

HOUSEHOLD COMBUSTION OF SOLID FUELS

Indoor emissions from household combustion of coal (Group 1)

Indoor emissions from household combustion of biomass fuel (primarily wood) (Group 2A)

For definition of Groups, see Preamble Evaluation.

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5. Summary of Data Reported

[The text of these Summaries and Evaluations may be edited for language and clarity during the checking of the main text of the Monographs.]

5.1 Exposure data

Use of solid matter as household fuel is widespread and affects approximately half of the human population, almost exclusively in low- and medium-resource countries. The use of biomass is much more frequent than that of coal in most parts of the world. Exposure to the emissions from the combustion of these fuels occurs as a result of cooking or heating, usually in poorly ventilated spaces. Women and young children especially may be exposed to extremely high levels of these emissions.

The factors that determine the use of solid fuels involve a combination of issues related to economics, social status, convenience and physical availability. Income and education play a major role in the selection of fuel, and the households that use solid fuels tend to have lower education and incomes. This is because biomass fuels are frequently collected from the local environment, whereas liquid fuels must be purchased at a local market or fuel retailer.

Access to both solid fuels and liquid fuels also plays a major role in the selection of fuels for household use. The ready availability of biomass from the local environment or from agricultural residues encourages people to use it as a cooking fuel, since cash expenditure is not required. When liquid fuels are not available in local markets or significant initial costs represent a barrier to their adoption, the probability that solid fuels will be used is greater.

Energy policies in specific countries that involve issues of access and energy prices, taxes or subsidies also play a role in the selection of the type of fuel to be used. Taxes on liquid fuels obviously reduce the probability that people will use them for cooking, whereas subsidies encourage their use. Additional factors that influence exposure to indoor air pollution from solid fuels include the type and quality of the fuel, the type and condition of stoves, the presence of a flue, the type of ventilation and housing, the task, the skill of the stove operator and weather condition, all of which play a role in determining the level of pollutants. These factors vary by day, season and year, and generalization of the levels of exposure that can occur from individual monitoring studies that are conducted under widely differing conditions is difficult.

Typical household combustion of biomass and coal diverts 10–30% of fuel carbon into products of incomplete combustion. Total emissions of these products from coal and biomass overlap, largely depending on the species of fuel and the type of stove. Thousands of chemical species have been

identified in the gas-phase and particle-phase of products of incomplete combustion. The mixture contains fine and ultrafine particles and a large number of semi-volatile and non-volatile organic compounds, including known carcinogens such as benzene, formaldehyde and benzo[*a*]pyrene. On the basis of results from a limited number of studies that measured emission factors, combustion of the same amount of coal and wood in household stoves generates relatively comparable amounts of benzene and benzo[*a*]pyrene. However, combustion of wood appears to generate larger amounts of formaldehyde and acetaldehyde than combustion the same amount of coal.

Virtually all of the rural population of China (about 740 million) uses solid fuels, and most rely on coal or a variety of biomass fuels for most of their energy needs. A considerable portion of the urban population (560 million) uses coal, which is increasingly in the form of briquettes. Improved biomass fuel stoves are very common, as are unvented portable coal stoves. Typical average indoor exposure levels of particulate matter < 10 µm in size range from several tens to several hundred micrograms per cubic metre and those for benzo[*a*]pyrene range from low single digits to more than 40 ng/m³. In some households, average exposure levels can be an order of magnitude higher. While gas fuels and electricity are progressively replacing solid fuels, the latter remain prevalent, even in wealthier rural households.

Exposure to indoor air pollutants that are associated with the combustion of solid fuels for cooking and heating is extensive in South Asia. Exposures are widespread and prevalent in half to three-quarters of the population in most countries of the region. In Latin America nearly 25% of the population live in rural areas where biomass fuels are most frequently used for cooking and heating. In Africa, biomass fuel is used almost exclusively in rural areas and is still widely used in most urban areas.

Although there is some variability in exposure levels as a result of a differential distribution of determinants, levels of pollutants that range from several hundreds of micrograms per cubic metre of particulate matter of varying size during the day to several thousands of micrograms per cubic metre during cooking have consistently been reported in many countries in these regions.

