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KEROSENE: A REVIEW OF HOUSEHOLD USES AND THEIR HAZARDS IN LOW- AND MIDDLE-INCOME COUNTRIES

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Abstract

Kerosene has been an important household fuel since the mid-19th century. In developed countries its use has greatly declined because of electrification. However, in developing countries, kerosene use for cooking and lighting remains widespread. This review focuses on household kerosene uses, mainly in developing countries, their associated emissions, and their hazards. Kerosene is often advocated as a cleaner alternative to solid fuels, biomass and coal, for cooking, and kerosene lamps are frequently used when electricity is unavailable. Globally, an estimated 500 million households still use fuels, particularly kerosene, for lighting. However, there are few studies, study designs and quality are varied, and results are inconsistent. Well-documented kerosene hazards are poisonings, fires, and explosions. Less investigated are exposures to and risks from kerosene's combustion products. Some kerosene-using devices emit substantial amounts of fine particulates, carbon monoxide (CO), nitric oxides (NO_x), and sulfur dioxide (SO_2). Studies of kerosene used for cooking or lighting provide some evidence that emissions may impair lung function and increase infectious illness (including tuberculosis), asthma, and cancer risks. However, there are few study designs, quality is varied, and results are inconsistent. Considering the widespread use in the developing world of kerosene, the scarcity of adequate epidemiologic investigations, the potential for harm, and the implications for national energy policies, researchers are strongly encouraged to consider collecting data on household kerosene uses in studies of health in developing countries. Given the potential risks of kerosene, policymakers may consider alternatives to kerosene subsidies, such as shifting support to cleaner technologies for lighting and cooking.

Since the mid-19th century, when it replaced the more expensive whale oil as a lighting fuel, kerosene (synonyms: kerosine, paraffin, paraffin oil, fuel oil no. 1, lamp oil) has become a major household, commercial, and industrial fuel. “Kerosene” started as a brand name but was later adopted (with a small “k”) as a general descriptor. In the first half of the 20th century, the prevalence of household kerosene lighting greatly reduced as electrification and availability of gas fuels spread, particularly in developed countries. However, in the developing countries of Africa, Asia, and Latin America, kerosene use for cooking and lighting remains widespread. Globally, an estimated 500 million households still rely on kerosene or other liquid fuels for lighting, corresponding to 7.6 billion liters consumed annually (Mills 2005).

Produced originally from coal (“coal oil”), but later from the fractional distillation of petroleum oil, kerosene is a transparent liquid fuel with a mixture of hydrocarbon chains 6 to 16 carbon atoms in length. Although kerosene has numerous commercial and industrial applications (e.g., aviation fuel, general solvent), the focus of this article is on household uses, for cooking, heating, and lighting, in low- and middle-income countries. These lead to the most widespread exposures to kerosene and its combustion products.

Kerosene is commonly used in countries where solid fuels—biomass (wood, agricultural residues, and animal dung) and coal—are major household energy sources, often burned indoors without chimneys or smoke hoods. Exposures to combustion products from solid fuels have been associated with a range of health effects, including lung cancer, chronic obstructive pulmonary disease (COPD), low birth weight, cataracts, pneumonia, and tuberculosis (Fullerton et al. 2008). Justifiably, pollution from solid fuels has provoked efforts to find alternative energy sources or ways of burning biomass more cleanly. Least polluting alternatives at the household level are solar power and electricity. Gaseous fuels, particularly liquefied petroleum gas (LPG) and natural gas, which burn with higher combustion efficiency, resulting in fewer products of incomplete combustion compared to solid fuels, are the cooking fuels of choice in most countries. Biogas made from anaerobic digestion of animal dung is used where conditions are suitable. These alternatives, however, are often unavailable or unaffordable, and kerosene is sometimes advocated as an alternative cleaner fuel for cooking and is often used for lighting where electricity is not available. Some countries, such as India and Nepal, subsidize its retail price to stabilize in-country prices and make it affordable to the poor. However, there is substantial black market diversion of subsidized kerosene, as it mixes easily with the often more expensive diesel fuel, and it is often sold illegally in surrounding countries that do not subsidize it (Parikh 2010; Shenoy 2010).

