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Title: Long-term exposure to tobacco smoke on alveolar macrophages phenotype with regard to genetic and age predisposition

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Body: Core role of tobacco smoke as a risk factor in chronic obstructive pulmonary disease (COPD) is proved, but COPD develops not in every smoker. Objective:to estimate the effect of long-term tobacco smoke on a functional phenotype of alveolar macrophages (A;M) with regard to genetically determined macrophages phenotype while aging. Methods: Experimental COPD (eCOPD) was simulated in vivo in two genetic mice strains:C57/BL6 (n=40)-predominant M1 phenotype,and Balb/c (n=40)-M2 phenotype. ECOPD groups included 30 mice, control–10 mice. Tobacco smoking lasted for 6 months-2 cigarettes t.i.d. COPD was verified histologically. AM functional phenotype was assessed by nitric oxide production (NO) spectrophometrically. Results.COPD was confirmed histologically in both eCOPD groups, changes were more expressed in C57/BL6. There was no significant difference in basal NO production (bNO) in eCOPD groups, but induced NO production (iNO) significantly decreased during 6 months and was much lower in C57/BL6 than in BALB/c: 3.80±0.21 vs 5.53±0.29 mkM ($p<0.05$). There were no differences in initial bNO in both controls; bNO significantly decreased in both controls during 6 months. Aging decreased iNO in both controls - from 26.40±3.2 to 6.60±0.45 mkM in C57/BL6, and from 19.21±1.20 mkM to 7.57±0.72 mkM in BALB/c, changes were more expressed in C57/BL6. Conclusions.We elicited genetic predisposition to COPD risk factor - tobacco smoke, associated with M1 macrophages phenotype and age-related transformation of AM phenotype towards antiinflammatory M2, increasing with long-term inhalation of tobacco smoke and more expressed in M1 phenotype.