid antibodies: Clinical and diagnostic problem as an intriguing notion on Immunology

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Antiphospholipid syndrome (APS) is defined by clinical manifestations that include thrombosis and/or fetal loss or pregnancy morbidity in patients with antiphospholipid antibodies (APL). Diagnostic Problem: Antiphospholipid antibodies are among the most common causes of acquired thrombophilia, but unlike most of the genetic thrombophilias are associated with both venous and arterial thrombosis. Antiphospholipid antibodies are directed primarily toward phospholipid binding proteins rather than phospholipid per se, with the most common antigenic target being β2-glycoprotein 1 (β2GPI) although antibodies against other targets such as prothrombin are well described. Laboratory diagnosis of aPL depends upon the detection of a lupus anticoagulant (LA), which prolongs phospholipid-dependent anticoagulation tests, and/or anticardiolipin and anti-β2-glycoprotein 1 antibodies. Indefinite anticoagulation remains the mainstay of therapy for thrombotic APS, although new strategies that may improve outcomes are emerging. Clinical Problem: While the clinical presentation of APS can be quite diverse because the disease can affect virtually any organ system, patients typically present with symptoms relating to joint, skin or mucosal inflammation, or with a varying degree of haematological abnormality or constitutional features. However, the lack of a gold standard test to confirm diagnosis often results in delays or misdiagnosis.

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Innovative Vaccine Strategy against HSV Infections

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Herpes simplex virus type 1 and type 2 (HSV-1 and HSV-2) infections would be controlled by the development of an effective vaccine. However, in spite of several clinical trials, starting as early as 1920s, no vaccine has been proven sufficiently safe and efficient to warrant commercial development. Recently, great advances in cellular and molecular immunology understanding have stimulated creative approaches in controlling herpes infections and diseases. Before moving towards novel vaccine strategy, it is required to answer the important questions: (i) why past herpes vaccines were unsuccessful? (ii) Why the majority of HSV seropositive individuals naturally control HSV infections and exhibit few or no recurrent herpetic disease, while few others have frequent herpes clinical episodes? We recently discovered that HSV-1 symptomatic and asymptomatic individuals develop distinct immunity to viral epitopes recognized by CD4+ and CD8+ T cells. These epitopes (protective vs pathologic) have provided a solid foundation for the development of novel herpes epitope-based vaccine strategy. In this presentation, I will provide an overview of past clinical vaccine trials and outline current progress towards developing a new generation “asymptomatic” clinical herpes vaccines and discuss future mucosal “asymptomatic” prime-boost vaccines that could optimize the protective immunity.

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Electron microscopic studies of brain tissue in fetuses from schizophrenic mothers

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The neurodevelopmental theory in the aetiology of schizophrenia is considered one of the most consistent at present. Evidence from epidemiological and neuropathological studies indicates that the pathogenic process that culminate in the development of schizophrenia are initiated early in life and has been associated with a variety of prenatal environmental insults to the developing brain, including infection. Although the infectious agents have been proposed as one of the risk factors for schizophrenia the data on the association of a specific infectious agent with prenatal brain evidence is absent. Understanding of the structural abnormalities would allow a better identification of neurodevelopmental processes that contribute to risk for schizophrenia. We have hypothesized that at ultra-high-risk fetuses would have alterations at cellular level that would let us differentiate them to the comparison subjects. A reappraisal of our ultrastructural studies carried out in samples of the left temporal lobe of foetuses at ultra-high risk of developing schizophrenia is presented. The findings obtained are compatible with an active infection of the central nervous system by herpes simplex hominis type I [HSV1] virus. The present results are the first direct evidence that demonstrate the presence of this virus in the central nervous system of foetuses from schizophrenic mothers in the critical period of foetal development. The importance of this finding can have practical applications in the prevention of the illness keeping in mind its direct relation to the aetiology and physiopathology of schizophrenia.

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The World Incidence and Prevalence of Autoimmune Diseases is Increasing

