

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 TRANSEPTIDASE IN CHILDHOOD OBESITY

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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 ± 5.8 vs 18.4 ± 8.8 IU/L, $p = 0.025$). Both groups were statistically significant with respect normal weight (12.2 ± 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WITHDRAWN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

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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/height²). The KSA charts were used as reference. Excess weight categories were defined as overweight (BMI-for age ≥ 85 th to < 95 th), obesity ≥ 95 th to < 97 th), and severe obesity ≥ 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

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Aims To investigate whether probiotics and/or prebiotics attenuate *Salmonella typhimurium* induced NF- κ B activation via Smad7 and I κ B α expression in the human colorectal epithelial CaCO₂ cells; to determine the molecular mechanisms of preventive effects of probiotics on intestinal infection.

Material and methods CaCO₂ 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 CaCO₂ cells were also transfected with plasmids containing Smads or NF- κ 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- κ B activation elevated by *S. typhimurium*. IL-8 mRNA was significantly lower in probiotics pretreated CaCO₂ cells compared with the cells infected with *S. typhimurium* alone. Synbiotics showed strongly suppressed effects on IL-8 and TNF- α gene transcriptions elevated by *S. typhimurium*. Pretreatment of probiotics increased I κ B α expression level. Consistent with I κ B α expression, pretreatment of probiotics increased 7 folds of Smad3/4 activity. The protein expressions of TGF- β 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.