Chapter 14
Indoor Biomass Burning and Health Consequences

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14.1 Introduction

Inefficient cooking with biomass fuels in poorly ventilated homes is a major source of exposure to indoor air pollution in the developing world. The levels of exposure to particulate matter (PM) from biomass smoke in such homes are often at least an order of magnitude higher than the highest concentrations that occur in the ambient air of the developed world (Bruce et al. 2000; Diette et al. 2012). Household air pollution from cooking and heating with biomass fuels also is an important contributor to outdoor air pollution, accounting for an estimated 10 % of ambient fine PM ($PM_{2.5}$) (Smith et al. 2014).

Biomass fuel refers to any recently living plant- and/or animal-based material that is deliberately burned by humans as fuel, including wood, crop residues, and animal dung (Bruce et al. 2000). The number of people reliant on biomass fuels is projected to increase to 2.6 billion by 2030 (Smith et al. 2013). Most of these people live in rural areas of lesser-developed countries (LDCs), where some four-fifths of households rely on biomass fuels as their major or only source of domestic energy for cooking and sometimes space heating (World Health Organization 2009).

Cooking with biomass fuels is generally done on unvented stoves typically consisting of such simple arrangements as three rocks, a U-shaped hole in a block of clay, or a pit in the ground (Smith et al. 2013). Combustion under such conditions is inefficient and therefore incomplete products of combustion are generated. This typically leads to extremely high pollutant concentrations in the vicinity of the

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stove. Pollutant concentrations are further exacerbated by the lack of ventilation that characterizes many of the kitchens in rural areas of developing countries.

The high levels of smoke from cooking indoors with biomass fuels commonly contain up to 1,000 μg/m^3 particulate matter with a diameter of 5 μm or less (PM_5) and much higher values have been reported (Smith et al. 2013; Balakrishnan et al. 2013). These concentrations are orders of magnitude higher than either the U.S. Environmental Protection Agency national ambient air quality standard for PM10 or the World Health Organization (WHO) guideline. In addition to PM, carbon monoxide (CO), nitrogen oxides, formaldehyde, and a number of toxic organic compounds [e.g., benzene; 1,3 butadiene; benzo[α]pyrene and other polycyclic aromatic hydrocarbons (PAHs)] are present in biomass smoke, depending on the type of fuel that is burned (Warwick and Doig 2004). Readers are referred to Chap. 6 for details on potential carcinogenic effects of these constituents. The combustion of wood and other biomass is qualitatively similar to the burning of tobacco in terms of emissions of PM and gases, although without the nicotine.

In rural areas of lesser developed countries, women often spend many hours a day cooking so duration of exposure to biomass smoke is often considerable. The potential for exposure is further increased by common cultural practices. Infants and toddlers are often carried on the mother's back while she cooks so that from early infancy children spend hours breathing smoke from cooking or heating fires (Armstrong and Campbell 2009). In temperate climates and highland areas, people spend more time indoors to protect themselves from the cold, and the cold temperatures that characterize these areas require fires that burn over extended periods and tighter house construction (i.e., less ventilation) for space heating (Smith et al. 2013). Thus, both pollution levels and exposure times increase.

The public health impact of the relatively high exposures to PM in homes where cooking is done with inefficient, poorly ventilated stoves using biomass fuels is great. The latest version of the Global Burden of Disease comparative risk assessment undertaken in 2010 found household air pollution from solid fuel use to be responsible for approximately 4.8 % of the total global burden of disease disability-adjusted life years and 3.9 million premature deaths per year (Lim et al. 2012; Smith et al. 2014). Household air pollution ranked third on the examined list of risk factors globally and was the most important environmental risk factor. The estimates are based on the strength of the evidence, primarily meta-analyses of epidemiological studies of acceptable scientific quality, although for cardiovascular disease the evidence is more inferential (see Sect. 14.8). The greatest burden of household air pollution-related premature deaths is in children with pneumonia exposed to biomass smoke. The greatest burden in adults is for cardiovascular disease, but for non-smoking women, chronic obstructive pulmonary disease (COPD) is also an important cause of disability and death. Household air pollution also contributes to the burden of cataracts, the leading cause of blindness, and lung cancer, especially among women. It is also worthy of note that an estimated 16 % of the global burden of disease attributed to ambient PM pollution comes from household combustion of solid fuel. The burden of disease due to household air pollution falls hardest on poor populations in Africa, Latin America, and Asia.

In some parts of the world, poorly burned solid fuels are commonly used for space heating and/or lighting, as well as cooking. The 2010 comparative risk assessment for household air pollution was explicitly limited to exposures related to cooking with solid fuels, and did not include other sources of exposure, largely because of lack of data. Because exposures to pollutants do occur from combustion for space heating and lighting as well as cooking with non-solid fuels, such as kerosene, future assessments will consider these sources as better data accumulate.

14.2 Mechanisms

The mechanisms by which biomass smoke causes adverse health effects in humans are likely similar to those involved in tobacco smoke and combustion-generated PM pathogenic processes. Oxidative stress in the airways and alveoli leads to stimulation of alveolar macrophages and injury to the epithelial lining, which in turn attracts inflammatory cells from the circulation. This local lung inflammatory reaction can spill over into the systemic circulation and contribute to adverse effects in other organs, such as the cardiovascular system, and the fetus in pregnant women. Alveolar macrophages laden with carbon particles from biomass smoke may contribute to increased risk of respiratory tract infections. The pathways involved in biomass smoke-induced lung carcinogenesis are probably identical to those by which tobacco smoke induces lung cancers.

