Biomass smoke exposure Increase Alveolar Macrophage in relation to respiratory Symptoms and Pulmonary Functions among the rural women in West Bengal: A Preliminary Study

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Abstract:

Alveolar macrophages (AM) are critical to the homeostasis of the inflammatory environment in the lung. To perform sputum analysis for verification of pulmonary changes in rural Indian women chronically exposed to biomass smoke during cooking. Three consecutive morning sputum samples were collected from 30 student (median age 17 years) cooking with biomass. Smears made on slides were stained with Papanicolaou and Perl's Prussian blue. Analysis of sputum samples revealed 64.76 AM/ 15 hpfs among the college students with different nuclear anomalies (cells with binuclei and micronuclei) were observed. It indicates increase of AM responsible for hypersensitivity for inflammation and allergic reaction.

Key Words: Alveolar Macrophage, sputum, pulmonary, chronically exposed, inflammation, allergic reaction,

Introduction:

More than 2 billion people of the world (Smith et al., 2004; Fullerton et al., 2008) and about 74% population of India (WHO, 2006) utilize biomass fuel as their primary source of domestic energy. Though it is the major household energy supply mostly in rural India, the poor urban dwellers have also started using it (Ramachandran et al., 2003). Biomass burning is a foremost risk to human health mainly due to the hundreds of incomplete combustion products that it generates. It have known human carcinogens like benzene, 1,3-butadine and benzo(a)pyrene (Sinha et al., 2006; Naeher et al., 2007; Straif et al., 2006; Danielsen et al., 2009), a wide spectrum of potentially health-damaging pollutants that include coarse, fine and ultrafine particles, carbon monoxide (CO), oxides of nitrogen and sulphur, transitional metals, polycyclic aromatic hydrocarbons, volatile organic compounds and bioaerosols (Smith, 2000; Morawska and Zhang, 2002). The concentration of respirable suspended particulate matter in Indian kitchens is 30 times higher than the WHO guideline while its outdoor concentration is 2.5 times more than the guideline (TERI, 1997; WHO, 1999). A typical 24-hr average concentration of

PM10 in biomass fuel-using homes ranges from 200 to 5000 g/m3 (Smith, 1993; Ezzati and Kammen, 2002) while that in major Indian cities, it ranges between 90 and 400 g/m3. This is mainly due to the absence of separate kitchens in the poor rural Indian households, left alone the presence of well-ventilated kitchens that is too far from what they can afford. Both chronic and acute exposures to air pollution have been shown to directly control the structural integrity of the respiratory system. Continued exposure can direct to sloughing off of airway cells. Microscopic examination of these exfoliated cells in spontaneously expectorated sputum provides important information about the pathophysiological changes in the lung tissue and development of lung disease including malignancy (Roby et al., 1990). Air pollution exposure was associated with cytological changes in the respiratory epithelium (Saccomanno et al., 1970), presence of abnormal columnar epithelial cells and squamous metaplasia in sputum of young adults and children (Plamenac et al., 1973, 1978), and several fold increase in the number of alveolar macrophage (AM) in sputum (Mylius and Gullvag, 1986; Kulkarni et al., 2005). Influx of activated inflammatory cells to the lung leading to pulmonary inflammation (Adler et al., 1992, 1994; Martin et al., 1997; Takizawa, 1998).Continuous insult by airborne pollutants leads to cellular damage that may direct to increase of airway diseases. Since most of the airway diseases, including cancer, take a long latent period to develop, the cellular changes are enormously helpful to identify persons at risk so that medical intervention can be initiated at an early stage for better therapeutic response. In view of this, we have made an effort to find out the cytological changes in sputum. Hence, we have undertaken the present study to explore the cytological and cytochemical changes in sputum.

Materials and methods:

Participants:

Rural College students) were recruited from Purba Medinipur Districts of West Bengal, a state in eastern India. Out of a total of 30 women, 23(median age: 19years, range:17-20) were biomass users and used cow dung cakes, crop residues, wood, etc. for cooking purposes.

This is a population based cross sectional study with age- and sex-matched comparison groups. Information on demographics (age, education, habits, occupation of the participants, average family income, cooking hours per day, cooking-years, kitchen and fuel type, family), occupation of the spouse and environmental tobacco smoke (ETS) was collected through personal interview using structured questionnaire.

