DOMESTIC SMOKE POLLUTION AND RESPIRATORY DISEASES IN NEPAL

MR Pandey[1]

Founder, Mrigendra-Samjhana Medical Trust, Kathmandu, Nepal

ABSTRACT
The main respiratory diseases caused by Domestic Smoke Pollution (DSP) are chronic bronchitis and chronic obstructive lung diseases. In our study conducted in Nepal, the increasing trend of the prevalence of chronic bronchitis as the hours of exposure to domestic smoke pollution increased (even after elimination of the age effect and also among the non-smokers) established the definite role of domestic smoke pollution in causing this disease. Another study showed significant correlation of DSP and airway obstruction in smokers but the association was not significant in non-smokers. Significant correlation has also been found of DSP with acute respiration infection in studies done in Nepal and several other countries. Apart from these, there is evidence that DSP can also cause interstitial lung disease, tuberculosis and asthma. Further research work is recommended in these areas.

INDEX TERMS
Domestic smoke pollution, Chronic bronchitis

INTRODUCTION
Indoor air pollution in rural locations in developing countries, where combustion of biomass fuels are the principal source, has now been recognized as an important and widespread problem (Pandey and Smith, 1989). Evidence from a number of countries, including Nepal (Pandey, Neupane and Gautam, 1990), India (Behera, Das and Malik, 1988), Papua New Guinea (Anderson, 1979) (Cleary and Blackburn, 1968), and Kenya (Boleji, Ruigewaard, Hoek et al., 1989) suggests that domestic fires for cooking and heating purpose can cause significant pollution with gaseous substances and suspended particulates in the home environment. It is estimated that about half the world's households cook daily with biomass fuels. Most of this cooking is done indoors with unvented stoves. The highest exposures are probably experienced by women, infants and young children. Measured levels of air pollution in these houses of developing countries greatly exceed indoor air and outdoor concentration found in developed countries (Smith, 1979) – personal communication.

Several studies have implicated domestic smoke pollution responsible for respiratory diseases. Of these the evidences are quite definite for chronic obstructive pulmonary disease (COPD) in adults and acute respiratory infection (ARI) in children. There is evidence of relationship with tuberculosis and interstitial lung disease. There is also some suggestion that domestic smoke pollution may be related to asthma. This is an important area for further research.

1. Chronic bronchitis and chronic obstructive lung disease (COLD) in adults
Chronic obstructive pulmonary disease (COPD) is a leading cause of death and disability of human life around the world. Early study done in Papua New Guinea (Anderson, 1979) showed a possible relationship between Domestic Smoke Pollution and Chronic Bronchitis.

[1] Contact author e-mail: msmt@healthnet.org.np

Definitive study was conducted in Nepal to determine the distribution and magnitude of these disease in different geographical regions of the country and to identify factors responsible for them (Pandey, Basnyat and Neupane, 1988). Four different sites were selected for this purpose, these sites were urban Kathmandu and the Sundarijal and Bhadrabas villages of Kathmandu district from the rural hill region, Parasauni of Bara district from the plain region, and Chandannath of Jumla district from the mountain region of the country. Analysis of data from different sites showed the crude prevalence rate of chronic bronchitis to be 11.3 percent in urban Kathmandu, 13.1 percent in Parasauni, 18.3 percent in Sundarijal and Bhadrabas and 30.9 percent in Chandannath.

An interesting fact of this study is that there was a similar high rate of prevalence of chronic bronchitis in women as in men, which is in contrast with the findings of most of the other studies which show a lower prevalence rate among women (Pandey, 1984). On the other hand, higher percentages of women as compared to men in all the four sites were either non-smokers or smoked lesser quantities. Such a state of affairs can be explained in light of the fact that a significantly higher proportion of women as compared to men in all the four areas were exposed to domestic smoke pollution for longer hours (Pandey, 1988). In Sundarijal and Bhadrabas the increasing trend of the prevalence of chronic bronchitis as the hours of exposure to DSP increased (even after elimination of the age effect and also among the non-smokers) further establishes the definite role of domestic smoke pollution in causing this disease (Pandey, 1984).