Although the toxicological literature for biomass smoke is much less rich than for tobacco smoke and PM, a number of animal and *in vitro* studies have been reported and were referenced in a recent review paper (Migliaccio and Mauderly 2010). Pulmonary outcomes have been best studied in animal models, including airway responsiveness, lung function, effects on surfactant, epithelial damage, inflammation, edema, and antibacterial activity. Extrapulmonary outcomes have included effects on the cardiovascular system, adjuvant activity, p450 enzyme activity, glutathione depletion, and tumor/cancer growth.

A growing number of reports of controlled human exposure studies of wood smoke have been published. These studies are limited to acute effects and are specific to the fuels and burn conditions used, but evidence of systemic oxidative stress and inflammation, airway inflammation, and arterial stiffness has been observed after short-term exposure to wood smoke (Barregard et al. 2008; Sehlstedt et al. 2010; Stockfelt et al. 2012; Unosson et al. 2013). Not all studies, however, have found adverse effects (Riddervold et al. 2012; Forchhammer et al. 2012; Stockfelt et al. 2013).

14.3 Low Birth Weight

Low birth weight was not one of the conditions considered in the 2010 Global Burden of Disease effort because of lack of data from many countries. That said, there is considerable evidence of an association between biomass smoke exposure and low birth weight. A systematic review on the risk of low birth weight and solid fuel use was published in 2010 (Pope et al. 2010). The meta-analysis was updated in 2012 for a WHO report on indoor air quality. One randomized controlled trial (RCT) (Thompson et al. 2011) and six observational studies (Mavalankar et al. 1992; Boy et al. 2002; Mishra et al. 2004; Siddiqui et al. 2008; Tielsch et al. 2009; Abusalah et al. 2011) on the risk of low birth weight (<2,500 g at term) were reviewed. The pooled odds ratio (OR) was 1.40 [95 % confidence interval (CI): 1.26–1.54].

The biological plausibility of a biomass smoke effect on birth weight is supported by a similar and well-documented effect of in utero exposure to secondhand tobacco smoke (SHS) as well as mechanistic evidence of association between components of household air pollution (HAP) (CO, PM, and PAHs) and low birth weight. The range of reported estimates are consistent with those for related exposures, including outdoor air pollution, second hand smoking (SHS) and active smoking. The mean decrease in birth weight for biomass smoke exposure (90–100 g) lies between published estimates for SHS and active smoking, as would be expected from the relative levels of exposure to $PM_{2.5}$ (Pope et al. 2010). The main deficiency of this literature is the lack of measurement of exposure to biomass smoke and thus the inability to demonstrate an exposure-response relationship. Nevertheless, the WHO report assessed the evidence of an association between biomass smoke exposure and low birth weight as moderately strong, applying Bradford Hill viewpoints, because of consistency, temporality, biological plausibility, and analogy. The one published RCT of a chimney stove provided some evidence in support of an intervention effect (Thompson et al. 2011).

14.4 Acute Lower Respiratory Infection (Childhood Pneumonia)

Acute lower respiratory infection (ALRI) is a leading contributor to the global burden of disease, accounting for 4.6 % of the total (Murray et al. 2012). Pneumonia is also the primary cause of death in children (1.4 million deaths in children younger than 5 years) and the incidence and mortality are generally highest in those countries and regions where solid fuel use is greatest (Nair et al. 2013). The relative risk of ALRIs for children exposed to household biomass smoke has been quantified in a number of studies, the majority from developing countries, but also from the United States (Torres-Duque et al. 2008). Most of these studies have used a case-control design, although several cohort studies have also been conducted. Taken

together, these studies show a consistent association between solid fuel use and an increase in the risk of ALRI in exposed children. The overall estimate of the risk of ALRI from 24 studies selected for a meta-analysis conducted in preparation for the 2010 Global Burden of Disease comparative risk assessment (Dherani et al. 2008) was a pooled OR of 1.79 (CI: 1.26–2.21) for children younger than 5 years. The OR for children younger than 2 years was 1.96 (CI: 1.36–2.82). Only three studies included direct measurement of exposure, the remainder used proxies (fuel type, etc.). Outcome definitions varied from parental recall of WHO signs of ALRI to chest X-ray confirmation, and included severe and fatal outcomes; included studies had to distinguish upper from lower respiratory infections. Some evidence of an increased risk for more severe pneumonia was found. Impaired respiratory tract defense mechanisms, such as decreased mucociliary clearance and alveolar macrophage function, provide biological plausibility for the observed association between biomass smoke exposure and ALRI risk in children (Siddiqui et al. 2008; Zelikoff et al. 2002).

Only one RCT of an intervention to reduce exposure to biomass smoke to prevent childhood ALRI has been completed (Smith et al. 2011), involving 514 Guatemalan children aged <16 months, randomized to use a chimney wood stove or traditional three-stone fire. The primary outcome was physician diagnosis, with pulse oximetry to define severity. Exposure was assessed for all children using repeated CO measurements, together with co-located kitchen CO and $PM_{2.5}$ measurements in a sub-sample to define the CO-$PM_{2.5}$ relationship. In intention to treat analysis, child exposure was reduced by 50 %, associated with a relative risk of 0.78 (CI: 0.59, 1.06) for all physician-diagnosed pneumonia, and of 0.67 (CI: 0.45, 0.98) for severe pneumonia (defined by low oxygen saturation). Adjusted exposure-response analysis found significant relationships for both all and severe pneumonia.