Sample collection:

Sputum cytology is a cost-effective, non-invasive way of airway change detection, and is free of investigating complications. Therefore, early morning spontaneously expectorated sputum samples were collected provided with sterile plastic sputum containers. The difference between sputum and spit was explained to the subjects. The participants were instructed to rinse their mouth with saline water and to cough vigorously to expectorate sputum. The sputum was

directly collected in the given container. Four smears were made on clean glass slides from the non-transparent high viscosity part of each sample. Two samples were for cytological analysis (qualitative and quantitative evaluation) using Papanicolaou's (Pap) procedure. Each specimen was labeled, fixed (30 minutes in ethyl alcohol for Papanicolaou staining.

Sample analysis:

The smears stained according to Papanicolaou's procedure(Hughes and Dodds, 1968) were evaluated using a light microscope (Dialux 20, Leitz, Germany). At least 20 high power fields at 400 x magnification were observed and the total and differential sputum cell count was scored (Grubb, 1994). Mean of the two slides was scored for each subject.

Results:

Sputum samples were analyzed for 30 students and the number of AM in the sputum was expressed as the mean number of cells present in 15 high power fields (hpfs) of stained slides. Analysis of sputum samples revealed 64.76 AM/ 15 hpfs among the college students. Different nuclear anomalies (cells with binuclei and micronuclei) were observed in the majority of AM.

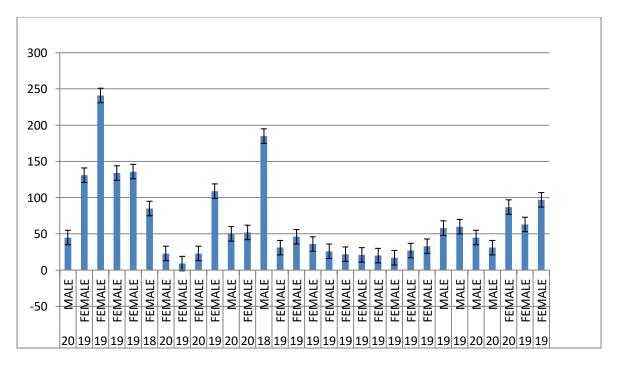


Fig: Distribution of AM among the Participants.



Fig: Study of alveolar macrophages (AM) and associated nuclear anomalies.

Discussion:

Examining sputum cytology in the participants of this study revealed elevated numbers of alveolar macrophages (AM) and airway inflammatory cells, multinucleated AMs of airway cells suggesting hypersensitivity, inflammation and abnormal cellular differentiation among exposed groups of this study. The findings suggest that air pollution exposures through respiration can alter the function of airway cells and lung defense. Abundance of iron-containing macrophages in sputum of the exposed female was also notably high suggesting covert pulmonary hemorrhage. Presence of atypical exfoliated epithelial cells in sputum is an independent risk factor for lung cancer (Fan and Watanabe, 2003). Sputum cytologic examination combined with other screening procedures therefore may play an important role in the early detection of lung cancer (Fan and Watanabe, 2003). Biomass smoke contains potent respiratory irritants; exposure may adversely affect bronchial smooth muscle tone or induce airway inflammation. By using sputum cytology as the first screening method, lung cancer can be detected at an early stage. In essence, altered sputum cytology and presence of metaplasia and dysplasia of airway epithelial cells predicts increased lung cancer risk. Thus, sputum cytology can help cancer surveillance and/or chemoprevention that may be clinically useful. There is a limitation of this study. We were not able to monitor personal exposures to particulate pollutants, nor were we able to measure all the constituent pollutants, which might have played a role in the ill health effects.

Conclusion:

In this study has shown that women continually exposed to pollution from biomass smoke have changed sputum cytology, increased pulmonary inflammation and airway oxidative stress. These may be the underlying key players involved in causing deterioration of the health of biomass-using women. Hence, intervention steps must be applied by the regulatory authorities and policy makers to ensure good health and living conditions to the poor underprivileged women of rural India.

References:

Adler, K.B., Akley, N.J. and Glasgow, W.C., 1992. Platelet-activating factor provokes release of mucin-like glycoproteins from guinea pig respiratory epithelial cells via a lipoxygenase-dependent mechanism. Am J Respir Cell Mol Biol, 6(5), pp.550-556.

