

# CHAPTER 6. HOUSEHOLD USE OF BIOMASS FUELS

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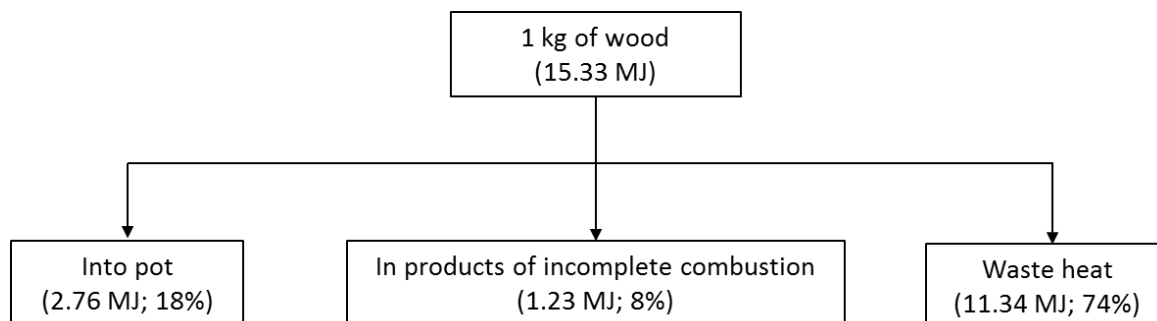
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Household use of solid fuels (biomass fuels and coal) is the most widespread source of indoor air pollution worldwide; solid fuels are used extensively for cooking and home heating in developing countries, especially in rural areas ([Perez-Padilla et al., 2010](#)). *Biomass fuel* usually refers to solid fuels that are derived from plants and animals and that are intentionally burned by humans for household energy. The solid fuels primarily include wood, but also agricultural residue, animal waste (dung), charcoal, and even leaves and grass. These fuels are often collected from the local environment in rural areas and purchased through markets in urban areas. Traditional biomass represents 10% of the world's primary energy use, with almost 2.7 billion people worldwide using biomass fuels for their household energy needs. By 2030, the population projected to use biomass will be 2.8 billion, indicating that the use of solid fuels is anticipated to remain relatively constant in the future ([IEA, 2010](#)). The percentage of the population relying on household use of solid fuels for cooking varies significantly among countries (urban and rural areas) and regions. Recent estimates based on national surveys representing 85% of the world's population have shown a decline from 62% to 41% between 1980 and 2010. This decline occurred in all regions, with a slower decline in

sub-Saharan Africa. Africa and South-East Asia have the highest proportion of households using solid fuels, with 77% and 61%, respectively. In the Western Pacific and Mediterranean regions, the proportion of use is estimated to 46% and 35%, respectively. ([Bonjour et al., 2013](#)). In Latin America and the Caribbean, 16% of households use solid fuels, with a large variation between countries. For example, in Guatemala 62% of the population uses solid fuels, with 88% in rural areas and 29% in urban areas, whereas in Mexico 15% of the population uses solid fuels, with 45% in rural areas and < 5% in urban areas. ([WHO, 2012](#)).

In the majority of industrialized countries, solid fuel use falls below the 5% mark. Greater use of solid fuels is associated with poverty in countries, in communities within a country, and in households within a community ([Perez-Padilla et al., 2010](#); [Lim and Seow, 2012](#)).

The World Health Organization (WHO) identified indoor smoke from combustion of solid fuels as one of the top 10 risks for worldwide burden of disease, accounting for 2.7% of the global burden of disease and 2 million premature deaths annually from acute lower respiratory infections, chronic obstructive pulmonary disease, and lung cancer (for coal smoke only),

**Fig. 6.1 Energy flow in a typical wood-fired cooking stove**

MJ, megajoules.

Source: [Holdren and Smith \(2000\)](#); reproduced with permission from the United Nations Development Programme.

mainly occurring in developing countries ([WHO, 2009](#); [Smith \*et al.\*, 2004](#)).

