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NOVEL IMMUNE REGULATORY PROPERTIES OF NAD+ AND ITS BENEFITS IN DISEASE SCENARIOS

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It is well known that MHC-TCR activation following pathogen invasion dictates CD4+ T cell differentiation. More recently, a second mechanism involving TLRs and NLRs pathways have been shown to regulate CD4+ T cell differentiation as well. Both pathways require antigen presenting cells in particular dendritic cells (DCs). Moreover, CD4+ T cell fate is tightly regulated by cytokine milieu (produced by DCs) and major transcription factors that give rise to specific T helper subset (Th1, Th2, Th17 and regulatory T cells (Tregs)). Alterations in DC-mediated CD4+ T cell regulation pathway leads to a myriad of diseases including atopic disorders, autoimmune, primary immunodeficiency, infections and cancer. In our studies, we demonstrated that NAD+ regulates CD4+ T cell differentiation independently of cytokine milieu and well established transcription factors. It is well established that the transcription factor T-bet is critical for Th1 differentiation. Our results demonstrated that in the presence of NAD+, the frequency of T-bet−/− CD4+IFNγ+ T cells was twofold higher than wild-type CD4+ T cells cultured in conventional T helper 1 polarizing conditions. Moreover, we showed a robust and unique immunoregulatory property of NAD+ that are independent of CD4+CD25+Foxp3+ Tregs, a unique T cell lineage that is essential for maintaining immune tolerance and homeostasis. Finally, our findings indicate that following NAD+ administration MCs, exclusively, promote CD4+ T cell differentiation, both in absence of antigen and independently of major APCs. Moreover, we found that MCs mediated CD4+ T cell differentiation independently of MHC-II and TCR signaling machinery. Collectively, our study unravels a novel cellular and molecular pathway that regulates innate and adaptive immunity via MCs, exclusively. This untapped novel and distinct pathway may serve as an alternative to bypass certain inflammatory conditions and pave the way for novel therapeutic approaches in the context of autoimmune diseases, transplantation, primary immunodeficiencies and antimicrobial resistance.

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THE IMPORTANCE OF LEARNING IDENTIFICATION OF LARVAE AND ADULT MOSQUITOES

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Malaria is one of the most serious health problems facing the world today and is one of the most common diseases affecting humans. Malaria causes more than 400,000 deaths a year according to the World Health Organization (actually 429,000 deaths in 2016). Unfortunately, 90% of deaths occur in children under the age of five years and in Pregnant women. Female Anopheles Mosquitoes (some species) are the main vector for malaria disease transmission from the infected patients with malaria plasmodium parasites to the other healthy persons, so it is essential to learn more about these mosquitoes especially how to identifye the genus and the species of Anopheles both the mosquitoes and the larvae. A around the world there are about 460 species of Anopheles mosquitoes whereas only 60 species of them can transmit malaria disease so we must know and learn how to distunguish between the different species and determined the area of their presence to follow up the suitable procedures when a malaria case is being detected at area in which there is presence for species of Anopheles mosquitoes capable to transmit malaria disease, so this will assist to avoid the spread of the disease.

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GUT AND INTEGRATED PATHOPHYSIOLOGY OF IMMUNE RESPONSE; OBSERVATIONS FROM EXPERIMENTS IN ORAL TOLERANCE IN MICE AND THE RESPONSE ASSOCIATED WITH A MODEL OF METABOLIC SYNDROME SURGERY

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The gut mucosa is the place that most contact with foreign antigenic proteins occurs and forms with the immune system an integrated, dynamic and adaptive complex that has evolved to provide effective digestion and defense. The whole intestinal area is 100-fold larger than the skin, presents the largest amount of lymphoid tissue of the body and the more number of activated lymphocytes. The Peyer´s patches and the lamina propria of the gut present a very large number of T cells. Immunoglobulin production, especially IgA, that is the only antibody secreted by mucosal, offers the first protection to neonates. For the experiment with Oral Tolerance proposed an protocol in adult Swiss mice by oral administration of a recombinant dermo-necrotic toxin of brown spider Loxosceles intermedia (LiRecDT1) and its mutated form (LiRecDT1H12A) for three weeks. Our results demonstrated evidences of tolerance induction through decrease in IgG anti-dermonecrotic toxin levels, paw edema reduction and increased survey in 24h after challenge. All statistical analysis was performed using ANOVA following Bonferroni's pos hoc test. Related to bowel surgery readjustment we observed that the removal of the greater omentum decreases the secretion of cytokines, particularly IL-6, regressing other diseases associated with obesity such as bronchitis. In conclusion, the intestine can be considered the main immune organ of the body and this association between immunity and digestion begin prior to birth and mediate allergic responses and/or tolerance throughout the life of the individual.

