time, 2.9 years, range 1.0 to 4.4). Annual decline of pulmonary function was analyzed retrospectively. Mean annual decline of FEV_1 ($dFEV_1$) was 0.033 L/yr. There was significant and positive correlation between current R35 and $dFEV_1$ (r=0.33, p=0.01), though current FEV_1 did not correlate with $dFEV_1$ (r=-0.14, p=0.31). Multiple regression analysis confirm that only current R35 and FEV_1 at baseline can predict $dFEV_1$ significantly (p=0.004), though smoking status and age did not contribute. These results suggest that high R35 reflects retrospective rapid decline of FEV_1 and also may predict rapid decline in the future. In the contrast to spirometry, measurement of central airway resistance using IOS can provide additional and useful information for evaluating COPD.

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Body mass index and health-related quality of life in patients with chronic obstructive pulmonary disease

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Aim: The aim of this study was to assess the correlation between the nutritional status and quality of life in COPD patients.

Method: Group of 72 COPD patients were analyzed. Body mass index (BMI) was used to estimate nutritional status. Dyspnoa level was measured by Borg scale and health-related quality of life (HRQL) using St George's Respiratory Questionnaire (SGRQ).

Results: Spirometric testing showed severe lung function damage. The mean BMI was 24.9 ± 6.0 kg/m^2, and Borg score was 3,5 ± 1,5. Total SGRQ score in the group was 72.58 ± 15,3. According to BMI all patients were divided into three groups: BMI< 18,5 kg/m^2 - underweight; 18.5 < BMI < 25 kg/m^2 - normal; BMI> 25 kg/m^2 - overweight and obesity. Analysis HRQL showed following results: 1) underweight group: symptoms 69.5 ± 14.8; activity 90,91 ± 12,5; impact 70,11 ± 15,26 and total score 76,0 ± 11,7. 2) normal weight group: symptoms 64,4± 18,8; activity 81,7 ± 17,4; impact 63,3± 19,3 and total score 68,8 ±16,1; 3) overweight and obesity group: symptoms 64,6± 18,8; activity 88,3± 18,7; impact 69,6± 17,5 and total score 74,2 ± 15,9. The highest scores were in the underweight group patients. Relationship between SGRQ (total and impact) scores and dyspnoa score was statistical significant.

Conclusion: Underweight is common in COPD and it is associated with clinically important reductions in health status. Dyspnoa is an important simptom of disease, which is reflecting to HRQL.

83. Biological correlates of COPD

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Clinical phenotypic differences between COPD and asthma in elderly patients

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Despite its relatively common occurrence in elderly subjects, late-onset asthma is usually mistaken for chronic obstructive pulmonary disease (COPD). The aim of this study was to describe clinical and functional features and atopy status of elderly asthma in comparison with age-matched COPD patients. Clinically stable 24 COPD patients (17 male, 7 female) and 22 elderly asthmatics (6 male, 21 female) were included in the study. Smoking was a more distinct feature of COPD patients. Atopy rate of asthmatics was significantly higher than that of the COPD group (37% versus 8.3%). Mean total IgE levels were more higher in asthmatics compared to COPD patients (248 kU/L, 130 kU/L, respectively).

Table. Characteristics of patients

	COPD (n: 24)	Asthma (n:27)	P
Mean age (yrs)	68.83±4.82	69.82±6.26	NS
FEV_1 (% pred)	43.79±20.08	63.48±15.76	P<0.001
ΔFEV_1 (% pred)	11.50±12.21	26.00±27.59	P=0.05
FRC (% pred)	184.46±76.43	139.69±51.49	NS
DLCO/VA (% pred)	79.47±24.65	103.10±17.21	P<0.01
PaO2 (mmHg)	54.86±11.22	61.61±6.53	P<0.01
PaCO2 (mmHg)	41.57±7.44	39.03±2.33	P<0.01

Conclusions: Although some clinical features may help distinguishing COPD and asthma in patients above 65 years of age, it is often underdiagnosed and undertreated. A history of heavy smoking, decreased diffusing capacity for carbon monoxide, the presence of more prominent lung hyperinflation and chronic hypoxemia favour the diagnosis of COPD whereas atopy and significant bronchodilator responsiveness favour the diagnosis of asthma.