A variety of interventions are already available, and new technologies and approaches are emerging. A small body of evidence shows that interventions can substantially reduce exposure and the incidence of lung cancer (chimney stoves, switching to cleaner fuels) and chronic obstructive pulmonary disease (chimney stoves). Levels of indoor air pollutants associated with the use of biomass and other solid fuels can be substantially reduced, particularly by stoves with flues, but experience shows that exposure levels remain high and people are exposed in the vicinity of their homes and from neighbours' homes. Biomass stoves that use secondary combustion may offer advantages due to greatly reduced emissions. Cleaner fuels, in particular liquefied petroleum gas and natural gas, offer the largest reductions in exposure, but cost and practical issues may result in lesser reductions being achieved in practice. Electricity is important for development, but is unlikely to contribute to substantive reductions in exposure as it is rarely used for cooking and heating in poor communities due to the high cost of supply, infrastructure and use. Behavioural changes can complement technical interventions, but appear to have limited potential alone.

5.2 Human carcinogenicity data

5.2.1 Lung cancer

(a) Combustion of coal

More than 20 case-control studies and one cohort intervention study reported on the association between exposure to coal smoke and the risk for lung cancer. The majority of them were conducted in China; in addition, a few studies were available from North America and Europe. Several studies that used different epidemiological designs originated from Xuan Wei County, China. Initially, an ecological study from this area showed a strong correlation between communities that used several different types of smoky coal and mortality from lung cancer. Two population-based case-control studies

reported a positive association between the use of smoky coal and an increased risk for lung cancer. A statistically significant exposure–response relationship between the amount of smoky coal used and risk for lung cancer was observed in both of these studies. In one of them, even though controls were matched on village and fuel type to cases, the amount of smoky coal used was still significantly associated with risk for lung cancer in an exposure–response manner. A cohort study carried out in Xuan Wei County that included more than 20 000 farmers who used smoky coal throughout their lifetimes and approximately 1300 lung cancer cases showed that transition to the use of a stove with a chimney was associated with a reduced risk for lung cancer in both men and women and that this became evident 10 years and more after the intervention.

Two case–control studies from northern China that used general population controls provided evidence for an association between exposure to indoor air pollution from coal smoke and the risk for lung cancer. The first, a large, well conducted case–control study in Shenyang, reported internally consistent, positive exposure–response associations for different metrics of exposure to coal smoke, including a cumulative index of indoor exposure to coal smoke from heating and cooking, that were adjusted for smoking and education. The second study, from Harbin, reported a strong exposure–response relationship among nonsmoking women for years of use of a coal stove in the bedroom and risk for lung cancer after adjustment for several potential confounders.

One hospital-based case–control study from Taiwan observed a statistically significant twofold increase in risk for adenocarcinoma of the lung with use of ‘coal or anthracite’ as cooking fuel and adjusted for smoking and socioeconomic status; no exposure–response results were provided. A population-based case–control study on lung cancer among women in Los Angeles reported a twofold increased risk for adenocarcinoma of the lung with the use of coal for heating or cooking in childhood and adolescence; results were adjusted for potential confounders, but exposure–response analyses were not provided.

(b) *Combustion of biomass*

To examine the role of biomass in the risk for lung cancer, the Working Group considered that four studies that collected information on the use of this fuel type for cooking and/or heating were the more informative, and that, among those, a case–control study conducted in Taiwan and a large well-conducted European multicentre case–control study were the most informative. In the study in Taiwan, compared with people who did not use wood, nonsmoking women who used wood for cooking showed a significant twofold increased risk for lung cancer. In a subsequent expanded study, use of wood was also associated with a significant threefold increased risk for squamous–cell carcinoma and adenocarcinoma of the lung. In the large European case–control study, compared with men and women who never used coal and/or wood for cooking or heating, a significant 20–30% increased risk for lung cancer was found among those who cooked or heated with wood but never with coal after adjustment for active tobacco smoking and other potential confounders. However, neither the Taiwanese nor the European studies provided information on duration of exposure to wood smoke and thus exposure–response relationships could not be examined.

The other two informative studies were in nonsmoking women, one in Japan and one in Mexico, and found an increased risk for lung cancer in relation to exposure to smoke from wood or wood and straw. No information on duration of exposure was available in the Japanese study and the significantly increased risk was restricted to women who had been exposed to wood smoke at the age 30 years. In the Mexican study, an approximate twofold increased risk was restricted to women who had used wood for > 50 years whereas the risks were not increased for those who had used wood for 1–20 and 21–50 years. Thus, the accumulated evidence suggests that exposure to smoke from wood that was used for heating and/or cooking may be associated with an increased risk for lung cancer but information on the effect of duration and intensity of exposure was lacking.

