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## **INDOOR AIR POLLUTION IN INDIA – A MAJOR ENVIRONMENTAL AND PUBLIC HEALTH CONCERN**

In many people's minds air pollution is associated with the contamination of urban air from automobile exhausts and industrial effluents. However, in developing countries, the problem of indoor air pollution far outweighs the ambient air pollution. There are four principal sources of pollutants of indoor air<sup>1</sup>: (i) combustion, (ii) building material, (iii) the ground under the building, and (iv) bioaerosols. In developed countries the most important indoor air pollutants are radon, asbestos, volatile organic compounds, pesticides, heavy metals, animal dander, mites, moulds and environmental tobacco smoke. However, in developing countries the most important indoor air pollutants are the combustion products of unprocessed solid biomass fuels used by the poor urban and rural folk for cooking and heating.

Approximately half the world's population and up to 90% of rural households in developing countries still rely on unprocessed biomass fuels such as wood, dung and crop residues<sup>2</sup>. A recent report of the World Health Organization (WHO) asserts the rule of 1000 which states that a pollutant released indoors is one thousand times more likely to reach people's lung than a pollutant released outdoors<sup>3</sup>. It has been estimated that about half a million women and children die each year from indoor air pollution in India<sup>4</sup>. Compared to other countries, India has among the largest burden of disease due to the use of dirty

household fuels and 28% of all deaths due to indoor air pollution in developing countries occur in India<sup>4</sup>.

The type of fuels used by a household is determined mainly by its economic status. In the energy ladder, biomass fuels namely animal dung, crop residues and wood, which are the dirtiest fuels, lie at the bottom and are used mostly by very poor people. Electricity, which is the most expensive, lies at the top of ladder and it is also the cleanest fuel. The 1991 National Census for the first time inquired about the fuel used for cooking. It revealed that about 90% of the rural population relied upon the biomass fuels like animal dung, crop residues and wood. A small portion used coal. Nation-wide about 78% of the population relied upon the biomass fuels and 3% on coal<sup>5</sup>.

### **MAJOR AIR POLLUTANTS RELEASED FROM BIOMASS COMBUSTION**

It has been estimated that more than half world's households cook their food on the unprocessed solid fuels that typically release at least 50 times more noxious pollutants than gas<sup>6</sup>. The stoves or *chullah* used for cooking are not energy efficient. The fuels are not burned completely. The incomplete combustion of biomass releases complex mixture of organic compounds, which include suspended particulate matter, carbon monoxide, poly organic material (POM), poly aromatic hydrocarbons (PAH), formaldehyde,

*etc.* The biomass may also contain intrinsic contaminants such as sulphur, trace metals, *etc.*

### **Particulates**

In recent years a large number of studies of health impact of suspended particulate air pollution have been undertaken in developing countries<sup>7</sup>. These studies show remarkable consistency in the relationship observed between changes in daily ambient suspended particulate levels and changes in mortality. Smith<sup>8</sup> estimated the health risk from exposure to particulate air pollution by applying the mean risk per unit ambient concentrations based on the results of some urban epidemiological studies<sup>9,10</sup>. The range of risk was found to be 1.2 - 4.4% increased mortality per 10 mg/m<sup>3</sup> incremental increase in concentration of respirable suspended particles (PM<sub>10</sub>). For the calculations of estimates, it was assumed that the health risk has linear relationship to exposure, the risk factors determined for urban centers of developed nations were used as standards; where the PM<sub>10</sub> data were not available, 50% of suspended particulate matter (SPM) levels were considered as equivalent. The above assumptions may add to inaccuracy already inherent in such estimates.

### **Carbon Monoxide**

Incomplete combustion of fuels produces carbon monoxide (CO). The CO and particle emission pose a serious problem when biomass fuels are used. Smith<sup>11</sup> has estimated that about 38, 17, 5 and 2 g/meal carbon monoxide is released during the household cooking, using dung, crop residues, wood and kerosene respectively. During the use of liquid petroleum gas (LPG) a negligible amount of CO is released. A study<sup>12</sup> by the National Institute of Occupational Health (NIOH), Ahmedabad reported indoor air CO levels of 144, 156, 94, 108 and 14 mg/m<sup>3</sup> air during cooking by dung, wood, coal, kerosene and LPG respectively. The short-term health effects of CO exposure are dizziness, headache, nausea, feeling of weakness, *etc.* The association between long-term exposure to carbon monoxide from cigarette smoke and heart disease and foetal development has been described by several authors<sup>13,14</sup>.

### **Poly Organic Material and Poly Aromatic Hydrocarbons**

Poly organic material is a loose term used to depict a group of chemicals having two or more rings. Of several chemicals included in this group, the PAHs have attracted interest for their possible carcinogenic effects. In addition

to PAH, azo and arino compounds have also been found to be potentially carcinogenic<sup>15</sup>. Most other categories of POM are of less environmental interest or are not found in large amounts in organic combustion products.

