

Published Literature on Woodsmoke and Cancer

Summary

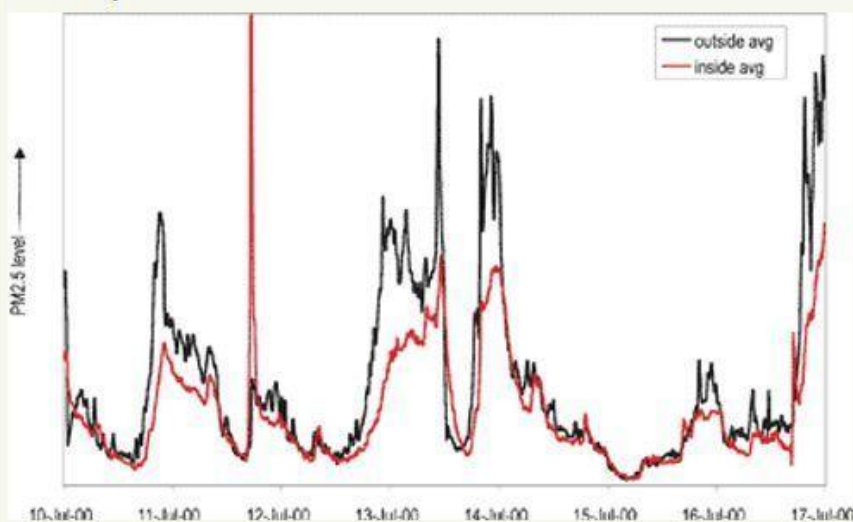
- In Launceston the increase in *indoor* PM_{2.5} levels from *outdoor* woodsmoke was similar to that from living with a 1-pack per day smoker.
- Woodsmoke contains several known human carcinogens, including benzene, benzo[a]pyrene and formaldehyde. Burning 1 kg of wood in a modern Australian heater produces more benzo[a]pyrene than in the smoke from 27,000 cigarettes and more benzene and formaldehyde than in the smoke of 6,000 cigarettes.
- In human cell lines, woodsmoke caused more DNA damage than traffic-generated PM per unit mass and was found to induce lung cancer in mice. Oncogene mutations in human patients with advanced non-small cell lung cancer were associated with exposure to wood smoke as well as tobacco smoking. Organic extracts of ambient particulate matter containing substantial quantities of woodsmoke were found to be 30- fold more potent than extracts of cigarette smoke condensate in a mouse skin tumour induction assay.
- Predominant wood (fuel) users in North America and Europe had a 21% higher risk of lung cancer.
- In developing countries, exposure to woodsmoke is associated with lung, mouth and throat cancers, and even cervical cancers in women who test positive for the HPV virus.
- In OECD countries, lung cancer increases by 14% for every additional 10 ug/m³ of annual PM_{2.5} exposure. Woodsmoke seems to be as dangerous as other PM_{2.5}, e.g. causing more DNA damage in human cell lines than traffic-generated particles. It is the largest single-source of PM_{2.5} emissions in most Australian cities - 67% of PM_{2.5} emissions in Canberra (where 3.9% of households have woodheaters) and 34% in Sydney (where 4.3% use wood as the main form of heating). With 7626 lung-cancer deaths in 2007, reducing wood heater use in major cities from 4% to 2% of households would reduce PM_{2.5} emission by about 15%, leading to about 2.1% fewer lung cancer deaths and eventually saving about 160 lives per year.

Indoor PM_{2.5} from woodsmoke in Australian Rural Towns is similar to living with a 1-pack per day smoker, even in households without wood heating

Neas et al. (1994) reported that living with a 1-pack per day smoker increases PM_{2.5} exposure by about 30 ug/m³. PM_{2.5} are so small they behave like gases, infiltrating homes in similar way to the air we need to breathe. CSIRO research (Fig 115 below) shows that indoor PM_{2.5} levels in winter track outdoor levels. Apart from the occasional spike from an indoor source, a large proportion of the indoor woodsmoke pollution is from outdoors. In Launceston, where woodsmoke accounts for the vast majority of PM_{2.5} pollution, indoor PM_{2.5} concentrations averaged 47.3 ug/m³ in winter, compared to 15.9 in summer (NHT 2004). The difference of 31.4 ug/m³ is more than expected from living with a 1-pack per day smoker.

