Of these 32 patients 29 had positive tTg. This gives a sensitivity of 90%. Thirteen patients had tTg greater than 100, 12 had positive intestinal biopsy; this gives a sensitivity of 92%.

Conclusion Although the sensitivity of tTg in our series is 90%, it is reasonable to assess small intestinal biopsy in before subjecting children to lifelong gluten free diet.

724 HLA DQ2/DQ8 TYPING IN CHILDREN DIAGNOSED WITH CELIAC DISEASE
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Background and Aims Genes encoding HLA DQ2/DQ8 are associated with celiac disease (CD) and testing for their presence has high negative predictive value for the diagnosis. The aim of this study was to assess the role of HLA typing in symptomatic individuals in whom the diagnosis of CD is uncertain.

Methods We proceeded a retrospective study leaded on a group of children investigated for CD in ‘Grigore Alexandrescu’ Emergency Children’s Hospital from 2007 to 2012 that underwent HLA typing. Inclusion criteria were all patient with mild enteropathy (Marsh I, 2, 3a), moderate elevated values of tisular tranglutaminase (tTG) antibodies (between cut off point and 5 times normal value) and poor response to gluten free diet. The medical records of all patients investigated for CD were reviewed.

Results 164 patients were performed HLA typing; 26 patients satisfied the inclusion criteria; 20 (76.9%) of these had HLA DQ2/DQ8 present and 6 (23.07%) had a negative test for HLA DQ2/DQ8. The mean age of our investigated group was 23.46 months and the mean age for HLA DQ2/DQ8 negative group was 21.08 months. Sex distribution indicated 9 boys and 17 girls. Gastrointestinal symptoms dominated: 17 children had diarrhea, 9 had failure to thrive and 13 patients had both chronic diarrhea and poor weight gain.

Conclusion Patients with clinical suspicion of CD that have moderate levels of tTG antibodies, mild biopsy changes and poor response to gluten free diet need to have HLA typing specially at younger ages (under 3 years old).

725 LYMPHOCYTE RESPIRATION IN CHILDREN WITH TRISOMY 21
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Aims This study aimed to measure lymphocyte mitochondrial O2 consumption (cellular respiration) in children with trisomy 21. Methods Peripheral blood mononuclear cells were isolated from whole blood of trisomy 21 and control children and immediately used to measure the respiratory rate. [O2] was determined as function of time from the phosphorescence decay rates (1/t) of Pd (II)-meso-tetra-(4-sulfonatophenyl)-tetrabenzoporphyrin. In sealed vials containing cells and glucose as a respiratory substrate, [O2] declined linearly with time, confirming the zero-order kinetics of O2 consumption (conversion to H2O) by cytochrome oxidase.

Results The rate of respiration (k, in mM O2 per min), thus, was the negative of the slope of [O2] vs. time. NaCN inhibited O2 consumption, confirming the oxidation occurred in the mitochondrial respiratory chain. For control children (age = 8±5.6 yr, n=26), the mean (± SD) value of k (in mM O2 per min per 107 cells) was 1.36±0.79 (coefficient of variation = 58%; median = 1.17; range = 0.60 to 3.12; -2SD = 0.61). For children with trisomy 21 (age = 7±3.4 yr, n=26), the value of k was 0.82±0.62 (coefficient of variation = 76%; median = 0.60; range = 0.20 to 2.80), p<0.001. Fourteen of 26 (54%) children with trisomy 21 had k values of 0.20 to 0.60 (i.e., < -2SD).

Conclusion Thus, it appears that some children with trisomy 21 have relatively reduced lymphocyte bioenergetics. The biological implication of this finding (variation) requires further studies.
Wilson’s disease. Detection of WD in children and young adults remains very difficult. The most important investigation is liver biopsy with the assessment of liver copper. Genetic analysis may help in doubtful cases.

**Results**

α frequencies of the IL-13 R polymorphism does not contribute to asthma or allergic rhinitis susceptibility, although the interleukin-13 receptor α subunit gene locus might be involved in the control of immunoglobulin E production. IL13 can be used as an immunological marker in atopic upper respiratory diseases and to differentiate between atopic and non atopic bronchial asthma.

**Background and Aims**

Phototherapy is the most common treatment for jaundice. One of the various endpoints that can be used to investigate the potential of blood as a predictor of radio sensitivity is DNA damage and apoptosis. The aim of this study was to investigate the possible relation between phototherapy and DNA damage and apoptosis, in neonates with hyperbilirubinemia.

**Methods**

The study included 15 healthy full-term newborns as control. The phototherapy group consisted of 30 non-physiologic jaundice that after 16 hours phototherapy, 1 ml peripheral venous blood were obtained from them.

**Results**

DNA damage immediately after phototherapy termination (IFT) was higher in jaundice infants than control one (p<0.01). After 24 hours cells repaired their damages, as there wasn’t any difference between these groups about this value. DNA damage in the phototherapy group was higher at IFT than 24 hours after that, but in the control one there weren’t any differences between them (respectively p<0.001, p<0.05). Apoptosis value at 24 hours after phototherapy termination was higher than the IPT in the phototherapy group was higher at IPT than 24 hours after that, but there was no significant difference between these groups about this value. DNA damage in the phototherapy group was higher at IPT than 24 hours after that, but in the control one there weren’t any differences between them (respectively p<0.001, p<0.05). Apoptosis value at 24 hours after phototherapy termination was higher at IPT than 24 hours after that, but there was no significant difference between these groups about this value.

**Conclusions**

Since repair proofreading and fidelity properties isn’t absolutely, then always this is probable that errors maybe occur during extensive DNA damages repair and finally these errors can cause mutation in DNA. If this event be in important and sensitive region of genome, harmful effects would menace phototherapy-treated infants’ later-life. So this is necessary to investigate of long-term genotoxic effects of phototherapy in phototherapy-treated neonates.

**Objectives**

To assess the value of serum interleukin (IL) 13 levels as an immunological marker in atopic upper respiratory diseases, to clarify its differences in atopic and non atopic bronchial asthma and to determine the role of an IL-13 R α gene single nucleotide polymorphism (SNP) (A1398G) in the pathogenesis of these diseases.

**Methods**

Seventy-five patients were compared with 25 age-matched healthy volunteers. Serum total immunoglobulin (Ig) E and IL-13 levels were measured by enzyme-linked immunosorbent assay and the IL-13 R α gene (A1398 G) was screened by specific polymerase chain reaction.

**Results**

There was a non significant association between G allele frequencies of the IL-13 R α (1398G) gene polymorphism as compared to in controls. There were a significant increase in the serum level of total IgE & IL-13 towards heterozygous AG and homozygous GG than homozygous AA in atopic asthma, non atopic asthma, and allergic rhinitis patients. There was a significant increase in the serum level of total IgE & IL-13 towards homozygous GG than heterozygous AG in atopic asthma non atopic asthma, and allergic rhinitis patients for IgE and in all groups for IL-13.

**Conclusion**

Interleukin-13 receptor α 1 subunit gene A1398G polymorphism does not contribute to asthma or allergic rhinitis susceptibility, although the interleukin-13 receptor α 1 subunit gene locus might be involved in the control of immunoglobulin E production, IL13 can be used as an immunological marker in atopic upper respiratory diseases and to differentiate between atopic and non atopic bronchial asthma.