Polycyclic aromatic hydrocarbons constitute a large class of compounds released during the incomplete combustion or pyrolysis of organic matter<sup>16</sup>. They are often called polynuclear aromatic (PNA) because they contain three or more aromatic rings that share carbon atoms. Benzo(a)pyrene (BaP) is one of the most important carcinogen of the group. Often it is measured to indicate the presence or absence of PAHs although the relationship between BaP content and actual carcinogenicity may be weak<sup>17</sup>. Anthracene and phenanthracene are not carcinogens but methyl additions may render them carcinogenic. PAHs are activated by the hepatic microsomal enzyme system to carcinogenic forms that bind covalently to DNA<sup>18</sup>.

Study by NIOH<sup>19</sup> showed that the indoor levels of PAH (total) during use of dung, wood, coal, kerosene and LPG were 3.56, 2.01, 0.55, 0.23 and 0.13 µg/m<sup>3</sup> of air respectively. These PAH were fluorene, pyrene, chrysene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, dibenz(ah)anthracene, benzo(ghi)perylene and indeno(1,2,3-cd)pyrene. All these PAHs except the first three have been classified as possible carcinogens.

### **Formaldehyde**

Patel and Raiyani<sup>12</sup> measured levels of formaldehyde in indoor environment during cooking by different fuels. The formaldehyde mean levels were 670, 652, 109, 112 and 68 µg/m<sup>3</sup> of air for cattle dung, wood, coal, kerosene and LPG respectively. The formaldehyde is well recognized to be an acute irritant and long-term exposure can cause a reduction in vital capacity and chronic bronchitis. The formaldehyde is well known to form cross-links with biologic macro-molecules. Inhaled formaldehyde forms DNA and DNA-protein cross-links in the nasal respiratory mucosa<sup>20</sup>. The formaldehyde has been shown to be carcinogenic in a dose dependent fashion in rodents<sup>21,22</sup>. The studies done in workers occupationally exposed to formaldehyde have consistently (11 of 13 studies reviewed) shown higher incidence of leukaemia<sup>23</sup>. In an epidemiological study in U.K., significantly excess mortality from lung cancer was observed in workers exposed to high levels of formaldehyde<sup>24</sup>.

## **Mutagenic Activity of the Smoke Particulate Extract**

Microbial tests are widely used as a screening tool for assessing mutagenic potential of chemical substances. The particulate matter in the smoke generated as a result of incomplete combustion of biomass fuels contains a number of organic compounds. To evaluate their carcinogenic potential, it is necessary to screen their mutagenicity through simple and rapid microbial assay as a first step.

Ames assay is simple and sensitive enough to measure mutagenicity of air-borne particulates, so that many researchers have applied this assay to demonstrate the ambient carcinogenic and mutagenic compounds in the extractable organic matter from air-borne particulates. Mutagenic response of complex mixtures of polycyclic organic matter from the combustion of biomass energy fuels was studied using tester strains TA 98 and TA 100 of *Salmonella typhimurium* which can detect the presence of frame-shift and base-pair mutagens<sup>25</sup>. The results indicated that the organic residues of smoke particulates of wood and cattle dung fuels contained direct acting frame-shift mutagens and cattle dung contained only direct acting base-pair mutagens while indirect acting frame-shift and base-pair mutagens were found to present in smoke particulates of both the energy fuels<sup>26</sup>.

## **SPECIFIC DISEASES ASSOCIATED WITH INDOOR AIR POLLUTANT EXPOSURE**

Respiratory illness, cancer, tuberculosis, perinatal outcomes including low birth weight, and eye diseases are the morbidities associated with indoor air pollution<sup>27</sup>.

### **Respiratory Illness**

The effect of air pollutants in general would depend on the composition of the air that is inhaled which will depend on the type of fuel used and the conditions of combustion, ventilation and duration for which the inhalation occur. The most commonly reported and obvious health effect of indoor air pollutants is the increase in the incidence of respiratory morbidity. Studies by the NIOH<sup>28</sup> on the prevalence of respiratory symptoms in women using traditional fuels (biomass) (n=175) and LPG (n=99), matched for economic status and age, indicated that the relative risk (with 95% C.I.) for cough, and shortness of breath (dyspnoea) was 3.2 (1.6-6.7), and 4.6 (1.2-18.2) respectively.

## ***Childhood acute respiratory infections***

### ***Acute lower respiratory infections***

Acute respiratory infections (ARIs) are the single most important cause of mortality in children aged less than 5 years, accounting for around 3-5 million deaths annually in this age group<sup>30</sup>. Many studies in developing countries have reported on the association between exposure to indoor air pollution and acute lower respiratory infections<sup>31,32</sup>. The studies on indoor air pollution from household biomass fuel are reasonably consistent and, as a group, show a significant increase in risk for exposed young children compared with those living in households using cleaner fuels or being otherwise less exposed<sup>33</sup>. Some of the studies carried out in India have reported no association between use of biomass fuels and ARI in children. In a case-control study in children under five years of age in south Kerala<sup>34</sup>, where children with severe pneumonia as ascertained by WHO criteria were compared with those having non-severe ARI attending out patient department, the fuel used for cooking was not a significant risk factor for severe ARI. Non-severe ARI controls may represent the continuum (predecessor) of the cases themselves. Sharma *et al*<sup>35</sup> in a cross-sectional study in 642 infants dwelling in urban slums of Delhi and using wood and kerosene respectively, did not find a significant difference in the prevalence of acute lower respiratory tract infections and the fuel type.