The randomized control trial to study the effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE) results support a causal association between biomass smoke exposure and early childhood pneumonia and show that the greatest risk reduction occurs at lower exposure levels. The RESPIRE exposure-response data suggest that ALRI incidence would be reduced by around one-third with interventions that bring average $PM_{2.5}$ down from several hundreds of $\mu g/m^3$ to levels experienced by "unexposed" groups in the majority of epidemiological studies (Smith et al. 2014) (see Fig. 14.1). Reduction of exposure to biomass smoke levels at or below the WHO annual average guideline of 10 $\mu g/m^3$ $PM_{2.5}$ would theoretically result in larger risk benefit.

14.5 Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease is a leading cause of morbidity and mortality (~4 million deaths/year) worldwide and results in an economic and social burden that is both substantial and increasing, especially among women (Global Initiative for Chronic Obstructive Lung Disease 2014; Lozano et al. 2012). In less developed countries, a substantial proportion of COPD occurs in people who have never

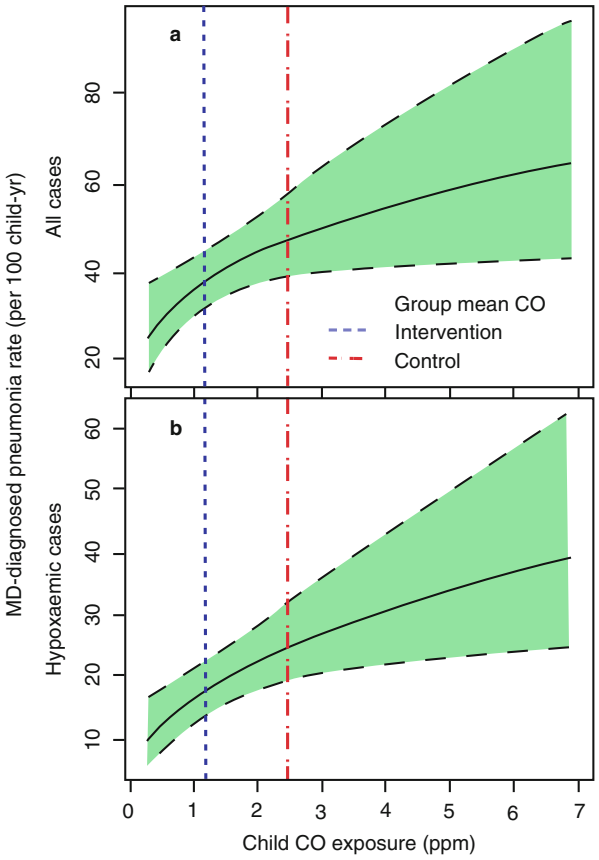

Fig. 14.1 Exposure-response from the RESPIRE study for wood smoke exposure as assessed by average personal monitoring data and the rate of physician-diagnosed severe pneumonia as assessed by oxygen saturation (Reprinted from Smith et al. (2011) with permission from Elsevier)

smoked tobacco products, especially among women cooking with stoves that emit high levels of a wide variety of pollutants similar to those present in tobacco smoke (Lim et al. 2012). Multiple case-control and cross-sectional studies have found an association between cooking with biomass fuel and chronic bronchitis or COPD when compared with lower exposure/cleaner fuels (Akhtar et al. 2007; Albalak et al. 1999; Caballero et al. 2008; Dutt et al. 1996; Ellegard 1996; Menezes et al. 1994; Qureshi 1994; Regalado et al. 2006; Saha et al. 2005; Dennis et al. 1996; Dossing et al. 1994; Ekici et al. 2005; Kiraz et al. 2003; Orozco-Levi et al. 2006; Perez-Padilla et al. 2006). Exposure was often assessed as present or absent or by daily hours spent by the stove. Most studies did not include direct measurements of specific pollutants. Studies that have measured kitchen particulate levels from biomass fuel use have confirmed very high concentrations (Albalak et al. 1999; Regalado et al. 2006), but personal measurements of exposure have not been used.

Only a few cross-sectional studies have measured lung function and these studies have shown an association between biomass fuel use and chronic airflow obstruction (Caballero et al. 2008; Regalado et al. 2006). Taken together, the results of previous studies show a strong association between cooking with biomass fuel and COPD among women (Balmes 2010).

Several systematic reviews and meta-analyses have been published, including the one conducted for the 2010 Global Burden of Disease comparative risk assessment (Smith et al. 2014; Kurmi et al. 2010; Hu et al. 2010; Po et al. 2011). In the Smith et al. (2014) review 25 studies were included in the final analysis. All but seven included in the meta-analysis were cross-sectional in design. One study was a retrospective cohort (Chapman et al. 2005) and six were case-control (Dennis et al. 1996; Dossing and Khan 1994; Orozco-Levi et al. 2006; Perez-Padilla et al. 2006; Sezer et al. 2006; Xu et al. 2007). Case-control studies recruited controls from various sources, including visitors to the hospital, patients from other hospital services without presence of pulmonary disease, and population-based selection. Heterogeneous exposure measures were used in these studies, including rural-urban comparisons (where data supported the use of place of residence as a proxy for fuel use), outdoor versus indoor cooking, fuel type for cooking and/or heating, stove type and time-exposed to biomass fuel combustion. The pooled OR was 1.94 (CI: 1.62–2.33). Stratified analysis by gender showed a stronger association between exposure to biomass smoke and COPD in women [OR, 2.30 (CI: 1.73-3.06)] than in men [OR, 1.90 (CI: 1.15–3.13)]. The association in women remained significant even after adjusting for both age and smoking, while this was not the case for men.