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The association between polymorphic genotypes of glutathione s-transferases and COPD in Turkhis population

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Chronic tobacco smoking is known as the major risk factor for chronic obstructive pulmonary disease (COPD), but only relatively small proportions of smokers actually develop airway obstruction.

Objective: We aimed to investigate the association between glutathione S transferase (GST) P1, GSTM1 and GSTT1 gene polymorphisms and COPD.

Methods: Blood samples were taken from 71 patients and 62 healthy controls. The detection of GSTT1, GSTM1 and GSTP1 polymorphisms were made by using Real-Time PCR.

Results: GSTP1105Ile/Ile genotype was found more frequently in the patients than in the controls (74% versus 37%, p=0.0001). GSTP1 Ile/Val and Val/Val genotypes were associated with a decreased risk of COPD (3.33-fold, 9.4-fold decrease, respectively). Furthermore, while individuals who have Val allele and nonsmoker have a 6.25-fold decreased risk for COPD, individuals who have the same allele and smoker have a 3.44-fold decreased risk, but the difference between the smoker and nonsmoker was not statistically significant. We also examined the risk of COPD associated with combinations of the three genotypes. The presence of GSTM1 present and GSTT1 present and GSTP1 Ile/Val or Val/Val genotype was associated with a 4.76-fold decreased risk of COPD. GSTM1 null and GSTT1 present and GSTP1 Ile/Val or Val/Val genotype has a 3.33 fold decreased risk of COPD.

Conclusions: It is likely that GSTP1 105Ile/Val or Val/Val genotypes have a protective effect against to COPD development and this effect may be partially decreased by cigarette smoking.

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Association of polymorphism of NAT2 gene and group factors of blood (ABO, MN, Gm) with risk of COPD

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Associative searching of genetic markers of the predisposition to COPD will permit to predict individual sensitivity in smokers and form risk groups of COPD among persons with some professions and teenagers. 146 smokers were examined (71 patients with COPD, 36 patients with chronic nonobstructive bronchitis, 39 persons without chronic pulmonary diseases and heavy accompanying pathology). All investigated persons were unrelated males, Slavs, St.Petersburg's inhabitants, comparable with age, social and smoking status. PCR amplification combined with diagnostic restriction enzyme digestion for genotyping of NAT2 polymorphism (II xenobiotics detoxication stage) and standard methods of blood groups definition (systems ABO, MN, Gm) have been performed. Protective associations for slow acethylators (genotype SS of gene NAT2) were found if described above individuals were carriers of I(0) blood group (OR=0,34; CI95% 0,88-0,13), factor G1m(1+) (OR=0,38; CI95% 0,99-0,15), phenotype MN (OR=0,36; CI95% 0,88-0,15). Combination of genotypes SS with G1m(1+) and MN was associated with decreased risk COPD (OR=0,16; CI95% 0,57-0,04). Obtained results can be useful to form the risk groups for the development of COPD in smokers without pulmonary disease and patients with chronic bronchitis.

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The use of probucol for correction of hydrolipoperoxidation disturbances in COPD patients who stopped smoking

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Aim: To study the influence of probucol on hydrolypoperoxidation (HLP) in COPD patients who stopped smoking.

Methods: 37 COPD patients, male, in age from 40 to 65 years old were divided in 2 groups. In each group distribution of patients with COPD I and COPD II was equal. All patients stopped to smoke, during quit attempt was used combination of nicotin-replacement therapy and antidepressants. In the first group besides medicines for tobacco dependence, was used conventional therapy of COPD: in case of COPD I – formoterol, in COPD II formoterol and inhaled glucocorticosteroids. In the second group besides conventional treatment of COPD and treatment of tobacco dependence was used probucol 500 mg per day orally. The control group consist from 20 healthy nonsmokers.

Results: all COPD patients had increased HLP. Patients with COPD I had normal activity of antioxidative enzymes (AE) in blood, patients with COPD II had decreased activity of AE in blood. After conventional treatment and smoking cessation the parameters of HLP in patients with COPD I became normal. There were a tendency to normalization of HLP parameters after such treatment in patients with COPD II. In the second group the period of normalization of HLP in COPD I patients was shorter, and there were normalization of HLP in COPD II patients

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