# WHO Indoor Air Quality Guidelines: Household Fuel Combustion

## Review 8: Household Coal Combustion: Unique Features of Exposure to Intrinsic Toxicants and Health Effects

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**Convening lead authors**: those authors who led the planning and scope of the review, and managed the process of working with other lead authors and contributing authors, and ensuring that all external peer review comments were responded to.

**Lead authors**: those authors who contributed to one or more parts of the full review, and reviewed and commented on the entire review at various stages.

**Disclaimer**

The work presented in this technical paper for the WHO indoor air quality guidelines: household fuel combustion  has been carried out by the listed authors, in accordance with the procedures for evidence review meeting the requirements of the Guidelines Review Committee of the World Health Organization.

Full details of these procedures are described in the Guidelines, available at: http://www.who.int/indoorair/guidelines/hhfc ; these include declarations by the authors that they have no actual or potential competing financial interests. The review was conducted in order to inform the development of recommendations by the Guidelines Development Group. Some of  the authors are staff members of, or consultants to, the WHO. The authors alone are responsible for the views expressed in this publication, which do not necessarily represent the views, decisions, or policies of the WHO.

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# Summary

**Background**

By mass, more coal is used than any other fuel in the world with current use dominated by power production and industry. Depending on local availability and climate, coal has been widely used around the world for cooking and heating in the past, but today household use is largely confined to low- and medium- income countries, particularly China. In general, coal used in households is supplied locally from small mines, although in some areas it is a by-product sold to local populations by large mining operations.

**Objectives and key questions**

The aim of this review was to identidy the unique characteristics of coal, including toxic contaminants, and its health risks in household use to supplement the material in Review 4 (Health effects of household air pollution) on solid fuels. This review addressed the following four questions:

1. What are characteristics of combustion emissions specific to coal?
2. What are the specific adverse health effects of household coal combustion, in addition to the effects of products of incomplete combustion shared with biomass?
3. What are the health risks from toxic contaminants in coal?
4. What are the impacts of interventions to reduce risk from household use of coal, including the history and extent of bans on household coal use (with focus on China)

**Methods**

A narrative review was prepared to address question 1; for other questions, we drew on existing recent systematic reviews (and meta-analyses) where available, or conducted new systematic reviews. Computer-based searches covering 1980 to December 2013 of PubMed, Web of Science, China National Knowledge Infrastructure and Google Scholar were used for reviewing the published literature on household emissions, exposures, health effects, and interventions related to household coal use. Websites and databases of relevant organizations around the world were searched for coal use data and intervention policies. The background material for a recent IARC monograph (#95) on household coal use was also reviewed. Searches were conducted for Chinese and English language publications only.

**Main Findings**

Coal burned in small stoves shares certain emissions with biomass fuels, for example, products of incomplete combustion (e.g., carbon monoxide, polycyclic aromatic hydrocarbons, and fine particles). Based on relative emissions, therefore, health effects can be considered to be similar for the disease categories in Review 3 4 for products of incomplete combustion in general. Coal combustion, however, also produces sulfur dioxide, nitrogen oxides and a range of toxic pollutants (e.g., fluorine, arsenic, selenium, mercury, and lead) from coals mined in certain geographical regions. In poor combustion conditions, coal also produces a different mixture of polycyclic aromatic hydrocarbons (PAHs) and PAH derivatives as well as other complex organic materials than does biomass. We find strong evidence for household coal use and lung cancer, limited evidence for cancers of other organs, emerging evidence on developmental effects (lung, skeletal, neurobehavioral, and fetal development), carbon monoxide poisoning ,and endemic diseases (e.g. arseniasis, fluorosis, selenosis) caused by "poisonous" coal, i.e. coals containing significant concentrations of toxic elements (arsenic, fluorine, and selenium). Lead and mercury emissions are also of potential concerns.

Interventions in terms of coal bans, behavioral incentives, and certain technologies (e.g. chimney stoves) are reviewed and discussed. On a larger scale, most developed countries have either banned or greatly restricted household coal use to mitigate mainly urban outdoor

air pollution. With similar motivations, most large Chinese cities have also banned household coal use in the last decade or so, although these bans are not yet completely enforced. On the other hand, coal use seems to be rising in rural areas in China. There have been no coal ban policies, ironically, that stem from concerns of indoor air quality, despite that the greater fraction of exposure occurs indoors where coal is combusted.

Assessments of technology interventions indicate that a high degree of processing of certain coals may make them sufficiently cleaner-burning to protect health, but few modern studies were found to document the health benefits in a consistent manner. Integrated behavior and technology interventions have been more effective than only deploying improved fuel/stove technologies to improve household indoor air quality and to reduce the rate of arsenicosis in households using coal with high arsenic content.

**Conclusions**
As with biomass, it is difficult to burn coal cleanly in household appliances, but the wide variety of coal characteristics make generalizations more difficult. Nevertheless, it is concluded that household use of raw coal, with or without contaminants, presents significant risks to health, even with use of chimneys. Poisonous coals, i.e., those with high levels of toxic contaminants, should be banned entirely for household use. At this point, we are not able to make a judgment about the potential of processed "non-poisonous" coal to be used as a household fuel without risks to health, and this should be the subject of further review, with new investigations if studies addressing these questions are not available. In addition, there is a need for both an international and historical assessment of the policies that have been used to reduce household coal use and their effectiveness in different contexts. Even though recommending more systematic research on processed coal alternatives, we believe that the available evidence and the long negative experience worldwide support policies that strongly discourage household use of coal, including cessation of raw coal use, to protect health.

# 1. Introduction

Coal has been the most important fossil fuel that propelled the industrial revolution. Today it is still the largest source of energy for the generation of electricity worldwide and is an important household fuel in some countries, although this role has diminished in many others. Household coal emissions of particulate matter and sulfur dioxide lead to local air pollution today as in the past. Indeed, household use of coal for heating was the principal source of the infamous air pollution episode in 1952, called the London "smog" (smoke plus fog), although today smog commonly refers to photochemical smog characterized by high concentrations of ozone. Excess mortality and morbidity associated with urban pollution episodes were significant enough to prompt modern clean air regulations in the United Kingdom and other developed countries more than a half century ago. To curtail these episodes, the U.K. introduced smoke control areas and cities in which only smokeless fuels were allowed in 1956 (effective in 1964). Similarly, raw coal use or sale was banned in cities of most other developed countries many decades ago, with the last conversion occurring in Dublin in the 1990s *(1)*. However, such coal is still being used as a household fuel in many parts of the developing world, including, for example, India where nearly 20 million people use it for cooking. The largest number of households still using coal, however, lie in China and elsewhere in the Western Pacific region where it makes up around 20% of total household fuel use, while 6%-7% of total fuel consumption is in the form of coal globally (see the 2010 WHO data in Figure 1). Over 400 million people in the world rely on coal for cooking and an additional but an uncertain number of people use coal for household heating. In China, about 44.2% of total households used coal for cooking and 35.6% of total households used coal for heating in 2011*(2)*.

**Figure 1: Global household coal use by region (2010): the bars represent percent of coal consumption in all household cooking fuels (biomass, coal, kerosene, LPG, gas and electricity).**
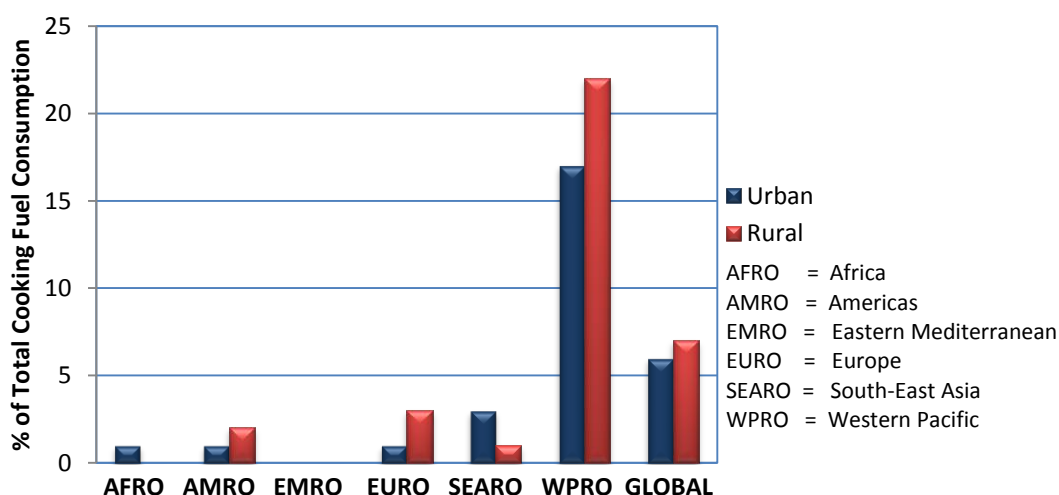


Source: WHO Global household energy database *(3)*

In common with biomass fuels, it is difficult to combust coal completely (efficiently) in simple household combustion devices such as stoves for cooking and/or heating. Thus, a significant fraction of fuel carbon is converted into products of incomplete combustion *(4)*. However, coal typically has a higher sulfur content than biomass; and hence burning coal emits more sulfur dioxide ($SO_2$) for the same amount of heat energy delivered to the pot or room *(4)*. Because combustion temperature for coal is often higher than for biomass, a coal

stove may emit more $NO_2$ than a biomass stove for delivery the same amount of heat *(4)*. The amount of other pollutants emitted from coal combustion largely depends on coal type or coal quality. For example, certain bituminous coals with high content of volatile constituents, which are widely used in Chinese households, generates so much smoke (particulate matter, or soot) that they are called "smoky" coals *(5)*.

Furthermore, depending on where coal deposits were formed, coal contains varying quantities of other toxic elements such as mercury, arsenic, fluorine, lead and selenium. Initially a total of 11 trace elements emitted during coal combustion were identified as hazardous air pollutants (U.S. National Committee for Geochemistry 1990). Recently, this number has been increased to 26. Some coals contain uranium, thorium and other radionuclides *(6)*. Based on toxicological properties of these toxic elements (and/or associated compounds), exposure to the emissions of such intrinsically "contaminated" coals is expected to result in adverse health effects. Household use of coals containing some of these toxic elements, especially under open fire burning conditions and some special behaviors such as drying foodstuff by coal smoke, has been associated with severe endemic health problems in some provinces of China *(6, 7)*.

These additional features of coal combustion in household settings have been associated with or imply additional or different health risks compared to household biomass combustion. We thus review the scientific literature on health effects specific to coal smoke exposure. Since the health effects of general household air pollution (HAP) from solid fuels including both coal and biomass are extensively reviewed in Review 4, here we focus on the carcinogenic effect of coal emissions for which there is stronger evidence than for biomass, on specific health issues related to coals intrinsically containing toxic elements, and on a few other health outcomes with emerging evidence (e.g., pneumonia death in adults, and children's lung and neurobehavioral development).

## 2. Key questions for reviews

The following reviews of evidence have been carried out in order to address four key questions relating to the formulation of recommendations on the household use of coal.

1. **What are characteristics of combustion emissions specific to coal?** A narrative review summarising information on global use of coal, with a focus on household use, and content of various coals in respect of key pollutant emissions, including toxic contaminants.

2. **What are the specific adverse health effects of household coal combustion, in addition to the effects of products of incomplete combustion shared with biomass?** A systematic review of respiratory and other health risks arising from exposure to the combustion mixtures from coal, drawing on the material reviewed for solid fuel pollution exposure in Review 4 (including three recently published systematic reviews of coal use and lung cancer), and a summary of the main findings from the International Agency for Research on Cancer (IARC) Monograph 95 *(8)* review on the carcinogenicity of household coal for cooking.

3. **What are the health risks from toxic contaminants in coal?** A systematic review of the exposure routes and health consequences of toxic contaminants in coal.

4. **What are the impacts of interventions to reduce risk from household use of coal, including the history and extent of bans on household coal use (with focus on China)?** A systematic review of interventions (including bans and other restrictions) aimed at reducing the risks and public health impacts of household coal

use and the extent to which these have, and can, reduce risks to an acceptable level based on air quality standards or guidelines.

These sections are followed by an assessment of the overall quality of available evidence, and conclusions regarding what needs to be done to reduce health risks.

# 3. Methods for Review

**Information Sources**
Literature published between 1980 and December 2013 was searched in the following peer review databases: PubMed and Web of Science (for the literature in English); China National Knowledge Infrastructure (CNKI) (for the literature in Chinese). Other sources such as the WHO household energy database and websites of relevant organizations (e.g. World Coal Association) were searched for coal use; IARC website was searched for evidence of cancer; government websites were searched for policies of coal banning or clean air act related information on intervention. Google Scholar was also searched for health effects of coal and interventions on coal use. No attempt was made to search databases in languages other than English and Chinese.

**Search Terms**
The following sets of search terms were used for the systematic reviews (and translated into Chinese when searching the Chinese literature).

*Health effects*
For health effects of household coal, we used "coal" in combination with "indoor" or "domestic" or "household" for exposure and in combination with "health effects". We also used "coal" in combination of one of the following health outcomes: cancer, neural tube defects, developmental effects, blood pressure, immune function, lung function, respiratory disease, COPD, tuberculosis, ALRL, CVD, cognitive development, asthma, cataract, CO poisoning.

*Toxic contaminants*
For exposure and health effects related to intrinsic toxicants, we used "coal" plus "fluorine"; "arsenic" ; "mercury" ; "lead " ;"selenium" ;"cadmium" ;"chromium" "element" for exposure and "health/poisoning" or "arseniasis"; "fluorosis"; "selenosis" for health effects. The selection of toxicants was based on the recommended hazardous elements by U.S. National Committee for Geochemistry *(9)* and the available information.

*Interventions and restrictions on use of coal*
For interventions, we combined various search term strategies from both policy and intervention related terms. For policies we use "coal" in combination with "ban", "policy", "and clean air". For intervention technologies, we used "coal" and "intervention" in combination with "fuel improvement"; "stove improvement" or "behavior change".