### ***Upper respiratory tract infections and otitis media***

Studies on the relationship between indoor air pollution and acute upper respiratory infections in children both from developed<sup>36,37</sup> and developing nations<sup>38,39</sup> have not been able to demonstrate the relationship between the two. However, there is strong evidence that exposure to environmental tobacco smoke causes middle ear disease. A recent meta-analysis reported an odds ratio of 1.48 (1.08-2.04) for recurrent otitis media if either parent smoked, and one of 1.38 (1.23-1.55) for middle ear effusion in the same circumstances<sup>40</sup>. A clinic based case-control study of children in rural New York state reported an adjusted odds ratio for otitis media, involving two or more separate episodes, of 1.73 (1.03-2.89) for exposure to wood-burning stoves<sup>41</sup>.

## ***Chronic pulmonary diseases***

### ***Chronic obstructive pulmonary disease and chronic cor pulmonale***

In developed countries, smoking is responsible for over 80% of cases of chronic bronchitis and for most cases of emphysema and chronic obstructive pulmonary disease. Padmavati and colleagues<sup>42,43</sup> pointed out to the relationship between exposure to indoor air pollutants and chronic obstructive lung disease leading to chronic cor pulmonale. These studies 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. Further analysis of the cases of chronic cor pulmonale in men and women showed that chronic cor pulmonale was more common in younger women. Chronic cor pulmonale seemed to occur 10-15 years earlier in women. The prevalence of chronic cor pulmonale was lower in the southern states than the northern states of India. This is attributed to higher ambient temperatures during most part of the year allowing for greater ventilation in the houses during cooking. The authors attributed this higher prevalence of chronic cor pulmonale in women 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. Subsequent studies in India confirmed these findings<sup>44,45</sup>. Numerous studies from other countries, including ones with cross-sectional and case-control designs, have reported on the association between exposure to biomass smoke and chronic bronchitis or chronic obstructive pulmonary disease<sup>46-52</sup>.

### ***Pneumoconiosis***

Pneumoconiosis is a disease of industrial workers occupationally exposed to fine mineral dust particles over a long time. The disease is most frequently seen in miners. Cases of respiratory morbidity who did not respond to routine treatment and whose radiological picture resembled pneumoconiosis have been reported in Ladakh<sup>52,53</sup>. However, there are no industries or mines in any part of Ladakh and therefore exposure to dust from these sources was ruled out<sup>54,55</sup>. Two factors considered responsible for the development of this respiratory morbidity were (i) Exposure to dust from dust storms. In the spring dust storms occur in many parts of Ladakh. During these storms the affected villages are covered by a thick blanket of fine dust, and the inhabitants are exposed to a considerable

amount of dust for several days. The frequency, duration and severity of these dust storms vary considerably from village to village; (ii) Exposure to soot – due to the severe cold in Ladakh, ventilation in the houses is kept at a minimum. The fire place is used for both cooking and heating purposes. To conserve fuel during non-cooking periods, the wood is not allowed to burn quickly but is kept smouldering to prolong its slow heating effect. The inmates are thus exposed to high concentrations of soot.

The clinico-radiological investigations of 449 randomly selected villagers from three villages having mild, moderate and severe dust storms showed prevalence of pneumoconiosis of 2.0, 20.1 and 45.3% respectively. The chest radiographs of the villagers showed radiological characteristics which were indistinguishable from those found in miners and industrial workers suffering from pneumoconiosis. The dust concentrations in the kitchens without chimneys varied from 3.22 to 11.30 mg/m<sup>3</sup> with a mean of 7.50 mg/m<sup>3</sup>. The free silica content of these dust samples was below 1%. Dust samples sufficient to allow measurement of the dust concentrations could not be collected during the periods of dust storms. A preliminary analysis of the settled dust samples collected immediately after the storms indicated that about 80% of the dust was respirable and the free silica content ranged between 60 and 70%. Detailed statistical analysis of the data showed that the frequency of dust storms, use of chimney in the houses and age were the most important factors related to the development of pneumoconiosis<sup>56</sup>. Thus, the results of medical and radiological investigations positively established the occurrence of pneumoconiosis in epidemic proportion. Exposure to free silica from dust storms and soot from domestic fuel were suggested as the causes of pneumoconiosis. Low oxygen levels or some other factor associated with high altitude may be an important contributory factor in causation of pneumoconiosis because it has been reported that the miners working at high altitude are more prone to develop pneumoconiosis than their counterparts exposed to the same levels of dust and working in the mines at normal altitude<sup>57,58</sup>.