Figure 115 shows simultaneous readings over four days of outdoor and indoor airborne fine particulate matter as measured by light scattering. In general, the indoor air can be modelled by assuming that peaks in the outdoor air will diffuse indoors. This means that the indoor peaks are generally lower, and later, than the outdoor peaks. Nevertheless, as shown during 11 July, there are times when indoor peaks are not related to outdoor events.

Figure 115: Nephelometer readings of outdoor and indoor particles over seven days.



Source: CSIRO Atmospheric Research

<http://www.environment.gov.au/soe/2001/publications/theme-reports/atmosphere/atmosphere04-6a.html>

Tumour potency – wood vs tobacco smoke

According to a recent review Naeher et al. 2007: “*Organic extracts of ambient particulate matter (PM) containing substantial quantities of woodsmoke are 30-fold more potent than extracts of cigarette smoke condensate in a mouse skin tumor induction assay* (Cupitt et al., 1994)”

Developed countries – 21% increased risk of lung cancer from burning wood

Hosgood et al. (2010) concludes: “*Predominant wood (fuel) users in North American and European countries (OR = 1.21; 95% CI, 1.06–1.38) experienced higher risk of lung cancer.*” This increase in lung cancer for using wood fuel in North America and Europe was similar to the estimated increase of 20-40% for environmental tobacco smoke.

Developing countries – 433% increase in lung cancer from cooking with wood

In third world countries, exposure to biomass smoke is associated with an even higher risk.

- Delgado et al. (2005) reported that approximately 38.7% of lung cancer patients had an association with wood smoke exposure. Adenocarcinoma was present in 46.7% of these patients
- Behera, D. and T. Balamugesh (2005) studied indoor air pollution as a risk factor for lung cancer in women. Among non-smokers, out of all the cooking fuels, the risk of development of lung cancer was highest for biomass fuel exposure with an odds ratio of 5.33 (95% CI 1.7-16.7). Use of mixed fuels was associated with a lesser risk (OR= 3.04, 95% CI 1.1-8.38). In multivariate logistic regression analysis biomass fuel exposure was still significant with OR of 3.59 (95% CI 1.07-11.97) even after adjusting for smoking and passive smoking.
- Non-smoking Mexican women with long periods of wood smoke exposure had about double the risk of lung adenocarcinoma (Hernández-Garduño et al. 2004).

Mouth and Throat cancer

Pintos, J., E. L. Franco, et al. (1998) concluded: “*The association of use of wood stoves with cancers of the upper aero-digestive tract is genuine and unlikely to result from insufficient control of confounding. Due to its high prevalence, use of wood stoves may be linked to as many as 30% of all cancers occurring in the region*”

Cervical Cancer

Velema, J., A. Ferrera, et al. (2002) concluded that “*Burning wood in the kitchen increases the risk of cervical neoplasia in HPV-infected women in Honduras.*” The odds ratio was 2.3 for 25-34 years of exposure, to wood smoke increasing to 9.5 for 35+ years compared with women who had 1-14 years of exposure (p = 0.017). Two recent studies have linked air pollution, and a third environmental tobacco smoke, to breast cancer. The study that linked PM2.5 exposure to breast cancer suggested that higher air pollution exposure at birth may alter DNA methylation, which may increase levels of E-cadherin, a protein important to the adhesion of cells, a function that plays an essential role in maintaining a stable cellular environment and assuring healthy tissues. More generally, chemicals in both wood and tobacco smoke can pass into the bloodstream where they can affect many different tissues.

Known human carcinogens in wood smoke

Burning 1 kg firewood creates more benzene, benzo[a]pyrene & formaldehyde than 6,000 cigarettes

Benzene, benzo[a]pyrene and formaldehyde are listed by the International Agency for Research on Cancer as known human carcinogens. Chemical analysis of the smoke from Australian wood heaters shows that burning 1 kg of wood produces more benzo[a]pyrene than in the smoke from 27,000 cigarettes and more benzene and formaldehyde than in the smoke from 6,000 cigarettes – see <http://woodsmoke.3sc.net/wood-vs-cigarette-smoke>.

Group 2A (probably carcinogenic to humans) chemicals includes dibenz[a,h]anthracene. Burning 1 kg of wood in an Australian wood heater produces more dibenz[a,h]anthracene than in the smoke from 225,000 cigarettes (<http://woodsmoke.3sc.net/wood-vs-cigarette-smoke>).