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Epidemiological data provide evidence of a steady rise in autoimmune disease throughout Westernized societies over the last decades. Multiple publications exist, describing past or actual incidences/prevalence of individual autoimmune diseases, however, long term studies on selected populations are scarce. Aims: to calculate the % increases per year of autoimmune diseases frequencies worldwide, analyse the differential increases per country and disease, and identify epidemiological trends. Results: The means ± s.d. of the net % increased/year incidence and prevalence of autoimmune diseases worldwide were 19.1±43.1 and 12.5±7.9, respectively. Rheumatic, endocrinological, gastrointestinal and neurological autoimmune diseases revealed the following annual % increases per year: 7.1, 6.3, 6.2, and 3.7, respectively. In all of these, differences between old vs new frequencies were highly significant ($p<0.0001$). Comparing various autoimmune diseases, celiac disease increased the most and the highest increase in incidence, comparing old to new surveys is allocated to myasthenia gravis. Despite considerable variations between the countries, celiac, type 1 diabetes and myasthenia gravis frequencies increased the most in Canada, Israel and Denmark, respectively. Frequencies of the autoimmune diseases increased significantly in the West and North when compared to East and South, respectively. Conclusions: Despite multiple reports on autoimmune diseases frequencies, long-term longitudinal follow-ups are scarce. Incidences and prevalence's have increased significantly over the last 30 years. Rheumatic, endocrinological and gastrointestinal autoimmune diseases in Israel, Netherlands, USA and Sweden increased the most. These observations point to a stronger influence of environmental factors as opposed to genetic factors on autoimmune disease development.

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New autoantibodies in spondyloarthritis

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Spondyloarthritis (SpA) is a common inflammatory disorder. because the lack of specific laboratory biomarker. The diagnosis of SpA is relying on clinical examination and x-ray of the spine which show characteristic spinal changes and sacroiliitis. However, abnormalities in x-ray often occur several years after the onset of the disease. Autoantibodies are widely used as biomarkers in many types of autoimmune diseases and other diseases. We used proteomic approach to screen new autoantigens in SpA patients. We detected one new autoantigen in SpA sera named as SPA1. Using ELISA technology, IgG autoantibody to SPA1 was found in 24.4% of the SpA patients. In the controls, the prevalence of the new autoantibody was 6.8% in health controls, 16.7% in systemic lupus erythematosus, 0% in psoriatic arthritis and gout. The titer of anti-SPA1 was close associated with disease activity of SpA. Antibody against SPA1 could provide an important additional tool for diagnosis of SpA.

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Sero-prevalence of Human and Bovine Brucellosis, and Molecular Detection of Brucella species in cattle in three selected pastoral districts of Borena zone, Southern Ethiopia East Africa

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Brucellosis is worldwide distributed disease with significant economic and zoonotic implication for the rural communities of developing countries. It is prevalent in Ethiopia in attributable to farmers and Pastoralist traditional life styles, feeding habits and disease patterns. Cross-sectional study was carried out in Borena zone Oromia regional state, southern Ethiopia to estimate prevalence of Bovine and human brucellosis; and molecularly detected Brucella species circulation in the area. Sero-epidemiological survey was applied on target population and a total of 503 and 161 sera samples were collected from cattle and human for serological analysis of brucellosis respectively. Structured questioner was also applied to assess associated risk factor of Brucellosis in Bovine and Human. Sera samples were serially tested by RBPT finally confirmed by c-ELISA. In this study, B.abortus detected from blood clot of cattle using molecular assay. This study revealed that human brucellosis prevalence was higher than animals. Prevalence of human brucellosis was 25.6% in pastoral association (PA) whereas 21.3% in hospital with febrile clinical sign. It also provides evidence of high importance of brucellosis in human and animal in the study area. In conclusion, existence of brucellosis, community daily practice, and uncontrolled movement of animals and livelihood nature of pastoralist suggests the need for farther investigation of brucellosis in human and animals to design/develop future prevention and control strategy of the disease in the area.

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Proteolytic enzymes in the pathogenesis of the influenza virus

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Deproteinization of a flu virus is necessary for its penetration into a cell and this occurs for the account of trypsin-like proteinases of the host's cell. We assumed that this enzyme has an important role in morphogenesis of flu virus and considerably defines its pathogenic and virulent properties.

Objective: to study the changes of proteinase and inhibitor activities in the development of influenzal infection at white mice previously infected with flu A virus.

Results: It has been being established that the level of trypsin-like proteinase and its inhibitor in the lungs and blood serum of not infected white mice were in balance at rather high level and did not change considerably during the whole period of supervision (6 days). At infection of white mice with virus of flu A/PR/8/34 (H1N1) there was a violation of proteinase-inhibitory balance. The most profound changes happened during the first hours after infection. There was the growth of proteinase activity and decrease of inhibitory activity. During the maximum accumulation of infectious titer of virus and its hemagglutinin, both proteinase and inhibitory activity was completely suppressed. The animals which didn't perish for 5-6 days increase of inhibitory activity and decrease in proteinase took place.

Conclusions: Increase of proteinase activity during the first hours after infection led to increase of infectious and hemagglutinating activity. The increase of inhibitory activity in 5-6 days after infection leads to some arresting of influenzal infection.