Other than some older studies of portable kerosene heating stoves in developed countries, there seems to have been little, if any, systematic study of the exposure implications and hazards of household kerosene combustion, possibly because kerosene is often assumed to be “cleaner burning” than biomass fuels for heating and cooking.

The purposes of this review are to (1) systematically examine what is known about household uses of kerosene, its combustion products and exposures to these, and their hazards; (2) identify gaps in knowledge and research needs; and (3) identify any policy implications of current knowledge. The emphasis is on kerosene use in developing countries, where it is widely used as a household fuel. However, developed-country experience is cited where it is informative. The hazards of poisoning and fire associated with kerosene are only briefly discussed, as these have been reviewed elsewhere (Peck

et al. 2008; Tshiamo 2009). For an assessment of the occupational literature and associated risks from exposure to fuel vapors and nonresidential combustion sources, readers are referred to an earlier review of kerosene and kerosene-based jet fuel by Ritchie et al. (2003).

SEARCH METHODS

Using the keywords “kerosene,” “kerosine,” and “paraffin,” combined with any of “epidemiology,” “toxicology,” “emissions,” “respiratory,” and “exposure,” all articles referenced in PubMed, Toxline, and Web of Science relevant to this review were sought. Further articles and publications were obtained from reference lists and Internet search engines, until we were satisfied that all substantive publications relevant to this review in publications indexed in major databases had been identified. Chinese-language bibliographic databases were searched using similar search terms, but no relevant publications were identified, which is probably because electrification and use of LPG have become widespread in China in recent decades. The review is thus limited to publications in English. Epidemiologic studies that combined kerosene with other fuels for the purposes of results presentation were not included.

HOUSEHOLD KEROSENE USES AND FUEL CHARACTERISTICS

Household Uses

Cooking, lighting, and heating are the main household services provided by kerosene, although there are kerosene refrigerators and other appliances in some areas. Kerosene heating is not widespread in temperate or highland areas of developing countries, mainly because of cost. Where heating fuel is needed, biomass or coal is usually used because it is cheaper or more available.

Portable kerosene room heaters are used primarily in developed countries, and some developing countries (e.g., Chile), although many countries have either prohibited or discouraged their use, particularly because of the risk of carbon monoxide (CO) poisoning (Long 1997).

Kerosene cooking is widespread in many developing countries, especially in urban populations, where biomass needs to be purchased, and electricity and LPG are expensive or unreliable. There are many kerosene stove designs, but they can be broadly categorized into two broad types depending on how the fuel is burned—wick stoves, which rely on capillary transfer of fuel, and the more efficient and hotter burning pressure stoves with vapor-jet nozzles that aerosolize the fuel using manual pumping or heat. In low-income households, wick stoves are more commonly used, because they are cheaper, they easily provide simmer heat for some staple foods, and they have no nozzles that can get clogged by soot.

Use of kerosene as a lighting fuel—either in wick lamps or brighter burning (but less common) pressure lamps—is common in some developing countries, particularly in regions where electricity supply is unaffordable, unreliable, or unavailable. An estimated one-fifth of the global population (approximately1.3 billion) in 2009 lacked access to electricity, while an even greater but unknown number had only intermittent access (IEA 2011). Detailed data on the source or frequency of lighting in houses are not as commonly collected as cooking fuel data in household surveys; however, fuel-based lighting is widely used in India and much of Africa (DGDA 2010). In India, in 2004–2005, an estimated one in three households reported kerosene as their primary source of lighting—44.4% of rural and 7.1% of urban households (NSSO 2007). In the lowest four socioeconomic deciles of India, 60% of households use kerosene for lighting (Parikh 2010). In several of the most populated African countries, including Uganda, Ethiopia, and Kenya, more than 60% of the population relies on kerosene

as the primary lighting fuel (Apple et al. 2010; IFC/WB 2008a; Uganda Bureau of Statistics 2010). Less is known of the quantity of kerosene used for lighting, since it is often difficult to differentiate kerosene used for lighting from that used for other purposes, particularly cooking. Based on existing surveys, reports, and local correspondence from 23 countries, monthly consumption has been reported to vary between 1 and 10 L per household (Mills 2005). These estimates may include the use of kerosene to illuminate businesses as well as residences, which would imply that the higher end of this scale is an overestimate. Recognition of the potential welfare benefits resulting from cleaner and more effective lighting technology has led to several large-scale government and private sector efforts to develop and disseminate solar lighting appliances in India and Africa (DGDA 2010; Palit and Singh 2011).