Although exposure to household combustion of biomass fuel (mainly wood) is listed only as a probable human carcinogen, this is probably due to a paucity of research. As the evidence mounts, e.g. a recent conference paper “Wood Smoke Stimulates Human Lung Cancer Cell Growth” (Tafur et al. 2011), it seems likely that wood smoke will be listed as a known human carcinogen.

Damage to human DNA from woodsmoke

A Norwegian study (Danielsen et al. 2009) compare the genotoxicity of wood smoke particulate matter, authentic traffic-generated particles, and standard reference material (SRM2975) of diesel exhaust particles in human A549 lung epithelial and THP-1 monocytic cell lines. DNA damage was measured as strand breaks (SB) and formamidopyrimidine DNA glycosylase (FPG) sites by the comet assay.

Wood smoke particulate matter generated more DNA damage than traffic-generated PM per unit mass in human cell lines, possibly due to the high level of polycyclic aromatic hydrocarbons in WSPM. The authors suggested that “*exposure to wood smoke particles might be more hazardous than PM collected from vehicle exhaust with respect to development of lung cancer*”.

Another study Danielsen et al. (2011) analyzed air samples from a Danish village where most homes used wood-burning stoves, and compared them to background particles outside the smoke area. The researchers concluded that wood smoke particulates were more powerful in damaging DNA, activating genes linked to disease and weakening immune response than were the background air pollution particles from a variety of sources. “*In this study, we found that wood smoke ... has similar toxicity and effects on DNA as that of vehicle exhaust particles,*” said University of Copenhagen researcher Steffen Loft, who led the project.

Professor Loft also found that in rural areas, *wood smoke particles were contaminating nearby fields of crops which, if eaten, caused DNA damage to liver cells.*

Woodsmoke induces lung cancer in mice

Mice were placed in an environment in which they inhaled coal smoke and wood smoke in indoor air for 15 to 19 months (Liang et al. 1988). The incidences of lung cancer in the control group and wood group were 17.0% (29/171), 45.8% (81/177).

Human population exposure and estimated number of cancers

Air pollution significantly increases the risk of lung cancer. For example Table 2 of Pope et al. (2002) show that an increase of 10 ug/m³ of PM2.5 pollution increases lung cancers by an average of 14%.

PM2.5 in Australian cities averages about 10 ug/m³, compared to less than 3 ug/m³ in country areas when no woodsmoke is present. A crude estimate is therefore that at least 70% of PM2.5 air pollution is man-made, and that the increase in lung cancer mortality from PM2.5 pollution is about 7*1.4 = 9.8%, i.e. about 740 deaths per year.

Wood smoke represents a surprisingly large proportion of PM2.5 emissions. For example, the NSW DECC emissions inventory for Sydney reports that domestic-commercial solid fuel combustion, virtually all of

Table ES10 Ten largest anthropogenic sources of particulate matter < 2.5 µm

Source Group	Source Type	Particulate Matter < 2.5 µm	
		Annual Emissions (tonnes/year)	Proportion of Annual Anthropogenic Emissions (%)
Sydney			
Domestic-Commercial	Solid Fuel Combustion	4,503	34.3
Off-Road Mobile	Industrial Off-Road Vehicles and Equipment	1,152	8.78
On-Road Mobile	Exhaust Emissions Light Duty - Diesel	840	6.40
Industrial	Crushing, grinding or separating works	807	6.15
On-Road Mobile	Exhaust Emissions Passenger Cars - Petrol	797	6.08
On-Road Mobile	Exhaust Emissions Heavy Duty Commercial - Diesel	681	5.19
Industrial	Ceramics production (excluding glass)	606	4.62
Industrial	Other land-based extraction	418	3.18
Commercial	Poultry Farming (Meat)	237	1.81
Industrial	Petroleum refining	237	1.80
All	Other	2,848	21.7

which is emissions from wood heaters comprises 34.3% of all PM2.5 emissions. ABS data shows that burning wood is the main form of heating in 4.3% of households, with another 2.2% using wood as a secondary source.

In Canberra, 67% of PM2.5 pollution is from wood heating, which is used by only 3.9% of households.

The average for all Australian capital cities is 4.2% (and 19.1% in the rest of Australia). Thus, woodsmoke could account for up to a third of all air-pollution related cancers per year.