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The key to manufacturing viral vaccines for individual human population

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Nowadays, subunit viral vaccine becomes the major choice for manufacturing viral vaccine with a thought of safety reason to prevent side effects. However, the success to use subunit viral vaccine to prevent a particular viral infection is very limit. This is different from the time when Cowpox virus was originally used for vaccination to prevent the smallpox viral epidemic over a century ago. Although the knowledge of immunity has been discovered a lot more than the Edward Jenner's period, the effectiveness of viral vaccine could not reach our accomplishment. Accordingly, we need to revise our knowledge and manipulate in the right direction for the viral vaccine production. Basically, to induce an immunity to prevent a viral infection, our body must produce a specific antibody which needs induction not only by a particular viral antigen but also the molecules called major histocompatibility complex (MHC). Each molecule of MHC alleles plays a key role in the immune response by forming a specific complex with its appropriate epitope to induce a specific T cell clone thru its specific receptor. MHC class I is required for inducing cytotoxic T cell while MHC class II is for helper T cell. Helper T cell plays a key role to induce an effective stage of acquired immunity especially a specific antibody which is believed to be a gearwheel to prevent an invasion of the particular viral particle. To produce the viral-specific antibody, MHC class II plays a key role to induce helper T cell and then B cell to synthesize a specific antibody. Since the MHC gene alleles are highly polymorphic so the possibility that individuals have the same gene alleles might be one in a million which, mostly, can be found in those who are an identical twin. Accordingly, a subunit viral vaccine, which contains a limit number of epitopes, would reduce a capacity of an antigen presenting cell, such as a dendritic cell, to process some epitopes to induce the particular helper T cell clones. Subsequently, the corresponding B cell clones cannot synthesize the specific antibody to neutralize the particular infectious viral particle. Accordingly, this presentation will present a different notion and principle to develop a viral vaccine for an individual human population.

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Neuroprotective, anti-inflammatory and immunomodulatory activities of *Ozoroa pulcherrima* and *Sida pilosa* extracts on murine model of neuroschistosomiasis

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Schistosomiasis (bilharziasis) is an infectious parasitic disease caused by blood flukes of the genus Schistosoma. Schistosomiasis is an important public health problem in Africa. After malaria, it is the second most prevalent tropical disease, affecting at least 258 million people worldwide and 90 % in Africa (WHO, 2017). The eggs released by the adult female worm are mainly responsible to the pathology where they are deposited in the liver, intestine, uro-genital or Central nervous system (CNS). The most severe clinical outcome associated with this parasite is the infection of the central nervous system (CNS) known as neuroschistosomiasis (NSM) and can affect the brain or the spinal cord occurring during all phases of schistosomiasis and resulting to severe complications. Chronic neuroschistosomiasis results from the host's immune response to the eggs and the resultant granulomatous reaction and fibro-obstructive disease. Once deposited into CNS, the mature embryo secretes immunogenic substances that causing inflammatory reaction leading to a periovular granulomatous reaction. In the early phase of schistosomiasis (the first 110 days) the immune response reaches maximum intensity (Pittella, 1997; Ferrari, 2008). The granulomas successfully destroy the ova but result in fibrotic deposition in the host tissue. The mass effect of thousands of eggs and the large granulomas concentrated within the brain or spinal cord leads to symptoms such as headache, focal or generalized seizures, ataxia, nystagmus, nausea and vomiting, intracranial hypertension and neurological deficit.

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Differential modulation of immune functions by food borne mycotoxins

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Mycotoxins are structurally diverse toxic secondary metabolites produced by the organisms of the Fungus kingdom. Citrinin (CTN) was first isolated as a secondary metabolite of Penicillium citrinum and can cause mycotoxic nephropathy, cytotoxicity and genotoxicity. Deoxynivalenol (DON) is a secondary metabolite produced by Fusarium fungi and a contaminate in wheat, barley and corn worldwide. DON has been implicated in inducing dysregulation of the immune response and is able to either enhance or suppress resistance to pathogens. Zearalenone (ZEN) is a non-steroidal estrogenic mycotoxin produced by various Fusarium species. ZEN is mainly known as a hormonal disrupter due to its estrogenic activities and consequent toxicity for reproduction. ZEN also displays hepatotoxicity, immunotoxicity and nephrotoxicity. Due to the widespread presence of fungi in the environment, CTN, DON and ZEN are regarded as an unavoidable contaminant in food products. However, the immunomodulatory effects of CTN, DON and ZEN in mice have not been yet fully elucidated. In the present study, we have investigated the immune modulatory effects of CTN, DON and ZEN in the female BALB/c mice.