Three other recently published systematic reviews and meta-analyses reported similar summary estimates for the effect of biomass smoke on risk of COPD. Kurmi et al. (2010) reported a summary OR (CI) for the use of solid fuels and COPD from 23 studies of 2.80 (CI: 1.85–4.0) and for biomass smoke and chronic bronchitis of 2.32 (1.92–2.80). Pooled estimates for different types of fuel showed that exposure to wood smoke carried a greater risk than coal (also seen in the Global Burden of Disease meta-analysis). Hu et al. (Hu et al. 2010) reported a summary OR from 15 studies of 2.44 (CI: 1.9–3.33) for the risk of developing COPD with biomass smoke exposure. Po et al. (Po et al. 2011) included data from 12 studies of female populations and reported a summary OR for chronic bronchitis of 2.52 (CI: 1.88–3.38) and for COPD of 2.40 (CI: 1.47–3.93). Applying the Bradford-Hill viewpoints (consistency, exposure-duration evidence, analogous evidence of the effect of smoking, and biological plausibility), the case for a causal association between biomass smoke and COPD is moderately strong, especially among women (Eisner et al. 2010).

Longitudinal studies of the impact of biomass smoke exposure on the development of COPD are lacking, but one study did follow women with COPD associated with biomass smoke exposure in terms of mortality risk (Ramirez-Venegas et al. 2006). Survival analysis over a 7-year follow-up period showed that women with COPD associated with biomass smoke exposure had mortality rates similar to those of men with COPD due to tobacco smoking.

Intervention studies to prevent COPD from biomass smoke exposure have been limited. A retrospective Chinese study found significant reductions in COPD incidence in homes where coal was used in improved stoves with chimneys, the effect increasing with time since adoption (Chapman et al. 2005). Two recently published studies of the efficacy of reducing wood smoke exposure with the use of an improved chimney stove were limited by short follow-up time (18 months) (Smith-Sivertsen et al. 2009; Romieu et al. 2009). Thus, although studies suggest lower biomass smoke exposure may be associated with lower COPD risk, several key questions remain, including can the development or progression of COPD be prevented by reducing biomass smoke exposure in these settings and what is the exposure-response relationship between biomass smoke and the rate of decline in lung function? Prospective cohort studies that have adequate statistical power, follow participants for sufficient duration to accurately measure rate of decline in lung function, and actual exposure measures are needed to answer these questions. Ideally, intervention studies with clean stoves will be conducted.

14.6 Lung Cancer

The emissions generated of combustion of the main solid fuels used for cooking and space heating, biomass and coal, may be associated with differential cancer risk because of differences in chemical composition. The International Agency for Research on Cancer (IARC) has concluded household use of coal is a Group 1 carcinogen, while biomass is classified as a Group 2(a) or probable carcinogen, due to more limited epidemiological evidence (International Agency for Research on Cancer 2010).

Two recent systematic reviews and meta-analyses of household use of coal and lung cancer risk have been conducted, including the one used as the basis for the 2010 Global Burden of Disease comparative risk assessment (Hosgood et al. 2011) and an additional effort by Kurmi et al. (2012). The Hosgood et al. review identified 25 case-control studies investigating household coal use with seven provided cooking-specific estimates. Exposure was assessed by fuel type, and lung cancer confirmed by pathology for most cases, otherwise by chest X-ray. Only five studies were conducted in countries other than mainland China and Taiwan. Household coal use for cooking and heating was associated with a pooled OR for lung cancer of 2.15 (CI: 1.61–2.89). The second review by Kurmi et al. pooled a total of 22 studies to generate an OR of 1.82 (CI: 1.60–2.06). In both reviews, the risk for women was somewhat greater than the risk for men.

Since the great majority of the 2.8 billion solid fuel users globally is exposed to biomass rather than coal smoke, the issue of the carcinogenic risk associated with the former is an important one. A systematic review for the 2010 Global Burden of Disease comparative risk assessment (Smith et al. 2014) identified 14 eligible

studies of cooking and/or heating with biomass. Most of the studies conducted in Asia, although there were studies from Europe and North America as well. Biomass fuel was defined as including wood, straw, grass, crop waste, animal dung, and charcoal; only household use was considered, whether for cooking or heating. Exposure was determined by fuel type and no study directly measured exposure. One European study had exposure-response data available (Lissowska et al. 2005). Most cases of lung cancer were confirmed by pathology. The pooled OR for all 14 studies was 1.18 (CI: 1.03–1.35), but the most reliable estimate OR of 1.22 (CI: 1.08–1.37) from well-adjusted studies with clean fuel comparisons. The review by Kurmi et al. pooled seven studies and only used adjusted estimates to generate an OR that was somewhat higher, 1.50 (CI: 1.17–1.94). (Kurmi et al. 2012). In both the 2010 Global Burden of Disease and Kurmi et al. meta-analyses, women had somewhat higher risk than men. Applying the Bradford-Hill viewpoints (including consistency, exposure-response, biological plausibility), there does appear to be a moderately strong case for a causal association.