In this review, we will briefly address the component of exposure to indoor smoke from burning biomass fuel, the mechanisms of carcinogenicity, and the epidemiological evidence on the relationship of biomass fuel to cancer.

## Indoor smoke from burning biomass fuel

Cooking and heating with biomass fuels on open fires or with traditional stoves results in high levels of health-damaging pollutants. Combustion of biomass fuel in households often takes place in simple, poorly designed and maintained stoves with no chimney for removing emissions and with poor ventilation. Biomass combustion is typically inefficient because it is generally difficult to pre-mix solid fuels sufficiently with air to ensure complete combustion in simple, small-scale devices such as those traditionally used for household needs ([Naehler \*et al.\*, 2007](#)). Even in households with chimneys, heavily polluting biomass fuel stoves can produce significant local outdoor pollution. This is particularly true in dense urban slums, where such neighbourhood pollution can be much higher than levels of average urban air pollution ([Holdren and Smith, 2000](#)). [Fig. 6.1](#) shows the energy flow

of a typical wood-fired cooking stove, in which a large fraction of the fuel energy is lost because of low combustion efficiency.

Although most biomass fuels are intrinsically free of contaminants, a substantial fraction of the fuel is converted to products of incomplete combustion. The smoke from burning biomass fuel contains thousands of chemicals, many of which have documented adverse health effects, including irritant, inflammatory, and carcinogenic properties. Few studies have been conducted to characterize detailed chemical speciation for biomass stoves in developing countries. The composition of the smoke varies with even minor changes in fuel quality, configuration of the cooking stove, or characteristics of the combustion. Although emission factors for specific compounds have been reported by different investigators, it is difficult to compare them as many of the reports are semiquantitative and the analytical methods used were not always validated for each analysis. Furthermore, variable combustion conditions (fuel type, moisture content, combustion device) were used and emission factors were reported in a variety of units ([Naehler \*et al.\*, 2007](#)). Some illustrative compounds of the different groups of pollutants generally present in the smoke are listed in [Table 6.1](#).

Particles emitted from biomass combustion are fine and ultrafine in size ( $< 1 \mu\text{m}$ ); some

**Table 6.1 Pollutants present in biomass smoke**

Group	Pollutants <sup>a</sup>	Emission rate (g/kg of wood burned)
Criteria pollutants	Inhalable particles PM <sub>10</sub>	
	Fine particles PM <sub>2.5</sub>	1.6–9.5
	Carbon monoxide	5.87–6.92
	Nitrogen oxides	1.16–2.78
Respiratory irritants	Phenols	
	Cresols	
	Acrolein	
	Acetaldehyde ( <i>IARC Group 2B</i> )	0.041–0.371
Carcinogenic organic compounds	Benzene ( <i>IARC Group 1</i> )	0.264–0.629
	Styrene ( <i>IARC Group 2B</i> )	
	Formaldehyde HCHO ( <i>IARC Group 1</i> )	0.042–0.261
	1,3-Butadiene ( <i>IARC Group 1</i> )	0.0008–0.001
Polycyclic aromatic hydrocarbons	Benzo[ <i>a</i> ]pyrene ( <i>IARC Group 1</i> )	0.0004–0.0007
	Benz[ <i>a</i> ]anthracene ( <i>IARC Group 2B</i> )	0.0006–0.0008
	Dibenz[ <i>a,h</i> ]anthracene ( <i>IARC Group 2A</i> )	0.0002–0.0006
	Chrysene ( <i>IARC Group 2B</i> )	
	Pyrene	
	Fluorene	
	Phenanthrene	
	Naphthalene ( <i>IARC Group 2B</i> )	0.004–0.039

<sup>a</sup> IARC Groups: Group 1, carcinogenic to humans; Group 2A, probably carcinogenic to humans; Group 2B, possibly carcinogenic to humans; Group 3, not classifiable as to its carcinogenicity to humans; Group 4, probably not carcinogenic to humans. Adapted from [Naeher et al. \(2007\)](#); [IARC \(2010\)](#); [Zhang et al. \(2000\)](#).