Breast cancer. Two recent studies have linked air pollution to breast cancer; a third linked environmental tobacco smoke to breast cancer.

1) [Exposure to air pollution early in life and when a woman gives birth to her first child may alter her DNA and may be associated with premenopausal breast cancer later in life.](#) A study, by researchers at

the University at Buffalo, is based on data from the Western New York Exposures and Breast Cancer (WEB) study, which collected information from 1,170 women with recently diagnosed breast cancer and 2,116 healthy women who lived in New York's Erie and Niagara counties between 1996 and 2001. "*The investigation looked for an association between exposure to pollution and alterations to DNA that influence the presence or absence of key proteins. Such genetic changes are thought to be major contributors to cancer development and progression, including at very early stages,*" said lead investigator Katharine Dobson, MPH – <http://www.sciencedaily.com/releases/2011/04/110420125508.htm>

2) **[The risk of breast cancer - the second leading cause of death from cancer in women – was linked to NO₂ pollution](#)** by researchers from The Research Institute of the MUHC (RI MUHC; Dr. Mark Goldberg), McGill University (Drs. Goldberg, Dan Crouse and Nancy Ross), and Université de Montréal (Dr. France Labrèche), (Crouse et al. 2010). "*We've been watching breast cancer rates go up for some time,*" says study co-author Dr. Mark Goldberg, a researcher at The RI MUHC. "*Nobody really knows why, and only about one third of cases are attributable to known risk factors. Since no-one had studied the connection between air pollution and breast cancer using detailed air pollution maps, we decided to investigate it.*" <http://www.sciencedaily.com/releases/2010/10/101006104003.htm>.

The paper talks about emissions, including "*gases, particles, volatile organic compounds, and polycyclic aromatic hydrocarbons (PAHs), many of which are accepted or potential carcinogens. Benzene ... is an accepted human carcinogen (International Agency for Research on Cancer 1987), and has been shown to cause mammary carcinomas in rodents (Huff et al. 1989; Maltoni et al. 1988, 1989). Exposure to other aromatic hydrocarbons including kerosene, toluene, and xylenes, has also produced increased rates of mammary cancers in female rats (Maltoni et al. 1997). Aromatic hydrocarbons are lipophilic and may therefore reach elevated concentrations in breast tissue and promote carcinogenesis in the cells of the breast (Morris and Seifter 1992)*". Wood heaters emit NO₂ and most of the other toxic chemicals listed above. The best estimate is about 0.44 grams of NO, 0.18 grams of NO₂ and 0.62 g of NO_x are emitted for every kg of firewood burned (Gras 2002) NO_x mission limits for petrol cars are 0.15 g/km (Euro-3), 0.08 g/km (Euro-4) and 0.07 g/km (Euro-5).

[Montreal banned the installation of new woodheaters because they account for 47% of Montreal's PM2.5 pollution](#), including 45 winter days from Nov 2008 to March 2009 with PM2.5 levels exceeding the standard - <http://woodsmoke.3sc.net/policies-elsewhere> If NO₂ is a proxy for benzene and other aromatic hydrocarbons, it seems likely that [woodsmoke exposure as well as exposure to the same and similar chemicals from traffic emissions](http://woodsmoke.3sc.net/woodheater-car-comparison) is a contributory factor <http://woodsmoke.3sc.net/woodheater-car-comparison>.

3) **Mexican women who do not smoke but are exposed to smoking are at three times higher risk for breast cancer than non-smoking women not exposed to passive smoking** (Chilian-Herrera et al. 2010) Although this focussed on environmental tobacco smoke, [research suggests that the pollutants in wood smoke have a much greater biological impact in the human body than cigarette smoke](http://woodsmoke.3sc.net/wood-vs-cigarette-smoke) (<http://woodsmoke.3sc.net/wood-vs-cigarette-smoke>) or even automobile exhaust on a per unit weight basis (Danielsen, Loft et al. 2009; Danielsen, Møller et al. 2011.)