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DISRUPTING THE NFAT-AP-1 TRANSCRIPTIONAL COMPLEX USING SMALL MOLECULES

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The physical interaction between the transcription factors NFAT and AP-1 is pivotal for both the effector immune response and for the exacerbated response that happens during autoimmune and inflammatory diseases. In the absence of AP-1, NFAT directs another program of gene expression, which resembles T cell tolerance, where the cells lose their effector function. We have screened ~200,000 small drug-like compounds using a FRET assay that allows identifying inhibitors of the NFAT-AP-1 complex on DNA. We identified 960 candidate inhibitors in the initial screen. 24 compounds were evaluated and one of them actually inhibits the *in vitro* assembly of the NFAT-AP-1 complex on DNA with no effect on the binding of NFAT or AP-1 individually to their consensus binding sites. This compound also inhibits the induction of cytokine genes that depend on NFAT-AP-1 interaction, such as IL2, but not of those regulated independently of NFAT-AP-1 cooperation, such as TNF. The differential effect on IL2 and TNF gene expression indicates that selective inhibition of NFAT-AP-1 complexes in preference to other NFAT transcriptional complexes may be achievable by small molecules. One caveat is that further experiments have shown that this compound binds directly to DNA and not to the interface between NFAT and AP-1 as desired. We are currently developing an ELISA assay to pinpoint inhibitors that bind at the NFAT-AP-1 interface, and plan to re-test the other 936 compounds identified in the initial high-throughput screen. A proper inhibitor targeting NFAT-AP-1 complexes might redirect T cell transcription from an effector program to a tolerance program, and might find practical applications in the treatment of autoimmune and inflammatory diseases.

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2005-2007 GLOBAL SPREAD OF H5N1: CAUSATIVE ROLE OF BODY BURDEN DIOXIN IN UP-REGULATING GENE OF INFLUENZA VIRUS NS1 PROTEIN IN CHICKEN AND HUMANS IN SOUTHEAST ASIA

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Agonist-induced recognition of a cognate DNA enhancer dioxin responsive element (DRE) does epitomize wide range of mammalian genes expression mediated via the Ah receptor pathway. The same was postulated also for viral DRE-containing genes expression caused by 2,3,7,8-TCDD (dioxin) in infected human cells. In this study, such mechanistic concept applied to type A influenza virus nonstructural protein 1 binding protein (NS1BP) induction in humans and chicken. The data are presented at genetic, cellular, and population levels. Primers for mutation analysis were constructed for two DRE identified within enhancer region of the IVNS1ABP gene. Treatment of HeLa cell line with 0.1 nM of dioxin resulted in substantial increase of NS1BP protein level. This might add to influenza virus A non-structural protein 1 (NS1) inhibitory effect on cellular interferons, which determines antiviral resistance of emerging H5N1 virus. 2005-2007 H5N1 outbreaks among poultry in China and Vietnam might partially relate to chicken NS1BP, as outbreaks occurred in areas highly contaminated by dioxin-like compounds. Minimal dose of TCDD upregulating human IVNS1ABP gene was estimated moderately above current TCDD blood level in general population. So, in human groups in Southeast Asia exposed to TCDD, its body burden might facilitate spreading of H5N1 if avian flu pandemic were to occur.

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THE NEW DOCTRINE OF ACUTE PNEUMONIA AND THE FIRST RESULTS OF PATHOGENETIC APPROACHES TO TREATMENT

