highly suspected TB pleurisy), 8 pericardic effusions (2 from non TB origin, 6 from TB or highly suspected TB etiology), and 2 peritoneal effusions (1 TB and 1 control). The results obtained by flow cytometry were highly discriminatory between TB and non-TB patients, providing a high sensitivity for TB diagnosis within 24 hrs, whereas the microbiological cultures were positive only 4 to 6 weeks later.

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S.30. Eosinophilic Esophagitis and Atopic Dermatitis Treated with Omalizumab in a Patient with NEMO Defect

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Alterations in NEMO (Nuclear Factor Kappa Beta Essential Modulator) cause ectodermal dysplasia with immune deficiency (EDI), a disorder characterized by absent or reduced sweat glands and hair follicles with severe immune deficiency. Here we report a two-year old-male patient with a NEMO defect who presented with elevated IgE, atopic dermatitis, and food hypersensitivity with eosinophilic gastroenteritis. Recognizing that broad immune suppression may make the patient more susceptible to pyogenic bacteria and mycobacterial infection, we chose to treat the patient with twice monthly anti-IgE antibody therapy (omalizumab) for six months. The treatment goals included: improvement in his atopic dermatitis, introduction of additional solid foods, and improved growth. During the six-month treatment period, the patient's atopic dermatitis was well controlled with marked decrease in the use of topical corticosteroids. Nevertheless, we were unable to advance his diet significantly and he remained below the 3rd precentile for age on his weight-height curve. Subsequently, tube feeding with an elemental formula was initiated and his nutritional status improved significantly. To our knowledge, this is the first report of EDI with severe atopic symptoms. Anti-IgE therapy has a role in the clinical management of such patients but the primary treatment for food allergies remains the avoidance of food triggers.

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S.31. Increased Superoxide Dismutase and Decreased Glutathione Peroxidase Activity in Plasma and Their Immuhistochemical Localize in Tissues of Diabetic Patients

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Oxidative stress (OS) has been implicated in the aetiology and progression of pancreatitis and other complications including diabetes. In this study, the enzymatic antioxidant activities of superoxide dismutase (SOD) and glutathione peroxidase (GPx) and their immuhistochemical localize in tissues of patients with type 2 diabetes mellitus (DM) and patients with chronic pancreatitis (CP) were assessed and compared with apparently healthy normal subjects to understand the involvement of OS in the subjects. The above-mentioned OS markers were measured in 30 subjects for each of the following groups: DM, CP and non-diabetic control (NC). Significant elevated activities of SOD (29.42%) and GPx (31.17%) were found in CP subjects compared with control. GPx activity were significantly reduced (12.82%) in contrast with elevated activity of SOD (105.88%) observed in DM subjects compared to the NC subjects. Level of glucose in plasma correlated positively with GPx, whereas a negative correlation was observed for SOD in DM. In the normal human pancreas we found only scare acinar cells staining positively for SOD and GPx. In slices of the pancreas, derived from patients with CP, a much stronger for SOD and stronger for GPx immunohistochemical reaction was noticed as compared with normal subjects. Interestingly, immunostanig of both antioxidant was different in tissues of diabetic patients. Expression of SOD was estimated as strong or much strong in contrast expression of GPx was negative or weak. Increased oxidation subsequent to diabetic conditions induces an over-expression of SOD activity suggesting a compensatory mechanism to prevent tissue damage in the subjects.

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S.32. Antibodies Against Sperm Associated Antigen 16 as a Novel Disease Marker for Multiple Sclerosis

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In multiple sclerosis (MS) research, there is a need for reliable and specific disease markers which could aid in disease diagnosis and prognosis prediction, as well as provide relevant information regarding underlying disease etiology and pathogenesis. We recently applied a phage cDNA display approach to identify antigenic targets interacting with antibodies present in the cerebrospinal fluid (CSF) of MS patients. A panel of 8 antigenic targets was identified with 86% specificity and 45% sensitivity for MS. The objective of this study was to characterize these novel antigen-antibody systems as biomarkers for MS. One of the novel identified antibody targets is sperm associated antigen 16 (SPAG16). Recombinant SPAG16 protein was produced and a protein ELISA was optimised for high-throughput screening of CSF and serum samples from MS patients and controls. Besides being a valuable CSF marker, we demonstrated anti-SPAG16 antibodies in the serum of 13% of MS patients with 100% specificity. Also, since little is known about the biological function of this protein, monoclonal antibodies against SPAG16 were produced and characterized by immunohistochemistry. Preliminary results show staining of cells in active chronic MS lesions. Future staining experiments will elucidate the expression pattern of the protein and its biological relevance in the MS disease process. In conclusion,

application of autoantibody profiling in MS CSF led to the discovery of SPAG16 as an antibody target in MS. Detection of antibodies against this target antigen can aid in the diagnosis of MS patients and will provide valuable information regarding underlying MS disease processes.