References

- Behera, D., Balamugesh, T., 2005. Indoor air pollution as a risk factor for lung cancer in women. *JAPI* 53.
- Chilian-Herrera, O.L., Cantor, K.P., Hernández-Ramírez, R.U., López-Carrillo, L., 2010. [Passive smoking increases the risk of breast cancer among pre- and postmenopausal Mexican women](#) Cancer Health Disparities Conference, Sept. 30-Oct.3, American Association for Cancer Research, Abstract A99
- Crouse, D.L., Goldberg, M.S., Ross, N.A., Chen, H., Labrèche, F., 2010. Postmenopausal Breast Cancer Is Associated with Exposure to Traffic-Related Air Pollution in Montreal, Canada: A Case-Control Study. *Environ Health Perspect* 118.
- Danielsen, P., Loft, S., Kocbach, A., Schwarze, P., Møller, P., 2009. Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines. *Mut. Res.-Genetic Toxicology and Environmental Mutagenesis* 674, 116-122.
- Danielsen, P.H., Møller, P., Jensen, K.A., Sharma, A.K., Wallin, H.k., Bossi, R., Autrup, H., Møhlhave, L., Ravanat, J.-L., Briedé, J.J., de Kok, T.M., Loft, S., 2011. Oxidative Stress, DNA Damage, and Inflammation Induced by Ambient Air and Wood Smoke Particulate Matter in Human A549 and THP-1 Cell Lines. *Chemical Research in Toxicology* 24, 168-184.
- Delgado, J., Martinez, L., Sanchez, T., Ramirez, A., Iturria, C., Gonzalez-Avila, G., 2005. Lung Cancer Pathogenesis Associated With Wood Smoke Exposure*. *Chest*, Am Coll Chest Phys. **128**: 124-131.
- Gras, J., 2002. Emissions from Domestic Solid Fuel Burning Appliances., Environment Australia Technical Report No. 5, March 2002.
- Hernández-Garduño, E., Brauer, M., Pérez-Neria, J., Vedal, S., 2004. Wood smoke exposure and lung adenocarcinoma in non-smoking Mexican women. *The International Journal of Tuberculosis and Lung Disease* 8, 377-383.

- Hosgood, H.D., III, Boffetta, P., Greenland, S., Lee, Y.-C.A., McLaughlin, J., Seow, A., Duell, E.J., Andrew, A.S., Zaridze, D., Szeszenia-Dabrowska, N., Rudnai, P., Lissowska, J., Fabianov, E.r., Mates, D., Bencko, V., Foretova, L., Janout, V., Morgenstern, H., Rothman, N., Hung, R.J., Brennan, P., Lan, Q., 2010. In-Home Coal and Wood Use and Lung Cancer Risk: A Pooled Analysis of the International Lung Cancer Consortium. *Environ Health Perspect* 118.
- Liang, C., Quan, N., Cao, S., He, X., Ma, F., 1988. Natural inhalation exposure to coal smoke and wood smoke induces lung cancer in mice and rats. *Biomed Environ Sci*.
- Naeher, L., Brauer, M., Lipsett, M., Zelikoff, J., Simpson, C., Koenig, J., Smith, K., 2007. Woodsmoke Health Effects: A Review. *Inhalation Toxicology* 19, 67-106.
- Neas, L., Dockery, D., Ware, J., Spengler, J., Ferris, B., Jr., Speizer, F., 1994. Concentration of indoor particulate matter as a determinant of respiratory health in children. *Am. J. Epidemiol.* 139, 1088-1099.
- NHT, 2004. Technical Report No 8. Personal Monitoring of Selected VOCs: The Contribution of Woodsmoke to Exposure, CSIRO Atmospheric Research, for the Natural Heritage Trust. (available at <http://www.deh.gov.au/atmosphere/airquality/publications/pem/>).
- Pope, C.A., 3rd, Burnett, R.T., Thun, M.J., Calle, E.E., Krewski, D., Ito, K., Thurston, G.D., 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* 287, 1132-41.
- Tafur, K.B., Ritzenthaler, J.D., Roman, J., Accinelli, R.A., 2011. Wood Smoke Stimulates Human Lung Cancer Cell Growth. *Am. J. Respir. Crit. Care Med.* 183, A3903-.

Appendix – Abstracts of Selected Papers

1) In-Home Coal and Wood Use and Lung Cancer Risk: A Pooled Analysis of the International Lung Cancer Consortium, H. Dean Hosgood, III et al. *Environ Health Perspect* [118\(12\) Dec 2010](#)

Background: Domestic fuel combustion from cooking and heating is an important public health issue because roughly 3 billion people are exposed worldwide. Recently, the International Agency for Research on Cancer classified indoor emissions from household coal combustion as a human carcinogen (group 1) and from biomass fuel (primarily wood) as a probable human carcinogen (group 2A).

