

**Conclusion** In children with malnutrition BMI, MUAC, TST and low serum albumin levels correlated with genotype GG and CG of the *IL-6* 572 gene.

**PS-078 CLINICAL RELEVANCE OF GAMMA-GLUTAMYL TRANSPEPTIDASE IN CHILDHOOD OBESITY**

<sup>1</sup>P Codoñer-Franch, <sup>1</sup>M Salamanca, <sup>2</sup>A Codoñer-Alejos, <sup>1</sup>M Porcar-Almela, <sup>1</sup>M Navarro-Solera, <sup>3</sup>J Carrasco-Luna. <sup>1</sup>Pediatrics, Hospital Universitario Dr Peset, Valencia, Spain; <sup>2</sup>Pediatrics Obstetrics and Gynecology, University of Valencia, Valencia, Spain; <sup>3</sup>Experimental Sciences, Catholic University of Valencia, Valencia, Spain

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**Background and aims** Metabolic risk leads to severe comorbidities in obesity. We evaluate the relationship between the values of gamma-glutamyl trans peptidase (GGT), a marker of hepatic involvement, and cardio metabolic risk factors in obese children.

**Methods** A prospective cross-sectional study of 147 children (aged 7 to 16 years) was carried out. Ninety-five children were obese with a body mass index standard deviation score (SDS-BMI) >2 and 52 children were normal weight. Patients with endocrine disease or syndromic obesity were excluded. We have analysed clinical parameters of adiposity (fat mass by bioelectrical impedance, waist and hip circumference), blood pressure, and classical biochemical parameters indicative of metabolic risk (lipid profile, glucose and insulin). Additionally, novel parameters related to metabolic risk such as uric acid, retinol binding protein (RBP4), cystatinC, homocysteine, thyrotropin, ultrasensitive C-reactive protein (CRP) and GGT were also determined. Statistical analysis was made ANCOVA test and Pearson partial correlation adjusting for gender, age, Tanner stage, and BMI.

**Results** GGT was higher in the children with SDS-BMI >4 with respect children with SDS-BMI between 2 and 4 ( $16.3 \pm 5.8$  vs  $18.4 \pm 8.8$  IU/L,  $p = 0.025$ ). Both groups were statistically significant with respect normal weight ( $12.2 \pm 2.9$  IU/L,  $p < 0.0001$  and  $p < 0.001$  respectively). GGT was correlated with SDS-BMI ( $p < 0.0001$ ), waist circumference ( $p < 0.001$ ), percentage of fat mass ( $p < 0.01$ ), SDS of systolic blood pressure ( $p < 0.010$ ), total cholesterol ( $p < 0.0001$ ), LDL cholesterol ( $p < 0.0001$ ), triglycerides ( $p < 0.0001$ ), RBP4 ( $p < 0.047$ ), thyrotropin ( $p < 0.019$ ) and CRP ( $p < 0.044$ ).

**Conclusion** GGT is a marker associated with several metabolic risk factors, which highlights the importance of considering hepatic impairment as a component of this syndrome.

**PS-079 WITHDRAWN**

**PS-080 PREVALENCE OF OVERWEIGHT IN PAEDIATRIC INFLAMMATORY BOWEL DISEASE IN SAUDI ARABIA**

<sup>1</sup>M El Mouzan, <sup>2</sup>A Mehaidib, <sup>3</sup>M Hasosah, <sup>4</sup>A Anazi, <sup>5</sup>A Al Hussaini, <sup>6</sup>K Noulji, <sup>7</sup>K Al Reheili. <sup>1</sup>Pediatrics, King Saud University, Riyadh, Saudi Arabia; <sup>2</sup>Pediatrics, King Faisal Specialist Hospital and Research Center, Riyadh, Saudi Arabia; <sup>3</sup>Pediatrics, National Guard Hospital, Jeddah, Saudi Arabia; <sup>4</sup>Pediatrics, King Fahad Specialist Hospital, Dammam, Saudi Arabia; <sup>5</sup>Pediatrics, King Fahad Medical City, Riyadh, Saudi Arabia; <sup>6</sup>Pediatrics, Dhahran Health Center, Dhahran, Saudi Arabia; <sup>7</sup>Pediatrics, Maternity and Children Hospital, Madinah, Saudi Arabia

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**Background and aim** Excess weight in inflammatory bowel disease (IBD) represents an additional morbidity, and yet the

prevalence has been rarely reported. The aim of this report is to establish the prevalence of overweight in children with IBD in the Kingdom of Saudi Arabia (KSA).

**Methods** Data from a cohort of children in the KSA diagnosed with IBD were analysed retrospectively. Growth parameters were recorded at diagnosis and body mass index (BMI) was calculated using the formula (weight/height<sup>2</sup>). The KSA charts were used as reference. Excess weight categories were defined as overweight (BMI-for age  $\geq 85$ th to <95th), obesity  $\geq 95$ th to <97th), and severe obesity  $\geq 97$ th percentile. Chi-square test was used and p-value of  $<0.05$  was considered significant.