### **Lung Cancer**

The link between lung cancer in Chinese women and cooking on an open coal stove has been well established<sup>59-61</sup>. Smoking is a major risk factor for lung cancer, however, about two-thirds of the lung cancers were reported in non-smoking women in China<sup>62</sup>, India<sup>63</sup> and Mexico<sup>64</sup>. The

presence of previous lung disease, for example tuberculosis which is common in Indian women, is a risk factor for development of lung cancer in non-smokers<sup>65</sup>. The smoke from biomass fuels contain a large number of compounds such as poly aromatic hydrocarbons, formaldehyde, *etc.* known for their mutagenic and carcinogenic activities, but there is a general lack of epidemiological evidence connecting lung cancer with biomass fuel exposure. The factors associated with rural environment may have a modulating effect on the occurrence of lung cancer and therefore the low incidence of lung cancer in Indian women should not lead to a final conclusion of no link between biomass exposure and lung cancer. It may be concluded that at present there is limited evidence of indoor exposure from coal fires leading to lung cancer and there is no evidence for the biomass fuels. Further investigations are needed to reach definite conclusions.

### **Pulmonary Tuberculosis**

Mishra *et al*<sup>66</sup> recently reported the association between use of biomass fuels and pulmonary tuberculosis on the basis of analysis of data collected on 260,000 Indian adults interviewed during the 1992-93 National Family Health Survey. Persons living in households burning biomass fuels were reported to have odd ratio of 2.58 (1.98-3.37) compared to the persons using cleaner fuel, with an adjustment for confounding factors such as separate kitchen, indoor overcrowding, age, gender, urban or rural residence and caste. The analysis further indicated that, among persons aged 20 years and above, 51% of the prevalence of active tuberculosis was attributed to smoke from cooking fuel. However, this study has inherent weakness that the cases of tuberculosis were self reported. There is strong possibility of false reporting as no investigation was done to confirm the reliability of the reporting. Gupta and Mathur<sup>67</sup> have reported similar findings from northern India. This study did not control for the confounding factors except for age.

There is experimental evidence to show that the exposure to wood smoke may increases susceptibility of the lungs to infections. Exposure to smoke interferes with the mucociliary defences of the lungs<sup>68</sup> and decreases several antibacterial properties of lung macrophages, such as adherence to glass, phagocytic rate and the number of bacteria phagocytosed<sup>69,70</sup>. Chronic exposure to tobacco smoke also decreases cellular immunity, antibody production and local bronchial immunity, and there is increased susceptibility to infection and cancer<sup>71</sup>. Indeed, tobacco smoke has been associated with tuberculosis<sup>72,73</sup>.

Although the evidence in favour of tuberculosis associated with biomass fuel exposure is extremely weak, there is a theoretical possibility of such an association and considering the public health importance of the problem further experimental and epidemiological studies are necessary.

### **Cataract**

During cooking particularly with biomass fuels, air has to be blown into the fire from time to time especially when the fuel is moist and the fire is smouldering. This causes considerable exposure of the eyes to the emanating smoke. In a hospital-based case-control study in Delhi the use of liquefied petroleum gas was associated with an adjusted odds ratio of 0.62 (0.4-0.98) for cortical, nuclear and mixed, but not posterior sub capsular cataracts in comparison with the use of cow dung and wood<sup>74</sup>. An analysis of over 170,000 people in India<sup>75</sup> yielded an adjusted odds ratio for reported partial or complete blindness of 1.32 (1.16-1.50) in respect of persons mainly using biomass fuel compared with other fuels after adjusting for socio-economic, housing and geographical variables; there was a lack of information on smoking, nutritional state, and other factors that might have influenced the prevalence of cataract. It is believed that the toxins from biomass fuel smoke are absorbed systematically and accumulate in the lens resulting in its opacity<sup>77</sup>. The growing evidence that environmental tobacco smoke causes cataracts is supportive<sup>76,77</sup>.

### **Adverse Pregnancy Outcome**

Low birth weight (LBW) is an important public health problem in developing nations attributed mainly to under-nutrition in pregnant women. Low birth weight has serious consequences including increased possibility of death during infancy. Exposure to carbon monoxide from tobacco smoke during pregnancy has been associated with LBW. Levels of carbon monoxide in the houses using biomass fuels are high enough to result in carboxyhaemoglobin levels comparable to those in smokers<sup>78,79</sup>. In rural Guatemala, babies born to women using wood fuel were 63 g lighter than those born to women using gas and electricity, after adjustment for socio-economic and maternal factors<sup>80</sup>. A study carried out in Ahmedabad reported an excess risk of 50% of stillbirth among women using biomass fuels during pregnancy<sup>81</sup>. An association between exposure to ambient air pollution and adverse pregnancy outcome has been widely reported<sup>82-84</sup>.

Considering the association of LBW with a number of disease conditions later in life, there is a need for further studies.

## **INTERVENTION**

Adequate evidence exists to indicate that indoor air pollution in India is responsible for a high degree of morbidity and mortality warranting immediate steps for intervention. The intervention programme should include (i) Public awareness; (ii) Change in pattern of fuel use; (iii) Modification in stove design; (iv) Improvement in the ventilation; and (v) Multisectoral approach.