Table 1. Prevalence of chronic bronchitis according to time per day spent near fire place

Average time/day (H)	Women			
	N	Cases	Prevalence (%)	
			Crude	Age adjusted
NON-SMOKERS				
0-0.9	64	4	6.25	6.99
1-1.9	142	6	4.23	4.91
2-3.9	85	29	15.67	17.68
4+	134	27	20.14	19.87
Total	525	66	12.57	13.76

(X^2 = 19.96, DF, P < 0.001)

In the urban Kathmandu and terai sites also, significant association between prevalence of this disease and hours of exposure to domestic smoke pollution was found (Pandey 1988). A large number of people exposed to domestic smoke pollution for more than eight hours in Chandannath of mountain region gave an opportunity to see the effect of longer hours of exposure versus shorter hours of exposure. A significant correlation was seen even beyond eight hours or exposure in both the sexes. Hence, there is a dose-response relationship between domestic smoke pollution and chronic bronchitis.

These studies tend to show that even non-smoking women who have cooked on biomass stoves for many years exhibit a higher prevalence of chronic bronchitis than might be expected in similar women who have had less use of biomass stove. Indeed, in rural Nepal nearly 15% of non-smoking women (20 years and older) had chronic bronchitis, a high rate for non-smokers.

Domestic smoke pollution and respiratory function among women
Another study was also conducted in Nepal to see the effect of domestic smoke pollution on respiratory function. The selected sample comprised of 6 groups - 25 subjects each of 3 exposure levels to domestic smoke pollution i.e. 0-1.0 hrs, 2-3.9 hrs and 4-6 hrs per day amongst smokers and non-smokers.

Variation of age, height, arm span and weight between the three different levels of exposure to domestic smoke in both the smokers and non-smokers were compared and results revealed no significant variation in all the variables mentioned above. So any differences noted on the respiratory function values in the three exposures could be attributed to the effect of domestic smoke pollution. There was a fall of mean FVC, FEVI and FMEF 25-75 as duration of exposure increased. This decline was found to be statistically significant (FVC and FEVI $p<0.025$, FMEF: $p<0.01$) amongst the smokers but not amongst the non-smokers. The results were again analyzed after adjusting for age and height as these two variables may have a compounding effect. Similar results were found even after adjusting for age and height (Pandey, Regmi, Neupane et al., 1985).

Some other recent quantitative studies have also shown positive correlation between biomass fuel consumption and chronic bronchitis (CB) and chronic air ways obstruction (CAO) diagnosed by internationally accepted criteria. In a case control study conducted in Mexico city (Perez-Padilla, 1996) found after multi variate analysis odds ratios for high exposure group (>200 hours years). CB = 15 (95% CI 5.6 – 40) and CB/CAO = 75 (95%CI 18-306)) and for exposure group 100 –200 hours per year it was found to be 9.3 and 10.3 respectively. Similarly a cross sectional study from Lucknow India (Gupta, 1997) using UKMRC and ATS questionnaire and after adjusting for age and sex, odds ratio was found to be 7.9 (95% CI 2.1 - 21.8)

A study in Ladakh (Narboo, Yahya, Bruce et al., 1990) has demonstrated an association between increase in exposure to pollution in individual subjects (measured by exhaled air carbon monoxide) between summer and winter, and a fall of about 10% in lung function (FEVI, but not in FVC) over the same period.

2. Acute Respiratory Infection (ARI)
ARI is the largest single disease category in the world contributing 8.5% of the total global burden of ill health (Murray and Lopez, 1996). More than five million children under five years of age die in the world each year from ARI and 80% of these deaths occur in developing countries. Several studies have shown positive correlation of indoor air pollution with ARI morbidity and mortality.

Studies done in developing countries
The most interesting studies on ARI and domestic smoke pollution (DSP) now available were done in South Africa, Nepal, Zimbabwe, Gambia, Nigeria, India, Malaysia, Papua New Guinea and Brazil. A study in Nepal conducted in 1990 measured the pollutant level and established dose response relationship between the exposure to domestic smoke pollution and acute respiratory infection in children. An average time spent near the fireplace per day by the children is taken as a measure of the extent of exposure to the domestic smoke pollution. A positive correlation was found between all grades of ARI and hours of exposure to domestic smoke pollution among infant below 2 years of age.