**Criteria for literature selection**
For health effects related to intrinsic toxicants, the following criteria are used for literature inclusion: (a) exposure from household coal combustion only (excluding exposure from drinking water, coal mining or industrial coal burning); (b) studies in humans (excluding studies in experimental animals or laboratory-based mechanistic studies and medical treatment cases); (c) health effect-related studies that have included exposure assessment; (d) intervention studies that have assessed health benefits (excluding those that have only assessed indoor air quality changes). In addition, studies with reliable quality assurance methods are preferred (see Table 1 and Figure 2).

Our review is focused on health effects associated with unique features of coal combustion. Hence the literature on general household solid fuel combustion, as covered in or overlapped with those in Review 4, are not reviewed here.

**Extraction of Results**
We extracted information on contents of toxicants in coal, indoor air levels for exposure and prevalence of disease, to the maximal extent based on the above criteria in order to summarize the effects of intrinsic coal toxicants and health.

For cancer risk, we draw on two published systematic reviews and a meta-analyses of case-control studies which were performed to assess the relationship between household coal use and lung cancer *(10) (11)*, and the IARC report in which emissions from household coal use are classified as Group 1 carcinogen *(8, 12)*.

For other health risks related to products of incomplete combustion, we added recent findings to those summarized in a previous narrative review in which Zhang and Smith *(5)* have critically evaluated individual publications prior to 2005 on exposure and health effects of household coal use in China.

**Methods for Individual Quality Assessment**
The quality assessment (QA) methods used for the individual studies consisted of evaluating evidence for health risk from coal exposure in comparison to the overall body of evidence of available studies covering the health topics as discussed in this review.

Individual studies were evaluated based on various criteria as established in general methods of quality assessment. Assessment criteria include:
- evaluation of the number of cases/participants studied;
- reviewing how subjects were assessed to be eligible within the given parameters of a study including screening methods and inclusion/exclusion criteria;
- specific study characteristics, e.g. length of follow-up, for any possible language barriers, and publication status;
- evaluation of outcome measures, looking at risk ratio as the principal summary measure, also looking at differences in means, percent effect of interventions, etc.;
- risk of bias in both study and outcome level looking for selected reporting and other possible evidence that may have affected the results, including adjustment for confounding.

Examples of how these have been applied, including allocation of a score (stars) out of a maximum of 10, are presented in Annex Table A.2. In choosing our selected papers for review, we used studies that (1) used higher quality exposure assessment involving clear descriptions of exposure contrasts or actual measurements where available; (2) used reliable health effects confirmation methods; and/or (3) implemented good quality assurance and control protocols for data collection and management.
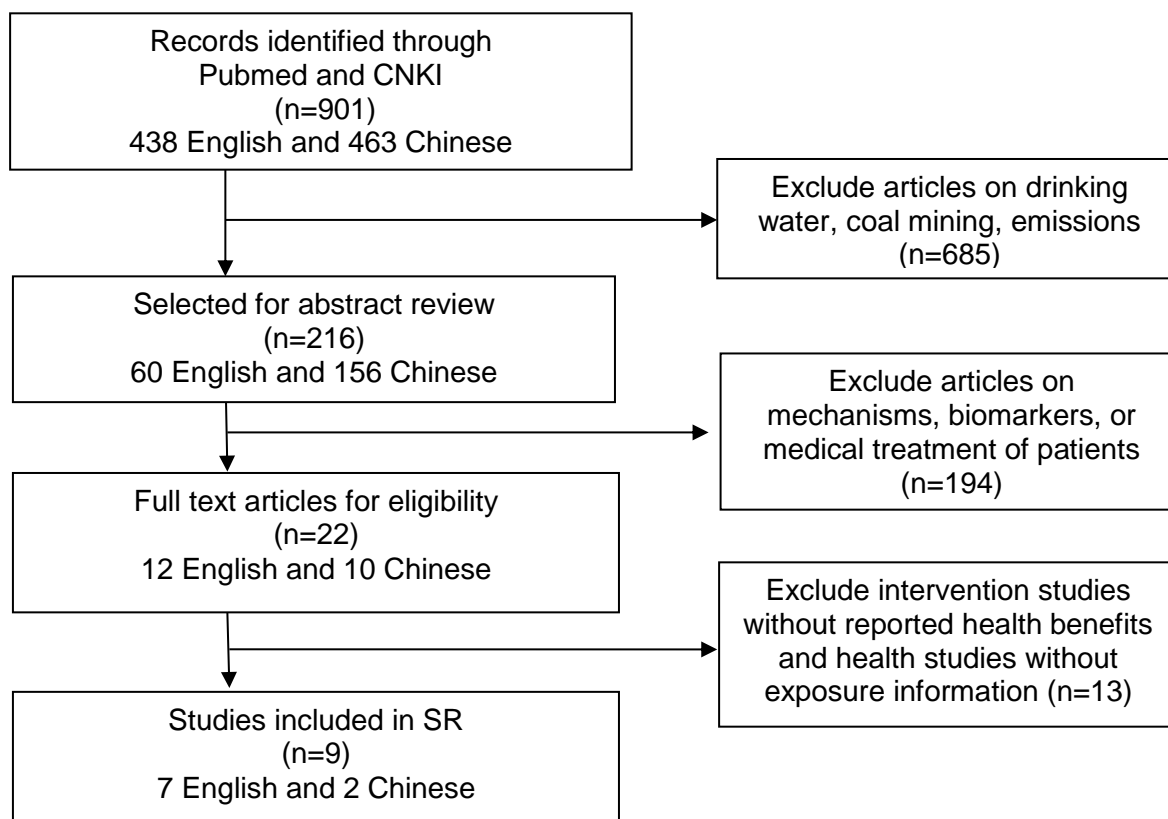
**Table 1: Methods for Systematic Reviews of Unique Features of Exposure to Intrinsic Toxicants and Health Effects**

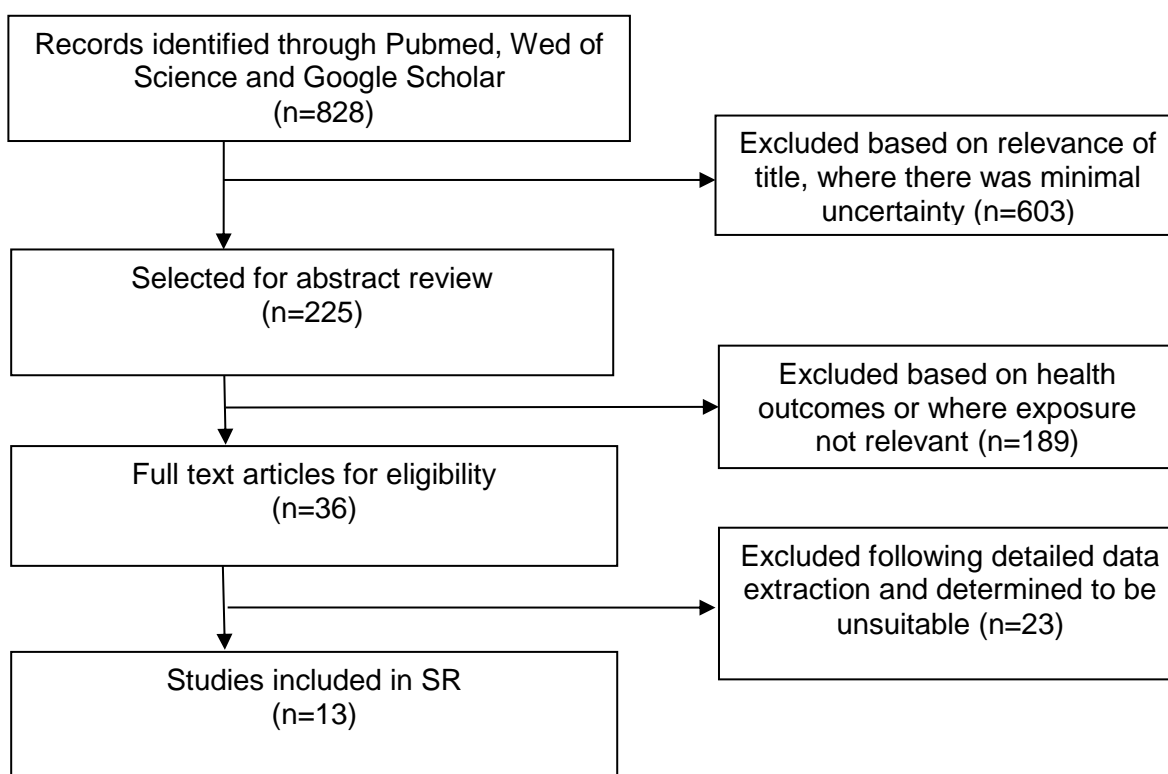| | | Effects related to intrinsic toxicants | Cancer | Other health effects |
|---|---|---|---|---|
| **Information sources** | **Literature databases** | English: PubMed, Web of Science, Google Scholar. Timeframe covered:1980 to December 2013 <br> Chinese: China National Knowledge Infrastructure (CNKI)* Timeframe covered: 1980 to December 2013 | | |
| | **Other sources** | IARC reports for evidence of cancer; WHO household energy database, websites of relevant organizations (e.g. World Coal Association) for coal use; government websites for policies of coal banning or clean air. | | |
| **Search Terms** | *Exposure* | "Coal" In combination with "fluorine"; "arsenic"; "mercury"; "lead "; "selenium"; "cadmium"; "chromium"; "element" | "Coal" in combination with "indoor/domestic/ household" if necessary | "Coal" in combination with "indoor/domestic/household" if necessary |
| | *Health Effects* | "health/poisoning" or "arseniasis"; "fluorosis"; "selenosis" for particular toxicants | "lung cancer" <br> Not searched intentionally for other cancers | neural tube defects, developmental effects, blood pressure, immune system impairment, lung function reduction, respiratory, COPD, tuberculosis, ALRL, CVD, cognitive development, asthma, cataract, CO poisoning |
| | *Intervention* | Search policies "coal ban"; "clean air" <br> Search intervention methods use "intervention" in combination with "fuel improvement"; "stove improvement" or "behavior change" if necessary | Not searched intentionally since Review 4 specifically covers this | Not searched intentionally since Review 4 specifically covers this |
| **Criteria for literature selection** | **Reviews** | References: *(5, 6, 13)* | Select the most recent high quality SR; references: *(10) (8, 12)* | Reference: *(5)* |
| | **Individual studies** | a) exposure from household coal combustion not geochemical drinking water problems; coal mining or industrial coal burning <br> b) human not animal studies, health studies not on mechanisms, biomarkers, or medical treatment health effect-related studies; that have quality exposure assessment where available, intervention studies with substantial health benefits outcomes using reliable confirmation methods. | Individual studies not included | Focus on the effects of coal particularly, distinct from biomass |

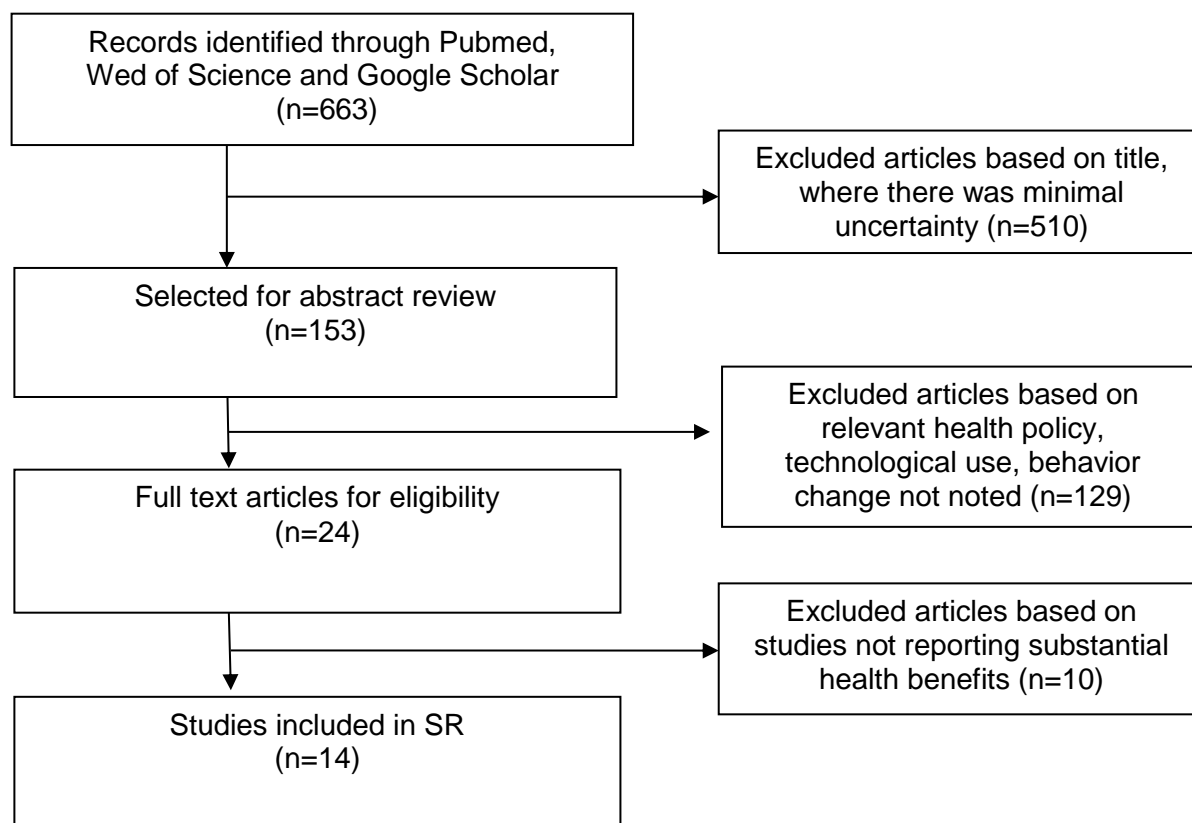*CNKI was searched for household poisonous coal use and effects only.