Objectives: We pooled seven studies from the International Lung Cancer Consortium (5,105 cases and 6,535 controls) to provide further epidemiological evaluation of the association between in-home solid-fuel use, particularly wood, and lung cancer risk.

Methods: Using questionnaire data, we classified subjects as predominant solid-fuel users (e.g., coal, wood) or nonsolid-fuel users (e.g., oil, gas, electricity). Unconditional logistic regression was used to estimate the odds ratios (ORs) and to compute 95% confidence intervals (CIs), adjusting for age, sex, education, smoking status, race/ethnicity, and study center.

Results: Compared with nonsolid-fuel users, predominant coal users (OR = 1.64; 95% CI, 1.49–1.81), particularly coal users in Asia (OR = 4.93; 95% CI, 3.73–6.52), and **predominant wood users in North American and European countries (OR = 1.21; 95% CI, 1.06–1.38) experienced higher risk of lung cancer.** The results were similar in never-smoking women and other subgroups.

Conclusions: Our results are consistent with previous observations pertaining to in-home coal use and lung cancer risk, support the hypothesis of a carcinogenic potential of in-home wood use, and point to the need for more detailed study of factors affecting these associations.

2) Delgado, J., L. Martinez, et al. (2005). Lung Cancer Pathogenesis Associated With Wood Smoke Exposure*. *Chest, Am Coll Chest Phys.* 128: 124-131.

Background: Tobacco is considered the most important cause of lung cancer, but other factors could also be involved in its pathogenesis. The aim of the present work was to establish an association between wood smoke exposure and lung cancer pathogenesis, and to analyze the effects of wood smoke on p53 and murine double minute 2 (MDM2) protein expression.

Design: Blood samples were obtained from 62 lung cancer patients, 9 COPD patients, and 9 control subjects. Of the 62 lung cancer patients, 23 were tobacco smokers (lung cancer associated with tobacco [LCT] group), 24 were exposed to wood smoke (lung cancer associated with wood smoke [LCW] group), and 15 could not be included in these groups. Western blot assays were performed to identify the presence of p53, phospho-p53, and murine double minute 2 (MDM2) isoforms in plasma samples. Densitometric analysis was used to determine the intensity of p53, phospho-p53, and MDM2 bands.

Results: **Approximately 38.7% of the lung cancer patients examined had an association with wood smoke exposure, most of them women living in rural areas. Adenocarcinoma was present in 46.7% of these patients.** The p53 and phospho-p53 proteins were significantly increased in LCW samples ($56,536.8 \pm 4,629$ densitometry units [DU] and $58,244.8 \pm 7,492$ DU, respectively [\pm SD]), in comparison with the other groups.

The 57-kD MDM2 isoform plasma concentration was very high in LCW and LCT samples ($75,696.4 \pm 11,979$ DU and $78,551.7 \pm 11,548$ DU, respectively). MDM2-p53 complexes were present in a high concentration in control and COPD subjects. This allows p53 degradation and explains the low concentrations of p53 found in these groups. MDM2-phospho-p53 complexes were observed in COPD but not in the other samples. This correlates with the low concentration of p53 observed in the COPD group ($13,657 \pm 2,012$ DU), and could explain the different clinic evolution of this smoker population in comparison with the LCT subjects.

Conclusion: This study suggests that there is a possible association of lung cancer with wood smoke exposure. Likewise, our findings demonstrate that wood smoke could produce similar effects on p53, phospho-p53, and MDM2 protein expression as tobacco.

3) Pintos, J., E. L. Franco, et al. (1998). "Use of wood stoves and risk of cancers of the upper aero-digestive tract: a case-control study." *Int J Epidemiol* 27(6): 936-40.