Fuel Characteristics

Kerosene is a middle distillate of the petroleum refining process, defined as the fraction of crude oil that boils between 145 and 300°C (U.S. Environmental Protection Agency [EPA] 2011). Kerosene can be produced from distillation of crude oil (straight-run kerosene) or from the cracking of heavier petroleum streams (cracked kerosene). Raw kerosene has properties that make it suitable for mixing with performance additives for use in a variety of commercial applications, including transportation fuel. Although this review focused on kerosene in the residential sector, which typically contains no performance additives, studies of kerosene-based aviation fuel are included where they are informative. Kerosene is a complex mixture of branched and straight-chain compounds, which can be generally categorized into three classes: paraffins (55.2% w/w), naphthenes (40.9%), and aromatics (3.9%) (U.S. EPA 2011). Relative proportions vary depending on source of the crude oil and the nature of the refining process. The American Society of Testing and Materials (ASTM) defines two kerosene grades, 1–K and 2–K, acceptable for household appliances. Grades are designated by impurity content, particularly sulfur and aromatics, which reduce combustion efficiency and increase noxious emissions during combustion. 1–K ("low-sulfur") kerosene contains no more than 0.04% sulfur by weight; 2–K has no more than 0.30%. ASTM considers 1–K as suitable for use in flueless appliances (e.g., portable heaters), while 2–K is suitable for flued appliances. Both grades are designated for use in "illuminating lamps" (ASTM 2008). Cleaner burning lamp oils (Fan et al. 2001), which are often deodorized kerosene or hydrocarbon mixes (e.g., 142 Flash), are not commonly available in developing countries. Unlike biomass fuels, which present no toxic risk prior to combustion, liquid kerosene fuel contains numerous compounds that potentially pose health risks, including *n*-hexane, naphthalene, and benzene.

HUMAN EXPOSURES AND POLLUTANT EMISSIONS

This section summarizes results of studies of human exposure to airborne pollution generated by household kerosene appliances. Kerosene heaters, cooking stoves, and lighting devices are considered separately.

The term "exposure" has differing interpretations. It is used here in the sense of the pollutant concentration at the interface between the human and their environment (e.g., in the breathing zone). Personal monitoring, for example, provides a good measure of exposure (exposure concentrations), while microenvironmental and area concentrations tend to provide less reliable estimates, since unmeasured factors (e.g., proximity and movement in and out of the measured environment) reduce the accuracy of exposure estimates. The term "emission factor" is used to refer to the mass of pollutant emitted per mass of fuel consumed or energy released and is abbreviated EF_x, where the subscript "x" is replaced by the pollutant abbreviation (e.g., EF_{CO}). The "emission rate" (ER_x) is the mass of

pollutant emitted per unit time—often used for modeling the impacts of indoor pollutant sources on air quality. The extent to which emissions from a source affect the pollutant concentration in a household microenvironment is determined by such characteristics as ventilation, indoor reactions, and air mixing.

The literature on human exposure and microenvironmental concentrations of airborne pollutants resulting from household kerosene combustion appliances is sparse. Household kerosene heaters have been most scrutinized, but are seldom used in the developing world and have different user–device interactions than lighting and cooking devices. The heating literature is nonetheless useful for identifying pollutant species from kerosene combustion and helps explain the potential influence of fuel quality and device setting.