larger particles can result from resuspension of ash and debris. Particulate matter (PM) small in size is most damaging to health. The composition of the PM varies with the specific fuel being burned and with the combustion conditions, but it generally consists of elemental carbon, levoglucosan, and hundreds of distinct organic compounds. The measured range of PM concentrations has been quite wide, starting with tens of  $\mu\text{g}/\text{m}^3$  and reaching into the hundreds or even thousands of  $\mu\text{g}/\text{m}^3$  for peak exposures during cooking. Fine particulate exposure measurement has been proposed as the best single indicator of the health impacts of most combustion sources ([Naeher et al., 2007](#)). Carbon monoxide is the single most important contaminant emitted during combustion of wood ([Viau et al., 2000](#)). Both the International Agency for Research on Cancer (IARC) Monograph Volume 95 ([IARC,](#)

[2010](#)) and [Naeher et al. \(2007\)](#) present summaries of indoor air quality studies reporting household pollution levels.

Burning of biomass fuel is a major source of volatile organic compounds, some of which are known for their carcinogenic effects, as listed in [Table 6.1](#). [Table 6.2](#) shows the measured levels of some of these pollutants compared with the WHO indoor air quality guidelines ([WHO, 2010](#)). Another approach for exposure assessment is the measurement of biological indicators or exposure biomarkers. Levels of biomarkers represent the absorbed dose of a chemical, integrated across all microenvironments and routes of exposure. Some biomarkers of exposure have been evaluated as metrics of biomass smoke exposures; for example, urinary polycyclic aromatic hydrocarbon (PAH) metabolites ([Viau et al., 2000](#); [Riojas-Rodriguez et al., 2011](#)) and

**Table 6.2 Measured indoor concentrations of organic pollutants present in biomass smoke**

Study location and population	Benzene ( $\mu\text{g}/\text{m}^3$ )	Formaldehyde ( $\mu\text{g}/\text{m}^3$ )	Benzo[a]pyrene ( $\text{ng}/\text{m}^3$ )	Naphthalene ( $\mu\text{g}/\text{m}^3$ )
WHO indoor air quality guidelines (WHO, 2010)	1.7 annual average	100 short-term (30 minute)	0.12 annual average	10 annual average
Bangladesh (Khalequzzaman et al., 2010), 42 urban households using biomass fuel, 24 hour kitchen monitoring	GM winter 54.2 GM summer 31.4	GM winter 9.9 GM summer 19.1		
India (Sinha et al., 2006), 55 rural households, monitored during cooking hours	Kitchen with ventilation 31.2 Kitchen without ventilation 45.0			
Sweden (Gustafson et al., 2007), 14 households, wood burning for space heating, 24 hour indoor monitoring	Median 2.6	Median 26		
Burundi (Viau et al., 2000), 16 rural homes, 8–12 hour indoor monitoring			Mean 70	Mean 28.7 Other low-weight PAHs Fluorene mean 8.6 Phenanthrene mean 3.4

GM, geometric mean.

the benzene metabolite *trans,trans*-muconic acid (Roychoudhury et al., 2012).

PAHs are important chemical components of combustion emissions. The smaller PAHs with 2 to 4 rings are volatile and are found in the gas phase to a higher degree than the 5- to 7-ring PAHs, which occur mainly or entirely as particles. Individual PAHs and specific PAH mixtures have been classified as carcinogenic by IARC. Benzo[a]pyrene (B[a]P), the most widely investigated PAH, has been classified as carcinogenic to humans (IARC Group 1). The lung cancer risk from inhalation exposure to a PAH mixture can be estimated by summarizing the individual PAH concentrations and taking into account the toxic equivalency factors denoting the cancer potency relative to the cancer potency of B[a]P (Boström et al., 2002). As summarized in IARC Monograph Volume 95, the concentration of PAHs in wood smoke emissions is generally lower than that for coal emissions. Other components of wood smoke may also be important for the observed health effects and a potential area for future research (Reid et al., 2012).