**Figure 2**

**a. Flowchart for study selection on household poisonous coal use and effects**

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┌─────────────────────────────────┐
│   Records identified through    │
│        Pubmed and CNKI          │         ┌──────────────────────────────┐
│           (n=901)               │────────▶│ Exclude articles on drinking  │
│  438 English and 463 Chinese    │         │ water, coal mining, emissions │
└─────────────────────────────────┘         │          (n=685)             │
                │                            └──────────────────────────────┘
                ▼
┌─────────────────────────────────┐
│   Selected for abstract review  │         ┌──────────────────────────────┐
│           (n=216)               │         │    Exclude articles on       │
│  60 English and 156 Chinese     │────────▶│  mechanisms, biomarkers, or  │
└─────────────────────────────────┘         │ medical treatment of patients│
                │                            │          (n=194)             │
                ▼                            └──────────────────────────────┘
┌─────────────────────────────────┐
│ Full text articles for eligibility│       ┌──────────────────────────────┐
│           (n=22)                │         │ Exclude intervention studies │
│  12 English and 10 Chinese      │────────▶│ without reported health      │
└─────────────────────────────────┘         │ benefits and health studies  │
                │                            │ without exposure information │
                ▼                            │          (n=13)             │
┌─────────────────────────────────┐         └──────────────────────────────┘
│     Studies included in SR      │
│           (n=9)                 │
│  7 English and 2 Chinese        │
└─────────────────────────────────┘
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**b: Flowchart for study selection on health effects due to coal use**

```
┌─────────────────────────────────┐
│ Records identified through Pubmed,│      ┌──────────────────────────────┐
│   Wed of Science and Google     │       │ Excluded based on relevance of│
│      Scholar (n=828)            │──────▶│ title, where there was minimal│
└─────────────────────────────────┘       │      uncertainty (n=603)      │
                │                          └──────────────────────────────┘
                ▼
┌─────────────────────────────────┐
│   Selected for abstract review  │        ┌──────────────────────────────┐
│           (n=225)               │        │   Excluded based on health   │
└─────────────────────────────────┘───────▶│ outcomes or where exposure   │
                │                           │    not relevant (n=189)      │
                ▼                           └──────────────────────────────┘
┌─────────────────────────────────┐
│ Full text articles for eligibility│      ┌──────────────────────────────┐
│           (n=36)                │        │Excluded following detailed data│
└─────────────────────────────────┘───────▶│ extraction and determined to be│
                │                           │      unsuitable (n=23)       │
                ▼                           └──────────────────────────────┘
┌─────────────────────────────────┐
│     Studies included in SR      │
│           (n=13)                │
└─────────────────────────────────┘
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**c: Flowchart for study selection on interventions on coal use**

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┌─────────────────────────────┐
│ Records identified through  │
│ Pubmed, Wed of Science and  │        ┌──────────────────────────────┐
│ Google Scholar              │───────▶│ Excluded articles based on   │
│ (n=663)                     │        │ title, where there was       │
└─────────────────────────────┘        │ minimal uncertainty (n=510)  │
              │                         └──────────────────────────────┘
              ▼
┌─────────────────────────────┐
│ Selected for abstract       │
│ review                      │        ┌──────────────────────────────┐
│ (n=153)                     │        │ Excluded articles based on   │
└─────────────────────────────┘───────▶│ relevant health policy,      │
              │                         │ technological use, behavior   │
              ▼                         │ change not noted (n=129)     │
┌─────────────────────────────┐        └──────────────────────────────┘
│ Full text articles for      │
│ eligibility                 │        ┌──────────────────────────────┐
│ (n=24)                      │        │ Excluded articles based on   │
└─────────────────────────────┘───────▶│ studies not reporting        │
              │                         │ substantial health benefits  │
              ▼                         │ (n=10)                       │
┌─────────────────────────────┐        └──────────────────────────────┘
│ Studies included in SR      │
│ (n=14)                      │
└─────────────────────────────┘
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# 4. Characteristics of Household Combustion of Coal

## 4.1 General products of incomplete combustion (PICs)

Under the ideal condition of 100% combustion efficiency, all the carbon in a carbonecous fuel (e.g., biomass, coal, kerosene, LPG, natural gas, diesel, and gasoline) would be completely converted to $CO_2$ while releasing the fuel chemical energy through oxidation as thermal and, radiant, t energy. However, the nominal combustion efficiency of simple household stoves burning solid fuels is generally in the range of 80% to 95%. This means a substantial fraction (5%-20%) of the fuel carbon is diverted into products of incomplete combustion (PICs) (see Review 2: Pollutant emissions). Like burning biomass, burning coal in typical household cooking and heating stoves generates PICs (e.g., CO and carbonaceous particles) in sufficient quantity to make indoor air concentrations of CO and $PM_{2.5}$ exceed health-based standards, as discussed in Review 2 and Review 5: Population levels of HAP and Exposure. Therefore, household coal combustion increases health risks associated with $PM_{2.5}$, CO, and the general combustion mixture evaluated in Review 4.

## 4.2 Emissions of pollutants other than general PICs from coal in households

Compared to biomass, coal usually contains higher levels of toxic elements and associated compounds such as sulfur and, in some coals, fluorine, arsenic, mercury, selenium, and lead, etc. (see Table 2).

**Table 2: Contents of Trace Elements in Coals**

**Fluorine**

| | Types of Coal | Sample Size | Contents (mg/kg) | |
|---|---|---|---|---|
| | | | Range | Average |
| Coals in Different Countries | Chinese | 305 | 47-347 | 152 |
| | US | 7367 | 0-4000 | 98 |
| | Australian | 74 | 20-300 | 110 |
| | Canadian | 57 | 31-580 | 154 |
| Selected Coals in China | Lignite | 25 | 71-889 | 223 |
| | Coking coal | 35 | 63-375 | 159 |
| | Anthracite | 49 | 38-520 | 136 |

Reference: *(14)*

**Mercury**

| | Types of Coal | Sample Size | Contents (mg/kg) | |
|---|---|---|---|---|
| | | | Range | Average |
| Coals in Different Countries | Coal | 1466 | 0.01-1.00 | 0.15 |
| | US-East | NA | 0.09-0.51 | 0.22 |
| | Australian | NA | 0.03-0.25 | 0.087 |
| | Poland | NA | 0.14-1.78 | 0.72 |
| Selected Coals in China | Lignite | NA | NA | 0.383 |
| | Coking coal | NA | NA | 0.268 |
| | Anthracite | NA | NA | 0.184 |

References: *(15)*, *(16)*

**Arsenic**

| | Types of Coal | Sample Size | Contents (mg/kg) | |
|---|---|---|---|---|
| | | | Range | Average |
| Coals in China (Guizhou and Shanxi) | High arsenic | 227 | 125.8-833.5 | NA |
| | Normal | 846 | 2.6-37.5 | NA |
| | Coking | NA | NA | 0.268 |
| | Anthracite | NA | NA | 0.184 |

Reference: *(14), (17)*

**Selenium**

| | Types of Coal | Sample Size | Contents (mg/kg) | |
|---|---|---|---|---|
| | | | Range | Average |
| Coals in Different Countries | US | 7563 | | 2.8 |
| Selected Coals in China | | 1123 | | 2.82 |

Reference: *(14), (7)*

**Lead**

| | Types of Coal | Sample Size | Contents (mg/kg) | |
|---|---|---|---|---|
| | | | Range | Average |
| Coals in Different Countries | US | 7469 | | 11 |
| Selected Coals in China | | 1123 | | 16.64 |

Reference: *(14), (7)*

NA=not available (i.e. not reported in the original study).

During combustion, these toxic elements are released into the air typically in their oxidized form. For example, sulfur is mainly released as sulfur dioxide ($SO_2$), a pollutant that is commonly regulated in many countries. Concentrations of $SO_2$ exceeding China's IAQ standard of 500 µg/m$^3$ for 1 hour (as high as 23,000 µg/m$^3$) have been often measured inside of coal-using households in both rural and urban settings [(see Figure 3 (a) and (b)].

Coal combustion takes place at a higher temperature than biomass combustion; and higher temperature favors the formation of nitrogen oxides from nitrogen contained in the fuel and

also nitrogen in the air. As a result, indoor $NO_2$ concentrations in coal-using households can be as high as or higher than $NO_2$ concentrations measured in biomass or gas using homes [(see Figure 3 (c) and (d)]. As shown in these figures, a larger fraction of coal-using homes appear to have kitchen $NO_2$ concentrations exceeding China's IAQ standard of 240 µg/m$^3$ (1 hour average) than that of gas-using homes in China. There has also been concern about indoor $NO_2$ exposures resulting from gas cooking *(18, 19)*, an issue which is the subject of a recently published systematic review described in Review 4. In contrast, biomass-using homes had kitchen $NO_2$ concentrations well below the standard.

**Figure 3: Concentrations of pollutants measured in solid-fuel-use households by location (bedroom, kitchen, livingroom, unspecified inside a home) and fuel type (coal, gas including natural gas and LPG, biomass, coal and gas mixed).**

**(a) Sulphur dioxide (rural)**



Note: Extremely high values may not be shown so that the plot scales are more appropriate for the vast majority of the data points. In the figure, n is number of data points, µ is arithmetic mean, and σ is the standard deviation. On the 'box plots', the lines and the box from bottom to the top represent 5th, 25th, 50th, 75th, and 95th percentile, respectively. The data were obtained from measurements made in Chinese households and summarized in a previous conference proceedings paper authored by Zhang and Smith, *(20)*.

## (b) Sulphur dioxide (urban)
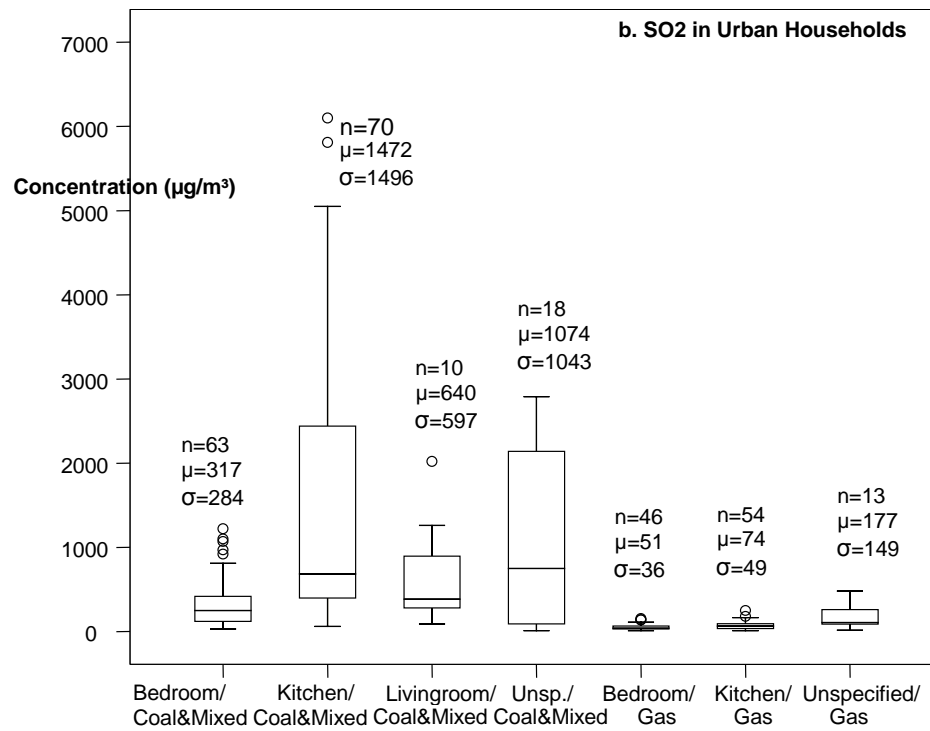


## (c) Nitrogen dioxide (rural)
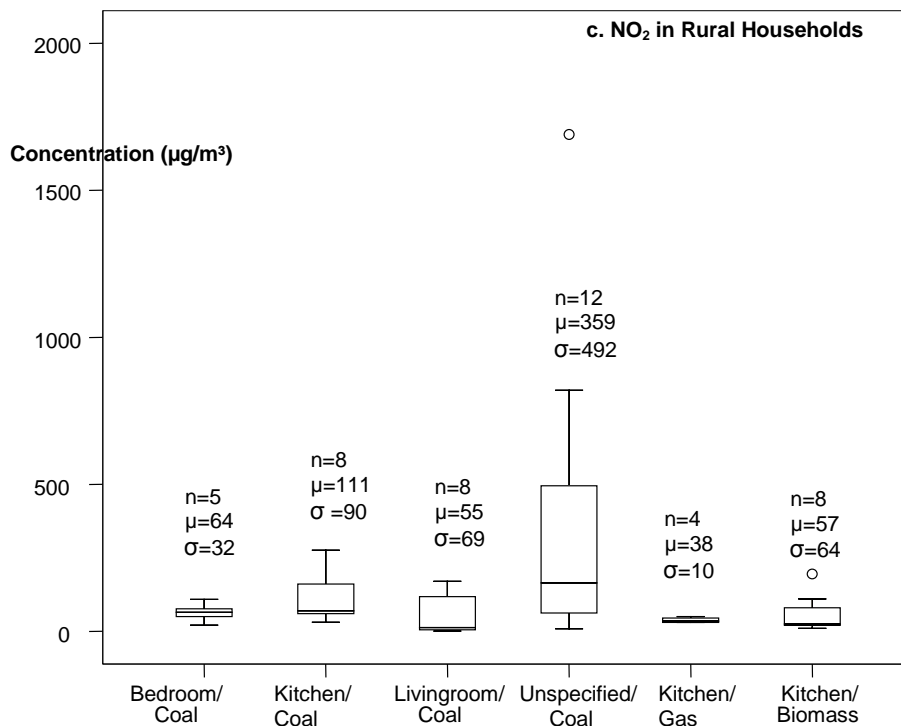


Note: Extremely high values may not be shown so that the plot scales are more appropriate for the vast majority of the data points. In the figure n is number of data points, $\mu$ is arithmetic mean, and $\sigma$ is the standard deviation. On the 'box plots', the lines and the box from bottom to the top represent 5th, 25th, 50th, 75th, and 95th percentile, respectively. The data were obtained from measurements made in Chinese households and summarized in a previous conference proceedings paper authored by Zhang and Smith, (20).

**(d) Nitrogen dioxide (urban)**



Note: Extremely high values may not be shown so that the plot scales are more appropriate for the vast majority of the data points. In the figure n is number of data points, μ is arithmetic mean, and σ is the standard deviation. On the 'box plots', the lines and the box from bottom to the top represent 5th, 25th, 50th, 75th, and 95th percentile, respectively. The data were obtained from measurements made in Chinese households and summarized in a previous conference proceedings paper authored by Zhang and Smith, *(20)*.

During coal combustion, other toxic elements are also released into the air, resulting in endemic coal poisoning. These coals are, hence, called 'poisonous' coals in China. Although the health effects of poisonous coals have been assessed (see Section 4.3), emissions and indoor concentrations of the toxic elements or compounds have been rarely measured in published studies.

## 4.3 Other features of coal combustion in the home

Another feature of coal combustion, relative to biomass combustion, is that it takes longer to start the fire, longer to reach to the steady full flame stage of the combustion, and longer to extinguish the fire. This means more prolonged exposure to pollution emitted from coal, especially during the 'initial' and 'extinguishing' stages of the combustion cycle. At both of these stages, combustion tends to be least efficient (often with visible smoke) *(21)*. Thus, although high-quality coal may be combusted relatively efficiently in large industrial or commercial facilities that employ so-called 'clean-coal' technologies, it is difficult to burn coal completely in household settings through intermittent combustion cycles, often coupled with simple stove technologies and low and sometimes varying fuel quality.

## 5. Health Effects of Household Coal Use

The effects of household coal use on lung cancer, as derived from recent published systematic reviews, are summarized in Table 3. The other (non-cancer) health effects as reviewed in this section including respiratory effects, developmental and reproductive effects, and acute CO poisoning are summarized in Table 4.

## 5.1 Cancer

### 5.1.1. Lung cancer

Exposure to HAP arising from coal combustion has long been recognized as a cause of lung cancer, and possibly for cancers at other organs *(10, 11, 22, 23)*. In 2010, IARC published a monograph on 'Household Use of Solid Fuels and High-temperature Frying' *(8)*, in which emissions from household coal use are classified as a Group 1 carcinogen (i.e., definitely carcinogenic to humans), mainly based on the available evidence on lung cancer. In contrast, emissions from household combustion of biomass were classified as a Group 2A carcinogen at that time *(8)* .

In a recent systematic review and meta-analysis, Hosgood et al. reviewed 16 English language and 9 Chinese language case-control studies published through 2009, using criteria that included specifying coal use distinct from biomass *(10)*. The overall pooled odds ratios (OR) for all studies are 2.15 (1.61, 2.89), 2.50 (1.56, 4.00) for women (8 studies) and 2.76 (1.44, 5.27) for men (3 studies). The risk estimates (ORs) reported in this review are comparable to that reported from an early review of studies including non-case-control studies for women and men combined, but higher when reported separately for women and men. In the earlier review, drawing on data from 19 studies investigating risks associated with exclusive coal or mixed coal and biomass use for cooking and/or heating. Smith et al. *(24)* derived pooled, adjusted odds ratios of 2.55 (1.58, 4.19) for men and women combined, 1.51 (0.97, 2.46) for men only, and 1.94 (1.09, 3.47) for women only.