Selected Pollutants Emitted From Kerosene Appliances

Kerosene, when burned in appliances, emits many potentially health-damaging pollutants. An exhaustive list would include hundreds of compounds. As a frame of reference for interpretation of concentrations reported in the following sections, short summaries of the best-established adverse health effects associated with some of these pollutants are presented. As applicable, guideline levels established by the World Health Organization (WHO) (2006, 2010) are included. WHO also provides interim target levels for some pollutants, which are higher than guideline levels and intended to provide incremental transition steps for situations with high baseline conditions, from which it is difficult to rapidly achieve guideline levels.

Particulate Matter (PM)

There is a strong and consistent body of evidence indicating that exposure to fine particulate matter (PM) increases the risk of respiratory and cardiovascular disease, cancer, and mortality (Krewski et al. 2005; Samet and Krewski 2007; Tsai et al. 2012; Yang 2008). Fine PM originates from both natural and anthropogenic sources and is emitted as a product of incomplete combustion. The median aerodynamic diameter of particles emitted from combustion is typically well below 2.5 μm ($PM_{2.5}$), the particle size below which the majority of PM deposits in the deep lung. The WHO has established $PM_{2.5}$ guideline concentrations for all nonoccupational environments (indoors and outdoors) of 10 $\mu g/m^3$ (annual) and 25 $\mu g/m^3$ (24 h) (WHO 2006). However, acknowledging the difficulty of achieving the annual guideline concentration in low- and middle-income countries, 3 interim targets for achieving the guideline values were provided—5, 25, and 35 μg $PM_{2.5}/m^3$. Combustion sources are known also to emit ultrafine particles (UFP), which have aerodynamic diameters <0.1 μm. There is some evidence from laboratory-based studies to suggest that UFP exhibit higher toxicity per unit mass than larger particles in the respirable size range (e.g., $PM_{2.5}$) (Peters et al. 1997). Due to their small diameters, ultrafine particles typically contribute little mass to traditional mass-based PM measurements but may constitute the predominant contributor to particle count and surface area. Surface chemistry and composition may be determinants of toxicity, but their importance is still uncertain (Stanek et al. 2011). Compared to larger respirable particles, UFP more easily evade lung-clearance mechanisms and enter the lung interstitium and vascular space. The toxicological mechanisms are not well characterized and epidemiological evidence that distinguishes the risk of UFP from other respirable particles is limited. No UFP guideline levels currently exist.

Carbon Monoxide

Carbon monoxide (CO) is a colorless and odorless gas generated by the incomplete combustion of hydrocarbon fuels. When inhaled, CO binds to hemoglobin in red blood cells to form carboxyhemoglobin, reducing oxygen-carrying capacity of the blood and increasing the risk of chronic and acute adverse health effects in adults, children, and fetuses. The effects of acute exposures include dizziness, muscle cramping, loss of consciousness, and, in extreme cases, death. Low-level chronic exposures have been associated with neurodevelopmental effects (Dix-Cooper et al. 2012; Garland and Pearce 1967; Hiramatsu et al. 1996; Long 1997) and cardiovascular diseases (Yang et al. 1998). WHO guideline levels reflect air concentrations at which a normal adult would not exceed 2% carboxy-hemoglobin. Several time-weighted average guideline levels were established to protect against both chronic and acute adverse effects of CO: 100 mg/m^3 (averaging time, 15 min), 35 mg/m^3 (1 h), 10 mg/m^3 (8 h), and 7 mg/m^3 (24 h) (WHO 2010).

Formaldehyde

Formaldehyde (HCHO) is produced by combustion sources as both a gas and adsorbed to particles. Being water soluble, over 90% of gas-phase formaldehyde is absorbed in the upper respiratory tract, unless it is bound to fine particles, which allow for deeper penetration into the lungs. Formaldehyde is classified as a Class 1 carcinogen (IARC 2006), because of sufficient epidemiologic evidence that it increases the risk of nasopharyngeal cancers and myeloid leukemia. Short-term effects include sensory irritation such as eye itching and frequent blinking at levels >0.38 mg/m^3. A guideline concentration of 0.10 mg/m^3 was established by WHO to protect against short- and long-term health effects (WHO 2010).