14.7 Cataracts

Cataract or severe lens opacification is one of the leading causes of blindness in the developing world. The 2010 Global Burden of Disease project (Murray et al. 2012) estimated that cataracts accounted for 0.2 % of the total disease burden, the majority of which is seen in the developing regions of sub-Saharan Africa and Southeast Asia alone where cooking with solid fuels is most common. The toxicological evidence from animal studies and epidemiological studies of other types of smoke (e.g., active smoking and secondhand smoke) suggest biological plausibility for an association between exposure to biomass smoke and risk of cataract. Condensates of both tobacco and biomass fuel smoke enhance the formation of superoxide radicals, which decrease antioxidants and cause lens discoloration and cataract. A component of biomass smoke is naphthalene, which has been shown to cause cataract in laboratory animal studies (van Heyningen and Pirie 1976).

The systematic review and meta-analysis for the 2010 Global Burden of Disease comparative risk assessment identified seven eligible studies, most of which had case-control designs, and all were from Southeast Asia (Smith et al. 2014). The pooled OR was 2.46 (CI: 1.74–3.50) and sensitivity analysis revealed no major confounding due to smoking or UV exposure. The results of the meta-analysis, the known risks from smoking, the consistency of results across studies, the toxicological data (especially regarding naphthalene), and some exposure-response evidence (Pokhrel et al. 2013) support causal inference.

14.8 Cardiovascular Disease

In contrast to the other health outcomes discussed above, at the time of writing this chapter, there were no epidemiological studies of the relationship between exposure to biomass smoke and cardiovascular disease. That said, a remarkably consistent, nonlinear relationship between estimated inhaled dose of combustion-sourced particles measured as $PM_{2.5}$ (particulate matter with aerodynamic diameter $\leq$2.5 μm) and cardiovascular disease mortality was first demonstrated by Pope et al. (Pope et al. 2009). This dose-response relationship was remarkable for a non-cancer outcome in that it covered at least three orders of magnitude of dose from lowest to highest (exposure to ambient air pollution, exposure to secondhand tobacco smoke exposure, and active cigarette smoking). It then struck Peel and Smith that exposures to $PM_{2.5}$ from solid fuel cooking lay in the range between those from secondhand smoke and active smoking (Smith and Peel 2010) and, given that tobacco is a kind of biomass, they postulated that the relative risk for CVD from biomass fuel smoke exposure would be in the order of 1.3–1.6 (Fig. 14.2).

Because the Global Burden of Disease project is designed to provide useful information for public health policy decisions regarding interventions to reduce important risks, the 2010 comparative risk assessment for household air pollution included identification of attributable risk for cardiovascular disease, the number one cause of death and disability on a global scale. If there were no available epidemiological studies that directly assessed the association between biomass smoke exposure and cardiovascular disease risk, then how could any risk be attributed to this exposure? An effort was made by an expert group to interpolate the risk due to biomass smoke by generating what has been termed an "integrated exposure-response curve." The curve derived for risk of ischemic heart disease in relation to combustion-sourced fine particles is similar to that suggested by Pope et al 2011 (Pope et al. 2009), i.e., supralinear in shape from low exposures through the household air pollution exposure range, where it reaches a relative risk of 1.5 for high biomass fuel smoke exposure and a maximum of 2.5 for high active smoking exposure (Smith et al. 2014; Burnett et al. 2014). The integrated exposure-response curve for stroke is similar, but appears to flatten off at levels well within the household air pollution range at a maximum relative risk of just over 2.0.

Subsequent to the preparation of the 2010 Global Burden of Disease comparative risk assessment, the results of a cross-sectional Chinese study involving over 14,000 men and women aged 18 and over were reported (Lee et al. 2012). Solid fuel use (biomass and coal) for heating and/or cooking was assessed by questionnaire, and categorized according to ever use, duration, total amount and lifetime use. Outcomes, including ischemic heart disease, stroke and diabetes mellitus, were assessed by self-report of physician-diagnosed conditions, while blood pressure was measured during the study. The results showed elevated adjusted odds ratios for ischemic heart disease forever vs. never use of solid fuels, and significant trends across duration of use for stroke, hypertension and diabetes. The OR for ischemic heart disease 2.6 is somewhat higher than predicted from the integrated exposure–response curve; the

