Theme: Adaptive Immunity

Time: Thursday 7 June 2018
Location: CSS, Øster Farimagsgade 5, Copenhagen K, Room 7.0.34
Organizer: Graduate School of Immunology and Infectious Diseases
Chairman: Mogens Holst Nissen

13.00	Welcome Mogens Holst Nissen
13.10	Andrea Cossarizza, University of Modena and Reggio Emilia: Aging of the immune system: Focus on inflammation and vaccination
13.55	Helen L. Leavis: University Medical Center Utrecht, the Netherlands: Microbial dysbiosis in common variable immune deficiencies: Evidence, causes, and consequences
14.40	Paul Blair, University College London, UK: Regulatory B cells: origin, phenotype and function
15.25	Coffee Break
15.45	Gerhild Wildner, Department of Ophthalmology, University Hospital, LMU Munich, Germany: The immunopathogenesis of chronic and relapsing autoimmune uveitis – Lessons from experimental rat models
16.30	Dr. Chris Wincup, University College London, UK: The Pathogenesis and Treatment of Systemic Lupus Erythematosus, diagnosis and treatment options
17.15	Closing remarks

Abstracts: Adaptive Immunity

Aging of the immune system: Focus on inflammation and vaccination

Andrea Cossarizza,
University of Modena and Reggio Emilia, Italy

The increase in the human lifespan that is occurring in most countries is due to major advances in preventing (vaccination), delaying (life style), or curing infections and individual pathologies. It is indeed quite frequent to reach the age of 90 years or more in good shape, an age that is not likely predicted by evolution. All scientific advances cannot prevent or delay the underlying cause of those physiopathological changes that invariably occur with time, including aging itself. An impairment of the immune system is considered one of the main causes of the aging process of the entire organism, and indeed several studies have described pivotal immunological changes which occur with age. My talk will present main theories regarding the importance of the immune system during the aging process, and will pay a particular attention to the so called “inflammaging" as well as to the dysregulation of innate immunity, altered T-cell or B-cell maturation and differentiation, and to the implications of immune aging for vaccination strategies in the elderly.

Microbial dysbiosis in common variable immune deficiencies: Evidence, causes and consequences

Helen L. Leavis,
University Medical Center Utrecht, The Netherlands

Common variable immunodeficiency disorder (CVID) is the most common primary immunodeficiency, but its etiology is not well understood. This humoral immunodeficiency is often accompanied by immune dysregulation phenomena, including autoimmunity, granulomas and lymphoproliferation. One of the most prevalent and invalidating immune dysregulation phenomena in CVID is granulomatous lymphocytic interstitial lung disease (GL-ILD). Better understanding of the cause of CVID and the immune dysregulation phenotype is key to improving care for these patients. Previous data indicate signs of immune exhaustion, and a causal role of microbial gut translocation and dysregulation has been hypothesized. In a cross-sectional cohort of 100 patients and 49 controls we studied associations between oral microbiome, fecal microbiome and clinical parameters to substantiate this. Based on identified associated pro- and anti-inflammatory bacterial species we aim to study the effect on patient immune cells and gut epithelium in functional assays.

Regulatory B cells: Origin, phenotype and function

Paul Blair
University College London, UK

Regulatory B cells have now been identified in humans and a number of different animal models. When functional these cells can support immune tolerance by suppressing the activation of pathogenic T cells and other inflammatory lymphocytes. In autoimmune settings, such as lupus or

rheumatoid arthritis, regulatory B cells have been found to be dysfunctional or reduced in number possibly contributing to the persistence of disease, while in the context of cancer regulatory B cells may inhibit immune clearance of tumour cells. The exact nature of these cells is however still a matter of debate. In this talk I will discuss the origin of these cells, from which stage of development they arise and what signals are necessary for their generation, whether there is a reliable phenotype for these cells, and the multiple ways, in addition to IL-10 expression, by which they may suppress inflammation.

The immunopathogenesis of chronic and relapsing autoimmune uveitis - Lessons from experimental rat models

Gerhild Wildner,
Section of Immunobiology, Dept. of Ophthalmology, University Hospital, LMU Munich, Germany

Autoimmune diseases usually have a relapsing-remitting or a chronic progressive course, while the underlying immune mechanisms and regulation are not yet known. We have established two experimental rat models of an intraocular inflammatory autoimmune disease (uveitis). These two models are either spontaneously relapsing-remitting or monophasic/chronic. They are induced in the same strain of rats and differ only in the autoantigen peptides used for immunization. Adoptive transfer of autoreactive T cells specific for these peptides induced the same types of disease, therefore we analyzed the differences between the T cell lines with respect of gene and cytokine expression to elucidate the mechanisms behind the disease courses.

The Pathogenesis and Treatment of Systemic Lupus Erythematosus, diagnosis and treatment options

Dr. Chris Wincup
University College London, UK

Systemic lupus erythematosus (SLE) is an autoimmune connective tissue disease characterised by the production of auto-antibodies directed against nuclear components. Over the last 20 years, there have been significant advances in the recognition and treatment organ threatening manifestations of the disease and this has been associated with improvements in morbidity and mortality rates. However, in spite of these advances there are still areas in which little is known and these areas represent major unmet needs in the context of SLE.

Clinical trials in the field of SLE often fail to reach primary endpoints and thus pose various challenges in the search for new therapeutic targets. In addition, patients with SLE are also at increased risk of cardiovascular disease and again the reasons for this are not fully understood. Furthermore, patients with SLE are often troubled with high levels of debilitating fatigue although the causes of this also remains a mystery. This is further compounded by the fact that standard immunosuppressive treatments do little to alleviate this disability symptom.