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Title: Effect of genetic polymorphisms of some cytokines and xenobiotic-metabolizing enzymes on the lung function in patients with COPD

Dr. Dimo 16042 Dimov dmdimov65@yahoo.com MD [1], Prof. Tatyana 16043 Vlaykova tvlaykova@abv.bg [2], Prof. Mateusz 16044 Kurzawski mkurz@op.pl [3], Dr. Vanya 16045 Ilieva ilvan@abv.bg MD [1], Dr. Atanas 16046 Koychev koychevn@abv.bg MD [1], Prof. Gospodinka 16059 Prakova prakova@hotmail.com MD [1], Prof. Maya 16061 Gulubova mgulubova@hotmail.com MD [4], Prof. Vladimir 16066 Maximov tvlaykova@hotmail.com MD [5], Prof. Marek 16068 Drozdzik drozdzik@sci.pam.szczecin.pl MD [4] and Prof. Vasil 16075 Dimitrov vas.dim488@gmail.com MD [5]. [1] Internal Medicine, Medical Faculty, Trakia University, Stara Zagora, Bulgaria ; [2] Chemistry and Biochemistry, Medical Faculty, Trakia University, Stara Zagora, Bulgaria ; [3] Experimental and Clinical Pharmacology, Pomeranian Medical University, Szczecin, Poland ; [4] Dept. General and Clinical 5Pathology, Medical Faculty, Trakia University, Stara Zagora, Bulgaria and [5] Clinical Center of Allergology, Medical University, Sofia, Bulgaria .

Body: COPD is a chronic inflammatory lung disease characterized by decreased expiratory flow rate. The decrease of lung function in COPD depends on tissue remodelling due to xonobiotic- and inflammation-induced ROS-mediated tissue damage and impaired proteinase/antiproteinase balance. Since the activity and/or the protein level of cytokines and enzymes involved in inflammation and xenobitic and antioxidant detoxification are found to be associated with some gene variants, we aimed to evaluate the role of gene polymorphisms of three xonobiotic-metabolizing enzymes and three proinflammatory cytokines as factors involved in decline of lung function in COPD. We genotype altogether 164 patients with COPD and 174 non-affected by the disease control individuals for the following SNPs: GSTP1+313A>G, IL6 -174G>C, TNFA -308G>A, IL1B -511C>T, IL1B +3953C>T and for the null polymorphisms in GSTM1 and GSTT1. Our results displayed that the carriers of A allele of GSTP1+313A>G showed a tendency for higher FEV1%. compared to the carriers of GG genotype (p=0.097). Patients COPD stage III/IV having GSTM1 null genotype demonstrated significantly lower FEV1% values (39.16%) than those with non-null genotype (43.91%, p=0.032). Moreover, patients with C containing genotypes of IL6 -174G>C SNP had significantly lower FEV1/FVC% (59.7%) compared to the patients with GG genotype (62.7%, p=0.034). The polymorphisms in GSTT1, TNFA and IL1B did not associate with respiratory indexes. In conclusion, we suggest that the polymorphisms in the genes of some cytokines and xenobiotic-metabolizing enzymes, such GSTP1, GSTM1 and IL6 are factors that may affect the lung functions in COPD.