### **Public Awareness**

The first and the most important step in the prevention of illnesses resulting from biomass fuels is to educate the public, administrators and politicians to ensure their commitment and promoting awareness of the long-term health effects on the part of users. This may lead to people finding ways of minimizing exposure through better kitchen management and infant protection.

### **Change in Pattern of Fuel Use**

The choice of fuel is mainly a matter of availability, affordability and habit. The gohar gas plant which uses biomass mainly dung has been successfully demonstrated to produce economically viable quantities of cooking gas and manure. Recently, the Government of Andhra Pradesh has introduced a programme called the Deepam Scheme to subsidize the cylinder deposit fee for women from households with incomes below the poverty line to facilitate the switch from biomass to LPG. Such schemes will encourage the rural poor to use cleaner fuels. The use of solar energy for cooking is also recommended.

### **Modification in Stove Design**

Use of cleaner fuels should be the long-term goal for the intervention. Till this goal is achieved, efforts should be made to modify the stoves to make them fuel efficient and provide them with a mechanism (*eg* chimney) to remove pollutants from the indoor environment. Several designs of such stoves have been produced. NIOH study showed significant decrease in levels of SPM, SO<sub>2</sub>, NO<sub>x</sub> and formaldehyde with specially designed smokeless stoves in comparison with traditional cooking stoves<sup>12</sup>. However, they have not been accepted widely. Large scale acceptance of improved stoves would require determined efforts. The most important barriers to new stove introduction are not technical but social<sup>85</sup>.

## **Improvement in Ventilation**

In many parts of the country poor rural folk are provided with subsidized houses under various government/international agencies aided schemes. Ventilation in the kitchen should be given due priority in the design of the houses. In existing houses, measures such as putting a window above the cooking stove and providing cross ventilation through the door may help in diluting the pollution load.

## **Multisectoral Approach**

Effective tackling of indoor air pollution requires collaboration and commitment between agencies responsible for health, energy, environment, housing and rural development.

## **CONCLUSIONS**

Indoor air pollution caused by burning traditional fuels such as dung, wood and crop residues causes considerable damage to the health of particularly women and children. There is evidence associating the use of biomass fuel with acute respiratory tract infections in children, chronic obstructive lung diseases, and pneumoconiosis in the residents of Ladakh villages. Lung cancer has been found to be associated with the use of coal in China, however, there is no evidence associating it with the use of biomass fuels. Cataract and adverse pregnancy outcome are the other conditions shown to be associated with the use of biomass fuels. The association of tuberculosis and chronic lung infections with the use of biomass fuels has not been proved.

Finally, there is enough evidence to accept that indoor air pollution in India is responsible for a high degree of morbidity and mortality warranting immediate steps for intervention. The first and the most important step in the prevention of illnesses resulting from the use of biomass fuels is to educate the public, administrators and politicians to ensure their commitment for the improvement of public health. There is utmost requirement to collect better and systematic information about actual exposure levels experienced by households in different districts and climatic zones and develop a model for predicting the exposure levels based on fuel use and other household data therein (exposure atlas) to protect the health of children, women and elderly persons.