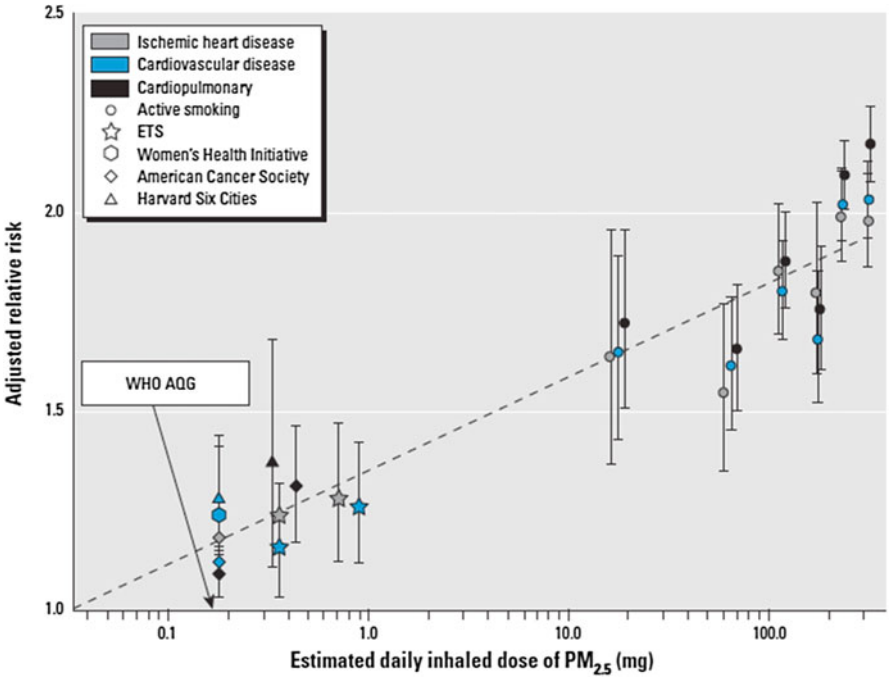

Fig. 14.2 Adjusted relative risks (95 % confidence intervals) of cardiovascular and cardiopulmonary mortality and estimated dose of PM2.5 across studies of outdoor air pollution, ETS, and active cigarette smoking (Adapted with permission from (Pope et al. 2009), their Fig. 2). Data on active smoking are from Pope et al. (Pope et al. 2009); on ETS are from the 2006 Surgeon General's Report (U.S. Department of Health and Human Services 2006) and INTERHEART study (Teo et al. 2006); on air pollution are from the Women's Health Initiative cohort (Miller et al. 2007), the American Cancer Society cohort (Pope et al. 1995, 2002, 2004), and the Harvard Six Cities cohort (Dockery et al. 1993; Laden et al. 2006). Exposure was measured as daily inhaled dose of $PM_{2.5}$ (plotted on a log scale), calculated assuming 18 m^3/day breathing rate. Active cigarette smoking was quantified as ≤3, 4–7, 8–12, 13–17, 18–22, and ≥23 cigarettes/day (relative to never-smokers). Also shown is the equivalent dose for the World Health Organization (WHO) (2006) Air Quality Guidelines (AQG) for $PM_{2.5}$ (10 μg/m3 annual average) (Smith and Peel 2010)

OR for stroke, 1.6, was in the range expected from the curve although it was not statistically significant. Sex-stratified analysis found stronger effects for women, consistent with their higher exposure, and among non-smokers.

Although there is little epidemiological literature on the effect of biomass smoke exposure on cardiovascular disease risk, there are studies that show effects on blood pressure (McCracken et al. 2007; Baumgartner et al. 2011) and the ST-segment of the electrocardiogram (ECG) (McCracken et al. 2011). For blood pressure, there are five studies, four observational and one intervention (the latter with both RCT and before and after components). All have found that increased exposure was associated with higher systolic and/or diastolic blood pressure, with most findings being statistically significant. In the intervention trial (RESPIRE), McCracken et al. found

the chimney stove intervention group to have statistically significant lower systolic (−3.7 mmHg) and diastolic (−3.0 mmHg) blood pressure in an intention-to-treat analysis, and similar sized effects when open-fire control subjects received the chimney stove at the end of follow-up (McCracken et al. 2007). In the same RESPIRE study subjects significant effects of the chimney stove intervention on subjects' ECGs were demonstrated. During the trial, the stove intervention was associated with a significant protective effect (OR 0.26) for ST-segment depression and a similar effect with the before-and-after comparison (McCracken et al. 2011).

The population attributable risk for ischemic heart disease associated with biomass smoke exposure estimated by the integrated exposure-response curve method was 18 % compared to 53 % for hypertension, 29 % for hypercholesterolemia, and 31 % for tobacco smoke (both active smoking and secondhand exposure) (Lim SS et al. 1990). Despite the dearth of epidemiological studies, the overall evidence, including from the related combustion-sourced particles of smoking (active and second-hand) and outdoor air pollution, together with the observed blood pressure and electrocardiographic effects, suggests that exposure to biomass smoke can increase cardiovascular disease risk.

14.9 Other Diseases (Tuberculosis, Adult Pneumonia, Asthma)

Three other diseases for which the weight of evidence was judged not strong enough to attribute risk of disability-adjusted life years to household air pollution in the 2010 Global Burden of Disease comparative risk assessment are tuberculosis, adult pneumonia, and asthma. Nevertheless, there is some evidence and biological plausibility for an association between exposure to biomass smoke and increased risk for each of these diseases.

14.9.1 Tuberculosis

Tuberculosis (TB) was responsible for 1.4 million deaths in 2011, and over 80 % of the global burden of TB lies in the Western Pacific, South-East Asia and Africa; China and India together have 40 % of the world's cases (Organization and Report 2013). The close association of tuberculosis with poverty, and use of solid fuel for cooking and heating in the home, makes the impact of exposure to household air pollution on risk of TB is a major concern. Because active tobacco smoking has been associated with increased risk for TB, it is again biologically plausible that exposure to biomass fuel smoke would also increase risk. Carbon particle–laden alveolar macrophages, for example, have been shown to be less effective in phagocytosis of microorganisms than those from subjects not exposed to smoke (Zhou and Kobzik 2007; Fick et al. 1984; Migliaccio et al. 2013).

Several reviews have been done over the past decade with the most recent, a systematic review and meta-analysis published in 2013 (Lin et al. 2007; Slama et al. 2010; Sumpter and Chandramohan 2013). In the 2013 review, 10 case-control and three cross-sectional studies were included. The outcome was sputum positive TB in all of the case-control studies, and two of the cross-sectional studies. None of the studies directly measured exposure to household air pollution so exposure assessment was only by survey of fuel type. Adjustment for smoking and crowding was done in most, but not all studies. Considerable study heterogeneity was found and study design issues were deemed problematic (e.g., hospital-based controls and over-representation of men). The pooled OR for the 10 case-control studies was 1.3. The six studies of women only had a pooled OR of 1.7.