**Results** There were 417 children from birth to 18 years of age, including 133 ulcerative colitis (UC) (32%), and 284 Crohn disease (CD) (68%). The prevalence of excess weight was 12/133 (9%) in UC and 23/284 (8.1%) in CD ( $p = 0.063$ ) much lower than in Western reports. However, the more common prevalence of excess weight in UC than CD, although not significant ( $p = 0.063$ ), was similar to patterns from other population. The commonest form of excess weight was overweight 20/35 (57%), followed by obesity 9/35 (26%), and severe obesity 6/35 (17%).

**Conclusion** The pattern of excess weight in KSA children with IBD is similar to Western literature. However, a much lower prevalence is demonstrated. Identification of factors associated with the low prevalence of overweight and obesity is needed.

**PS-081 LACTOBICILLUS ACIDOPHILUS ATTENUATED SALMONELLA-INDUCED INTESTINAL INFLAMMATION VIA TGF-BETA/SMADS SIGNALLING**

<sup>1</sup>JF Huang, <sup>2</sup>YC Liu, <sup>2</sup>PF Liu, <sup>2</sup>CW Shu. <sup>1</sup>Pediatrics, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan; <sup>2</sup>Medical Education and Research, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan

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**Aims** To investigate whether probiotics and/or prebiotics attenuate *Salmonella typhimurium* induced NF- $\kappa$ B activation via Smad7 and I $\kappa$ B $\alpha$  expression in the human colorectal epithelial CaCO<sub>2</sub> cells; to determine the molecular mechanisms of preventive effects of probiotics on intestinal infection.

**Material and methods** CaCO<sub>2</sub> cells were administered probiotic (*Lactobacillus acidophilus*) and/or prebiotic (inulin supplemented with oligofructose). Subsequently, the cells were infected with *S. typhimurium*. The culture supernatants and cell lysates were collected for cytokine determination and western blot analysis. The CaCO<sub>2</sub> cells were also transfected with plasmids containing Smads or NF- $\kappa$ B responsive reporter luciferase. After transfection, supernatants from cells were collected for luciferase assay. Involvement of miR-21 (Smad7 silencer) from supernatants of infected cells in the presence or absence of probiotics was determined.

**Results** The probiotics significantly suppressed NF- $\kappa$ B activation elevated by *S. typhimurium*. IL-8 mRNA was significantly lower in probiotics pretreated CaCO<sub>2</sub> cells compared with the cells infected with *S. typhimurium* alone. Synbiotics showed strongly suppressed effects on IL-8 and TNF- $\alpha$  gene transcriptions elevated by *S. typhimurium*. Pretreatment of probiotics increased I $\kappa$ B $\alpha$  expression level. Consistent with I $\kappa$ B $\alpha$  expression, pretreatment of probiotics increased 7 folds of Smad3/4 activity. The protein expressions of TGF- $\beta$  and Smad7 in *S. typhimurium* infected cells with or without probiotics were determined by immunoblotting. Compared to *S. typhimurium* infection alone, pretreatment with probiotics and synbiotics induced 20 and 4 folds of miR 21 expressions, respectively.

**Conclusions** The experimental results showed that probiotics effectively attenuated *Salmonella*-induced intestinal inflammation in human intestinal CaCO<sub>2</sub> cells via TGF- $\beta$ 1/Smads and TGF- $\beta$ 1/miR21 signalling pathway.

**PS-082 COEXISTENCE OF COELIAC DISEASE AND ATOPIC DERMATITIS**

<sup>1</sup>O Uibo, <sup>2</sup>K Ress, <sup>3</sup>T Annus, <sup>3</sup>U Putnik, <sup>3</sup>K Luts, <sup>2</sup>R Uibo. <sup>1</sup>Department of Pediatrics, Children's Clinic of Tartu University Hospital, Tartu, Estonia; <sup>2</sup>Department of Immunology, Institute of Biomedicine and Translational Medicine, Tartu, Estonia; <sup>3</sup>Department of Pediatrics, Tallinn Children's Hospital, Tallinn, Estonia

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Coeliac disease (CD) is an autoimmune disorder of the small intestine with highly variable clinical presentation, frequently associated with various diseases and conditions, autoimmune and non-autoimmune.

We aimed to study the association of childhood CD and atopic dermatitis (AD) in Estonia studying the frequency of AD cases in newly diagnosed CD patients and CD cases in active AD.

**Methods** We investigated 152 consecutive children with CD (45% boys, mean age 2.3 years) and 351 consecutive children with AD (57% boys, mean age 5.8 years). CD diagnosis was made according to the ESPGHAN diagnostic criteria and AD was diagnosed by UK Working Party's Diagnostic Criteria for AD.