5.2.2 *Aerodigestive tract cancers and combustion of coal or biomass*

Several studies investigated the relationship between the use of coal or biomass and the risk for nasopharyngeal carcinoma (the majority of which were conducted in Chinese populations and one in India). One study of nasopharyngeal carcinoma among Hung Chinese reported a statistically significant fivefold increased risk associated with current use of wood as fuel after adjustment for consumption of salted fish during weaning; however, no information on an exposure–response relationship was presented, except for some assessment of ventilation conditions. In other studies of nasopharyngeal carcinoma, assessment of exposure was also crude, the baseline comparison group was not clearly specified or included people who used coal or fuels other than coal and wood and no adjustment was made for consumption of salted fish.

Only one or two studies by cancer site investigated the relationship of exposure to emissions from the combustion of coal or biomass and other cancers of the aerodigestive tract, including the oral cavity, pharynx, larynx, nasal cavities and oesophagus. These studies were not very informative because they were very small, the baseline comparison group was not clearly specified, mixed exposures were investigated or the exposure was based on a dichotomized variable with no information on exposure–response relationships.

5.3 **Animal carcinogenicity data**

5.3.1 *Coal*

In one study, inhalation exposure to a high concentration of emissions generated from coal burned under conditions similar to those of human exposures in Xuan Wei County, China, increased the incidence of various types of malignant lung tumours (squamous-cell carcinomas, adenosquamous carcinomas and adenocarcinomas) in male and female Kunming mice and that of squamous-cell carcinomas in male and female Wistar rats. In another study in Kunming mice exposed by inhalation to an unspecified concentration of coal emissions from an unspecified source in Harbin City, China, the incidence of adenocarcinoma was increased. A veterinary epidemiological study of dogs also showed an association between exposure to coal emissions and sinonasal cancer.

Intratracheal administration of extracts of coal-derived soot from Xuan Wei County induced an increase in the incidence of lung adenocarcinomas. In two studies, subcutaneously administered extracts of coal emissions in Xuan Wei County increased the incidence of various types of malignant pulmonary tumours (squamous-cell carcinomas, adenosquamous carcinomas and adenocarcinomas) in Kunming mice. These extracts were used in a complete carcinogenesis study by dermal application and induced an increase in the incidence of skin carcinomas in SENCAR mice. Extracts of coal emissions from the same region increased the incidence of benign skin papillomas in two tumour initiation–promotion studies by dermal application in Kunming and SENCAR mice.

5.3.2 *Wood smoke*

In one study, inhalation exposure to a high concentration of emissions generated from wood burned under conditions similar to those of human exposure in Xuan Wei County increased the incidence of lung adenocarcinomas in male and female Kunming mice. The same inhalation exposure failed to increase the incidence of lung tumours in either sex of Wistar rats. Wood smoke generated from oak of mixed species that was burned in an uncertified wood stove over a simulated cycle induced no increase in tumour formation in Strain A mice exposed for 6 months and held for a 6-month period with no exposure.

Subcutaneously administered extracts of wood smoke in Xuan Wei County increased the incidence of pulmonary adenocarcinomas in male Kunming mice. Extracts of wood smoke from the same region increased the incidence of benign skin papillomas in two tumour initiation–promotion studies by dermal application in female Kunming and SENCAR mice. Similar regional extracts used in a com-

plete carcinogenesis study by dermal application induced a non-statistically significant increase in the incidence of skin carcinomas in female SENCAR mice. Extracts of relevant particulate matter from wood smoke generated from a wood stove in which hardwood and softwood were burned increased the incidence of benign skin papillomas in tumour initiation–promotion studies in female SENCAR mice following multiple topical applications to the skin.

5.4 Mechanistic and other relevant data

Emissions from combustion of organic materials, such as coal or wood, are complex mixtures that contain numerous different gases, aerosols and chemical compounds admixed with and/or adsorbed onto particulate matter.

The primary mechanisms for deposition of airborne particles in the respiratory tract are sedimentation, impaction and diffusion. Deposition by sedimentation and impaction depends on the aerodynamic diameter of the particle, whereas deposition by diffusion depends on its thermodynamic diameter. Following inhalation, particles may either deposit in the extrathoracic, tracheobronchial or pulmonary airways or remain in the air stream and be eliminated upon exhalation. The deposition of particles in the respiratory tract depends primarily on the size of the inhaled particle, the route of breathing (i.e. through the nose and/or mouth) and the breathing pattern (e.g. volume and frequency).