## References

1. Behera, D. Health effects of indoor air pollution due to domestic cooking fuels. *Indian J Chest Dis Allied Sci* 37: 237, 1995.
2. World Resources Institute, United Nations Environment Programme, United Nations Development Programme and World Bank,. 1998-99 *World Resources: A Guide to the Global Environment*. Oxford University Press, Oxford, 1998-99.
3. *Health and Environment in Sustainable Development. Five Years After the Earth Summit*. World Health Organization, Geneva, p.84, 1997.
4. Smith, K.R. Indoor air pollution implicated in alarming health problems. In: *Indoor Air Pollution – Energy and Health for the Poor*. Newsletter published by World Bank, p.1, 2000.
5. *National Family Health Survey (MCH and Family Planning): India 1992-93*. International Institute for Population Sciences, Mumbai, 1995.
6. Smith, K.R. Indoor air quality and the population transition. In: *Indoor Air Quality*. Ed. H. Kasuga. Springer Verlag, Berlin, p.448, 1990.
7. *Air Quality Guidelines for Europe*. World Health Organization European, Regional Office for Europe, Copenhagen, Series No.23, 1997.
8. Smith, K.R. Indoor air pollution in developing countries: Growing evidence of its role in the global disease burden. In: *Indoor Air '96 : Proceedings of 7th International Conference on Indoor Air and Climate*. Ed. K. Ikeda, and T. Iwata. Institute of Public Health, Tokyo, p.33, 1996.
9. *A Methodology for Estimating Air Pollution Health Effects*. WHO/EHG/96.5. World Health Organization, Geneva, 1996.
10. Hong, C.J., Corvalan, C. and Kjellstrom, T. Air Pollution. In: *Quantifying Global Health Risks: The Burden of Diseases Attributed to Selected Factors*. Eds. C.J.L. Murray and A.D. Lopez. Harvard University Press, Cambridge M.A., 1997.
11. Smith, K.R. Managing the risk transmission. *Toxicol Ind Health* 7: 319, 1991.
12. Patel, T.S. and Raiyani, C.V. Indoor air quality: Problems and perspectives. In: *Energy Strategies and Green House Gas Mitigation*. Ed. P.R. Shukla. Allied Publishers, New Delhi, p.72, 1995.
13. *The Health Consequences of Smoking – Cardiovascular Diseases. A Report of the Surgeon General*. US Department of Health and Human Services, Rockville, MD, 1983.
14. Wynder, E.L. Forum: Workshop on carbon monoxide and cardiovascular disease. *Prev Med* 8: 261, 1979.
15. U.S. National Research Council Committee on Medical and Biologic Effects of Environmental Pollutants. *Particulate Organic Polycyclic Matter*. National Academy of Sciences, Washington, D.C., 1972.
16. International Programme on Chemical Safety. *Environmental Health Criteria 202: Selected Non-heterocyclic, Polycyclic Aromatic Hydrocarbons*. World Health Organization, Geneva, p.63, 1998.
17. Schwarz-Miller, J., Rom, W.N. and Brandt-Rauf, P.W. Polycyclic aromatic hydrocarbons. In: *Environmental and Occupational Health*. Ed. R.N. Rom. Little Brown & Co., London, p.873, 1991.
18. Perera, F.P., Hemmlinki, K., Young, T.L., Brenner, D., Kelly, G. and Santella, R.M. Detection of poly-aromatic hydrocarbon-DNA adducts in white blood cells of foundry workers. *Cancer Res* 48: 228, 1988.
19. Raiyani, C.V., Jani, J.P., Desai, N.M., Shah, S.H., Shah, P.G. and Kashyap, S.K. Assessment of indoor exposure to polycyclic aromatic hydrocarbons for urban poor using various types of cooking fuels. *Bull Environ Contam Toxicol* 50: 757, 1993.
20. Swenberg, J.A., Barrow, M. and Starr, T.B. Nonlinear biologic responses to formaldehyde and their implications for carcinogenic risk assessment. *Carcinogenesis* 4: 945, 1983.
21. National Institute for Occupational Safety and Health. *Formaldehyde: Evidence of Carcinogenicity*. U.S. Department of Health and Human Services, Cincinnati, 1981.
22. Swenberg, J.A., Kerns, W.D., Mitchell, R.I., Gralla, E.J. and Parkov, K.L. Induction of squamous cell carcinomas of the rat nasal cavity by inhalation exposure to formaldehyde vapour. *Cancer Res* 40: 3398, 1980.
23. Blair, A., Saracci, I., Stewart, P.A., Hayes, R. and Shy, C. Epidemiologic evidence of the relationship between formaldehyde exposure and cancer. *Scand J Work Environ Health* 16: 381, 1990.
24. Acheson, E.D., Barnes, H.R., Gardner, M.J., Osmond, C., Pannet, B. and Taylor, C.P. Formaldehyde process, workers and lung cancer. *Lancet* i: 1066, 1984.
25. Maron, D.M. and Ames, B.N. Revised methods for the *Salmonella* mutagenicity test. *Mutat Res* 113: 172, 1983.
26. Gokani, V.N., Doctor, P.B., Raiyani, C.V., Kulkarni, P.K. and Saiyed, H.N. Monitoring the presence of mutagenic compounds from smoke particulates of biomass energy fuels by Ames Assay. Compendium of Research Papers presented at the National Conference on Environmental Pollution Prevention and Control for Healthy Living at the University College of Medical Sciences and GTB Hospital, New Delhi, November 21, 2000.
27. Bruce, N., Perez-padilla, R. and Albalak, R. Indoor air pollution in developing countries: A major environmental and public health challenge. *Bull World Health Organ* 78: 1078, 2000.
28. Domestic source of air pollution and its effects on respiratory system of house wives in Ahmedabad. In: *Annual Report*. National Institute of Occupational Health, Ahmedabad, p.32, 1982.
29. Behera, D., Dash, S. and Yadav, S.P. Blood carboxyhaemoglobin in women exposed to different cooking fuels. *Thorax* 46: 344, 1991.
30. Stansfield, S. and Shepherd, D. Acute respiratory infection. In: *Disease Control Priorities in Developing Countries*. Eds. D. Jameson, W. Mosle, A. Mesham and J. Bobadilla. Oxford University Press, Oxford, p.67, 1993.
31. Robin, L.F. Less, P.S.J. Winger, M., Steinhoff, M., Moulten, L.H., Santoshom, M. and Correa, A. Wood-burning stoves and lower respiratory illness in Navajo children. *Paediatr Infect Dis J* 15: 859, 1996.