Polycyclic Aromatic Hydrocarbons (PAH)

Polycyclic aromatic hydrocarbons (PAH) are a class of multiringed compounds present in indoor environments in both the gas and particle phases, depending on molecular size and environmental conditions. PAH are present as constituents of some hydrocarbon fuels, including kerosene, and also generated as products of incomplete combustion. Benzo[a]pyrene (BaP) is commonly used as an indicator of the toxic potency of PAH mixtures and is the only PAH considered a Class 1 carcinogen (IARC 2010), although more than a dozen PAH compounds have been associated with cancer and other adverse health outcomes. There is emerging evidence in animal models that phenanthrene, which is present in many petroleum-based fuels, is a potent immunosuppressant in animals and possibly in humans (Nadeau et al. 2010). However, noncancer risks are not currently considered in WHO guidelines for these compounds. Specific PAH and their relative proportions vary by source. Using BaP as an indicator, WHO has established a unit risk for lung cancer as 8.7×10^{-5} per ng BaP/m^3. WHO also provides a separate guideline for naphthalene ($C_{10}H_8$), a two-ring compound and the simplest PAH, which partitions almost entirely in the gas phase. Naphthalene is considered by IARC to be a Class 2 carcinogen (possibly carcinogenic to humans), with sufficient evidence of carcinogenicity from animal studies but lacking evidence from human populations (IARC 2002). A guideline level of 0.01 mg/m^3 was established (WHO 2010). This is approximately one order of magnitude above background levels (0.001 mg/m^3) in the absence of emission sources.

Sulfur Dioxide

Sulfur dioxide (SO_2) is generated from the sulfur content of fuels during combustion. The majority of sulfur emitted indoors exists as SO_2, but is later converted to secondary sulfur-containing compounds in the atmosphere (e.g., sulfate). Acute effects attributed to SO_2 exposure include changes in pulmonary function and respiratory symptoms, while chronic exposures at levels <20 $\mu g/m^3$ have been associated with increases in all-age mortality and childhood respiratory disease. WHO established a precautionary 24-h indoor guideline level of 20 $\mu g/m^3$, with interim target levels at 50 and 125 $\mu g/m^3$ (WHO 2010). To protect against acute adverse effects, a 10-min guideline level was set at 500 $\mu g/m^3$.

Nitrogen Oxides (NO_x)

Nitrogen oxide (NO) and nitrogen dioxide (NO_2) are formed in reactions between atmospheric nitrogen and oxygen during the combustion process, particularly at higher combustion temperatures. There is strong evidence linking NO_2 with adverse respiratory health effects in adults and children. These effects include inflammation, asthma, and reduced immune defenses that lead to exacerbation of, or susceptibility to, existing or new respiratory infections. WHO 1-h and annual guideline levels for NO_2 are 200 $\mu g/m^3$ and 40 $\mu g/m^3$, respectively (WHO 2006).

Heating

In response to rising electricity prices, portable household kerosene heaters became popular in developed countries in the early 1980s. They could be moved from room to room as needed and were less expensive than central heating. To reduce heat losses, these heaters were often used under low-ventilation conditions, raising concerns about indoor air quality (Leaderer 1982; Leaderer et al. 1986). Despite improvements in design and public education, exposure risk from kerosene heating is still a current topic in several countries, including Japan and Chile (Ruiz et al. 2010). Knowledge of emissions is dominated by results from laboratory-based chamber tests, while the majority of field-based measurements are from monitoring of a single microenvironment to infer exposure to all household inhabitants. Only one study of household exposures that used personal sampling devices was found (Adgate et al. 1992).

Two primary kerosene heater types are evaluated in the literature: convective and radiant heaters. Relatively cleaner burning “dual-stage” heaters are mentioned briefly, but only limited information regarding their emissions was found (Apte et al. 1989; Lionel et al. 1986). Both convective and radiant heater designs use wick capillary action to transfer kerosene to the burner unit. Radiant heaters generate infrared heat, reflected by a polished metal shield; convective heaters warm room air with a fan to force air through a steel tube containing the burner and typically generate more heat and consume more fuel.