Biomass fuel smoke can contaminate more than only the indoor air; it may also settle on the walls, floors, clothing, and food. PAHs can be absorbed through the respiratory tract, gastrointestinal tract (diet is the main route of exposure to PAHs in the general population), and skin. Most studies to date have not considered all routes of exposure (IARC, 2010; Reid et al., 2012).

## Mechanisms of carcinogenesis

The main compounds of interest in biomass fuel smoke with regard to carcinogenicity are PM and PAHs. Fine particles are deposited in the central and peripheral airways, where they may exert toxic effects. When the inhaled concentration of PM is high, the mechanism of lung overload with impairment of particle clearance has been observed. The response to chronic lung overload is a sustained increase in neutrophilic inflammation and the subsequent release of reactive oxygen species (ROS). ROS within cells may damage DNA directly and induce mutations and also promote cell turnover and proliferation (Lim

[and Seow, 2012](#); [IARC, 2010](#); [ILSI Risk Science Institute Workshop Participants, 2000](#)).

High-molecular-weight PAHs are clearly carcinogenic, as shown in both *in vitro* and *in vivo* studies. Activated PAH metabolites can form adducts with DNA and if these adducts are not repaired, misreplication converts them to mutations. Accumulation of additional mutations in key genes within stem cells, together with epigenetic and/or non-genetic changes, can result in tumour formation. Phase I and phase II enzymes are involved in the metabolic activation and detoxification of PAHs. Genetic polymorphism in these enzymes may confer susceptibility to individuals exposed to solid fuel smoke. Some gene–environment interactions have been explored, including *CYP1A1*, *GSTM1*, and *GSTT1* genotypes and use of coal, but fewer studies have included such susceptibility biomarkers considering biomass smoke exposure ([IARC, 2010](#)). A multicentre case–control study of nonsmokers in eight countries evaluated the association between *GSTM1* and *GSTT1* and risk of lung cancer ([Table 6.3](#)). Compared with subjects with < 20 years of wood smoke exposure, subjects with > 20 years of exposure had a higher risk of lung cancer among those with the *GSTM1* null genotype (odds ratio [OR], 6.2; 95% confidence interval [CI], 1.5–25). No gene–environment interactions were observed for the *GSTT1* null genotype ([Malats et al., 2000](#)).

PAH metabolites also increase cell proliferation through interaction with some signalling pathways, including the epidermal growth factor receptor pathway and the serine/threonine kinase Akt pathway ([Lim and Seow, 2012](#)). Compared with liquefied petroleum gas (LPG) users, women using biomass for cooking in India showed upregulation of phosphorylated Akt proteins in airway epithelial cells, suggesting that cumulative exposure to biomass smoke increases the risk of carcinogenesis via oxidative stress-mediated activation of the Akt signal transduction pathway ([Roychoudhury et al., 2012](#)).

Whereas the indoor emissions from wood combustion contain lower levels of PAHs, available data suggest that the mechanisms described for the carcinogenicity of coal may also be plausible for the lung cancer risk associated with biomass fuel emissions. Data from coal smoke are consistent with a carcinogenic mechanism including at least six major pathways disrupted by a mixture of genetic and epigenetic changes for a normal cell to be transformed to a tumour cell. Lung tumours had mutations in the *KRAS* gene, affecting cell growth and signalling, and in the *p53* gene, affecting cell growth and replication ([IARC, 2010](#)).

The genotoxicity of human exposure to biomass combustion emissions has been examined. Compared with LPG users, women using biomass for cooking in India had increased micronucleated buccal and airway epithelial cells, as well as comet tail% DNA and tail length in comet assay. The exposure to biomass fuel smoke stimulated the DNA repair mechanisms and increased ROS generation. A depletion of superoxide dismutase and total antioxidant status was also reported ([Mondal et al., 2010](#)).