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Treatment of acute pneumonia (AP) in recent decades focused solely on antibiotic therapy,does not include pathogenetic, specific methods of assistance and repeats the principles of treatment of other inflammatory diseases. Reducing the effectiveness of antimicrobial drugs,the emergence and the increasing number of antibiotic-resistant pathogens and a gradual increase in the frequency of purulent complications attach importance and urgency to the solution of this problem. The first step in this decision is a revision of ideas about the nature and mechanisms of AP. This work has been done and tested in a clinical setting in the years 1976-1984 in Novokuznetsk State Institute for postgraduate doctors (USSR, Russia). The basis of the new doctrine AP was based on the following scientific medical axioms, already having previous scientific justification: The body's response to any stimulus, including the initiation of inflammation, is highly individual and unique; the basis for the inflammatory transformation of the body tissue is a vascular reaction with a specific stage sequence; small and big circles of blood circulation not only have a direct relationship, but an inverse relationship; among the nonspecific forms of inflammation, AP is the only process occurring in the system of lesser circulation and the same medical procedure can have different effects on inflammation in the small or big circles of blood circulation. Following private studies were additionally performed: Experimental model of AP(4 series of experiments, 44 animals) obtaining a model of pleural complications; X-ray examination 56 lung anatomical preparations with different forms of the AP, taken from the dead patients; record comparative rheopulmonography before and after performing medical procedures (36 patients) and analysis of the observation and treatment of 994 children with AP and its various destructive and pleural complications. The revised treatment guidelines were applied in 101 patients in the initial period of aggressive forms of AP. The received results allow to speak about possibility of the guaranteed prevention of suppurative and destructive complications of the disease.

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FIFTY SHADES OF STRESS: A COLOURFUL SCREENING PLATFORM FOR DETECTING BACTERIAL CELL STRESS

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Antimicrobial resistance poses a serious threat to global public health. The number of drug-resistant pathogenic bacterial strains is rising at an alarming rate, while only a handful of new marketable antibiotics have been introduced in the past three decades. Current drug discovery efforts suffer from tunnel vision: screening is limited to lethal compounds. This method overlooks a goldmine of compounds that do not directly kill the bacterial cell, but do induce stress. iGEM Leiden aims to develop an innovative screening platform, enabling rapid discovery of compounds which stress bacterial cells: Fifty Shades of Stress. This will allow high-throughput screening of compound libraries, which ultimately can be utilized to establish novel combination therapies. Fifty Shades of Stress will thereby help safeguard humanity against multi-drug resistant disease outbreaks.

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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 producing a direct damage of 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 finding differences at cellular level in relation to controls.

Results: In these studies we have observed within the nuclei of neurons the presence of complete and incomplete viral particles 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 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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FROM BASIC RESEARCH TO DRUG DEVELOPMENT: THE STORY OF COPAXONE (GLATIRAMER ACETATE) IN THE TREATMENT OF MULTIPLE SCLEROSIS AND POTENTIAL APPLICATIONS FOR ADDITIONAL PATHOLOGIES

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Multiple sclerosis (MS) is currently recognized as complex diseases in which inflammatory autoimmune reactivity in the central nervous system (CNS) results in demyelination, axonal and neuronal pathology. Treatment strategies aim to reduce the detrimental inflammation and induce neuroprotective repair processes. The synthetic copolymer Copaxone (glatiramer acetate, GA), an approved drug for the treatment of MS, is the first and so far the only therapeutic agent to have a copolymer as its active ingredient. Using the animal model of MS -experimental autoimmune encephalomyelitis (EAE), the immunomodulatory mechanism of action of GA was elucidated. It was found that GA treatment induces immunomodulatory shift from the inflammatory towards the anti-inflammatory pathways, such as Th2-cells that cross the blood brain barrier (BBB) and secrete *in situ* anti-inflammatory cytokines, as well as T-regulatory cells (Tregs) that suppress the disease. Furthermore, recent studies revealed neuroprotective and repair consequences of GA treatment in the CNS. These include elevation in neurotrophic factors expressions, remyelination and neurogenesis. Based on its immunomodulatory mode of action, additional potential applications of GA were investigated, such as prevention of immune rejection, improvement of stem cells engraftment and amelioration of inflammatory bowel diseases (IBD).

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THE POWER OF PROTEIN AND ANTIBODY ARRAYS IN STEM CELL RESEARCH

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Stem cells are uni-, multi- or pluripotent eukaryotic cell types which function to generate most of the basic body plan and tissues of a multicellular organism during development and even late into adulthood. The potency of stem cells is most often marked by the unique signature of proteins manufactured by these cells, which serves as a valuable tool for both cell-type identification and the discovery of new stem cell-based signaling cascades driving cell signaling, commitment and differentiation. These protein signatures may be rapidly and comprehensively characterized by the application of protein and antibody arrays.

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