expression patterns. Ethical approval was granted by the Regional Committees for Medical and Health Research Ethics in Norway.

Results: CYP450 gene expression was significantly altered about 6 weeks postnatally between preterm infants (n=18) born at early (GA 25.2-28.6 wks) versus late (n=23) GA (29.0-33.6). Thirteen CYP450 genes, including those metabolizing commonly used drugs, such as CYP1A2 (p=0.0112) and CYP3A4 (p=0.0424), differed in expression levels. No significant differences in CYP450 gene expressions among preterm males and females were found.

Conclusion: We identified significant differences in CYP450 gene expression levels in preterm infants 6 weeks postnatally depending on GA. In particular CYP1A2 and CYP3A4 are interesting candidates for explaining variable drug response in preterm infants. Further investigation is needed to explore the role of these differences related to drug metabolism.

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ASSOCIATION OF MATRIX METALLOPROTEINASE 2 AND TIMP-2 GENE POLYMORPHISMS WITH DEVELOPMENT OF BRONCHOPULMONARY DYSPLASIA IN PRETERM INFANTS

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Background and aims: Bronchopulmonary Dysplasia (BPD) is considered to be a consequence of prematurity, pulmonary baro/volutrauma, hyperoxia and inflammation. Increased inflammatory response and lung repair with impaired alveolarization and vascularisation are largely determined by genetic variability. Metalloproteinases are endopeptidases with key role in extracellular matrix remodeling as occurs during lung development and repair after injury. The aim of this study was to assess the influence of MMP-2 and TIMP-2 genetic polymorphisms on the risk of BPD.

Methods: 95 preterm neonates with a gestational age of ≤32 weeks and birth weight ≤1500g were

included in this prospective study. For the evaluation of phenotypic effect 215 term healthy neonates were additionally included. Out of 95 premature infants 47 developed BPD according to NIH criteria. 7 neonates died before time of assessment presenting severe lung damage. Using SNP TaqMan assays -1306C/T MMP-2 and +853G/A TIMP-2 polymorphisms were genotyped in DNA extracted from blood samples.

Results: Analysis of genetic variability in preterm neonates showed difference in distribution of genotypes at -1306C/T MMP-2 position. C/T and T/T genotypes were more frequent in neonates developing BPD or in those who died. Additionally T allele presence was associated with prolonged ventilation time and hospitalization duration at NICU. No difference in +853G/A TIMP-2 polymorphism frequency was found between tested groups.

Conclusions: Based on presented data and multigenetic background of BPD, genetic variability of MMP-2 may be one of the contributory factors in development of bronchopulmonary dysplasia.

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PROGRESSIVE MUSCULAR DYSTROPHY: CLINICAL AND GENETICAL STUDY

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Background and aims: Progressive muscular dystrophies represent a group of genetic diseases with insidious onset in childhood; they are characterized by muscle fatigue associated with pseudohypertrophy of the muscles, high level of creatin kinase; electrophysiological, histological and molecular biology studies distinguish the primary disorder of the muscles.

Methods: Prospective study that included 16 patients (1-5 years old) in a period of 2 years (2006-2008). Patients were evaluated clinically, functionally, paraclinical (muscle enzyme), neurophysiology. In all patients muscle biopsy was performed and in 8 cases genetic tests.

Results: In this trial 12 children (75%) had pseudohypertrophy of calf muscles, 8 children (50%) had signs of muscular disease (the weakness of proximal muscles, a positive Gowers's sign), 5 children (31.25%) had increased

levels of transaminases, creatinkinase and lactic dehydrogenases. Genetic studies were performed in 8 of the patients, 6 presenting deletions and point mutations 2.

Conclusions: Progressive muscular dystrophy is a relative frequent disorder in children, which can be recognized early; if the high levels of transaminases are found, without other signs of hepatic disease, is necessary to determine the creatinkinase level. Genetic studies are required to specify the type of mutation in the initiation of therapies to slow evolution.

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AUTOMATED ECO-GENETIC SYSTEM PROGNOSIS OF BRONCHIAL ASTHMA IN GEORGIAN POPULATION

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Background: According to the actual direction of nowadays allergology more attention is paid to the prevention in early age period significantly decreases morbidity of allergic diseases and improve quality of life. Hereditary predisposition plays the most important role in the developing of disease, but it is not everything recognizable, it may be not demonstrated clinical symptoms in parents and other close relatives of affected individual.

Aim: To elaborate the new automated assessment cost-effective system which would allow early diagnostic and prevention BA by using "formalized cards". This system is based on the primary and secondary prognosis of BA.

Methods: HLA- markers (Terrasaki microlymphocytotoxic tests) typing was performed with 2nd class (DR, DQ) and 1st class (A, B, C) antigens locus's; Erythrocyte markers: ABO, Rhesus and MNS systems, Clinical-genealogic method (Falconer D., 1965): study of genealogical tree of investigated families; Genetic - statistic analysis "Informative Criteria" of different signs: ontogeny, phenotypic, environmental factors (by Kulback, Goubler E.B, 1990). Method of multi-dimensional phenotyping: agglomerative- hierarchical cluster analyse of chosen systems of signs. Analyse was performed in the interactive order.

Results: Multi-dimensional phenotyping shows phenotype classes: Classes of basic predisposition,

including up to 80% of individuals with manifested asthma; Classes of relative predisposition, including less than 80% of individuals with manifested asthma; Phenotype classes including a few patients; Phenotype classes not predisposed to BA, basically including healthy persons.

Conclusion: Elaborated computing table is enough simple and opportune for comprehensive use. It is proposed, for primary prognostic of BA. This method gives

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CYTOGENETIC RESULTS OF 153 PATIENTS WITH MENTAL RETARDATION IN MIDDLE ANATOLIA IN TURKEY

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Mental retardation (MR), that is a major social, educational, and health problem, occurs with the prevalence of 1-3% of general population. The etiologies of MR are complex the cause remains unknown in about 50% of cases and include teratogenic and environmental factors, perinatal asphyxia and genetic causes. Cytogenetic studies have been reported to determine the contribution of chromosomal abnormalities in parents with mental retardation from various other countries. As far as we know, no enough studies have been done in the middle Anatolia in Turkey. The aim of the present study was to evaluate the frequency and nature of chromosomal aberrations in Turkish patients that contribute to mental retardation.

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POLYMORPHISM OF SURFACTANT PROTEIN B GENES AND THE RISK OF RESPIRATORY DISTRESS SYNDROME AND CHRONIC LUNG DISEASE IN PRETERM NEONATES

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Objectives: To determine whether SP-B iΔ4 gene polymorphism is associated with respiratory distress syndrome (RDS) and chronic lung disease (CLD) etiology.