32. Armstrong, J.R. and Campbell, H. Indoor air pollution exposure and lower respiratory infections in young Gambian children. *Int J Epidemiol* 20: 424, 1991.
33. Smith, K., Samet, J.M., Romieu, I. and Bruce, N. Indoor air pollution in developing countries and acute respiratory infections in children. *Thorax* 55: 518, 2000.
34. Shah, N., Ramakutty, V., Premila, P.G. and Sathy, N. Risk factors for severe pneumonia in children in south Kerala: A hospital-based case-control study. *J Trop Paediatr* 40: 201, 1994.
35. Sharma, S., Sethi, G.R., Rohtagi, A., Chaudhary, A., Shankar, R., Bapna, J.S., Joshi, V. and Sapir, D.G. Indoor air quality and acute lower respiratory infection in Indian urban slums. *Environ Health Perspect* 106: 291, 1998.
36. Samet, J.M., Marbury, M.C. and Spengler, J.D. Health effects and sources of indoor air pollution. Part I. *Am Rev Respir Dis* 136: 1486, 1987.
37. Samet, J.M., Marbury, M.C. and Spengler, J.D. Health effects and sources of indoor pollution (state of the art). *Am Rev Respir Dis* 137: 221, 1988.
38. Anderson, H.R. Respiratory abnormalities in Papua New Guinea children. The effects of locality and domestic wood smoke pollution. *Int J Epidemiol* 7: 63, 1978.
39. Boleiz, J.S. Domestic air pollution from biomass burning in Kenya. *Atmos Environ* 23: 1677, 1989.
40. Strachan, D.P. and Cook, D.G. Parental smoke, middle ear disease and adenotonsillectomy in children. *Thorax* 53: 50, 1998.
41. Daigler, G.E., Markello, S.J. and Cummings, K.M. The effect of indoor air pollutants on otitis media and asthma in children. *Laryngoscope* 101: 293, 1991.
42. Padmavati, S. and Pathak, S.N. Chronic cor pulmonale in Delhi. *Circulation* 20: 343, 1959.
43. Padmavati, S. and Joshi, B. Incidence and etiology of chronic cor pulmonale in Delhi: A necropsy study. *Dis Chest* 46: 457, 1964.
44. Malik, S.K. Exposure to domestic cooking fuels and chronic bronchitis. *Indian J Chest Dis Allied Sci* 27: 171, 1985.
45. Behera, D., Jindal, S.K. and Malhotra, H.S. Ventilatory function in nonsmoking rural Indian women using different cooking fuels. *Respiration* 61: 89, 1994.
46. Qureshi, K. Domestic smoke pollution and prevalence of chronic bronchitis/asthma in a rural area of Kashmir. *Indian J Chest Dis Allied Sci* 36: 61, 1994.
47. Dutt, D. Effect of indoor air pollution on the respiratory system of women using different fuels for cooking in an urban slum of Pondicherry. *Natl Med J India* 9: 113, 1996.
48. Ellegard, A. Cooking fuel smoke and respiratory symptoms among women in low income areas in Maputo. *Environ Health Perspect* 104: 980, 1996.
49. Master, K.M. Air pollution in New Guinea. Cause of chronic pulmonary disease among stone-age natives in the highlands. *J Am Med Assoc* 228: 1635, 1974.
50. Pandey, M.R. Prevalence of chronic bronchitis in rural community of the hill region of Nepal. *Thorax* 39: 331, 1984.
51. Pandey, M.R. Domestic smoke pollution and chronic bronchitis in a rural community of the hill region of Nepal. *Thorax* 39: 337, 1984.
52. Norboo, T. Domestic pollution and respiratory illness in a Himalayan village. *Int J Epidemiol* 20: 749, 1991.
53. Norboo, T., Angchuk, P.T. and Yahya, M. Silicosis in a Himalayan village population: Role of environmental dust. *Thorax* 46: 341, 1991.
54. Saiyed, H.N., Shirma, Y.K., Sadhu, H.G., Norboo, T., Patel, P.D., Venkaiah, K. and Kashyap, S.K. Non-occupational pneumoconiosis at high altitude villages in central Ladakh. *Br J Industr Med* 48: 825, 1991.
55. Saiyed, H.N., Sharma, Y.K., Norboo, T., Sadhu, H.G., Majumdar, P.K. and Kashyap, S.K. Clinico-radiological and PFT profile in non-occupational pneumoconiosis. *Indian J Industr Med* 38: 148, 1992.
56. Venkaiah, K., Saiyed, H.N., Sharma, Y.K., Sadhu, H.G. and Kashyap, S.K. Multiple logistic model to assess the non-occupational pneumoconiosis risk. *Indian J Occup Health* 36: 103, 1993.
57. Odinaev, F.I. The characteristics of development and course of pneumoconiosis under the conditions of a mountain climate. *Grig Tr Prof Zabol* 7: 13, 1992 (Russian).
58. Odinaev, F.I. The mechanism of the formation of pneumoconiosis under high altitude conditions. *Grig Tr Prof Zabol* 7: 11, 1992 (Russian).
59. Smith, K.R. and Liu, Y. Indoor air pollution in developing countries In: *Epidemiology of Lung Cancer. Lung Biology in Health and Disease*. Ed. J Samet. Marcel Dekker, New York, p.151, 1993.
60. Mumford J.L. Human exposure and dosimetry of polycyclic aromatic hydrocarbons in urine from Xuan Wei, China with high lung cancer mortality associated with exposure to unvented coal smoke. *Carcinogenesis* 16: 3031, 1995.
61. Xu, Z., Kjellstrom, T., Xu, X., Lin, U. and Daqlan, Y. *Air Pollution and Its Effects in China*. Ed. B. Chen. World Health Organisation, Geneva, p.47, 1995.
62. Gao, Y.T. Risk factors for lung cancer among nonsmokers with emphasis on life-style factors. *Lung Cancer* 14(Suppl.1): S39, 1996.
63. Gupta, R.C. Primary bronchogenic carcinoma: Clinical profile of 279 cases from mid-west Rajasthan. *Indian J Chest Dis Allied Sci* 40: 109, 1998.
64. Medina, F.M. Primary lung cancer in Mexico City. A report of 1019 cases. *Lung Cancer* 14: 185, 1996.
65. Wu, A.H. Previous lung disease and risk of lung cancer among lifetime nonsmoking women in the United States. *Am J Epidemiol* 141: 1023, 1995.
66. Mishra, V.K., Retherford, R.D. and Smith K.R. Biomass cooking fuels and prevalence of tuberculosis in India. *Int J Infect Dis* 3: 119, 1999.