BACKGROUND: Incidence rates for cancers of the upper aero-digestive tract in Southern Brazil are among the highest in the world. A case-control study was designed to identify the main risk factors for carcinomas of mouth, pharynx, and larynx in the region. We tested the hypothesis of whether use of wood stoves is associated with these cancers. **METHODS:** Information on known and potential risk factors was obtained from interviews with 784 cases and 1568 non-cancer controls. We estimated the effect of use of wood stove by conditional logistic regression, with adjustment for smoking, alcohol consumption and for other sociodemographic and dietary variables chosen as empirical confounders based on a change-in-estimate criterion. **RESULTS:** After extensive adjustment for all the empirical confounders the odds ratio (OR) for all upper aero-digestive tract cancers was 2.68 (95% confidence interval [CI] : 2.2-3.3). Increased risks were also seen in site-specific analyses for mouth (OR = 2.73; 95% CI: 1.8-4.2), pharyngeal (OR = 3.82; 95% CI: 2.0-7.4), and laryngeal carcinomas (OR = 2.34; 95% CI: 1.2-4.7). Significant risk elevations remained for each of the three anatomic sites and for all sites combined even after we purposefully biased the analyses towards the null hypothesis by adjusting the effect of wood stove use only for positive empirical confounders. **CONCLUSIONS:** The association of use of wood stoves with cancers of the upper aero-digestive tract is genuine and unlikely to result from insufficient control of confounding. Due to its high prevalence, use of wood stoves may be linked to as many as 30% of all cancers occurring in the region.

4) Behera, D. and T. Balamugesh (2005). "Indoor air pollution as a risk factor for lung cancer in women." *JAPI* 53.

Objectives: Tobacco smoking is the most common risk factor for lung cancer. But a significant proportion of lung cancer occurs in non-smokers. Indoor pollution due to domestic fuels has been recently implicated as a causative agent in lung cancer especially in women. We conducted a case control study to find out the role of indoor air pollution due to domestic cooking fuels in Indian women.

Methods: In a case control study 67 women with proven lung cancer were recruited. Forty-six females having a non-malignant respiratory disease constituted the control group. The patients and controls were asked about the exposure in various cooking fuels using a questionnaire.

Result: There were 50(74.6%) non-smokers and 17(25.4%) smokers among the female cancer cases ($p=0.016$). Adenocarcinoma was the commonest histological type of malignancy ($n=26$, 38.8%) in the whole group and was the predominant form in the nonsmoking females. Tobacco smoking was the most important risk factor for lung cancer with OR of 4.87 (95% CI 1.34-17.76). **Among non-smokers out of all the cooking fuels the risk of development of lung cancer was highest for biomass fuel exposure with an odds ratio of 5.33 (95% CI 1.7-16.7). Use of mixed fuels was associated with a lesser risk (OR= 3.04, 95% CI 1.1-8.38). In multivariate logistic regression analysis biomass fuel exposure was still significant with OR of 3.59 (95% CI 1.07-11.97) even after adjusting for smoking and passive smoking.**

5) Wood smoke exposure and lung adenocarcinoma in non-smoking Mexican women.

[Hernández-Garduño E, Brauer M, Pérez-Neria J, Vedal S.](#) *Int J Tuberc Lung Dis.* 2004 Mar;8(3):377-83.

Department of Tuberculosis Control, British Columbia Centre for Disease Control, Vancouver, British Columbia, Canada. eduardo.hernandez@bccdc.ca

Abstract

OBJECTIVE: To determine the association between long-term exposure to wood smoke from cooking and lung adenocarcinoma in non-smoking Mexican women.

METHODS: We reviewed records of hospitalized patients at a chest referral hospital in Mexico City and

identified 113 histologically proven lung adenocarcinoma cases in non-smoking women. Four control groups of non-smoking women were also selected: 99 patients with pulmonary tuberculosis (PTB), 110 with interstitial lung disease (ILD), 64 with miscellaneous pulmonary conditions (MISC), and the three control groups combined (COMB) (n = 273).

RESULTS: Exposure was assessed on the basis of questionnaire responses at the time of hospital admission. Exposure to wood smoke for more than 50 years, but not for shorter periods, was associated with lung cancer after adjusting for age, education, socio-economic status and environmental tobacco smoke (ETS) exposure. Adjusted odds ratios from the multivariable logistic regression models were 1.4 (95% CI 0.6-2.0) for cases vs. TB controls, 1.9 (95% CI 0.9-4.0) for cases vs. ILD controls, 2.6 (95% CI 1.0-6.3) for cases vs. MISC controls and 1.9 (95% CI 1.1-3.5) for cases vs. COMB controls.

CONCLUSION: These findings suggest that long-term exposure to wood smoke from cooking may contribute to the development of lung cancer.

