PUD-11

TUMOR NECROSIS FACTOR-ALPHA, β_2 ADRENERGIC RECEPTOR, GLUTATHIONE-S-TRANSFERASE GENE POLYMORPHISMS, AND INFLAMMATION PARAMETERS BLOOD LEVEL IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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BACKGROUND/AIMS: Smoking is the major risk factor causing COPD, but only 15-20 % of smokers will develop COPD. The reason for this is not clear at present. Inflammatory cytokine gene polymorphisms were the possible cause of this discrepancy in general population.

METHODS: This is prospective, case-control study. Measurements of polymorphisms of TNF-α, the ß$_2$-AR and GST coding block were delineated using an allele-specific polymerase chain reaction approach. The allele-specific PCR technique was verified by direct dideoxy sequencing of PCR products. Serum level of TNF-α, c-reactive protein, eosinphil cationic protein, absolute eosinophil count and immunoglobulin E were also analyzed at the same time. Pulmonary function tests were performed in all patients.

RESULTS: We enrolled 63 COPD patients and 10 healthy controls with smoking habit with normal PFT. In comparison of COPD patients and control subjects, Gln27 of B2AR and GSTM1 deletion odd ratio were 18.0 (3.0-345.2) and 6.8 (1.5-46.9) respectively ($p<0.05$). Polymorphism of TNF-α was not significantly different between COPD and controls. PEFR and FEV_1% of predicted Gln 27 (50 ± 20 and 198 ± 102) were significantly lower than Glu/Gln27 (63 ± 22 and 284 ± 165). Polymorphism of GST in COPD patients, FVC% of predicted GSTM1 deletion (62 ± 20) was significantly lower than normal (72 ± 19) type.

DISCUSSION /CONCLUSIONS: Gln27 of B2AR and GSTM1deletion may increase the patient's susceptibility to the development of COPD in smokers.

Key words: COPD, TNF-α, ß$_2$-adrenoreceptor, Glutathion-S-transferases, c-reactive protein, eosinphil cationic protein, absolute eosinophil count and immunoglobulin E.