## Exposure to biomass fuel smoke and cancer (excluding coal smoke)

Indoor air pollution has been associated with cancers of the lung, upper aerodigestive tract, and cervix. Lung cancers are the most studied and well characterized. Although biomass fuel is much more widely used than coal, the adverse health effects, including the association between biomass smoke and lung cancer, have been less studied. Burning of coal appears to be a stronger risk factor for lung cancer than wood burning ([Lim and Seow, 2012](#); [IARC, 2010](#); [Reid et al., 2012](#); [Kurmi et al., 2012](#)). But because exposure to biomass fuels is much more prevalent, the adverse health effects are likely large even if the risks associated are smaller than for coal.

**Table 6.3 Summary of case–control studies on the risk of cancer associated with biomass smoke exposure**

Location, period (Reference)	Study population (cases/controls)	Exposure assessment	Outcome	Odds ratio (95% CI)	Adjusted covariates and comments
Osaka, Japan 1986–1988 ( <a href="#">Sobue, 1990</a> )	144/731 nonsmoking women Hospital-based unmatched controls	Self-administered questionnaire Used straw or wood for cooking at age 15, at age 30, and at time of interview	Newly diagnosed lung cancer (78% ADC, 8% SCC, 5% small cell, 5% large cell, 4% other)	Straw or wood for cooking at age 30 1.77 (1.08–2.91)	Age, education, other household members smoked in adulthood, mother smoked in childhood Controls were younger with higher education Reference category was not clearly defined
Shanghai, China 1984–1986 ( <a href="#">Gao et al., 1987</a> )	672/735 women Population-based controls	Oil used, frequency of stir-frying, deep-frying, boiling Type of fuel used for cooking	Newly diagnosed lung cancer, mainly ADC	Wood for cooking 1.0 (0.6–1.8)	Age, education, smoking Type of fuel used for cooking unrelated to risk
Guangzhou, China 1983–1984 ( <a href="#">Liu et al., 1993</a> )	316/316 women and men Hospital-based matched (sex, age, residential district) controls	In-person interview Type of fuel (coal and wood) used for cooking during three periods of time	Newly diagnosed lung cancer	Wood for cooking 1.19 (0.46–3.11)	Smoking, passive smoking, education, occupation, history of tuberculosis and chronic bronchitis, family history of cancer, size of living area Very few wood users, mostly coal
Taiwan, China 1992–1993 ( <a href="#">Ko et al., 1997</a> )	117/117 nonsmoking women Hospital-based matched (age) controls	In-person interview No cooking or use of gas, coal, or wood for cooking during three age-stages	Newly diagnosed lung cancer Information on histological type not provided	Wood or charcoal versus gas or none < 20 years old 2.5 (1.3–5.1) 20–40 years old 2.5 (1.1–5.7) > 40 years old 1.0 (0.2–3.9) After further adjustment 20–40 years old 2.7 (0.9–8.9)	Socioeconomic status, education, residential area Use of fume extractor in the kitchen (protective), history of tuberculosis, vegetable consumption, living near industrial district
Brazil 1987–1989 ( <a href="#">Pintos et al., 1998</a> )	784/1568 women (13%) and men (87%) Hospital-based matched (sex, age, hospital, admission) controls	In-person interview Wood stove use for cooking and heating	Newly diagnosed mouth, larynx, and pharynx cancer	Use of wood stove versus no use All sites 2.45 (1.84–3.26) Pharynx 3.82 (1.96–7.42)	Smoking, alcohol consumption, rural residence, schooling, ethnicity, income, consumption of smoked meat and other foods

Table 6.3 (continued)