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Adenosine and Adenosine Receptors in the Immunopathogenesis and Treatment of Cancers

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Identification of the precise mechanisms behind the robust immunosuppression exerted by tumor cells can help us to design new therapeutic approaches for cancer therapy. The generation of adenosine is one of the main immunosuppressive mechanisms by which tumor cells not only inhibit anti-tumor responses, but also induce suppressive cells such as regulatory T cells (Treg). Two cell surface expressed molecules including CD73 and CD39 catalyze the generation of adenosine from adenosine triphosphate (ATP). The generation of adenosine can be enhanced under metabolic stress like tumor hypoxic conditions. Adenosine exerts its immunoregulatory functions through four identified adenosine receptors (ARs) including A1, A2A, A2B and A3 which are expressed on various immune cells. So, blocking the adenosine generating enzymes or ARs can be considered as an important therapeutic approach for cancer therapy. It is demonstrated that signalling of A2A receptor (A2AR) and A2BR in the tumor microenvironment can lead to induction and expansion of immunosuppressive cells such as Treg and MDSC. On the other hand, reports regarding the effect of A1R and A3R signalling in tumor biology are controversial. It seems that tumor promoting or tumor limiting effects of these two receptors depend on the tumor type and tumor condition. Several ARs directed agonists and antagonists have been developed and used for treatment of various tumors. We think the use of these agents as monotherapy or in combination with other conventional cancer drugs may lead to promising outcome in near future.

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Molecular Biology of Oral Cancer and Role of Cyclin D1 in Oral Premalignant Lesions and Oral Squamous Cell Carcinoma – A review of original Study

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Carcinogenesis is a complex, multi-step process in which genetic events within signal transduction pathways governing normal cellular physiology are quantitatively or qualitatively altered (Vogelstein and Kinzler, 1993). The genetic basis of cancer is now well-established. Under normal conditions, these tightly controlled excitatory and inhibitory pathways regulate oral keratinocyte biology. Aberrant expression of the proto-oncogene epidermal growth factor receptor (EGFR)/c-erb1, members of the ras family, as well as cmyc, int-2, HST, Cyclin- D1, and bcl-1, is believed to contribute to oral cancer development. Cyclin D1 is a protein derived from PRAD1 or CCND1 or Bcl-1gene located on chromosome 11q13 and it acts as a positive regulator of the cell cycle in normal cells as well as in neoplasia. Over expression of cyclin D1 may lead to shortening of G1 phase, increased cell proliferation and reduced dependency on growth factors. Our immunohistochemical study results showed that the alteration of cyclin D1 is frequent in oral precancer and oral squamous cell carcinoma. Expression of cyclin D1 was significantly altered from oral epithelial dysplasia to oral squamous cell carcinoma indicating that over expression of cyclin D1 may be an early event in oral cancer development.

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Class IV semaphorin checkpoints regulate allergic asthma

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Two class IV semaphorins, Sema4A and Sema4D, belong to a family of neuron guidance proteins which were also found to be expressed and function in the immune system. They both act as immune checkpoints by either directly or indirectly regulating T cell activation. We defined the in vivo function of Sema4 molecules in allergic asthma using OVA challenges of corresponding semaphorin-deficient mice. We found that Sema4A and 4D molecules play the opposite roles in disease. Whereas Sema4A-/- mice demonstrated a selective increase in airway eosinophilia accompanied by bronchial epithelial cell hyperplasia as compared to WT mice, these asthma parameters were decreased in Sema4D-/- mice. The enhanced inflammatory response in Sema4A-/- mice was associated with a selective increase in bronchoalveolar lavage IL-13 content, augmented airway hyperreactivity (AHR), and lower Treg cell numbers. In contrast, lower Th2 cytokine levels and higher number of Treg cells were found in the lungs of Sema4D-/- mice, whereas AHR was not affected. Allergen-primed Sema4A-/- CD4+ T cells were more effective in transferring Th2 response to naive mice as compared with WT CD4+ T cells. T-cell proliferation and IL-13 productions were upregulated in $OVA_{323-339}$-restimulated Sema4A-/- cell cultures and downregulated in Sema4D-/- cultures as compared to similarly challenged WT cells. Generated bone marrow chimeras showed an equal importance of both lung-resident cell and inflammatory cell Sema4A expression in optimal disease regulation. These data provide a new insight into Sema4 biology and define Sema4 molecules as important regulators of Th2-driven lung pathophysiology and prospective targets for disease immunotherapy.

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