The second recent systematic review, published recently *(11)*, examined risk with exposure to both coal and biomass. For coal, 22 studies (28 estimates) including Chinese language studies, were included. While all study designs were eligible, most were case-control and inclusion criteria required that effect estimates were adjusted. The overall pooled OR was 1.82 (1.60, 2.06). Sensitivity analysis reported pooled ORs of risks 1.54 (1.25, 1.88) for men (3 studies) and 1.70 (1.40, 2.06) for women (10 studies), and a larger effect in the studies combining men and women.

Findings from these reviews indicate that although risks of lung cancer with household coal use may vary by location and type, elevated risks are seen with coal use across a wide range of settings. The estimates show roughly a 2-fold increased risk of lung cancer associated with household coal use and a higher risk for women than men, possibly reflecting higher exposures in women who normally cook for their families.

### 4.1.2. Cancers of other organs

Compared to the evidence supporting the lung cancer risk, the evidence for a link between household coal use and cancers of other organs is more limited. Non-lung cancers examined to date, in relation to coal (and/or solid fuel including both coal and biomass), include cancers of the upper aero-digestive tract (UADT: the larynx, oropharynx, hypopharynx, nasopharynx, paranasal sinuses, and middle ear), cancer of the uterine cervix, and cancer of the kidney (renal cell carcinoma) *(8)*. The associations of household coal use and these cancers are biologically plausible given that coal smoke contains many carcinogens (e.g., polycyclic aromatic hydrocarbons) found in tobacco smoke that has been linked to these cancers. Ingestion of food contaminated with coal smoke may also contribute to cancer risks *(5)*. However, cancers of other organs have been studied in fewer studies, generating risk estimates with large heterogeneity across studies. As such, the Global Burden of Disease study (GBD) 2010 project comparative risk assessment (CRA) included updating evidence on cancers of UADT and uterine cervix, but excluded these cancers from the GDB estimates given the insufficiency of the evidence (see Review 4).

## 5.2 Other health effects

### 5.2.1 Respiratory effects

Given that a large body of literature exists on the respiratory effects of $PM_{2.5}$ and other combustion-generated pollutants (see Review 4), a direct link between household coal use and adverse respiratory outcomes is expected. As summarized in the review of Zhang and Smith (2007), household coal use has been associated with wheeze, asthma, rhinitis, faucitis, tonsillitis, cough, bronchitis, and pneumonia in children. In adults, household coal use has been associated with chest pain, cough, phlegm, shortness of breath, and, importantly, COPD (5). After this 2007 review article, new evidence has been presented that indoor air pollution from unvented coal burning is a risk factor for pneumonia death in adults (25). The evidence was based on the findings of a retrospective study that used a large cohort (n=42,422) with a long follow-up period to identify all deaths from 1976 through 1996 in Xuanwei County, Yunnan Province of China. The study found not only a positive association between household coal use and pneumonia mortality in adults but also a 50% reduction in pneumonia deaths associated with stove improvement by installing a chimney (25).

### 5.2.2 Developmental and reproductive effects

#### 5.2.2.1 Lung development

Household coal use has been associated with retarded lung development of children, measured as decreased lung function, forced vital capacity (FVC), forced expiratory volume in 1 sec (FEV1), or peak expiratory flow rate (PEFR). This has been demonstrated when lower lung function values were measured in Chinese children whose households used coal for cooking and/or heating than in children living in natural gas or LPG use households (5). The detrimental effect of coal smoke exposure has been further demonstrated in a longitudinal examination of lung function growth rate (26). In this study of 3273 children aged 6-13 years living in four Chinese cities, use of coal as a household fuel was associated with 16.5 ml/year lower (33%, P < 0.001) and 20.5ml/year lower (39%, P < 0.001) growth in children's FEV1 and FVC, respectively, in reference to the use of gas or LPG as a fuel. Among children living in houses where coal was used as a fuel and no ventilation devices were present, covariate-adjusted FVC and FEV1 growth, respectively, were 37% and 61% that of the average growth per year in the full cohort. The findings provide direct evidence that household coal use may cause deficits in lung function growth and that venting coal smoke outdoors is protective of children's lung development. As retarded lung function growth in childhood is associated with a higher risk in many diseases throughout the lifetime, it is important to avoid early life exposure to coal smoke (26).

The associations between household coal use and lung function reductions has also been found in adults in several studies conducted in China, as reviewed by Zhang and Smith (2007). It is noteworthy to mention a study that compared the effects of smoky coal, smokeless coal, and wood on lung function (27). The results show that the strongest risk factor for lowered lung function (measured as peak expiratory flow rate) was smoky coal, followed by smokeless coal, then by wood. This study also found that the use of coal stoves with chimneys was associated with higher lung function values compared with the use of open fire pits (with no chimneys).

#### 5.2.2.2 Early childhood height (skeletal) growth

Exposure to ambient (outdoor) $PM_{2.5}$ has been associated with slower growth in children's height in a handful of studies (28, 29). In a recent prospective longitudinal study conducted in the Czech Republic, a total of 1133 children were followed from birth to 36 months old. Adjusted for covariates, at age of 36 months, indoor coal use was associated with a reduction in height of about 1.34 cm (95% CI: 0.51, 2.16) for boys and1.30 cm (95% CI:

0.50, 2.10) for girls raised in homes that used coal. These findings suggest that HAP from coal use may impair early childhood skeletal growth at least up to 36 months old *(30)*.

### 5.2.2.3 Neurobehavioral development

Evidence has also emerged suggesting that prenatal exposure to coal-burning pollutants adversely affects children's neurobehavioral development *(31, 32)*. In a study conducted in Tongliang, Chongqing, China, where a seasonally operated coal-fired power plant was the major source of ambient PAHs and also contributed lead and mercury to the air, a cohort of non-smoking women and their newborns were enrolled between March 2002 and June 2002 *(32)*. In the study, measured levels of PAH-DNA adducts, lead, and mercury in umbilical cord blood were used as internal dose markers of coal exposure; and developmental quotients (DQs) in motor, adaptive, language, and social areas were obtained to assess neurobehavioral development status of children at 2 years of age. The study found that decrements in one or more DQs were significantly associated with cord blood levels of PAH-DNA adducts and lead. Increased adduct levels were associated with decreased motor area DQ (p = 0.043), language area DQ (p = 0.059), and average DQ (p = 0.047) after adjusting for cord lead level, environmental tobacco smoke, sex, gestational age, and maternal education. In the same model, high cord blood lead level was significantly associated with decreased social area DQ (p = 0.009) and average DQ (p = 0.038).

In a follow-up study, Perera et al. (2008) evaluated the benefit to neurobehavioral development from the closure (in May 2004) of the same coal-fired power plant in Tongliang. A new cohort of non-smoking women and their newborns were enrolled in 2005 (after shutdown) and were compared with the original cohort described above (enrolled before shutdown) regarding the association between PAH-DNA adduct levels and neuro-developmental outcomes. Significant associations previously seen in 2002 between elevated adducts and decreased motor area developmental quotient (DQ) and average DQ were not observed in the 2005 cohort (p = 0.546 and p = 0.146). However, the direction (i.e., negative or positive association) of the relationship did not change. The findings from this follow-up "intervention" study increases the certainty about the causal relationship between retarded neurobehavioral development and exposure to coal combustion pollutants. Given that household coal combustion emits the same pollutants (especially PAHs) and that pregnant women in coal-use households are likely to have higher exposures than the Tongliang women, it is reasonable to expect similar effects in children exposed to household coal emissions. However, there have been no neurobehavioral development studies carried out specifically among households using coal.

### 5.2.2.4 Neural tube birth defects

Emerging evidence suggests that household coal use is a potential risk factor for neural tube defects (NTDs). In a population-based case-control study, 610 NTD cases and 837 normal controls between November 2002 and December 2007 were identified in a rural population in Shanxi Province, China. Compared with women with no exposure, women with coal smoke exposure had a 60% increased risk of having a child with an NTD (adjusted OR=1.6, 95% CI: 1.1, 2.1). An increased NTD risk was linked to both residential heating (adjusted OR = 1.7, 95% CI: 1.1, 2.4) and cooking (adjusted OR=1.5, 95% CI: 1.1, 2.1). Moreover, the NTD risk increased with increasing exposure estimates, showing a dose-response trend (P <0.001) *(33)*. In another case-control study conducted in a coal-using population in Shanxi Province, placental levels of selected PAHs were used as an internal dose marker of coal combustion exposure to explore the dose-response relationship for NTD risks. The study found that the median concentration was significantly higher in case placentas than in controls. PAH concentrations above the median were associated with a 4.52-fold [95% CI: 2.10, 9.74) increased risk for any NTDs, and 5.84-fold (95% CI: 2.28,14.96) and 3.71- fold (95% CI, 1.57, 8.79) increased risks for anencephaly and spina bifida, respectively. A dose–response relationship was observed between PAH levels and the risk of NTDs, with odds ratios for the second, third, and fourth quartiles, compared with the first, of 1.77-fold (95%

CI: 0.66, 4.76), 3.83-fold (95% CI: 1.37, 10.75), and 11.67-fold (95% CI: 3.28, 41.49), respectively. Similarly, a dose–response relationship was also observed for anencephaly and spina bifida subtypes *(34)*. Because active or passive exposure to tobacco smoke and ingestion of char grilled meats also contribute to internal dose of PAHs, making an exclusive inference of the observed PAH-NTD risk being an adverse effect of coal requires an assumption that the sources other than coal combustion were negligible or remained constant across the case and control subjects, and this should be assessed in any future studies.

### 5.2.2.5  Low birth weight

A recent study in India examined the impact of maternal use of different household cooking fuels in India on low birth weight (LBW defined as smaller than 2500 g) and neonatal mortality (death within 28 days of birth) *(35)*. Using cross-sectional data from India's National Family Health Survey (NFHS-3), Epstein et al separately analyzed the prevalence of these two outcomes in households utilizing three types of high-pollution fuels for cooking (biomass, coal, and kerosene) in reference to low-pollution fuels (gas and biogas) as the "control" group. The results indicate that household use of high-pollution fuels was significantly associated with increased risk of LBW and neonatal death. Compared to households using cleaner fuels (in which the mean birth weight was 2901 g), the primary use of coal, kerosene, and biomass fuels was associated with significant decreases in mean birth weight (of −110 g for coal, −107 g for kerosene, and −78 g for biomass). Results also suggest an increased risk of neonatal death strongly associated with household use of coal (OR = 18.54; 95% CI: 6.31,54.45) *(35)*.

### 5.2.3  Acute CO Poisoning

Because coal can be burned for a relatively long time, even in simple household stoves, without the need to attend the fire, coal is often preferred over biomass for space heating. The prolonged coal burning, often under poor ventilation conditions during the sleeping time, can generate carbon monoxide (CO) concentrations high enough to cause acute CO poisoning, particularly for low-volatile coals such as anthracite, which do not produce sufficient volatile emissions to warn occupants of the rising CO levels *(5)*. Acute CO poisoning from coal use for heating, especially in urban households, has been long recognized in China and other countries. By analyzing data on CO poisoning collected from the emergency medical service system during August 1, 2005, to July 31, 2007, in Beijing, a recent study showed that over 88% (3331 patients) of CO poisoning cases had occurred during winter months when coal use for heating was most prevalent *(36)*. However, the rest (about 12%) of the cases have also occurred during non-heating seasons presumably from coal combustion for cooking. In all the cases, the poisoning was the result of extremely high concentrations of CO inside homes where the coal smoke was not efficiently vented.

**Table 3: Summary of Lung Cancer Effects Due to Coal Use, derived from three published systematic reviews (see also Review 4 for further details on methods and quality of these reviews)**

| Type of review | Author | Design of individual studies | Fuel | OR | 95% CI | Subgroups | Number of studies | Ref. |
|---|---|---|---|---|---|---|---|---|
| Systematic review and meta-analysis | Hosgood *et al.* | case-control | coal | 2.15 | 1.61-2.89 | overall | 25 | *(10)* |
| | | | | 2.50 | 1.56-4.00 | women | 8 | |
| | | | | 2.74 | 1.44-5.27 | men | 3 | |
| Systematic review and meta-analysis | Kurmi *et al.* | case-control | Coal | 2.19 | 1.74-2.76 | overall | 12 | *(11)* |
| | | | | 1.70 | 1.40-2.06 | women | 10 | |
| | | | | 1.54 | 1.25-1.88 | men | 3 | |
| Systematic review and meta-analysis | Smith *et al.* | case-control | Coal | 2.55 | 1.58-4.19 | overall | 5 | *(24)* |
| | | | | 1.94 | 1.09-3.49 | women | 9 | |
| | | | | 1.51 | 0.97-2.46 | men | 3 | |

## Table 4: Summary of non-cancer Health Effects Due to Coal Use

| Health outcome | Author and Year | Location | Study design | Population | | Exposure | Outcome | | Ref. |