Location, period (Reference)	Study population (cases/controls)	Exposure assessment	Outcome	Odds ratio (95% CI)	Adjusted covariates and comments
Sweden, Germany, France, Italy, the Russian Federation, Romania, Poland, Brazil ( <a href="#">Malats et al., 2000</a> )	122/121 nonsmoking women and men Population- and hospital-based controls	In-person interview Use of wood or coal for cooking or heating, duration of exposure	Newly diagnosed lung cancer (69% ADC, 21% SCC, 6% small cell, 4% large cell)	All subjects > 20 years indoor wood combustion versus no wood use 2.5 (1.0–6.2) GSTMI null > 20 years indoor wood combustion versus no wood use 6.2 (1.5–25)	Sex, age, centre Nonsmokers were defined as never-smokers and occasional smokers (up to 400 cigarettes in a lifetime) Controls were not matched by age and sex Controls were younger than cases Study period not specified
Chandigarh, India 1995–1997 ( <a href="#">Gupta et al., 2001</a> )	265/525 women and men Hospital-based matched (sex, age) controls	In-person interview Coal or wood for heating and cooking	Newly diagnosed lung cancer	Cumulative exposure in women of > 45 years to indoor air pollution from use of coal or wood for cooking or heating 1.43 (0.33–6.30)	Age, socioeconomic status, smoking, sex Mixed coal and biomass use
Taiwan, China 1993–1999 ( <a href="#">Le et al., 2001</a> )	527/805 women and men Hospital-based matched (sex, age) controls	In-person interview No cooking or use of gas, coal, or wood for cooking when women were 20–40 years old	Newly diagnosed lung cancer (SCC and small cell 28.2%, ADC 47.7%)	For women Wood or charcoal versus gas or none SCC 3.1 (1.0–9.2) ADC 3.0 (1.4–6.4)	Smoking, residential area (urban, suburban, rural), socioeconomic status, and education Long-term residence near industrial district increased lung cancer risk Fume extractor in the kitchen decreased lung cancer risk Only 7% of men reported cooking
Mexico City, Mexico 1986–1994 ( <a href="#">Hernández-Garduño et al., 2004</a> )	113/273 nonsmoking women Hospital-based controls	Medical records Ever used wood fuel for cooking Years of exposure	Newly diagnosed ADC of the lung	Years of cooking with wood versus no use of wood fuel 1–20 years 0.6 (0.3–1.2) 21–50 years 0.6 (0.3–1.3) > 50 years 1.9 (1.1–3.5)	Age, ETS exposure, socioeconomic status, education Different control groups were used

**Table 6.3 (continued)**

Location, period (Reference)	Study population (cases/controls)	Exposure assessment	Outcome	Odds ratio (95% CI)	Adjusted covariates and comments
Czech Republic, Hungary, Poland, Romania, the Russian Federation, Slovenia, United Kingdom 1998–2002 ( <a href="#">Lissowska et al., 2005</a> )	2861/3118 women and men Multicentre study Population- and hospital-based matched (sex, age, and area) controls	In-person interview Fuels used for cooking and for heating	Newly diagnosed lung cancer	Ever used wood for cooking versus no solid fuel use 1.23 (1.00–1.52) Ever used wood for heating versus no solid fuel use 1.31 (1.06–1.61)	Age, sex, education, smoking, centre Coal was the most commonly used fuel for heating (50%) and cooking (44%) Associations due to mixed wood and coal exposures could not be ruled out
Montreal, Canada 1996–1997 ( <a href="#">Ramanakumar et al., 2007</a> )	1205/1541 women and men Population-based matched (sex, age) controls	In-person or next-of-kin interview Fuels used for cooking and for heating and duration of exposure	Newly diagnosed lung cancer	In women versus never exposed Only traditional heating 1.8 (1.0–3.2) Only traditional cooking 1.2 (0.7–1.9) Both 2.5 (1.5–3.6)	Age, ethnic group, family income, smoking, place of birth, type of interview, schooling, occupational exposures, ETS Mixed coal and biomass use
India 2001–2004 ( <a href="#">Sapkota et al., 2008</a> )	799 (lung)/718 (upper digestive)/718 Women and men Multicentre study Hospital-based matched (sex, age, area of residence) controls (19% hospitalized, 81% visitors)	In-person interview Solid fuel use and duration of exposure	Newly diagnosed lung and hypopharyngeal/laryngeal cancer	Always wood use versus always modern fuels  Hypopharynx: 1.56 (1.09–2.25) Larynx: 1.06 (0.74–1.53) Lung: 1.06 (0.77–1.47) Years of wood usage Hypopharynx > 0–30: 0.85 (0.50–1.45) > 30–50: 1.59 (1.06–2.38) > 50: 1.45 (0.96–2.19) $P_{\text{trend}} = 0.03$	Centre, age, socioeconomic status, cumulative tobacco consumption
North America, Europe, Asia International Lung Cancer Consortium ( <a href="#">Hosgood et al., 2010</a> )	5105/6535 Pooled analysis Population- and hospital-based matched (at least age and sex) controls	In-person interview Solid fuel use, wood use, coal use	Newly diagnosed lung cancer	Wood use in Europe/North America versus nonsolid fuel user All studies 1.21 (1.06–1.38)	Age, sex, education, smoking status, race/ethnicity, study centre Cases tended to be older and more educated