A study by Pokhrel et al. also examined the risk of TB associated with the use of kerosene as well as solid biomass fuel (Pokhrel et al. 2010). Kerosene use was shown to have a much stronger effect on TB risk, for cooking with kerosene, the OR was 3.36 (CI: 1.01–11.22) and for lighting the OR was 9.43 (CI: 1.45–61.32). For cooking with a biomass fuel stove, the OR was a non-significant 1.21.

As noted above, the most up-to-date review and meta-analysis of the solid fuel-TB risk association reported a significantly increased pooled OR. However, both exposure-response evidence and intervention studies are lacking. Nevertheless, together with the well-established evidence that smoking increases risk of TB, findings from studies of animals exposed to solid fuel smoke (Zhou and Kobzik 2007; Fick et al. 1984; Migliaccio et al. 2013), and the plausible mechanism through impaired immunity from solid fuel exposure (Dutta et al. 2012), there is now a moderately strong case for a causal association between exposure to household air pollution and TB risk. Given the high prevalence of both exposure to household air pollution and TB across the developing world (and in the same geographic regions), reduction in household air pollution could make an important contribution to control of TB. Further research is needed to confirm that the association is causal rather than due to confounding by poverty-related factors, to better quantify exposure-response, and to assess potential interventions to reduce exposure.

14.9.2 Adult Acute Lower Respiratory Infection (Pneumonia)

In the 2010 Global Burden of Disease report, acute lower respiratory infections caused 2.8 million deaths annually (Lozano et al. 2012). Slightly over half of these deaths occur among adults (15 years and above), and most in those aged 60 years and over. While the majority of the adult deaths due to ALRI occur in developed countries, a substantial proportion of adult deaths in developing countries is due to ALRI/pneumonia. Given the strong evidence that exposure to biomass smoke causes ALRI in children (most probably through reducing immune defense mechanisms), as well as the established association between tobacco smoking and ALRI in adults, a biomass smoke-ALRI in adults is plausible.

A systematic review of adult ALRI was carried out for the 2010 Global Burden of Disease comparative risk assessment (Smith et al. 2014). From the many papers reviewed, only three were selected to have adequate data to assess the potential association of exposure to household air pollution and ALRI, and there is considerable heterogeneity among the three (Ezzati and Kammen 2001; Levesqu et al. 2001; Shen et al. 2009). The study by Ezzati and Kammen is a small cohort study in rural Kenya where various forms of biomass are used (wood, charcoal); home visits were used for assessment of community-acquired pneumonia (Ezzati and Kammen 2001). The data analysis also demonstrated a significant exposure-response relationship. The study by Levesque et al. is from Quebec, where biomass is used for household heating rather than cooking and exposure is much lower; the ALRI outcomes were self-reported and likely subject to some misclassification (Levesqu et al. 2001). The study by Shen et al. used a case–control design to assess risks for pneumonia deaths in a coal-using area of China (Shen et al. 2009). All three studies reported significant ORs for exposed groups in the range of 2.0–3.0. Because of differences in outcome definitions and assessment, pooling was not done.

Although the epidemiological data are limited, the exposure-response relationship from the study by Ezzati and Kammen (2001), strong evidence of a biomass smoke effect in children, and the known effects of other sources of combustion-sourced particle exposure (e.g., smoking and ambient air pollution), together suggest that exposure to solid fuel smoke increases the risk of adult ALRI. Further research is required to confirm and quantify this risk.

14.9.3 Asthma

Given the airway irritating effects of biomass smoke, it is reasonable to suspect that exposure might increase risk for asthma, especially because exposure to secondhand tobacco smoke has been associated with the development of asthma in children (Vork et al. 2007). On the other hand, there is a rural-urban gradient for asthma. The disease is generally much more prevalent in urban areas than in the largely rural areas where exposure to biomass smoke is more common (Addo-Yobo et al. 2007). The protective effect of living on a farm, especially with livestock, has been well documented (Ege et al. 2011). Because the evidence for an effect of biomass smoke on COPD is strong, and both asthma and COPD involve airway inflammation, the relationship between exposure to biomass smoke and risk of asthma remains a topic of considerable research interest.

A systematic review and meta-analysis of the association between exposure to biomass smoke and asthma in children and women was published in 2011 (Po et al. 2011). Nine studies were selected for the meta-analysis, four involving children and five involving women. The pooled OR for the four studies on asthma in children exposed to biomass smoke was 0.50 (CI: 0.12–1.98) (Fagbule and Ekanem 1994; Noorhassim et al. 1995; Behera et al. 2001; Melsom et al. 2001). For the five studies of women, the pooled OR was 1.34 (CI: 0.93–1.93) (Qureshi 1994; Behera et al. 2001; Golshan and Faghihi 2002; Uzun et al. 2003; Mishra 2003).

Since the Po et al. review was published, the results of several new studies have been reported. A survey of 508 adults in southeastern Kentucky found a significant association with asthma when wood and coal were burned for cooking (Barry et al. 2010). A much larger population survey from India found that adult women living in households using biomass and solid fuels had a significantly higher risk of asthma than those living in households using cleaner fuels, even after controlling for the effects of a number of potentially confounding factors (Agrawal 2012).