|---|---|---|---|---|---|---|---|---|---|
| **Respiratory Effects** | Zhang J. & Smith K., 2007 | Wushan, China | Cross-sectional | N = 2,075 | Children w/colds | Coal burning for cooking/heating | Increased the risk of wheezing | OR (95% CI): 1.57 (1.07, 2.29) | *(5)* |
| | | | | N = 2,075 | Children w/out colds | Coal burning for cooking/heating | Increased the risk of wheezing | OR (95% CI): 1.44 (1.05, 1.97) | |
| | | Shunyi, China | Case-control | N = 394 | Children | Coal stove heating | Increased risk for asthma | OR (95% CI): 1.5 (1.1, 1.9) | |
| | | | | N = 129 | Children | Coal for cooking with no ventilation | Increased risk for asthma | OR (95% CI): 2.3 (1.5, 3.5) | |
| | Shen M. et al., 2009 | Xuanwei, China | Retrospective | N = 2,980 | Men & Women (Farmers) | Smoky coal | Pneumonia mortality positively associated. Stove improvement led to 50% reduction in pneumonia death. | OR (95% CI): .521 (.340, .521) | *(25)* |
| | | | | N =12,605 | | Smokeless coal | | OR (95% CI): .449 (.215, 0.937) | |
| **Lung Development** | Roy, A. et al, 2012 | (Chongqing, Guangzhou, Lanzhou, Wuhan) China | Longitudinal | N = 791 | Children (ages 6-13) | Coal for cooking and heating with no household ventilation devices. | FVC growth was 39% lower for children from homes that used coal compared to homes that used gas/LPG as a fuel | OR (95% CI): 20.5ml/year (12.7, 28.3) | *(26)* |
| | | | | | | | $FEV_1$ growth was 33% lower for children from homes that used coal compared to homes that used gas/LPG as a fuel | OR (95% CI): 16.5ml/year (9.3, 23.6) | |
| | Jin Y. et al, 1995 | China | Case-control | | | Household coal use (smoky, smokeless, wood) | Peak Expiratory Flow Rate | Strongest risk factor for lowered PEFR was smoky coal, smokeless and then wood. Coal stoves chimneys resulted in higher lung function compared to open fire pit | *(27)* |
| **Early Childhood Height and Skeletal Growth** | Ghosh, R., et al., 2011 | Teplice & Prachatice, Czech Republic | Longitudinal | N = 576 | Boys | Indoor coal combustion for heating. | Reduction in height (cm) was significantly associated at age 36 months for age/sex. | OR (95% CI): 1.34 (.51, 2.16) | *(30)* |
| | | | | N = 557 | Girls | | | OR (95% CI): 1.30 (.50, 2.10) | |
| | Calderon, L., 2012 | Mexico City | Prospective | N = 35 | Children (age 6) | Air pollution | Children found to have significantly higher concentrations of IL-6, reductions in total blood neutrophils, increase in monocytes, and insufficient Vitamin D intake. | These systemic changes, vitamin D insufficiency, air pollution may have long-term detrimental bone outcomes, increasing risk of low bone mass and osteoporosis. | *(29)* |

| Health outcome | Author and Year | Location | Study design | Population | | Exposure | Outcome | | Ref. |
|---|---|---|---|---|---|---|---|---|---|
| **Neuro-behavioral Development** | Perera F., 2008 | Tongliang, Chongqing, China | Prospective | N = 107 | Children born to Chinese non-smoking women | Prenatal exposure to PAHs, mercury, lead as released by combustion of coal | Significant associations previously seen between elevated adducts and decreased motor area (p = 0.043) and average DQ (p = 0.047) were not observed in the 2005 cohort (p = 0.546 and p = 0.146). | The findings indicate that neurobehavioral development benefited by elimination of PAH exposure from the coal-burning plant, consistent with the significant reduction in PAH–DNA adducts in cord blood of children in the 2005 cohort. | *(31)* |
| | Tang D., 2008 | Tongliang, Chongqing, China | Prospective | N = 110 | Children born to Chinese non-smoking women | Prenatal exposure to PAHs, mercury, lead as released by combustion of coal | Decrements in one or more DQs were significantly associated with cord blood levels of PAH-DNA adducts and lead, but not mercury. Decreases found in: | motor area (-16.01: -31.30, -0.72) language area (-16.63: -33.73, 0.46); high cord blood lead level significantly associated with decreased social area DQ (-6.08: -10.53, -1.63) and average DQ (-4.24: -8.20, -0.29). | *(32)* |
| **Neural Tube Birth Defects** | Li., et al. 2011 | Shanxi, China | Case-control | N = 1099 | Infants (liveborn, stillborn), electively aborted fetuses of mothers | Coal smoke exposure (compared to none) | 60% increased risk of child w/ NTD. The risk increased with increasing exposure estimates, showing a dose response. | OR (95% CI): 1.6 (1.1, 2.1) | *(33)* |
| | | | | N = 1052 | | Coal smoke exposure w/ cooking | | OR (95% CI): 1.5 (1.1,2.1) | |
| | | | | N = 198 | | Coal smoke exposure w/ residential heating | | OR (95% CI): 1.7 (1.1.2.4) | |
| | Ren A., 2011 | Shanxi, China | Case-control | N = 130 | Women (placentas) | Persistent organic pollutants | Increased risk for spina bifida, dose response for subtypes | OR (95% CI): 3.71 (.1.57, 8.79) | *(34)* |
| | | | | | | | Increase risk for anencephaly, dose response for subtypes | OR (95% CI): 5.84 (2.28, 14.96) | |
| | | | | | | | PAH concentration associated with higher risk of NTDs, also a dose response relationship | OR (95% CI): 4.52 (2.10, 9.74) | |
| **Low Birth Weight** | Epstein M.B. et al., 2013 | India | Cross-sectional | Women and children | | High-pollution cooking fuel (coal, kerosene, biomass) | Neonatal-death | Coal showed a strong association to mortality: OR (95% CI): 18.54 (6.31, 54.45) | *(35)* |
| | | | | | | | Low Birth Weight | Significant decreases in mean birth weight (-110g coal, -107g kerosene, -78g biomass). | |
| **Acute CO Poisoning** | Du et al, 2010 | Beijing, China | Case-control | N = 3,331 | All (industrial-, fire- and work-related cases) | Household coal | Incidence of acute domicile-related CO poisoning | 88% of Co poisoning during winter months (when coal is used for heating). | *(36)* |

## 5.3. Health effects from intrinsic toxicants

A summary of health effects resultant from arsenic- and fluorine-rich coal are provided in Table 5.

### 5.4.1 Introduction

Perhaps the most distinct health problems associated with household coal combustion (as opposed to the general incomplete combustion of hydrocarbons in biomass and fossil fuels) arise when the coal contains high levels of toxic elements (e.g., As, F, Se, Hg, and Pb). The relative abundance of these contaminants in coal varies substantially from place to place (see Table 2). Hence the severity of the health problems related to these toxic contaminants varies as well. Although contaminated coals may have been used globally (see Figure 1), most of the reported health impacts stem from certain areas of China. Therefore our discussion of this issue is based on studies that have been conducted in China.

In China, there are approximately 100 counties (of nearly 3000 total) that have been deemed "endemic" in Chinese literature because local coal deposits have high contents of toxic elements (5). Such coals have been called "poisonous" coals, most noticeably due to their high content of arsenic (As) and that of fluorine (F) and resulting arsenic poisoning and fluorosis endemics (5, 7). Since the publication of three review articles in which urgent research and intervention efforts are recommended towards understanding and reducing devastating health effects of poisonous coal (5-7), several new studies have been published on this topic. Below we summarize the findings of these new studies along with the main findings of the review articles.

### 5.4.2 Arsenic-rich coal

Chronic As poisoning, or arseniasis, in some regions in China has been reported for more than three decades. This problem is most prevalent in the southwest prefecture of Guizhou province where some 33,000 people, among some 200,000 people in the whole of China, are estimated to be affected (6, 37, 38). Because the coal is burned indoors in open pits for cooking and crop drying, affected people are exposed to arsenic not only through inhalation of coal smoke but also through As-contaminated food. High As levels have been found in coal-smoke-dried corns (5-20 ppm) and chili peppers (100-800 ppm), which makes food ingestion exposure the most important route of As exposure (50-80% of total exposure, compared to inhalation exposure (10-20%) and drinking water exposure (1-5%) (6). Reported illness includes symptoms of arseniasis (39) and damage to internal organs (e.g., lung dysfunction, neuropathy, hepatomegaly, and nephrotoxicity). In several endemic arseniasis areas (8 provinces in China) surveyed in a recent study, the average rate of occurrence of arseniasis at advanced stages (moderate, severe and skin cancers) was 1.2% of the 135,492 examined, mainly attributed to As exposure from drinking high As well water. This rate, however, increased to 2.7% when indoor burning of As-rich coal was also present (40).

Although cirrhosis, ascites, skin cancer, and liver cancer are the most serious outcomes of arsenic poisoning (37, 38), substantially more commonly reported are skin lesions including keratosis of the hands and feet, pigmentation on the trunk, and skin ulceration (38). For example, more than 2,400 cases of arsenic-related skin lesions (as of 2002) have been reported in several villages of the arsenic-coal endemic area of Guizhou Province (41). Furthermore, arsenic exposure can increase an individual's risk for dying from cancer. For example, in a study directed to ascertain the mortality of a group of arseniasis patients in an endemic rural township in southwestern China, a significantly increased mortality due to lung cancer or non-melanotic skin cancer was found in those patients who had been exposed for

decades to indoor combustion of high arsenic coal. However, no significant elevation of mortality due to liver cancer or bladder cancer was observed. The study also found that male arseniasis patients diagnosed with severe skin lesions had higher risks of malignancies and of non-melanotic skin cancer in particular in the following years *(42)*.

More recent studies conducted in the endemic arseniasis areas have used body burden levels of As to ascertain the exposure, employed biomarkers of pathophysiologic pathways to mechanistically examined the health effects, and analyzed genetic polymorphisms to assess individuals' susceptibility to arsenic exposure from household coal burning. For example, urinary As levels and malondialdehyde (MDA, a biomarker of oxidative stress) were significantly higher in villagers who had lived in an arseniasis endemic area than those who had lived in neighboring non-endemic villages. Furthermore, MDA levels were correlated with As exposure levels but not with age or smoking habit. The findings suggest that one of the pathophysiologic pathways of As health effects was increased body burden of oxidative stress. Directly or indirectly related to the oxidative stress pathway, other biomarkers of molecular-level damage have also been associated with the household use of As contaminated coals in another study conducted in Guizhou province *(43)*. Results from this study also showed an association between biomarkers of As exposure (urine and hair As levels) and indices for damage to chromosomes and DNA, gene mutations, gene deletions, and alterations of DNA synthesis and repair ability *(43)*. Moreover, studies of volunteers living in endemic areas have also identified several functional genetic polymorphisms that can predispose an individual to increased risks of arseniasis *(41, 43, 44)*. These studies have provided more mechanistic evidence on adverse effects of household combustion of coal intrinsically containing arsenic.

### 5.4.3 Fluorine-rich coal

During the combustion of coal containing fluorine (F), the element is emitted mainly in the form of HF (85-90%) and also in the form of $SiF_4$, and $CF_4$. Based on toxicity tests, HF is a highly toxic compound (10–100 times more toxic than $SO_2$). However, studies of F-contaminated coals have not attempted to identify which form of fluorine was mainly responsible for the endemic fluorine-rich coal poisoning, nor the extent to which the fluorine is derived from the clay used as a binder for making coal briquettes in some areas. The concerns have mainly arisen from high prevalence and incidence of dental fluorosis in many places of China where household coal has a high F content. Household combustion of fluorine-rich coal affects far more people and more regions than burning arsenic-rich coal in China. It is estimated that more than 30 million people living in 14 provinces, mainly northwestern Guizhou, southern and southeastern Sichuan and northeastern Yunnan, southwestern and northwestern Hubei, have suffered from various forms of fluorosis *(6)*.

According to a study in which three areas of Shaanxi province were compared, prevalence rates for primary dental fluorosis, permanent dental fluorosis, and severe fluorosis were all substantially (5 to 30 times) higher in the high-F coal region and the high-F drinking water region than the 'normal' low-F coal and low-F water region *(45)*. Strikingly, in the high-F coal region (average coal F content =713 mg/kg, and average room air F concentration =57 µg/m$^3$), nearly every child (97%) had dental fluorosis in permanent teeth and 47% had severe dental fluorosis. In contrast, none of the children were diagnosed with severe dental fluorosis and 20% had dental fluorosis in permanent teeth in the 'normal' region (average coal F content = 36 mg/kg and average room air F concentration = 3.8 µg/m$^3$) *(45)*. We did not identify any systematically conducted studies across all the dental fluorosis endemic areas in China, however, which would allow an assessment of the 'dose-response' relationship between indoor air F concentrations and dental fluorosis prevalence on a geographical basis.

In the dental fluorosis areas, a higher prevalence of osteosclerosis in skeletal fluorosis patients was also reported, compared to non-fluorosis areas in China and Japan, more than a decade ago *(46)*. More recently, a cross-sectional study was carried out in 1,616 students between 7 and 16 years old to assess the prevalence and pathogenic stage of skeletal fluorosis among children and adolescents residing in a severe coal-burning fluorosis endemic area of Guizhou province *(47)*. In this study, any student who lived in a household that burned coal, used an open-burning stove, or baked foodstuffs over a coal stove was deemed high-risk participants. About 23% (370) of all surveyed students were identified at high-risk and further examined by X-ray. Subsequently, one-third of the 370 high-risk participants were diagnosed with skeletal fluorosis. Overall prevalence of child skeletal fluorosis attributable to household coal use was 7.5%. Four types of skeletal fluorosis were identified: constrictive (60.7%), raritas (15.6%), mixed (16.4%), and soft (7.4%). Although most diagnosed cases (91%) were mild or moderate in severity, even a few severe cases can have devastating effects. In addition, about 97% of the 370 high-risk children were also diagnosed with dental fluorosis. Moreover, dental fluorosis was highly correlated with skeletal fluorosis in this study. Childhood skeletal fluorosis is a risk factor for adulthood bone health (e.g. bone deformation). In addition, skeletal fluorosis among children may contribute to poor health and reduced productivity when they reach adulthood *(47)*.