ADC, adenocarcinoma; CI, confidence interval; ETS, environmental tobacco smoke; SCC, squamous cell carcinoma.



As part of the Global Burden of Disease analysis, the literature relating solid fuel use exposure to different health outcomes was qualitatively evaluated ([Desai et al., 2004](#)). At that time, there was strong evidence of lung cancer from exposure to coal smoke in adult women, but only moderate evidence for this same association for biomass smoke exposure. Four investigations, one in Japan ([Sobue, 1990](#)), two in China ([Gao et al., 1987](#); [Liu et al., 1993](#)), and one in Taiwan, China ([Ko et al., 1997](#)), assessing the relation between biomass fuel use and lung cancer in women, were considered and a relative risk of 1.5 (95% CI, 1.0–2.1) was obtained ([Table 6.3](#)).

In the evaluation of the carcinogenicity of biomass fuel by IARC ([Straif et al., 2006](#)), four new studies were considered. A study conducted in Taiwan, China, reported that women who burned wood for cooking had a 3-fold increase in risk of lung cancer after adjusting for potential confounders, reporting stronger associations with squamous cell carcinoma and adenocarcinoma than with other histological subtypes of lung cancer ([Le et al., 2001](#)). In a case–control study from Japan, smoke exposure from wood or from wood and straw was associated with lung cancer only among those exposed before the age of 30 years ([Sobue, 1990](#)). Exposure to wood smoke for 50 years or more was associated with adenocarcinoma in a case–control study from Mexico ([Hernández-Garduño et al., 2004](#)). In addition, a large multicentre European case–control study ([Lissowska et al., 2005](#)) recorded an adjusted 20–30% increased risk of lung cancer in people who burned wood but not coal, compared with people who never used solid fuels for cooking or heating. However, information on any exposure–response relationship could not be determined as data on duration and intensity of exposure were lacking. These studies, conducted across several geographical regions, supported the conclusion of the IARC Working Group that evaluated indoor emissions from household combustion of biomass fuel (mainly wood) as

a possible carcinogen (Group 2A) ([Straif et al., 2006](#); [Table 6.3](#)).

Since the IARC Working Group evaluated the evidence in 2006, a pooled analysis from the International Lung Cancer Consortium, including seven case–control studies from Europe, Asia, and the USA, reported a modest association between lung cancer and wood smoke exposure (OR, 1.2; 95% CI, 1.06–1.38) ([Hosgood et al., 2010](#)). More recently, a review listed 13 studies reporting lung cancer risk estimates for biomass fuels ([Lim and Seow, 2012](#)). A systematic review and meta-analysis including seven of these studies ([Sobue, 1990](#); [Ko et al., 1997](#); [Lissowska et al., 2005](#); [Hernández-Garduño et al., 2004](#); [Le et al., 2001](#); [Sapkota et al., 2008](#); [Liu et al., 1991](#)) estimated an overall OR of 1.50 (95% CI, 1.17–1.94) for biomass use (predominantly wood) and lung cancer ([Kurmi et al., 2012](#)), similar to the pooled estimate obtained previously.