The most robust evidence supporting an increased risk of asthma from cooking with solid fuels was reported by the International Study of Asthma and Allergy in Children (ISAAC) which surveyed almost 513,000 children from 1999–2004 (Wong et al. 2013). The sole use of an open fire for cooking (assessed by questionnaire) was associated with an increased risk of wheeze in the past year in both young children (ages 6–7 years), OR 2.17 (CI: 1.64–2.87), and in older children (ages 13–14 years), OR 1.35 (CI: 1.11–1.64). The ORs for wheeze in the past year and the use of open fire in combination with other fuels for cooking were slightly lower, but still significant for both age groups.

Not all recent studies have reported positive associations between exposure to biomass smoke and asthma outcomes. For example, a recent study of 299 children from rural villages in Nigeria, reported smoke exposure was not associated with an increased risk of asthma symptoms or airway obstruction (Thacher et al. 2013). Because the published evidence is mixed, further research is needed to better understand the nature of the relationship between exposure to solid fuel smoke and asthma in both children and adults and for both new onset and exacerbations of disease. Studies that actually measure exposure and which are thus able to develop an exposure-response relationship would be particularly helpful.

14.10 Research Gaps

While one RCT of the efficacy of a chimney stove compared to an open fire for the prevention of early childhood pneumonia has been published (Smith et al. 2011) and related studies have suggested benefit regarding low birth weight, cognitive development, and respiratory symptoms in adult women, more studies of stove interventions are needed. In particular, the exposure-response data from the RESPIRE trial provide evidence that stoves which are cleaner than the chimney stove studied would provide greater reduction in childhood pneumonia. Several other ongoing RCTs of cleaner stove interventions in Nepal and Africa should contribute data to fill the gap in our knowledge about the added benefit of cleaner burning stoves beyond those with chimneys.

Another gap is the lack of a well-designed longitudinal study of the impact of improved stoves on the development of COPD in adults exposed to biomass smoke form cooking and heating. One published study from Mexico conducted in the context of a RCT designed to prevent childhood pneumonia found a protective effect of a chimney stove on rate of decline of lung function in women who actually

used the stove (Romieu et al. 2009), but this study suffers from the limitation of too short of a follow-up period (1 year). Another paper from RESPIRE reports evidence that reduced exposure from use of a chimney stove is associated with decreased expression of metalloproteinase genes (Guarnieri et al. 2014).

Perhaps the greatest research gap involves the lack of epidemiological evidence to support the concept that biomass smoke exposure leads to increased risk of cardiovascular disease consistent with other combustion-sourced particle exposures, outdoor $PM_{2.5}$, secondhand tobacco smoke, and active smoking.

More research is needed with regard to the associations between biomass smoke exposure and adult pneumonia, asthma, and tuberculosis, either because of paucity of data (adult pneumonia) or conflicting results. Another unresolved issue is the toxicity of kerosene as an alternative fuel, especially given the recently published evidence that it substantially increases the risk of tuberculosis (Pokhrel et al. 2010)

Because adoption of new stoves can be a difficult transition for households in the developing world due to both financial and cultural factors, more research is needed to improve our understanding of these potential barriers to the effectiveness of intervention programs.

Toxicological studies (*in vitro*, animal, and controlled human exposure) are needed to improve our mechanistic understanding of the pathways by which biomass smoke causes or contributes to disease pathogenesis. Such studies can also assist in the development of biomarkers of both exposure and effects (Rylance et al. 2013).

14.11 Opportunities for Intervention

For many years, the major obstacle to implementing programs to reduce biomass smoke exposure from domestic cooking and heating with inefficient, poorly ventilated stoves was lack of data proving benefit. Now that at least one RCT has proved efficacy in reduction of early childhood pneumonia, comparable to that of vaccination, with results from several other trials on the way, the major issues are cost and effectiveness of stoves that can substantially reduce biomass emissions. Improved stoves still cost more than many households in the developing world can afford, maintenance can be difficult, and many households may not be willing to adopt new stoves that require changes to traditional cooking styles.

An exciting program that was started with the help of Hillary Clinton when she was US Secretary of State is the Global Alliance for Clean Cookstoves (GACC), under the auspices of the United Nations Foundation. The GACC is a public-private partnership that "seeks to save lives, improve livelihoods, empower women, and protect the environment by creating a thriving global market for clean and efficient household cooking solutions."(Global Alliance for Clean Cookstoves 2014) Its mission is to mobilize high-level national and donor commitments toward the goal of universal adoption of clean cookstoves and fuels. The primary goal of the GACC is to foster the adoption of clean cookstoves and fuels in 100 million households by 2020. Multiple demonstration and research projects have been funded by the

organization since it was established in 2010. In 2012, the following countries were prioritized for immediate GACC engagement: Bangladesh, China, Ghana, Kenya, Nigeria, and Uganda.

Although the GACC represents a major breakthrough for efforts to improve the health of the 45 % of the world's population that still cooks with solid fuel, even the most efficient stoves that burn biomass emit more $PM_{2.5}$ than the gas and electric stoves that are used by developed world households. This has led some experts to suggest that electrification of developing countries and the use of relatively inexpensive induction cookers may be the most effective way to reduce exposure to household air pollution.

A final policy note is that black carbon generated from biomass combustion is an important short-lived climate-forcing agent (Jacobson 2001). Interventions to improve the efficiency of combustion of biomass fuels or better, that eliminate combustion of biomass fuels altogether, would provide a major climate change mitigation co-benefit in addition to the public health benefits described above.

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