Table 2. ARI episodes according to time spent per day near the fireplace (February-June 1984) (Pandey, Neupane and Gautam, 1989)

Average time per day (in h)	0-1 year				1-2 years			
	ARI episodes				ARI episodes			
	N	I	II	III/IV	N	I	II	III/IV
0-0.9	33	40 (1.21)	9 (0.27)	1 (0.03)	17	42 (2.47)	4 (0.24)	1 (0.06)
1-1.9	90	120 (1.33)	33 (0.37)	6 (0.07)	64	118 (1.84)	15 (0.23)	8 (0.13)
2-3.9	94	170 (1.81)	48 (0.15)	16 (0.17)	95	179 (1.88)	32 (0.34)	11 (0.12)
4+	16	30 (1.88)	13 (0.81)	9 (0.56)	40	81 (2.03)	31 (0.78)	11 (0.28)
Total	233	360 (1.55)	103 (0.44)	32 (0.14)	216	420 (1.94)	82 (0.38)	31 (0.14)

Grades I, II, III and IV are according to Goroka classification of ARI.

There are many other studies indicating that indoor air pollution is a definite cause of ARI in children. In a study of 500 children in Gambia, girls under 5 years carried on their mother's back during cooking (in smoky cooking huts) were found to have 6 times higher risk of ARI, a substantially higher risk factor than parental smoking. There was no significant risk, however, in young boys, the risk estimate were adjusted by using multivariate analysis to minimize the effect of some other factors like ethnic group, socioeconomic status, access to health care and nutrition (Armstrong and Campbell, 1991) Sumargono (Sumargono, 1988) and Achmadi (Achmadi, 1990) from Indonesia observed during a three-month cohort study of acute respiratory infection (ARI) among children under five, that the episodes of acute respiratory tract infection were closely related to independent variables such as the degree of indoor air quality, number of people per square meter (density), and socio-economic status. A South African hospital based study of 132 infants with severe lower respiratory tract disease found that 70 per cent had a history of heavy smoke exposure from cooking and/or heating fires (Kossove, 1982).

However, a recent hospital based study of children under five from Kerala, India failed to show a relationship of pneumonia with stove smoke after multivariate analysis (Shah, Ramankutty, Premila and et al., 1994). There have also been reports from Malaysia and Papua New Guinea, which do not show any effects of domestic smoke on ARI. But these studies were done with school-age children who are at relatively low risk. Most of these studies, provide extremely suggestive indication that exposure to wood smoke is an important risk factor of pneumonia.

K. Smith on his informal 'meta-analysis' of 10 studies in developing countries concludes that the exposed young children have 2-3 times more risk of serious ARI than unexposed children (Smith et al., 1999).

3. Interstitial lung disease

In our study in mountainous regions of Jumla, where there is prolonged intense exposure to wood smoke, we found .5% prevalence of interstitial lung disease. There was no other obvious cause to account for the same. The diagnosis was confirmed by chest x-ray and spirometry, which showed purely restrictive disease pattern (Pandey, Basnyat and Neupane, 1988). Recently, a study in Mexico has also shown similar interstitial lung disease with pulmonary hypertension associated with prolonged intense exposure to wood smoke. It affected mostly country women in their 60s. the chest x-ray showed a diffuse bilateral reticulo endothelial pattern and pulmonary function tests showed mixed restrictive and obstructive pattern. The patient had severe Pulmonary Arterial Hypertension in which, as in other chronic lung disease, chronic alveolar hypoxia may play the main pathogenetic role. However, Pulmonary Arterial Hypertension in wood-smoke inhalation-associated lung disease

(WSIALD) appears to be more severe than in other forms of interstitial lung disease and tobacco related COPD (Sandoval et al., 1993).

4. Tuberculosis

Although domestic smoke has been suggested to be a risk factor of tuberculosis, there has been no direct evidence of the same. Recently Mishra and Smith have shown on the basis of analysis of 260, 162 persons age 20 and over in India's 1992-93 National Family Health Survey, that persons living in households that primarily use biomass for cooking fuel have substantially higher prevalence of active tuberculosis than persons living in households that use cleaner fuels (odds ratio = 3.56, 95% confidence interval = .282-4.50). This effect is reduced somewhat when availability of a separate kitchen, house type, indoor crowding, age, gender, urban or rural residence, education, religion, caste or tribe, and geographic region are statistically controlled OR = 2.58, 95% CI = 1.98-3.37) (Mishra, Retherford and Smith, 1999). This is an important study but has to be replicated in other parts of the world.