Other types of cancer have also been related to biomass fuel. A study conducted in Brazil observed an increase in risk of upper aerodigestive tract cancer in women ([Pintos et al., 1998](#)). IARC Monograph Volume 95 mentions studies of nasopharyngeal cancer and other upper aerodigestive tract cancers, but the existing evidence precluded the Working Group from drawing conclusions about these cancers ([IARC, 2010](#)). A subsequent case–control study in India reported an association between the use of wood as a solid fuel and hypopharyngeal cancer, but not lung and laryngeal cancer ([Sapkota et al., 2008](#)).

A study conducted in Honduras suggested that wood burning could increase the risk of cancer among women infected with human papillomavirus (HPV) ([Velema et al., 2002](#)). Since the 2006 IARC review, a case–control study of Colombian women reported that the risk of cervical cancer was stronger among HPV-infected women exposed to wood smoke in the kitchen for 16 or more years than in HPV-infected women without wood smoke exposure (OR, 5.3; 95% CI, 1.9–14.7) ([Sierra-Torres et al., 2006](#)).

## Interventions

The use of biomass fuels in developing countries is likely to remain stable in the near future. The *fuel-switching* approach has turned out to be too simplistic to describe the household fuel use, particularly within rural and suburban areas. Many households follow a *multiple fuels* strategy, taking advantage of both traditional and modern practices. Improved biomass cook stoves have been identified as an option to reduce negative impacts of cooking with traditional open fires ([Masera et al., 2005](#); [Ruiz-Mercado et al., 2011](#)). Improvements to biomass stoves have focused on combustion efficiency and the venting of emissions outdoors. Reductions in concentrations of PM < 2.5 µm in diameter (PM<sub>2.5</sub>) and carbon monoxide have been reported due to the use of improved stoves in Mexico ([Zuk et al., 2007](#)) and Guatemala ([Smith et al., 2010](#)). Despite being substantially lowered, the concentrations remain high compared with those in households using gaseous fuels and health-based guideline values ([IARC, 2010](#)). While an intervention study conducted in Mexico provides some evidence that lowering exposure to biomass fuel through clean stove intervention decreases lung function decline comparably to smoking cessation ([Romieu et al., 2009](#)), no data are available on the impact of stove intervention on the risk of lung cancer. After an intervention with an efficient biomass stove, a reduction of PAHs exposure biomarkers has been reported in Mexico and Peru ([Riojas-Rodriguez et al., 2011](#); [Li et al., 2011](#); [Torres-Dosal et al., 2008](#)). However, levels of these exposure biomarkers remained higher than those reported for the general population.

## Conclusion

There is widespread use of biomass fuel, involving almost 40% of the human population. Exposure occurs during cooking or heating, typically in poorly ventilated and crowded spaces.

Women and children are often most exposed to extremely high levels of health-damaging pollutants. This chronic exposure to toxic pollutants has severe health consequences and may increase the risk of cancer. Although further studies are needed, available data suggest that exposure to biomass fuel smoke increases the risk of lung cancer as well as other types of cancer. One of the main limitations of the epidemiological studies reported so far has been the exposure duration and intensity assessment, precluding the demonstration of an exposure–response relationship. Considering information about household characteristics, such as room partitions and extent of ventilation (air exchange), could provide more accurate measurement of the exposure. Most of the studies controlled for confounding to a reasonable extent, but few considered the potential confounding by socioeconomic status, a determinant of both disease and fuel use. Intervention studies can provide strong evidence for causal inference, showing how the exposure cessation or reduction decreases the risk of cancer, but such studies have not been performed to date for biomass fuel smoke exposure and lung cancer ([Perez-Padilla et al., 2010](#); [Lim and Seow, 2012](#); [IARC, 2010](#)).

Opportunities for research on indoor air pollution (due to domestic use of biomass fuels) and cancer include studies of the effect on cancers, other than lung, and studies of genetic factors that modify susceptibility. Studies exploring gene–environment interaction have been performed among populations exposed to coal, but similar studies are needed in populations with exposure to smoke from wood and other biomass fuels. Such research areas could be incorporated into intervention studies to maximize the information that can be obtained and to identify susceptible subgroups ([Reid et al., 2012](#)).

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