

Chronic Obstructive Pulmonary Disease

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Indoor air pollution and COPD

D Behera

Postgraduate Institute of Medical Education & Research, India

More than 400 million people worldwide suffer from COPD. The estimated burden in India is about 15 million cases (~9 million males and ~6 million females) [INSEARCH study IJTLD 2012]. Besides smoking air pollution, particularly indoor air pollution is one important risk factor for COPD in India and other developing countries. Women who cook with solid fuels have increased respiratory symptoms including chronic cough and phlegm. Decrease in lung function and COPD is present in 7% of biomass using women who never smoked in their life. In contrast, only 1.8% of never-smoking LPG users had COPD. A number of studies have showed that in India, the incidence of chronic cor pulmonale is similar in men and women despite the fact that 75% of the men and only 10% women are smokers. In women, chronic cor pulmonale was found to be more common in younger age. This has been attributed to domestic air pollution as a result of the burning of solid biomass fuels leading to chronic bronchitis and emphysema which result in chronic cor pulmonale. A number of studies by us have shown that exposure to biomass fuel produces various respiratory symptoms including COPD, impaired lung functions (Lung function, particularly FVC is affected by indoor air pollution due to domestic cooking more so with biomass fuel), high levels of blood carboxyhemoglobin levels and respiratory symptoms in children (Mixed fuel and kerosene fuel had worst effects on respiratory system in children whose households used these fuels).

dirlrsi@gmail.com

Genetic determinants of chronic obstructive pulmonary disease in south Indian male smokers

K K Reddy¹, A Cholendra¹, R Premananda² and A Chandrasekar³¹Sri Venkateswara University, India²Premananda Allergy and Chest Hospital, India³Anthropological Survey of India, India

The development of chronic obstructive pulmonary disease, upon exposure to tobacco smoke is the cumulative effect of defects in several genes. With the aim of understanding the genetic structure that is characteristic of our patient population, we selected forty two single nucleotide polymorphisms of twenty genes based on previous studies and genotyped a total of 382 samples, which included 236 patients and 146 controls using Sequenom Mass ARRAY system. Allele frequencies of rs2276109 (MMP12) and rs1800925 (IL13) differed significantly between patients and controls ($p=0.013$ and 0.044 respectively). Genotype analysis showed association of rs2276109 (MMP12) under additive and dominant models ($p=0.017$, $p=0.012$ respectively), rs1800925 (IL13) under additive model ($p=0.047$) and under recessive model, rs1695 (GSTP1; $p=0.034$), rs729631, rs975278, rs7583463 (SERPINE2; $p=0.024$, 0.024 and 0.012 respectively), rs2568494, rs10851906 (IREB2; $p=0.026$ and 0.041 respectively) and rs7671167 (FAM13A; $p=0.029$). The minor alleles of rs1695 (G), rs7671167 (T), rs729631 (G), rs975278 (A) and rs7583463 (A) showed significant negative association whereas those of rs2276109 (G), rs2568494 (A), rs10851906 (G) and rs1800469 (T; TGF- β) showed significant positive association with lung function under different genetic models. Haplotypes carrying A allele of rs2276109, G allele of rs1695 showed negative correlation with lung function. Haplotypes carrying major alleles of rs7671167 (C) of FAM13A and rs729631 (C), rs975278 (G), rs7583463 (C) of SERPINE2 had protective effect on lung function. Haplotypes of IREB2 carrying major alleles of rs2568494 (G), rs2656069 (A), rs10851906 (A), rs965604 (C) and minor alleles of rs1964678 (T), rs12593229 (T) showed negative correlation with lung function. In conclusion, our study replicated the results of most of the previous studies. However, the positive correlation between the minor alleles of rs2568494 (A) and rs10851906 (G) of IREB2 and lung function needs further investigation.

kanalakr@yahoo.com