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**ALPHA-1-ANTITRYPSIN (A1AT) FECAL CONCENTRATION IN INFANTS WITH ATOPIC DERMATITIS AND IN HEALTHY INFANTS WITH ATOPIC PARENTS.**  
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In food allergy the intestinal permeability from the lumen to the gut wall increases, probably due to mucosal damage. No data are available concerning the intestinal permeability from gut wall to lumen (IPGWL). We studied 24 infants (mean age 6 mos; range 4-18) with untreated atopic dermatitis related to food allergy (Rast and/or Prick test positive); 39 healthy infants with at least one atopic parent (mean 5,5 mos; range 1-4); 25 healthy infants as controls (mean 5,5 mos; range 1-20). As a marker of IPGWL we studied the excretion of endogenous A1AT in random lyophilised feces samples determined by a nephelometric method. Infants with atopic dermatitis showed significantly higher fecal excretion of A1AT than the controls ( $1,43 \pm 0,74$  S.D. mg/g dry weight versus  $0,74 \pm 0,23$  S.D. -  $p < .001$ ). Also in healthy infants with atopic parent(s) the A1AT excretion was significantly increased ( $1,32 \pm 0,88$  -  $p < .001$ ). These babies were followed every three months for the first year of life: 10 of them subsequently became affected with atopic dermatitis. We conclude that in infants with untreated atopic dermatitis there is an increase in IPGWL which seems to begin before the dermatitis develops and could predispose infants to such disease.

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**CORRELATION OF IgE ANTIBODIES TO ASPERGILLUS FUMIGATUS WITH BRONCHODILATOR SENSITIVITY IN CYSTIC FIBROSIS (CF)** Thomas Nicolai, Sissi Arleth, Rose Marie Bertele, Karsten H. Harms, Elke Miller  
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Bronchial hyperreactivity is often present in CF patients, but little is known about its causes. Therefore, in 80 outpatients with CF (age 5-34y) Aspergillus IgE antibodies, basic lung function parameters and bronchial reactivity to salbutamol inhalation were measured. Results were analysed using multiple regression for age, sex, weight as percentile for height (indicating general condition) and Aspergillus RAST.

In 55% of patients Aspergillus IgE was present, 22,5% had RAST Class 3 or 4. In addition to the known good correlation between basic lung function parameters and weight percentiles ( $p < .01$ ) we found an independent negative influence of age on FVC, FEV<sub>1</sub>, MEF<sub>50%VC</sub> and sRAW ( $p < .05$ ), and of Aspergillus RAST on FEV<sub>1</sub>, MEF<sub>50%VC</sub> RV, RV/TLC. Bronchodilator sensitivity did not correlate significantly with age and weight ( $p > .05$ ). However, Aspergillus RAST did significantly correlate with bronchodilator reactivity of sRAW ( $p < .01$ ) and MEF<sub>50%VC</sub> ( $p < .05$ ).

These results would indicate that bronchial reactivity in CF is often caused by hypersensitivity against Aspergillus and not related to general condition or age.

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**MONOCYTE (Mo) FUNCTION IN CHILDREN WITH JUVENILE RHEUMATOID ARTHRITIS (JRA).** Teresa Szydłowska, Wojciech Uracz, Irena Ruggiero, Jacek J. Pietrzyk, Marek Zembala,  
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A sample of 20 children with juvenile rheumatoid arthritis and 23 matched controls were studied in an attempt to verify null hypothesis that Mo function in children with JRA did not differ from that observed among healthy controls. The suppressor activity of Mo was studied by inhibition of 3H-thymidine incorporation in coculture with control PBMC stimulated with PHA. Functional expression of Fc receptor (FcR) was determined by EA rosettes, whereas expression of MHC class II determinants was assessed with HAK-75 and 14-4-4s mAb. It was shown that patients Mo provided no appreciable suppression of PBMC stimulated with PHA. A significant ( $p < .05$ ) decrease of Mo (FcR<sup>+</sup>) in patients blood was observed. The number of Mo revealing HLA-DR determinants reacting with HAK-75 mAb was similar in both groups of children. However, the population of Mo with Ia.7 receptor identified by 14-4-4s mAb was significantly ( $p < .05$ ) diminished among the patients. A decreased Mo suppressor activity combined with decreased expressivity of Fc and Ia.7 receptors justify the rejection of null hypothesis and imply an abnormal regulatory function of Mo in children with JRA.