Particles are frequently aggregates or agglomerates of smaller primary particles. The aerodynamic and thermodynamic properties of these aggregates (rather than the primary particles) affect their behaviour in the air and their probability of deposition in the respiratory tract. Once deposited, properties such as the size and surface area of both the aggregate and primary particle can potentially affect the kinetics of clearance.

The deposition and clearance of particles vary among individuals for a number of reasons, including age, gender, tobacco smoking status and health status. Pre-existing lung diseases or conditions such as asthma or chronic obstructive pulmonary disease can influence the efficiency and pattern of deposition within the respiratory tract. Deposition also depends on the level of activity and breathing patterns. Deposition and retention determine the initial and retained dose of particles in each region and may, therefore, influence the risk for developing diseases specific to those regions of the respiratory tract.

Studies in rodents (primarily rats and mice) have shown that, depending on the concentrations and durations of exposure, the long-term retention of particles in humans can be greater than that predicted from rodent studies that used lower concentrations or shorter durations of exposure.

A cascade of events proposed to describe the biological process that starts from some particle deposition on critical target cells or tissues within the rat lung and results in rat lung tumours includes sustained inflammation, production of reactive oxygen species, depletion of antioxidants and/or impairment of other defence mechanisms, cell proliferation and gene mutations. These individual steps comprise an overall mode of action that can be used to compare responses of rats with those of other species, including humans. Particle surface area is a better predictor of lung tumours than particle mass in rats exposed to various poorly soluble particles of fine or ultrafine size.

Among other compounds, polycyclic aromatic hydrocarbons are important chemical components of combustion emissions. These compounds are absorbed through the respiratory tract, gastrointestinal tract and skin, and smaller molecules (two to three rings) are absorbed more rapidly than larger ones. Active transport and passive diffusion are both involved, and, once absorbed, polycyclic aromatic hydrocarbons are distributed widely to most organs and tissues and tend to accumulate in fatty tissue. They are metabolized rapidly to more soluble (and in some cases more reactive) metabolites, such as epoxides, phenols, dihydrodiols, phenol dihydrodiols, dihydrodiol epoxides, quinones and tetrols. At least three pathways of metabolism are involved: the cytochrome P450 pathway, the cytochrome P450/aldo-keto reductase (oxidative) pathway and a cytochrome P450/peroxidase (radical cation) pathway. Beyond these Phase I metabolic pathways, polycyclic aromatic hydrocarbon metabolites

may bind with macromolecules, which can lead to toxic, mutagenic or carcinogenic effects, or they may be eliminated in a conjugated form via Phase II metabolism.

Polycyclic aromatic hydrocarbons may be metabolized to their bay- and fjord-region diol epoxides or undergo cyclopenta-ring oxidation. These can be electrophilic and bind to DNA and proteins, which results in genotoxic effects—primarily through the formation of DNA adducts. Polycyclic aromatic hydrocarbons also have non-genotoxic effects that may include the interruption of gap-junctional communication and changes in gene expression; radical cations, *ortho*-quinones and reactive oxygen species may also be formed by their metabolism. They may also operate through receptor-mediated mechanisms that involve the aryl hydrocarbon receptor. These compounds can have immunological and haematological effects and can also be phototoxic.

Several studies evaluated populations who are exposed to indoor air pollution from coal, wood, other biomass or cooking oil fumes for associations between polymorphisms in genes that are involved in xenobiotic metabolism and risk for lung cancer. However, multiple comparisons and generally small sample sizes could have resulted in both false-positive and false-negative findings. There is some evidence that the *GSTM1* null genotype was associated with increased risk for lung cancer in some studies in which at least part of the study population was definitely or probably exposed to indoor air pollution, particularly where exposure to polycyclic aromatic hydrocarbons was suspected to be a contributing agent. However, results for polymorphisms in other genes are inconsistent or have been analysed in only one study. Therefore, no firm conclusion can be made regarding the effect of polymorphisms of genes other than *GSTM1* on risk for lung cancer in these populations.