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S.33. The Development of a Rapid Diagnostic Test for Sepsis

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The microbiological diagnosis of bacterial infection as the cause of a systemic inflammatory response in patients on the intensive care unit can take two days. The aim of this project is to develop a rapid diagnostic test for bacterial infection that will focus antibiotic treatment only for patients with infection. The hypothesis is that specific markers upregulated on blood neutrophils or monocytes can be used to diagnose a bacterial cause of an inflammatory response in hospital patients. The *in vitro* study focuses on the functional differences between neutrophils expressing high levels of Toll-like receptor 2 (TLR2) versus TLR4 in dealing with bacteria. Sixty patients have been recruited and screened from Accident and Emergency department in a double blinded fashion, and then divided into pathological controls and confirmed infections. A number of adhesion molecules appeared to be upregulated in all patients compare to normal (members of beta-1 and beta-2 integrins, CD64, TLR2 and 4, CD14 etc.), yet some of them showed lower expression pattern in confirmed infection patients group (CD49d, CD49e, CD49f, CD11c, CD14). The *in vitro* experiments were aimed at creating TLR2high or TLR4high neutrophils using normal cells incubated with cytokines (IFN-γ, GM-CSF, TNF-α, IL-4 and IL-17), bacterial products (LPS and LTA) or whole bacteria (E.Coli and MRSA). The results so far showed that only the IFN-γ treated neutrophils (15 h) increases the expression of TLR2 (125%↑).

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S.34. Regulatory T Cells Increase During Renal Allograft Rejection

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Alloreactive memory and regulatory T-lymphocytes may influence graft outcome by mediating rejection or promoting tolerance. The clinical relevance of post-transplant monitoring of memory and regulatory T-cell subsets to graft outcome is not well defined. We characterized the phenotype of sequential posttransplant blood samples (~5 time points/ patient) from 133 renal allograft recipients (22 rejectors, 111 non-rejectors), transplanted between May 2007 and December 2008. The frequencies of naïve, central memory (TCM) and effector memory (TEM) T cells were assessed using the adhesion/homing markers CD45RA, CD62L,CD49a. Regulatory (Treg) and suppressor cells were distinguished by the expression of CD4+CD25hiCD127dim and CD8+CD28. Allograft rejection was confirmed by renal biopsy and classified by modified Banff 2007 criteria. Immunophenotyping data were correlated with incidence of graft rejection to determine clinical relevance. Patients with biopsy proven acute cellular rejection showed higher percentages of CD4 T regulatory cells in the periphery compared to non-rejectors, indicating a strong relationship between T-regulatory cells and episodes of rejection ($P=0.02$). The average ratios of CD4 TEM+ CD4 TCM cells to Tregs was significantly lower in rejectors compared to non-rejectors ($P=0.002$). We conclude that increased frequencies of Tregs may serve as an informative biomarker of acute-rejection. Posttransplant monitoring of Tregs may offer a noninvasive strategy to improve prediction of graft rejection in renal transplants.

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S.35. Quantification of Soluble Cytokines in Tear Film of Sjögren Disease, Non Sjögren Dry Eye and Healthy Subjects

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Purpose: To quantify soluble cytokine concentrations in tear samples of patients with Sjögren disease, non Sjögren dry eye and healthy subjects. Methods: We classified severity of dry eye according to tear film break-up time (BUT)$<$7 sec, Schirmer II test (ST)$<$7 mm, symptoms$<$12 points in a validated questionnaire by Donate, et al. and oral mucosa biopsy or SS-A, SS-B Antibodies in the Sjögren disease patients. We obtained a tear sample of both eyes and performed a quantification of protein concentration. Using the Cytometric Bead Arrays (CBA) Cytokine Detection Kit we performed IL-2, TNF-a, IL-10, IL-4, IL-5 and IFN-g detection in tear samples. Descriptive statistics were performed for demographic variables and T test for identification of differences between groups. $P<0.05$ was considered statistically significant. Results: 65 patients were included, mean age 44 years (18-83), 37% male (n=24) and 63% female (n=41). Sjögren disease group showed the greatest concentration of TNF-a and IFN-g concentration ($p<0.05$). We did not find statistically significant difference between non Sjögren dry eye and healthy subjects. Conclusions: The Sjögren patients showed a higher cytokine concentration of TNF-a and IFN-g. without significant differences in IL-2, TNF-a, IL-10, IL-4 and IL-5. Interesting, Sjögren patients showed the clinically most severe dry eye, it could be possible that the clinical status corresponds to raised TNF-a concentrations in the tear film. Acknowledgments: This work was partially