Danielsen, P.H., Loft, S., Kocbach, A., Schwarze, P.E. and Møller, P., 2009. Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines. Mutation Research/Genetic Toxicology and Environmental Mutagenesis, 674(1-2), pp.116-122.

Ezzati, M. and Kammen, D.M., 2002. The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs. Environmental health perspectives, 110(11), pp.1057-1068.

Fullerton, D.G., Bruce, N. and Gordon, S.B., 2008. Indoor air pollution from biomass fuel smoke is a major health concern in the developing world. Transactions of the Royal Society of Tropical Medicine and Hygiene, 102(9), pp.843-851.

Kulkarni, N.S., Prudon, B., Panditi, S.L., Abebe, Y. and Grigg, J., 2005. Carbon loading of alveolar macrophages in adults and children exposed to biomass smoke particles. Science of the Total Environment, 345(1-3), pp.23-30.

Martin, L.D., Rochelle, L.G., Fischer, B.M., Krunkosky, T.M. and Adler, K.B., 1997. Airway epithelium as an effector of inflammation: molecular regulation of secondary mediators. European Respiratory Journal, 10(9), pp.2139-2146.

Morawska, L. and Zhang, J.J., 2002. Combustion sources of particles. 1. Health relevance and source signatures. Chemosphere, 49(9), pp.1045-1058.

Mylius, E.A. and Gullvåg, B., 1986. Alveolar macrophage count as an indicator of lung reaction to industrial air pollution. Acta cytologica, 30(2), pp.157-162.

Naeher, L.P., Brauer, M., Lipsett, M., Zelikoff, J.T., Simpson, C.D., Koenig, J.Q. and Smith, K.R., 2007. Woodsmoke health effects: a review. Inhalation toxicology, 19(1), pp.67-106.

Plameenac, P., Nikulin, A. and Pikula, B., 1973. Cytologic changes of the respiratory tract in young adults as a consequence of high levels of air pollution exposure. Acta Cytologica, 17(3), pp.241-244.

Ramachandran, G., Adgate, J.L., Pratt, G.C. and Sexton, K., 2003. Characterizing indoor and outdoor 15 minute average PM 2.5 concentrations in urban neighborhoods. Aerosol Science & Technology, 37(1), pp.33-45.

Roby, T.J., Swan, G.E., Sorensen, K.W., Hubbard, G.A. and Schumann, G.B., 1990. Discriminant analysis of lower respiratory tract components associated with cigarette smoking, based on quantitative sputum cytology. Acta cytologica, 34(2), pp.147-154.

Saccomanno, G., Saunders, R.P., Klein, M.G., Archer, V.E. and Brennan, L., 1970. Cytology of the lung in reference to irritant, individual sensitivity and healing. Acta cytologica, 14(7), pp.377-381.

Sinha, S.N., Kulkarni, P.K., Shah, S.H., Desai, N.M., Patel, G.M., Mansuri, M.M. and Saiyed, H.N., 2006. Environmental monitoring of benzene and toluene produced in indoor air due to combustion of solid biomass fuels. Science of the total environment, 357(1-3), pp.280-287.

Smith, K.R., 1993. Fuel combustion, air pollution exposure, and health: the situation in developing countries. Annual Review of Energy and the Environment, 18(1), pp.529-566.

Smith, K.R., 2000. Indoor air pollution implicated in alarming health problems In: Indoor air pollution energy and health for the indoor for the poor. Newsletter published by World Bank.

Smith, K.R., Mehta, S., Maeusezahl-Feuz, M., 2004. Indoor smoke from household solid fuels. In: Ezzati, M., Rodgers, A.D., Lopez, A.D., Murray, C.J.L. (Eds.), Comparative Quantification of Health Risks: Global and Regional Burden of Disease due to Selected Major Risk Factors. World Health Organization, Geneva, pp. 1437–1495.

Straif, K., Baan, R., Grosse, Y., Secretan, B., El Ghissassi, F. and Cogliano, V., 2006. Carcinogenicity of household solid fuel combustion and of high-temperature frying. The lancet oncology, 7(12), pp.977-978.

Takizawa, H.A.J.I.M.E., 1998. Airway epithelial cells as regulators of airway inflammation. International journal of molecular medicine, 1(2), pp.367-445.

World Health Organisation. Fuels for life: Household Energy and Health. Rehfuess, E., WHO Library Cataloguing-in-Publication Data, 2006.