The available information on the mutagenicity and genotoxicity of smoky coal emissions from Xuan Wei County includes a wide range of end-points, that encompasses mutations in *KRAS* and *TP53* genes in lung tumours from nonsmokers who were exposed to smoky coal emissions and whose tumours are linked epidemiologically to exposure to the emissions. In addition, studies show that such an exposure results in the excretion of several polycyclic aromatic hydrocarbon metabolites, and that exposed individuals exhibit elevated levels of cytogenetic damage (micronuclei, sister chromatid exchange and chromosomal aberrations), as well as DNA adducts and accumulation of TP53 protein. There is only limited information on the genotoxicity and mutagenicity of emissions from combustion of other types of coal.

The available information on the genotoxicity and mutagenicity of emissions from wood combustion includes a number of human studies that show the induction of cytogenetic damage in exposed individuals, including micronuclei, sister chromatid exchange and chromosomal aberrations. Also, exposed individuals have an elevated level of DNA adducts, DNA damage and accumulation of TP53 protein. In cultured cells, extracts of the emissions (mostly from wood) induce DNA strand breaks and sister chromatid exchange.

Extracts or condensates of emissions from coal and wood are mutagenic in *Salmonella*. In strain TA98 in the presence of a metabolic activation system, the geometric mean potency in terms of revertants per milligram of particle is approximately 3000 for smoky coal and 550 for wood. Thus, smoky coal emissions are five times more mutagenic than those from wood in terms of activity per milligram of particle. In contrast, the mutagenic potencies of these emissions expressed as revertants per cubic metre of air are approximately 15 000 for smoky coal and 1300 for wood. This much larger range of potencies reflects the range of the amounts of organic compounds (together with the potency of the organic compounds) emitted under the test conditions by the two combustion processes.

Bioassay-directed fractionation studies with *Salmonella* have identified that, for smoky coal, most of the mutagenic activity is due to polycyclic aromatic hydrocarbons and methylated polycyclic aromatic hydrocarbons. For wood, these compounds contribute 10–50% of the activity and polar aromatic compounds (aromatic amines and ketones) and nitropolycyclic aromatic hydrocarbons contribute to some of the remaining activity.

Findings from a study of exposure to wood smoke in Guatemala suggest an effect on all acute lower respiratory infections that is about one-third as strong as that reported from the meta-analysis of observational studies. Several studies have reported an association between exposure to biomass smoke and general acute respiratory illnesses in children, most of which are probably upper respiratory illness. Exposure to emissions from solid fuel is associated with an increased risk for chronic bronchitis, partially reversible airways obstruction and emphysema. There is also some evidence of a link with lung fibrosis and interstitial lung disease, although, in some of the latter studies, the subjects were also exposed to environmental dusts with a high silica content. A small number of observational studies have identified an association between use of biomass fuel and tuberculosis. The evidence to date suggests important and fairly consistent effects of indoor air pollution on both birth weight and still-birth. One of the commonest complaints among people who use biomass fuel, especially in unvented environments, is that the smoke causes stinging and watering of the eyes, which is unpleasant and impacts on the quality of life.

6. Evaluation and Rationale

6.1 Combustion of coal

There is *sufficient evidence* that household combustion of coal is carcinogenic to humans. Household combustion of coal causes cancer of the lung.

There is *sufficient evidence* in experimental animals for the carcinogenicity of emissions from combustion of coal.

There is *sufficient evidence* in experimental animals for the carcinogenicity of extracts from coal-derived soot.

Overall evaluation

Indoor emissions from household combustion of coal are *carcinogenic to humans (Group 1)*.

6.2 Combustion of biomass

There is *limited evidence* in humans for the carcinogenicity of household combustion of biomass fuel (primarily wood). Household combustion of biomass fuel (primarily wood) is associated with cancer of the lung.

There is *limited evidence* in experimental animals for the carcinogenicity of emissions from combustion of wood.

There is *sufficient evidence* in experimental animals for the carcinogenicity of wood smoke extracts.

Overall evaluation

Indoor emissions from household combustion of biomass fuel (primarily wood) are *probably carcinogenic to humans (Group 2A)*.

In reaching this evaluation, the Working Group considered mechanistic and other relevant data. These data include (a) the presence of polycyclic aromatic hydrocarbons and other carcinogenic compounds in wood smoke, (b) evidence of mutagenicity of wood smoke and (c) multiple studies that show cytogenetic damage in humans who are exposed to wood smoke.