67. Gupta, B.N. and Mathur, N. A study of the household environmental risk factors pertaining to respiratory disease. *Energy Environ Rev* 13: 61, 1997.
68. Houtmeyers, E., Gosselink, R., Gayan-Ramirez, G. and Decramer, M. Regulation of mucociliary clearance in health and disease. *Eur Respir J* 13: 1177, 1999.
69. Fick, R.B., Pant, E.S., Merrill, W.W., Reynolds, H.Y. and Loke, J.S.O. Alterations in the antibacterial properties of rabbit pulmonary macrophages exposed to wood smoke. *Am Rev Respir Dis* 129: 78, 1984.
70. Beck, B.D. and Brain, J.D. Prediction of the pulmonary toxicity of respirable combustion products from residential wood and coal stoves. *Proceedings of the Residential Wood and Coal Combustion Special Conference*. Air Pollution Control Association, Pittsburg, 1982 (SPU5).
71. Johnson, J.D., Houchens, D.P., Kluwe, W.M., Craig, D.K. and Fisher, G.L. Effects of mainstream and environmental tobacco smoke on the immune system in animals and humans: A review. *Crit Rev Toxicol* 20: 369, 1990.
72. Altet, M.N., Alcaide, J., Plans, P., Taberner, J.L., Salto, E., Folguera, L.I. and Salleras, L. Passive smoking and risk of pulmonary tuberculosis in children immediately following infection. A case-control study. *Tuber Lung Dis* 77: 537, 1996.
73. McKenna, M.T. The association between occupation and tuberculosis. A population-based survey. *Am J Respir Crit Care Med* 154: 587, 1996.
74. Mohan, M., Spereduto, R.D. and Angra, S.K. India-US case control study of age-related cataracts. *Arch Ophthalmol* 107: 670, 1989.
75. Mishra, V.K., Retherford, R.D. and Smith, K.R. Biomass cooking fuels and prevalence of blindness in Indian. *J Environ Med*. 2000 (In Press).
76. Shalini, V.K. Lothra, M., and Srinivas, I. Oxidative damage to the eye lens caused by cigarette smoke and fuel wood condensates. *Indian J Biochem Biophys* 31: 262, 1994.
77. West, S. Does smoke get in your eyes? *J Am Med Assoc* 268:1025, 1992.
78. Dary, O., Pineda, O. and Belizan, J. Carbon monoxide in dwellings in poor rural areas of Guatemala. *Bull Environ Contam Toxicol* 26: 24, 1981.
79. Behera, D., Dash, S. and Malik, S.K. Blood carboxyhaemoglobin levels following acute exposure to smoke of biomass fuels. *Indian J Med Res* 88: 522, 1998.
80. Boy, E., Bruce, N. and Delgado, H. Birth weight and exposure to kitchen wood smoke during pregnancy. In: *Child and Adolescent Health*. World Health Organization, Geneva, 2000 (In Press).
81. Mavlankar, D.V., Trivedi, C.R. and Gray, R.H. Levels and risk factors for perinatal mortality in Ahmedabad, India. *Bull World Health Organ* 69: 435, 1991.
82. Dejmek, J., Selevan, S.G., Benes, I., Solansky, I. and Sram, R.J. Foetal growth and maternal exposure to particulate matter during pregnancy. *Environ Health Perspect* 107: 475, 1999.
83. Wang, X., Ding, H., Ryan, L. and Xu, X. Association between air pollution and low birth weight: A community based study. *Environ Health Perspect* 105: 514, 1997.
84. Bobak, M. and Leon, D. Pregnancy outcomes and outdoor air pollution: An ecological study in districts of the Czech Republic 1986-88. *Occup Environ Med* 56: 539, 1999.
85. Gopalan, H.N.B. and Saxena, S. Domestic environment and health of women and children. In: *Biofuels*. Tata Energy Research Institute, New Delhi, p.77, 1999.

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