A study across the region of China where dental fluorosis is prevalent showed that there appeared to be a significant relationship between the concentration of fluorine in local coal and the prevalence of dental fluorosis (R=0.94, P<0.01, see Figure 4), the recommended threshold of fluoride content in coal was recommended to be 250 mg/kg *(48)*.

**Figure 4: Prevalence of dental fluorosis and concentration of fluorine in coal *(48)***



$$y = 0.0751x - 14.281$$
$$R^2 = 0.8897$$
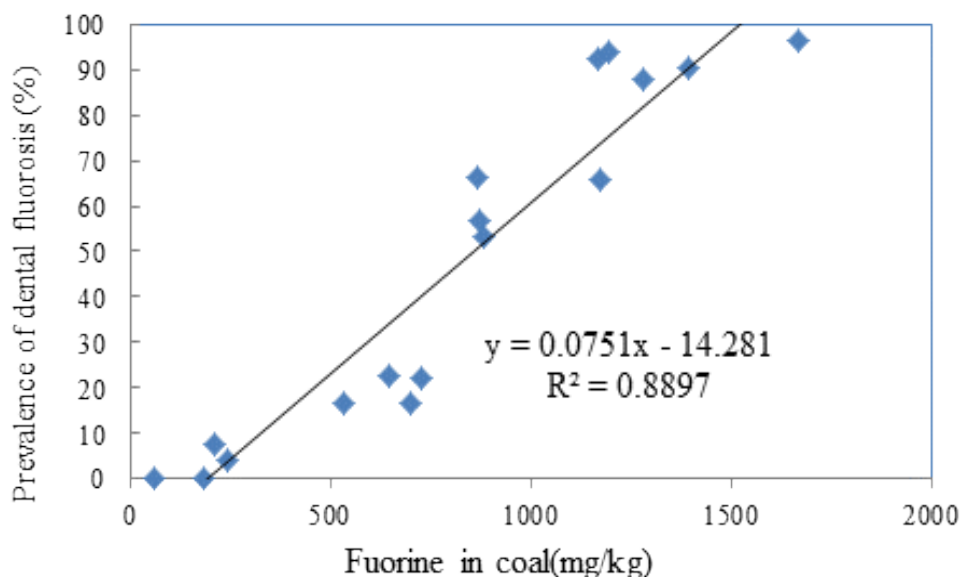
Reproduced with permission

### 5.4.4 Selenium, lead, and mercury from household coal

In addition to F and As, several other elements (e.g., selenium, lead, and mercury) released from coal combustion have also raised health concerns. However, few studies have specifically assessed the direct health effects of these elements in household use, although there is a growing literature on widespread environmental contamination by coal combustion generally, particularly for mercury.

**Selenium**. Although selenium (Se) metal has low toxicity, selenide, selenite, and other Se compounds such as $SeF_2$ have high toxicity. During coal combustion, Se has similar characteristics to those of As and F, in that it is easily volatilized and can be inhaled or ingested, leading to Se poisoning (selenosis). Room air concentrations of Se[1] have been reported as high as 1.2 mg/m$^3$ during coal combustion in households of selenosis cases. The first selenosis cases in China were diagnosed at a village in Hubei province in 1963. Nineteen of 23 residents in the village developed symptoms including hair and nail loss, sometimes in association with disorders of the neural system. Similarly, in another five villages in the area, about 49% of the residents suffered from selenosis. Altogether about 477 regional selenosis cases have been reported from 1923 to 1987 in Hubei province alone. Selenosis cases have also been reported in other parts of China such as villages in Shanxi province *(6)*.

**Lead**. Coal combustion has been widely recognized as a source of lead (Pb) measured in urban atmospheres worldwide *(49, 50)*. For example, after phasing out of leaded gasoline, a study found that blood lead level of children strongly correlated with the coal consumption but not with gasoline consumption in China *(51)*. Another study, carried out in 2010 concluded that coal combustion emissions dominated anthropogenic Pb sources during the past century contributing from 52% to 69% of total Pb released into the atmosphere in the greater Shanghai region *(51, 52)*.

In a study conducted in Poland, blood lead concentrations were 9.39 ±4.70 μg/dL in 24 children whose households had used coal stoves, 37% higher than the concentration (6.83 ± 3.95 μg/dL) measured in 46 children whose households had not used coal stoves. . These blood Pb levels (even in the non-coal use children) were higher than 5 μg/dL, above which the US CDC considers exposure to be unsafe. However, studies have shown that lead can impair child development even at blood lead level below 5 μg/dL *(53) (54)*. Exposure of adults to lead may result in anemia, nervous system dysfunction, weakness, hypertension, kidney problems, decreased fertility and increased level of miscarriages, and (where women are exposed) low birth weight and premature deliveries *(55)*.

Although no specific studies assessing health effects of lead released from household coal combustion have been conducted, a link has been established between adverse biological outcomes and lead as a constituent of the pollution mixture attributable to coal combustion. As discussed earlier, lead levels in cord blood samples were significantly associated with neurobehavioral development metrics in the Tongliang cohort *(32)* In another study conducted in Silesia, Poland, where emissions from coal-burning stoves were important contributors to the total exposure to lead (and PAHs), the mean blood lead level in 74 children was 7.69 μg/dL, reflecting their relatively high exposure. In these children, cytogenetic biomarkers were significantly associated with blood lead concentrations, suggesting coal smoke (Pb) induced chromosomal abnormalities that are risk factors for cancer and other diseases *(56)*.

**Mercury.** Coal combustion contributes to environmental mercury (Hg). Based on an analysis of the 1999 data *(57)*, coal combustion is the second largest source of Hg emissions, following non-ferrous metal smelting, in China. Given the rapid increase in coal-fired power plants in China since 1999, coal combustion is most likely to be the leading source of Hg now *(58, 59)*. Atmospheric deposition of Hg into the aquatic system enables Hg to enter,

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[1] According to the most recently published occupational exposure limits for "selenium and its inorganic compounds (as SE)" by the Commissionfor the Investigation of Health Hazards of Chemical Compounds in the Work Area (MAK Commission) in Germany, limits should not exceed 0.02 mg/m$^3$ (based on a typical 8 hour day, 40 hours week) (see http://onlinelibrary.wiley.com/book/10.1002/9783527675128). No population based air-quality guideline value for Selenium was available.

convert into methyl-Hg (the most toxic form), and bio-accumulate in the aquatic food chain. Hg is responsible for multiple toxic effects on human health. A case study showed the male residents of Hong Kong who consumed more than four or more meals of fish per week tended to contain higher Hg in their hair, which was linked to their sub-fertility *(57)*. There has also been increasing evidence showing that skin disorders and autism in Hong Kong children were related to their high Hg body loadings (hair, blood and urine), through prenatal methyl-Hg exposure *(57)*. With close proximity to the coal emissions, members of coal use households may be exposed to elemental or inorganic Hg through inhalation and ingestion of coal-smoke dried foodstuffs, but this is unlikely to have been methylated. To the best of our knowledge, however, no studies have attempted to specifically link Hg from household coal use to health outcomes.

**Table 5: Summary of Health Effects Related to Poisonous Coals containing (a) arsenic and (b) fluorine**

**(a) Arsenic (As)**

| No | First author and year of publication | location | Study design | Population | | Exposure | Outcome | | Ref |
|---|---|---|---|---|---|---|---|---|---|
| 1 | Dong An, 2009 | Guizhou,China | Intervention | N=47000 | | Reduce exposure by improve stove with chimney and behavior change of not eating coal dried food, shut up coal mine pit. without measurement of indoor air levels | | Urinary As concentrations in the region decreased from 0.198 ± 0.300 mg/L (n = 144) to 0.049 ± 0.009 mg/L (n = 50) after intervention | (60) |
| 2 | Baoshan Zheng, 2005 | Guizhou,China | cross-sectional | N=2634 | In families with arseniasis patient | As in coal is 523.8±601.5 mg/kg, in corn is 4.13±2.76 mg/kg , chili is 2.32±1.26 mg/kg and 0.46±0.30mg/m3 in kitchen on cooking in indoor air. | The prevalence rate of arseniasis is up to 98.6% in the village with highest As (3360mg/k) content in coal. There is a higher prevalence with increasing As concentration in coal. | The urinary As was up to 1.34±0.67mg/L in the village with the highest As in coal. There was an increasing urinary As with increasing As concentration in coal. | (61) |
| | | | | | In comparison group | As in coal is 10.8±10.0mg/kg. | | | |
| 3 | Amjad Shraim,2003 | Guizhou,China | cross-sectional | | exposed group | As in coal was 56.39±42.5mg/kg | The prevalence rate of arseniasis is 30% in exposure group. | In exposed group the urinary As was 71.49±37.1ug/g creatinine. | (39) |
| | | | | | control group | As in coal was 10.8±10.0mg/kg. | Not mention prevalence in control group. | In control group, the level was 0.005-0.009mg/L. | |
| 4 | Guanglu Bai, 2006 | Shaanxi, China | cross-sectional | 8 villages | | As in coal is 92.61 ± 90.70 mg/kg, 0.80 ±2.43mg/kg in corn, and 4.73 ±15.69mg/kg in chili. | The prevalence rate of arseniasis is 19.26%, 43.51% skin lesions, and 6.79% neuro damage in exposure group. | | (62) |

## (b) Fluorine (F)

| No | First author and year of publication | Location | Study design | Population | | Exposure | Outcome | Ref |
|---|---|---|---|---|---|---|---|---|
| 1 | Mitsuru Ando, 2001 | Sichuan, China; Jiangxi, China and Nagaiio Prefecture, Japan | cross-sectional | | Fluorosis area | F is 656.4±133.4mg/kg in coal, 367.5±428.5mg/kg in chilli; | The prevalence rate of dental fluorosisis 42.10% in exposure group. | (46) |
| | | | | | Non-fluorosis area | F is 152.01m g/kg in coal,1.68 mg/kg in chilli. | The prevalence rate of dental fluorosisis 0% in exposure group. | |
| 2 | Kun-li Luoa, 2011 | Yunnan, Guizhou Sichuan province, China | cross-sectional | 405 children | | F in coal is 98±60 mg/kg in coal, 1116±635mg/kg in clay and 313±127mg/kg in coal mix. | The prevalence rate of dental fluorosisis 100% in exposure group (< 6 years old) | (63) |
| 3 | Xianghui Qin, 2009 | Guizhou, China | cross-sectional | 1,616 students aged between 7 and 16 years | high-risk group for skeletal fluorosis (n=370) | Students lived in a household that burned coal, used an open-burning stove, or baked foodstuffs over a coal stove | Overall prevalence of child skeletal fluorosis due to indoor burning of coal was 7.5%. The prevalence rate of dental fluorosisis is 97%. | (47) |
| 4 | Jie Zhang, 2009 | Chongqing, China | cross-sectional | 10388 | Fluorosis area | F is 12.77±8.08μg/m$^3$ in indoor air | The prevalence of dental fluorosis is 82.77% | (64) |
| | | | | | Non-fluorosis area | F 1.16±1.08μg/m$^3$ in indoor air | | |
| 5 | Ping He, 2009 | Guizhou,China | cross-sectional | | High fluorosis area | F emission from coal consumption is 1.3 times of low fluorosis area | The prevalence of dental fluorosis is 98% and 73% | (65) |
| | | | | | Low fluorosis area | | The prevalence of dental fluorosis is 25.5% | |

# 6. Interventions

A summary of the intervention methods and results including coal bans, technological advancements and behaviour change is provided in Table 6.

## 6.1 Trends in Urban Household Coal Use Bans

Historically, household coal combustion in cities has contributed to outdoor air pollution, including leading to severe pollution episodes especially during cold calm weather. Such conditions persist today in some areas, for example Ulaanbaatar, Mongolia *(66, 67)*. Health concerns stemming from these episodes have prompted laws and regulations banning the use or sales of coal in cities worldwide. The Clean Air Act 1956 was an Act of the Parliament of the United Kingdom passed in response to London's Great Smog of 1952. The Act introduced a number of measures to reduce air pollution, especially by introducing 'smoke control areas' in some towns and cities in which only so-called 'smokeless' fuels could be burnt. By shifting homes' sources of heat towards cleaner coals, electricity, and gas, it reduced the amount of 'smoke' (particulate matter) pollution and $SO_2$ from household fires.

The sale of bituminous (smoky) coal was banned in parts of Ireland in the early 1990's in an effort to address this issue. 'Winter smog' was a growing problem in urban areas at the time, due to the widespread use of bituminous coal, and this, in turn, had given rise to serious health effects in the population. The ban on smoky coal has greatly reduced levels of particulate matter ($PM_{10}$) in major towns and cities *(1)*. Black smoke decreased significantly in Dublin after the ban in 1990, in Cork in 1995 and in Limerick 1998. A 70% reduction in the average smoke levels in winter in Dublin was achieved since October 1990. Following the Dublin ban, adjusted non-trauma death rates decreased by 5.7% (95% CI 4%–7%, p<0.0001), respiratory deaths by 15.5% (12%–19%,p<0.0001), and cardiovascular deaths by 10.3% (8%–13%, p<0.0001). Respiratory and cardiovascular standardized death rates fell coincident with the ban on coal sales. About 116 fewer respiratory deaths and 243 fewer cardiovascular deaths were seen per year in Dublin after the ban. Reductions in respiratory and cardiovascular death rates in Dublin suggest that control of particulate air pollution could substantially diminish daily death. The benefit of the reduced death rate was greater than predicted from results of previous time-series studies *(1)*, possibly adding to the evidence that PM from coal combustion is more toxic than PM from other sources *(68)*. The burning of bituminous coal by households in open fires is now banned in all cities of Ireland.

Even during periods without well-defined severe air pollution "episodes", household coal use can contribute significantly to urban air pollution especially during heating seasons. For example, a case study carried out over a 3-week period in 2005 in a typical major city situated in a European coal combustion region (Krakow, Poland), the major culprit for the extreme pollution levels (up to 8 times the European Air Quality Standard for $PM_{10}$) was demonstrated to be residential heating by coal combustion in small stoves and boilers (>50% for $PM_{10}$ and >90% B(a)P), whereas road transport (<10% for $PM_{10}$ and <3% for B(a)P), and industry (4-15% for $PM_{10}$ and <6% for B(a)P) played lesser roles. In addition, indoor $PM_{10}$ and B(a)P concentrations were at high levels similar to those of outdoor concentrations and were found to have the same sources as outdoors *(69)*.

Realizing the similar significant contribution of household coal combustion to urban air pollution, China started to ban the use or sales and uses of coal in large cities with the revision of the Law of the People's Republic of China on the Prevention and Control of Atmospheric Pollution in 2000. According to the basic law, the state government designated 113 "key cities" (large and medium sized cities, including 31 capital cities of each province) in China. In each of these cities, the local governments are required to define "restricted areas" where the sale and use of high-pollution fuels are prohibited and smoky coal should be replaced with natural gas, LPG, electricity or other clean energy.