5. Asthma

The incidence of asthma is rapidly increasing in the developing world. In our recent study of 2330 schoolchildren in Kathmandu, Nepal, the risk of asthma was higher in subjects exposed to passive smoking [OR 1.9 (95% CI 1.0 to 3.9)] and indoor use of smoky fuels [OR 2.2 (95% CI 1.0 to 4.5)] (Melsom T, Brinch T, Hessen J O and et al. 2001).

REFERENCES

Achmadi U F, 1990, The risk of analyses of the adverse effect of air pollutants (CO and PB) to the urban pollution of Jakarta. *Journal of Indonesia Medical Association.*

Anderson H R, 1979, Chronic lung disease in the Paupa New Guinea Highlands. *Thorax,* 34: 647-653.

Armstrong J R M and Campbell H, 1991, Indoor air pollution exposure and lower respiratory infection in young Gambian children. *Int. J. Epidemiology,* 20 (2)M 424-428.

Behera D, Dash S, Malik S K, 1988, Blood carboxyhemoglobin levels following acute exposure to smoke and biomass fuel. *Indian J Med Res,* 88:522-524.

Boleji J S, Ruigewaard P, Hoek F et al., 1989, Domestic air pollution from biomass burning in Kenya. *Atmospheric Environment,* 23: 1677-1681.

Cleary G J and Balckburn C R B, 1968, Air pollution in native huts in the highlands of New Guinea. *Arch Env Health,* 17: 785-794.

Gupta B N et al., 1997, A study of household environmental risk factors pertaining to respiratory disease. E*nergy Environment Monitor,* 13 (2): 61-67.

Melsom T, Brinch T, Hessen J O and et al. 2001, Asthma and indoor air environment in Nepal. *Thorax,* Vol. 56, No. 6, pp 477-481.

Mishra V, Retherford R D, 1997, Cooking smoke increases the risk of acute respiratory infections in children. *National Family Health Survey Bulletin # 8,* Mumbai, and East-West Center, Honolulu: International Institute for Population Sciences.

Mishra V, Retherford R D and Smith K, 1999, Biomass cooking fuels and prevalence of tuberculosis in India. *International J of Infections Diseases,* Vol.3, No. 3.

Murray CJL, Lopez AD, 1996, Global burden of disease, Cambridge MA: *Harvard University Press.*

Narboo T, Yahya M, Bruce N G et al., 1990, Domestic pollution and respiratory illness in a Himalayan village. *Int J. Epidemiol.*

Pandey M R, 1984, Prevalence of chronic bronchitis in a rural community of the hill region of Nepal. *Thorax,* 39: 331-336.

Pandey M R, 1984, Domestic smoke pollution and chronic bronchitis in a rural community of the hill region of Nepal. *Thorax,* 39: 337-339.
Pandey M R, Regmi H N, Neupane R P et al., 1985, Domestic smoke pollution and respiratory function in rural Nepal. *Tokai J. Exp. Clin. Med.*, 10, 4, 471-481.
Pandey M R, Basnyat B and Neupane R P, 1988, Chronic bronchitis and cor pulmonale in Nepal. Monograph: *Mrigendra Medical Trust.*
Pandey M R, Neupane R P and Gautam A, 1989, Domestic smoke pollution and acute respiratory infection in rural community of the hill region of Nepal. *Environment International,* Vol. 15.
Pandey M R, Smith K R, Bolejo J S M, et al., 1989, Indoor air pollution in developing countries and acute respiratory infection in children. *Lancet,* Feb 28, 427-429.
Pandey M R, Neupane R P, Gautam A et al., 1990, The effectiveness of smokeless stoves in reducing indoor air pollution in a rural hill region of Nepal. *Mountain Research and Development,* Vol.10, No.4, pp. 313-320.
Perez-Padilla R et al., 1996, Exposure to biomass smoke and chronic airways disease in Mexican women a case-control study. *Am J Respir Crit Care Med,* 154 (3 Pt 1): 701-706.
Shah N, Ramankutty V, Premila P G et al., 1994, Risk factors for severe pneumonia in children in South Kerela: a hospital bases case-control study. *J Trop Ped* 40:201-206.
Smith K R, 1987, Biofuels, air pollution and health. *A global review, New York; Plenum.*
Smith K et al., 1999, Air pollution and acute lower respiratory infections in children, draft submitted to *Thorax.*