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**ENDOTOXIN LEVELS IN MENINGOCOCCAL INFECTIONS**  
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We studied prospectively plasma endotoxin (lipopolysaccharides, LPS) levels in forty-five consecutively admitted patients with bacteriologically verified systemic meningococcal disease (SMD). Using a semi-automated, Limulus / chromogenic substrate test (detection limit = 25 ng/L, 25 patients (56%) had detectable LPS on admission. LPS > 700 ng/L seemed to be critical in developing: 1) Severe, persistent septic shock (15/15 versus 3/30) ( $p < .0001$ ). 2) Pathologically elevated serum creatinine (15/15 versus 3/30,  $p < .0001$ ), 3) Adult respiratory distress syndrome (5/15 versus 1/30,  $p = 0.01$ ) and 4) Dying (8/15 versus 1/30,  $p = 0.0002$ , Fisher's exact test). LPS were cleared from the circulation with T/2 = 1-3 hours after initiation of antibiotic therapy. Increasing LPS levels were never observed. Blood exchange or plasmapheresis did not significantly increase the LPS clearance. Conclusion: LPS quantitation is of importance as a prognostic marker in meningococcal infections

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**PLASMA LACTOFERRIN IN PIGS. AN EVALUATION OF LACTOFERRIN AS A PARAMETER OF SEPTICAEMIA.**

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The levels of plasma lactoferrin in response to endotoxin and Escherichia coli infusion in piglets were studied to give exact time relation of plasma lactoferrin increase in relation to start of endotoxin and Escherichia coli infusion. Swine lactoferrin was determined by a new enzyme linked immunoassay. In 13 piglets there was a ten-fold increase of plasma lactoferrin concentrations 2 hrs after the start of 0.25 mg/kg/2hrs endotoxin intravenous infusion. The initial rise was 3.4 mg/l/hr, thereafter it levelled off. Fourteen piglets, receiving a bolus of 10<sup>11</sup> Escherichia coli intravenously, had a twenty-fold increase of plasma lactoferrin concentrations, amounting to 6-9 mg/l/hr. Fourteen piglets receiving the same bolus of Escherichia coli intraperitoneally had an increase of 0.5 mg/l/hr.

These data indicate that plasma lactoferrin was an immediate marker of septicaemia and endotoxaemia, giving rise to twenty-fold increments of plasma lactoferrin within 2 hrs.

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**BACTERIAL MENINGITIS IN NEWBORNS: SONOGRAPHIC FEATURES**

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9 newborns (B.W. 2812 ± 742 gr; G.A. 36.8 ± 3.8 wks) having proven bacterial meningitis were studied with repeated cranial ultrasounds examinations to detect and follow-up possible complications. The aetiological germs were group B streptococcus (1), E. Coli (1), Pseudomonas Aeruginosa (1), Listeria monocytogenes (4), Streptococcus Pneumoniae (1) and Klebsiella (1). Early sonographic abnormalities were observed in 8 of 9 patients including bright convolitional markings (3), diffused increased echogenicity (3), smaller and non-visible ventricles (3), focal areas of increased echogenicity (1), ventricular dilation (2), ventricular debris (2). Late sonographic abnormalities were detected in 2 of 9 newborns: ventricular dilation (2) and encephalomalacia (1). 2 babies died. 5 survivors were followed-up: at 1 year of age, 3 had a normal psycho motor development, 2 had moderate mental retardation. Cranial ultrasound examination is now proved to be an alternative and effective method in monitoring the infective process of bacterial meningitis, besides CT scan. By serial sonographic studies it is easy to detect the early and sequential structural changes of meningitis: arachnoiditis, brain edema, cerebritis, infarction, hemorrhages, brain abscess, ventriculitis, ventricular dilation, encephalomalacia and cerebral atrophy. Therefore cranial sonography is very useful in the appropriate management and prognosis of meningitis. We suggest the routine use of cranial ultrasounds during neonatal bacterial meningitis.