In the non-restricted urban areas of large or medium-sized cities, only sulfur-fixed coal briquettes and other cleaner fuels can be used as household cooking fuels; and the direct use of raw coal will be gradually eliminated within a prescribed time limit. In coal-heating areas, district-wide central heating systems shall be developed. In areas covered by central heating pipelines or networks, no new coal heating boilers may be installed. The law also prohibits mining coal with toxic or harmful substances, such as arsenic, that exceed the prescribed limits. By 2013, nearly all of the 113 cities had implemented coal-ban policies in the restricted areas. However, to date, there are no available data to assess the compliance or impact of these city-specific regulations/laws.

## 6.2 Interventions for Rural Households

Ironically, while urban household coal use is being banned or discouraged by governments in China, biomass fuels have been increasingly substituted with coal in rural areas, principally driven by increased incomes that make coal, as a commercial fuel (as opposed to self-collected biomass fuels), more affordable to rural residents *(70)*. Considering general health risks associated with solid fuel use and additional health problems associated with coal emissions, reducing exposures from household coal combustion, especially substituting cleaner fuels for the poisonous coals, is recommended as a high public health priority *(5)*.
While a switch to clean fuels is likely to be the most effective alternative, a number of solid-fuel interventions have been evaluated for efficacy (in terms of indoor air quality improvement), including improved coals or coal-derived fuels, improved stove technology, or improved emission ventilation such as adding a chimney. These are discussed below.

### 6.2.1 Fixing agents for toxic contaminants

Attempts have been made to mix agents into raw coal contaminated by fluorine (F) and arsenic (As) during briquette making. These fixing agents change the chemical form of F or As in coal so that it cannot readily vaporize during combustion, diverting the majority of F or As into the coal ash. Field measurements conducted in a fluorosis endemic area in Yunnan province have shown that burning the briquettes reduced F and As concentrations in coal-smoke dried corns by 65% and 75%, respectively, compared to burning the raw coals *(71)*. Because the fixing agents were readily available local calcium-based materials, the cost for making the coal briquettes is minimal when considering the marked reduction in F and As emissions, but there is little literature describing either cost or effectiveness in terms of reducing human exposures and health impacts.

### 6.2.2 Sulphur-retaining materials

Another coal-derived solid fuel is the so-called coal-biomass briquette that contains sulphur-retaining materials. When this fuel was used in households in Chongqing, China, the average 8-hr and 24-hr $SO_2$ concentrations indoors were nearly equal to or less than the World Health Organization's 40 ppb guideline. In contrast, $SO_2$ concentrations measured in households using traditional coal briquettes exceeded the guideline, despite the same sulphur content for the coal-biomass briquettes and for the traditional coal briquettes. However, PM and other toxic elements emissions of the new solid fuel were not assessed *(72)*.

### 6.2.3 Honeycomb Coal Briquettes

Burning coals in the form of briquette (especially honeycomb briquette) usually emit less PM and $SO_2$ than in the unprocessed form *(4)*. Hence the use of coal briquettes, along with improved stoves, has been encouraged by the Chinese government by providing a subsidy for the briquette users, as a means of reducing air pollution and health impacts. Experimental measurements have shown that the combination of the fuel improvement and the stove improvement can lead to emission reductions in PM by 63±12% and black carbon by 98 ±1.7%. The more substantial reduction in black carbon (than PM) also has policy

implications for climate change mitigation *(73)*. Interestingly, potential climate benefits from switching 'dirtier' coals to 'cleaner' coals seem to be receiving increasing attention. For example, another study concluded that if low-quality medium-volatile bituminous coals are prohibited as household coal, coupled with further promotion of briquettes, black carbon emissions from household coal use in China could be reduced by approximately 80% *(74)*.

### 6.2.4  Chimney Stoves

The highest indoor concentrations of PM, $SO_2$, and other pollutants have been measured in households using unvented stoves (fire pits) *(5)*. Adding a chimney to coal stoves can substantially reduce indoor pollutant concentrations. A few measurements made in households of Xuanwei County, China, showed that the use of chimneys led to significant decreases in indoor air concentrations of $PM_{10}$ by 66% and of benzo[a]pyrene (BaP) by 84%, but no personal measurements were made. The reduction of indoor pollution levels by the installation of a chimney supports the epidemiology findings on the health benefits of this simple intervention, such as significantly reduced lung cancer and COPD risks *(75)*. However, even in the presence of a chimney, both $PM_{10}$ and BaP concentrations still exceeded the indoor air quality standards of China *(75)*. Furthermore, the principal function of a chimney is to divert a significant fraction of coal smoke from indoors to outdoors, facilitating so-called neighborhood pollution. In a densely populated village or neighborhood especially under an atmospheric inversion condition, ambient outdoor concentrations of pollutants can be elevated, mimicking a urban smog episode at a smaller spatial scale *(5)*.

### 6.2.5  Integrated Behavior and Technology Interventions

Experiences from many stove/fuel intervention programs worldwide indicate that the efficacy and sustainability of an intervention largely depends on participants' cultural and social acceptance of improved stoves, normally requiring some social behavioral changes (see Review 7). Studies conducted in four Chinese provinces, for example, indicate that technology improvement in stove/fuel, with insufficient health risk education, led to non-significant reductions in HAP concentrations *(76, 77)*. These studies called for the combined technology and behavioral interventions in order to achieve reductions in HAP concentrations and health risks. The effectiveness of such a coordinated approach has been demonstrated in a case study below.

In three counties of Guizhou province where arsenicosis endemics had been reported, a coordinated mitigation effort was made to reduce arsenic exposure resulting from the consumption of As-contaminated chili peppers and corn dried over unvented stoves. Before the mitigation, a baseline survey of 45,364 residents in 2004 identified more than 2,800 individuals with arsenicosis. The survey found that many residents had little knowledge about the link between coal use and arsenicosis, explaining why > 95% of the residents had used high-As coal. This survey provided the basis for a health education campaign that promoted lifestyle changes coupled with the shutdown of local coal mines and the installation of approximately 10,000 new stoves with chimneys. The cost of the mitigation was financed mostly by the government. A post-mitigation response survey in 2005 found that > 85% of the residents were well aware of the cause of arsenicosis; > 90% knew how to correctly operate the new stoves; and > 90% dried corns and chili peppers outdoors in the sun. The survey found that the average urinary As concentration in the individuals with arsenicosis decreased by 75% ($0.198 \pm 0.300$ mg/L ($n = 144$) in 2004 to $0.049 \pm 0.009$ mg/L ($n = 50$) in 2005) from pre-mitigation to post-mitigation. In the survey, however, no new assessment of health outcomes was conducted to examine whether the reduction in body burden would be associated with an improvement in arsenicosis related health problems. Such an assessment was difficult to do given that cumulative exposure from past decades might continue to result in new cases of arsenicosis emerging in the decades to come *(78)*.

## Table 6: Summary of Interventions

| Type | Ways of Intervention | | Place | Effects Evaluation/Outcome | | Ref |
|---|---|---|---|---|---|---|
| **Policy** | Ban on coal sale | | Dublin, Ireland | Black smoke concentrations | 70% reduction | *(1)* |
| | | | | Adjusted non-trauma death rates | 5.7% reduction | |
| | | | | Respiratory deaths | 15.5% reduction | |
| | | | | Cardiovascular deaths | 10.3% reduction | |
| | Coal vs. other sources | | USA | PM | PM from coal was found to be more toxic than from other sources | *(68)* |
| | | | Krakow, Poland | Household coal contribution to PM10 | >50% compared to other sources | *(69)* |
| | | | | Household coal contribution to B(a)P | >90% compared to other sources | |
| | Suggested Policy: banning of low-quality medium-volatile bituminous coals in combination with promotion of briquettes | | China | Black carbon | Potential to reduce emissions by 80% | *(74)* |
| **Technology** | Fuel | Fixing agents for toxic contaminants | China | F and As in coal-smoke dried corns | 65% and 75% reduction respectively | *(71)* |
| | | Sulfur-retaining materials | China | $SO_2$ | Found to be equal to or less than WHO 40 ppb guidelines. | *(72)* |
| | | Honeycomb Coal Briquettes | China | $SO_2$ and PM | Are emitted less compared to unprocessed form. | *(4)* |
| | | | | Black carbon | 98% reduction | *(73)* |
| | | | | PM | 63% reduction | |
| | Stove | Chimney Stoves | China | Indoor air concentrations of $PM_{10}$ | 66% reduction | *(75)* |
| | | | | Indoor air concentrations of (BaP) | 84% reduction | |
| | | | | Lung cancer and COPD risks | Reduced | |
| | | Improvement on Stove/Fuel (intervention program) | China | Hazardous Air Pollutants | Reductions (non-significant) in HAP concentrations. Outcomes would be better combined with behavior education. | *(76, 77)* |
| **Behavior** | Shutting down coal mine as well as educational program on people's behaviour change reduce arsenic exposure resulting from the consumption of As-contaminated chili peppers and corn dried over unvented stoves | | China | Urinary As concentration in the individuals with arsenicosis | 75% reduction (0.198 ± 0.300 mg/L (*n* = 144) in 2004 to 0.049± 0.009 mg/L (*n* = 50) in 2005) | *(78)* |

## 7. Discussion and assessment of overall quality of evidence

The overall quality of the scientific evidence is an important factor to consider in developing evidence-based policies. Therefore, we based this assessment on systematic reviews where possible protocols. Given that the nature of many of the available studies, notably that very few randomized trials and high quality cohort and case-control studies have been conducted, it was impractical to conduct meta-analysis or even to perform inter-study comparisons due to substantial heterogeneity in study type, design and quality.

Accordingly, the robustness of evidence for certain aspects of the health risks associated with the unique feature of household coal combustion is based primarily on quality assessment of individual studies as summarized in Annex Table A.2. These specific studies were selected for the quality assessment summary table to highlight a wide range of interventions and health effects due to various exposures from household coal use. In the absence of formal methods for combining studies and evaluating factors such as publication bias, an overall assessment of the quality of evidence drawing together the various sources is provided below.

### 7.1 Carcinogenicity

The carcinogenicity of emissions from household coal combustion was reviewed by IARC *(8)*, using the standard assessment methodology involving review of exposure data, studies of cancer in humans, studies of cancer in experimental animals, and mechanistic and other relevant data. It was concluded that for household use of coal:

- There is *sufficient evidence* in humans for the carcinogenicity of household combustion of coal. 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.

The overall evaluation concluded that indoor emissions from household combustion of coal are *carcinogenic to humans (Group 1)*. This evidence on the carcinogenicity, hence, is assessed to be of **high** quality. Although this evidence confirms that emissions from household coal combustion are carcinogenic, this does not provide risk estimates for the use of coal compared to the use of an alternative clean fuel.

### 7.2 Health risks from incomplete combustion of coal

The systematic reviews, reported in Review 4, examined evidence supporting that household air pollution from solid fuels can cause a range of disease outcomes. The reviews also provided estimates of disease risk and intervention impact. Most studies investigated risks of biomass fuel use, however, or did not differentiate types of solid fuels clearly. Separate risk estimates for coal use were available only for lung cancer and COPD, although for the latter there were too few studies to carry out grading of the evidence.

Annex Table A.1 reports the GEPHI assessment for coal use and lung cancer, which provided an intervention effect estimate of 0.46 (95% CI: 0.35, 0.62) and was assessed to be of **moderate** quality. The IER function for risk of lung cancer, although not specific to coal exposure, indicates that risk from $PM_{2.5}$ exposure remains elevated right down to the counterfactual level of 7.5 $\mu g/m^3$ of $PM_{2.5}$; this evidence was assessed as of **moderate** quality.

The current review sought additional studies on non-cancer health outcomes specifically related to coal use in the home, summarised in Table 4. Studies reporting risks for a total of seven health outcomes were included, but due to the low numbers of studies and heterogeneity of outcomes, no attempt was made to conduct meta-analysis. Accordingly, an assessment using GRADE domains as a guide for determining the quality of the body of evidence has been made (see below).

| Domain | Comment |
|---|---|
| *Number of studies* | Few studies were found for each outcome, with one (two outcomes), two (four outcomes) and three (one outcome). |
| *Study designs* | All studies were observational designs, although one (Shen 2009) is a cohort study based on more than 10 years of follow-up of a cohort with users and non-user of improved chimney stoves. |
| *Risk of bias* | Several of the studies were graded as poor to moderate quality (based on stars rating), due to selection bias and concerns about confounding. Overall, it is concluded there is some risk of bias, especially as there are few studies for each outcome. |
| *Heterogeneity* | There is heterogeneity in how outcomes are defined. Formal testing ($I^2$ statistic) was not carried out |
