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Chronic Obstructive Pulmonary Disease (COPD)

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Editorial

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Editorial

Chronic Obstructive Pulmonary Disease (COPD) is thought to be the result of environmental triggers in genetically susceptible individuals. COPD is characterized by coughing, shortness of breath, sputum production, rapid breathing, wheezing, and weight loss due to the energy required for labored breathing. COPD includes chronic bronchitis and emphysema chronic bronchitis-inflammation of the lining of the bronchial tubes emphysema - permanent destruction of the alveoli [1].The research work on this topic has been carried out at CSIR-North East Institute of Science and Technology, Jorhat, Assam to study the relative prevalence of the disease amongst the people residing in the vicinity of Open-cast coal mine areas in Assam and also to trace out the genetic susceptibility to the disease in the population. Extensive survey was carried out in the Open-cast coal mine and data were recorded in Questionnaire formats by close interaction with the local people with their consent. Blood samples were collected (random sampling) from large number of villagers residing very near to the coal mine through health camps conducted in the area; and spirometry was carried out. Genotyping for GSTT1 and GSTM1 were also carried out by multiplex PCR reaction. The major finding of our study is that the variables namely polluted environment of coal mine area, smoking, male sex have a highly significant contribution towards the development of COPD [2,3].

COPD is unique among complex genetic diseases in that the environmental inducer of the disease is usually completely obvious, and that the level of exposure can usually be documented with some precision. The high mortality and morbidity associated with COPD, and its chronic and progressive nature, has prompted the use of molecular genetic studies in an attempt to identify susceptibility factors for the disease [4]. The eventual aim of such studies is to develop effective therapy. The biomedical literature is now filling rapidly with candidate gene studies. Unfortunately, many reports of positive results fail to be reproduced. In interpreting such studies it is important to realize that any gene contains many polymorphisms (usually single nucleotide polymorphisms or SNPs) that occur approximately every 500 base pairs [5].

The developments of coal fields in Assam were initiated in the year 1870 and are now operated in the name North Eastern Coalfields, Coal India Limited (NECF-CIL), Margherita. There are about 1.00 billion tons coal reserves estimated in this coal bearing zone of North-East (NE) India, which is 0.5% of the country's total reserve of about 200 billion tones. The generation of mine tailings in an opencast coalmine as waste rock to coal is approximately 1:14, which is a peculiar character of NE coals [6]. The environmental degradation as a result of the dumping of mine tailings known as overburden (OB) is enormous. These materials are generally dumped in an identified area as artificial hills with distinct strips, coal dust, onset of chronic bronchitis symptoms, and decreased lung function.

Chronic Obstructive Pulmonary Disease (COPD) is thought to be the result of environmental triggers in genetically susceptible individuals. Although cigarette smoking is the main environmental risk factor, only about 15% of smokers develop clinically significant disease (ATS, 1996) suggesting other influences on disease expression. This is supported by family studies showing

ancestral aggregation of spirometric abnormalities both in general population and in the relatives of patients with COPD. Moreover, differences in rate of decline of lung function between smokers suggest a gene-environment interaction. Oxidative stress occurs when Reactive Oxygen Species is produced in excess of the antioxidant defense mechanisms and results in harmful effects, including damage to lipids, proteins, and DNA. There is increasing evidence that oxidative stress is an important feature in COPD. Inflammatory and structural cells that are activated in the airways of patients with COPD produce ROS, including neutrophils, eosinophils, macrophages and epithelial cells. Mechanisms of coal dust toxicity can be arbitrarily subdivided into two major pathways, involving the production of reactive oxygen species and related antioxidant protection [7]. Both pathways are based on the key concepts of macrophage activation and lung inflammation, and are considered to be crucial mediators in the respiratory effects that are observed in chronic exposure to mineral dusts. The glutathione system is the major antioxidant mechanism in the airways. The GSTs, a super family of enzymes consisting of alpha, mu, pi, theta, kappa, zeta, sigma, omega and delta families, are critical in the conversation of many reactive electrophilic compounds to less reactive metabolites. These genes are expressed in the respiratory tract and have common functional variant alleles that result in either a total absence or a substantial change in enzyme activity.

Chronic obstructive pulmonary disease (COPD) may be influenced by race, ethnicity, gender, and genetic factors. Limited data exist that compare COPD in different racial/ethnic groups; however, the available data suggest that differences in COPD may exist [8]. Potential differences in COPD between racial/ ethnic groups include genetic and biological differences; disparities in diagnosis and treatment; increasing exposure to cigarette in nonwhite populations worldwide; and a lack of enrollment of minorities in epidemiological and clinical trials.

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