**Results** Among CD patients, AD was diagnosed in 8 (5.3%) children, and in the AD group CD was confirmed in 5 (1.4%) patients. All patients with both diseases had histologically characterised small intestinal damage as Marsh IIIa-IIIc stages, two of them had silent CD. The risk for developing CD was revealed to be four times higher in AD patients (OR = 4.18; 95% CI: 1.12–15.64) when compared to general children population in Estonia (frequency 0.34%; Lillemäe *et al.*, Eur J Gastroenterol Hepatol 2012). Two patients with CD and AD had no symptoms to suspect CD, in spite of extensive histological changes in small intestinal mucosa.

**Conclusions** CD and AD coexist more frequently than could be expected. Therefore, our study emphasises the need for evaluating the cost-effectiveness for CD screening of AD patients alongside with other immune-mediated diseases in order to diagnose CD in time.

**PS-083 WITHDRAWN**

**PS-084 DIFFERENTIAL REGULATION OF INTERLEUKIN-8 AND HUMAN BETA-DEFENSIN-2 IN INTESTINAL EPITHELIAL CELLS TO PSEUDOMONAS INFECTION**

H Fu-Chen. Pediatrics, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University, Kaohsiung, Taiwan

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**Background and aims** *Pseudomonas aeruginosa* (*P.aeruginosa*), carriers the highest case fatality rate of all gram-negative infections and antimicrobial therapy has not been demonstrated to improve clinical outcome. Moreover, the emergence of multi-drug resistant *P. aeruginosa* has become a major concern in the

hospital setting. Fever and diarrhoea were the 2 most common initial symptoms in *P. aeruginosa* sepsis in previously healthy infants and children. This implied that intestinal epithelial cells (IECs) contacting with the pathogen may play an important role on innate immunity to *P.aeruginosa* infection. Therefore, we aim to investigate the intestinal epithelial IL-8 and hBD-2 expression to *P. aeruginosa* infection and its regulators.

**Methods** We applied ELISA for IL-8 and hBD-2 protein secretion, RT-PCR for IL-8 and hBD-2 mRNA expression, Western blot for signal pathway as well as inhibitors and siRNA to investigate the involved proteins in *P. aeruginosa*-induced IL-8 and hBD-2 expression in SW480 cells.

**Results** We demonstrated after prolonged infection by *P. aeruginosa*, secreted IL-8 protein was suppressed but hBD-2 protein was enhanced though both mRNAs were increased in SW480 cells. Interactions between the PI3K/Akt pathway and ERK kinase result ultimately in post-transcriptional effects that decrease *P. aeruginosa*-induced IL-8 production while NOD1 protein is involved in *Pseudomonas*-induced hBD-2 expression in SW480 cells.

**Conclusions** *P. aeruginosa* induced pro inflammatory response and antimicrobial peptide in IECs. The antimicrobial peptide in IECs has been shown to continuously protect the host against prolonged infection while modulation of pro inflammatory response prevents the host from the detrimental effects of overwhelming inflammation.

## Hematology and Oncology

**PS-085 CLINICAL CHARACTERISTICS AND TIME TO DIAGNOSIS IN CHILDREN WITH SHWACHMAN DIAMOND SYNDROME (SDS)**

<sup>1</sup>A Silwal, <sup>2</sup>SE Kinsey, <sup>3</sup>J Puntis. <sup>1</sup>Department of Paediatrics, Leeds General Infirmary, Leeds, UK; <sup>2</sup>Department of Paediatric Haematology, Leeds General Infirmary, Leeds, UK; <sup>3</sup>Department of Paediatric Gastroenterology, Leeds General Infirmary, Leeds, UK

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Shwachman Diamond syndrome (SDS) is a multisystem disorder with heterogeneous clinical presentation. We examined medical records of 11 patients (now age 9–30 years, with confirmed SDS gene mutation) attending SDS multidisciplinary clinic in Leeds (UK) to understand their clinical characteristics, time to diagnosis and progression over time.

**Results** Seven children in our group had one affected SDS relative; all had variable presentation, severity and progression; genotype-phenotype correlation did not exist even within affected siblings in same family. Median age at initial presentation was 3 months (0–7 m) with median delay to clinical diagnosis at 17 months (2 m–12 yr). Failure to thrive or steatorrhoea together accounted for initial concern in 81%. Most (7/11) had documented negative sweat test at an early age with proven exocrine pancreatic insufficiency (EPI) in 3/11, yet suspicion of SDS was not raised despite SDS being the second commonest cause of EPI after cystic fibrosis. The severity of symptoms at presentation and rate of progression influenced the recognition and suspicion of the condition.

Over time, all patients developed recurrent infections, haematological abnormalities, dental/skeletal abnormalities and neuropsychological/ developmental issues. During follow up, growth and pancreatic insufficiency improved, bone marrow became dysplastic in 30% with decrease in the frequency/severity of