| *Indirectness* | The majority of these studies directly investigate the relationship between use of coal as a household fuel and the stated health outcome. |
| *Imprecision* | Some of the individual studies reported found statistically significant increased risk of the outcome, but not all. Pooled odds ratios were not calculated as explained above. |
| *Publication bias* | Not formally tested through funnel plot asymmetry, but possible. |

Based on these considerations and in particular the observational designs, the overall quality of evidence for the outcomes reported is assessed to be **low**. The systematic review and meta-analysis (SRMAs) in Review 34, which report risk estimates for a number of these outcomes with solid fuel use (mainly biomass) lend some additional strength to the findings, for example for impaired child growth, low birth weight, etc., as there is currently little reason to suspect effects of PICs from coal combustion differ markedly from solid fuels in general. For outcomes not included in Review 4, that is child lung development and neural tube defects, the evidence cited here is of **low** quality at best (albeit plausible), and should be the subject of future research.

## 7.3  Toxic contaminants in coal

The systematic review identified studies reporting on health risks of five toxic contaminants in coal, arsenic, fluorine, selenium, mercury and lead. These studies provide evidence on various aspects of health risk, including toxin content of coals, combustion and emissions chemistry, exposure routes (i.e. food, air and water), air pollution and exposure levels, and health impacts. The risks and impacts for arsenic and fluorine have been most comprehensively reported. For all of these contaminants, the majority of available evidence derives from China.

The assessment of evidence for health risks from toxic contaminants of coal is based on an overall evaluation of the available studies covering all of the aspects noted above. The data obtained on the content of toxic contaminants in coal, the fact that these are released into the air during combustion, measurements of toxins in air and food (which are the main routes of exposure), and some dose measurement (e.g. in blood) make a clear case that those using contaminated coals in the home are at risk.

Epidemiological studies that have included toxin-specific health outcomes have all been observational, and generally compared the prevalence of health outcomes caused by the contaminants (i.e. arsenicosis, dental and skeletal fluorosis, etc.) in areas or homes using contaminated coals with those in areas using fuel with lower levels of contamination. No studies directly investigating the risk of adverse health effects of mercury or lead with household coal use were reported, although indirect evidence based on exposure biomarkers was available for lead. For example, after phasing out of leaded gasoline, a study found that blood lead level of children strongly correlates with the coal consumption but not with gasoline consumption in China.

Intervention studies provide evidence that exposure can be reduced but not eliminated by processing of the coal, through use of chimney stoves, and/or behaviour interventions to encourage drying of food outside of the home. These studies have demonstrated reductions in indoor concentrations and/or urinary levels of some of these toxins, but not so far included evaluation of health impacts.

In summary, the available evidence clearly shows important toxins are widely distributed in coal, present important health risks where coal is used in the home. While toxic contaminants in coal clearly result in serious adverse health consequences in addition to the health risks associated with general HAPs from coal combustion, the evidence has been mainly derived from studies conducted in affected areas of China. Taking all of this evidence together, including the specificity of effect linking coal containing toxins, emissions of the toxin, high measured levels in air, food etc. (routes of exposure) and disease outcomes specific to the toxins, the quality of evidence is assessed to be **moderate**.


## 8. Conclusions

In the near term, more effective and widespread interventions are vitally important to mitigate the severe health problems resulting from poisonous coals and reduce exposures to extremely high HAP released from unvented coal stoves. However, household coal combustion, even using cleaner coals and 'advanced' stoves typically found in cities, still produces large emissions of fine particles, $SO_2$, carcinogenic compounds such as PAHs and PAH derivatives, and black carbon, prompting the ban of household coal use in nearly all urban areas worldwide. Partly due to longer combustion duration of coal for cooking and continuous combustion of coal for space heating, rural coal-using residents may experience higher indoor $PM_{2.5}$ exposures than urban residents even though rural outdoor air is generally cleaner than polluted urban atmosphere. Along the economic development trajectory, the rural energy structure has begun or is beginning a transition from self-collected biomass and raw coal to commercial fuels in many countries. Considering the evidence presented above on various health risks associated with household coal use, even commercial coals in the form of briquettes, for example, should be discouraged in order to reduce pollution exposures. Research on new cleaner and affordable fuel technologies for rural households should be a priority, while immediate effective mitigation efforts are being implemented as a short-term solution to this large public health problem. Although this is an enormous challenge, strong political wills for improving health and mitigating black carbon emissions, along with leapfrog technology opportunities, can facilitate this transition.

As with biomass, it is difficult to burn coal cleanly in household appliances, but the wide variety of coal characteristics make generalizations more difficult. Nevertheless, it is concluded that household use of raw coal, with or without contaminants, presents significant risks to health,

even with use of chimneys. Poisonous coals, i.e., those with high levels of toxic contaminants, should be banned entirely for household use. At this point, we are not able to make a judgment about the potential of processed "non-poisonous" coal to be used as a household fuel without risks to health, and this should be the subject of further review, with new investigations if studies addressing this question are not available.

In addition, there is a need for both an international and historical assessment of the policies that have been used to reduce household coal use and their effectiveness in different contexts. Even though recommending more systematic research on processed coal alternatives, we believe that the available evidence and the long negative experience worldwide support policies that strongly discourage household use of coal, including the complete cessation of raw coal use, to protect health.

## Acknowledgements

**Annex Table A.1: GEPHI assessment of quality of evidence on risk of lung cancer with exposure to household coal use, based on the systematic review and meta-analysis described in Review 4.**

This assessment uses the grading of evidence for public health (GEPHI) methodology (for full description and discussion of these methods, see: Methods used for evidence assessment)

| Design | No. of studies | Risk of bias | Inconsistency (heterogeneity) | Indirectness (external validity) | Imprecision (power) | Publication bias | Other considerations (specify)* | Number of events | | Relative effect and 95% CI | Quality |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Intervention | Control | | |
| Observational | 25 | No | Yes (-1) | No | No | No | Large effect (+1) D/response (but can't upgrade) | 10,142 cases | | 0.46 (0.35, 0.62) | LOW |
| Intermediate score | | | | | | | | | | Low | |
| Final score | | Evidence that risk does vary by setting, possibly due in part to type of coal | | | | | | | | | |
| | | Evidence on effects of exposures to other sources of coal combustion pollution (+1) | | | | | | | | **Moderate** | |

## Table A.2: Quality Assessment Review (Selected References)

| Study [Reference] | Study Design | Outcome Type | Health Effects Evaluation | Quality Assessment and Issues [max stars = 10] |
|---|---|---|---|---|
| **Shen M., 2009** *(25)* | Retrospective cohort | Respiratory Effects | Pneumonia mortality positively associated. Stove improvement led to 50% reduction in pneumonia death.<br><br>Smoky coal users: 0.521 (0.340, 0.521)<br>Smokeless coal users: 0.449 (0.215, 0.937) | Stars = 7/10<br>Thorough adjustments made for many confounding factors.<br>Exposure-response relationships are well reported but assessment of exposure methods/data are not validated with other studies.<br>Potential bias as some pneumonia mortality cases may also have had undiagnosed lung cancer, affecting causality |
| **Roy A. 2012** *(26)* | Longitudinal | Lung Development | $FEV_1$ growth was 33% lower for children from homes that used coal vs homes that used gas or LPG as a fuel:<br>16.5ml/year (9.3, 23.6)<br><br>FVC growth was 39% lower for children from homes that used coal vs homes that used gas or LPG as a fuel:<br>20.5ml/year (12.7, 28.3) | Stars = 6/10.<br>Good description of study population and exposure methods explanation. Comparison of ambient air pollution between all study sites is a plus. Possible selection bias due to retention issues over the study period and possible exposure misclassification (non-quantitative responses for indoor air pollution). |
| **Ghosh R., 2011** *(79)* | Longitudinal | Early Childhood Height and Skeletal Growth | Reduction in height (cm) was significantly associated at age 36 months for age/sex for children that were raised in homes that used coal, compared to children raised in homes that did not use coal.<br>Boys: 1.34 (0.51, 2.16)<br>Girls: 1.30 (0.50, 2.10 | Stars = 4/10<br>Self-reporting bias cause for concern for quantifying indoor coal smoke. Exposure assessment defined moderately.<br>Of available participants, only 0.6% used coal as primary type of fuel for cooking or heating (small for the primary exposure group for analysis).<br>Potential confounder of prenatal/postnatal smoking more than likely unreliable (big difference between pre-post natal self-reporting). |
| **Tang D., 2008** *(32)* | Prospective cohort | Neurobehavioral Development | Developmental quotients were significantly associated with cord blood levels of PAH-DNA adducts and lead resulting from both household and non-household coal combustion in the study area. Decreases found in:<br>motor area (-16.01: -31.30, -0.72);<br>language area (-16.63: -33.73, 0.46); high cord blood lead level significantly associated with decreased social area DQ (-6.08: -10.53, -1.63) and average DQ (-4.24: -8.20, -0.29). | Stars = 8/10<br>A generally well conducted study with many efforts to reduce biases (good adjustment).<br>The exposure and outcome measures are very well defined.<br>Small sample size for the study but there was good follow up and retention.<br>Biomarker of internal dose was used to measure individual-level exposure.<br>Potential concern is that maternal groups not included in the study had significant differences in education. |

| Study [Reference] | Study Design | Outcome Type | Health Effects Evaluation | Quality Assessment and Issues [max stars = 10] |
|---|---|---|---|---|
| Li Z., 2011 (33) | Case-control | Neural Tube Birth Defects | 60% increased risk of child w/ NTD. The risk increased with increasing exposure estimates, showing a dose-response.<br><br>Coal smoke exposure (compared to none): 1.6 (1.1, 2.1)<br>Coal smoke exposure w/ cooking: 1.5 (1.1,2.1)<br>Coal smoke exposure w/ residential heating: 1.7 (1.1, 2.4) | Stars = 6/10<br>Recall bias is a potential concern.<br>OR for true cooking exposure may not be accurate as ventilation during cooking was not measured.<br>Exposure is defined well but the index used was not validated in prior studies or in different settings. |
| Epstein M.B. 2013 (80) | Cross-sectional | Low Birth Weight | Significant decrease in mean birth weight (-110g coal, -107g kerosene, -78g biomass).<br><br>Coal showed a strong association to mortality: 18.54 (6.31, 54.45). | Stars = 3/10<br>Comprehensive description of study population.<br>Analysis only includes infants with birth weight on cards, excluding those not weighed immediately after birth, in rural areas, low social economic status, or with poorer nutrition (selection bias).<br>Authors cautious over generalizability of results due to bias across fuel types in population surveyed.<br>Poor adjustments of OR, with evidence that key confounding variables were associated with the outcome. |
| Clancy L., 2002 (1) | Case-control | Intervention Policy: ban on coal sale | Black smoke concentrations: 70% reduction<br>Adjusted non-trauma death rates: 5.7% reduction<br>Respiratory deaths: 15.5% reduction<br>Cardiovascular deaths: 10.3% reduction | Stars = 8/10<br>Exposure assessment is very good along with good timeline for study. However, this is not household level measurements. The concentration was made at city-level.<br>Thorough adjustments made for relevant factors.<br>Seasonal effect taken into account. |
| Yamada K., 2008 (72) | Intervention household study | Fuel technology (briquettes): sulfur-retaining materials | 8-hr $SO_2$ concentrations found to be equal to or less than WHO 40 ppb guidelines | Stars: 4/10<br>Clearly defined outcome and measurements, specific to gaseous pollutants. However, exposure group characteristics not defined clearly. Only one study group used so no comparisons among different households. |
| Baoshan, Z. 2005 (61) | Cross-sectional | Toxic Coal: Arsenic poisoning | Arseniasis: up to 98.6% in the village with highest As (3360mg/k) content in coal; Urinary As: up to 1.34±0.67mg/L in the village with the highest As in coal.<br>A rising arseniasis prevalence and increasing urinary As with increasing As concentration in coal. | Stars = 6/10<br>Sampling and analysis methods explained in very good detail.<br>This study was primarily concerned with arsenic content in coal and subsequent arsenic content in environment (food, water, air, etc), not focusing on health effects. |
| Tian L., 2009 (75) | Prospective cohort | Chimney Stove | Indoor air concentrations of PM10 - (66% reduced)<br>Indoor air concentrations of (BaP) – 84% reduced<br>Lung cancer and COPD risks - reduced | Stars = 5/10<br>Exposure assessment defined moderately.<br>Limited adjustment (study design inherently controlled for certain confounders). |

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