6) Velema, J., A. Ferrera, et al. (2002). "Burning wood in the kitchen increases the risk of cervical neoplasia in HPV-infected women in Honduras." *International Journal of Cancer* 97(4).

There is suggestive evidence that the use of wood for cooking increases the risk of invasive cervical cancer. We investigated this association in women with cervical neoplasia in Honduras. Women aged 20-64 years with cervical intraepithelial neoplasia (CIN) grade I (n = 44), CIN II (n = 36) or CIN III (n = 45) were recruited from screening programs in Tegucigalpa City and each was matched by age and clinic to 2 controls (241 total) without cervical abnormalities. The clinics selected women of low socioeconomic status. Cervical scrapes were tested for the presence of human papillomavirus (HPV) DNA using a general primer set directed against the L1 open reading frame, and HPV genotyping was performed. Odds ratios (ORs) were computed through conditional logistic regression; p-values were from tests for linear trend of risk with increasing exposure. HPV DNA was detected in 48% of women with CIN I, 67% with CIN II and 89% with CIN III. The ORs were 1.5, 2.5 and 38.3 respectively. At univariate analysis, age at first intercourse was consistently lower among cases than controls. Risk was reduced by 50% or more in all 3 CIN classes when initiation of sexual activity at age 20 years or older was compared with initiation before age 16 years (p = 0.013 for CIN I). No effect was observed for smoking, oral contraceptives or previous cytologic screening. Effects for number of sexual partners, parity, age at first pregnancy and education were in the expected directions but never persisted after adjustment for HPV. Chronic exposure to wood smoke significantly increased the risk of CIN III (p = 0.022). However, women who said No when asked if they ever used wood in the kitchen had a higher risk than those with low or intermediate exposure. This was taken as evidence that the initial screening question had either been misunderstood or that answers were biased. Restricting the analysis to women who reported exposure yielded positive associations in all CIN classes with for CIN III ORs of 2.3 for 25-34 and 9.5 for 35+ years compared with women who had 1-14 years of exposure (p = 0.017). A multivariate analysis of the complete dataset (n = 366) allowed for separate ORs for HPV in each CIN class. Inclusion of age at first intercourse significantly improved this model (p = 0.021). Adding exposure to wood smoke further improved the model only if an interaction between woodsmoke and HPV was allowed for. If, as the data suggest, it was assumed that wood smoke had its effect among HPV-positives only, there was a significant linear dose-response relationship between exposure to woodsmoke and risk of CIN (p = 0.026). This association was independent of other risk factors including education, parity and number of sexual partners. ORs in the final model were 0.37 for age at first intercourse 20 years or higher and 5.69 for more than 35 years of exposure to wood burning in the kitchen. The present study suggests that the use of wood for cooking is a risk factor for cervical neoplasia that deserves further study, given its high prevalence in developing countries.

7) K-RAS Oncogene Mutation in Patients with Advanced Non-Small Cell Lung Cancer (NSCLC) Associated With Exposure to Wood Smoke and Tobacco Smoking: Therapeutic Implications. Wood smoke has been described as a human carcinogen and an important risk factor for the development of NSCLC. The frequency of exposure to wood smoke in patients with NSCLC is 28%. Current data indicates that lung cancer associated with tobacco smoking and the lung cancer associated with wood smoke exposure present different clinical characteristics, which suggests that they might also have different genetic alterations, which are a consequence of tumor etiology. Source: <http://www.bioportfolio.com/resources/trial/70281/K-ras-Oncogene-Mutation-In-Patients-With-Advanced-Non-small-Cell-Lung.html>

8) CK, L., Q. NY, et al. (1988). "Natural inhalation exposure to coal smoke and wood smoke induces lung cancer in mice and rats." *Biomed Environ Sci*.

In a rural area with a high mortality rate of lung cancer in humans, mice and rats were placed in an environment in which they inhaled coal smoke and wood smoke in indoor air for 15 to 19 months. The incidences of lung cancer in mice in the control group, wood group, and coal group were 17.0% (29/171), 45.8% (81/177), and 89.5% (188/210), respectively: in rats the incidences were 0.9% (1/110), 0 (0/110), and 67.2% (84/125), respectively. In addition, the pollutants in the air were analyzed. The results indicate that coal smoke is a highly significant risk factor for lung cancer in humans in Xuan Wei County of Yun Nan Province in China.