Table 1 presents pollutant emission factors and Table 2 microenvironmental and personal monitoring results for criteria health pollutants measured in studies of kerosene heating. All studies were performed in developed countries using local kerosene (typically 1–K grade).

TABLE 1

Emission Factors: Heating Devices

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[a]WMC, well-mixed chamber; HCB, emissions collection hood using carbon balance.
[b]Reported as μg/kg N of NOx. Table emission factors for NO_2 and NO are estimated by assuming all reported N is emitted as the respective pollutant and are therefore upper estimates.
[c]Emission factors from a maltuned convective heater are reported.
[d]Study country/region shown when fuel grade is not reported.

TABLE 2

MicroEnvironmental Concentrations of Selected Pollutants: Heating Devices

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Note. Values in parentheses represent one standard deviation; NS = not specified, *n* = number of households.
Symbols:

[*]Kerosene heaters added approximately 12 μg/m^3 to residential background concentrations of $PM_{2.5}$ and 6 μg/m^3 of SO_42- during the average use period (6.9 h) over the 24-h sampling period.
[^]Converted from mL/m3 using reported temperature and 1atm.
[#]Experiments were conducted in a test house; sample size (*n*) represents the number of tests.
[a]Converted from ppm assuming 25°C, 1 atm pressure.
[b]$n = 74$.

Laboratory chamber studies of kerosene heater emissions began in the early 1980s (Leaderer 1982; Traynor et al. 1983; Yamanaka 1984). Emission factors for fine particulate matter (PM), CO, SO_2, and NO_X (NO_2 and NO), were estimated using a well-mixed-room mass balance model (single-box model). The estimates of EF_{NO2} and EF_{SO2} suggested indoor concentrations of NO_2 and SO_2 could exceed ambient U.S. air quality standards, while emitted CO could pose health risks under conditions of low ventilation and small room volume (Leaderer 1982). The average CO level in mobile homes using kerosene heaters, for example, was 8.5 ± 1.6 mg/m^3—approximately 7 times greater than in homes without kerosene heaters and sometimes exceeding the WHO guideline level. Field measurements confirmed that kerosene heaters increased indoor concentrations of NO_2, SO_2, $PM_{2.5}$, and PM_{10} above ambient levels (Adgate et al. 1992; Leaderer et al. 1999; Mumford et al. 1991), and personal exposures to NO_2 in households using kerosene heaters were five to six times greater than in homes without these heaters (Adgate et al. 1992). Within apartment buildings in Santiago, Chile, a city known for high ambient air pollution levels, kerosene heaters were shown to increase average 24-h $PM_{2.5}$ concentrations by 44 μg/m^3 above background (Ruiz et al. 2010). UFP, SO_2, and NO_2 were also

elevated relative to outdoor levels and levels in houses using compressed natural gas (CNG) or liquid petroleum gas (LPG) for heating (Ruiz et al. 2010). Aggregated UFP concentrations were 163,800 particles/cm^3, approximately 10-fold higher than levels measured in houses with electric heaters and 3-fold higher than in houses with CNG or LPG heating.

Elevated levels of carcinogenic polycyclic aromatic hydrocarbons (PAH) and nitro-PAH have been measured in mobile homes and apartments with kerosene heating devices (Mumford et al. 1991; Ruiz et al. 2010), and identified species were consistent with results from controlled chamber experiments (Girman et al. 1982; Traynor et al. 1990). Using chamber experiments, formaldehyde (HCHO) emission factors ranged from 2 to 36 μg/g kerosene, depending on heater type (Girman et al. 1982; Traynor et al. 1983). An indoor mass-balance model that accounts for loss by surface reactions suggests that these emission rates are unlikely to lead to indoor HCHO concentrations exceeding 0.10 ppm, or 0.12 mg/m^3 at standard conditions, slightly above the WHO guideline level of 0.10 mg/m^3 (Girman et al. 1982).

Numerous studies demonstrated that heater design (radiant or convective) is influential on emissions of pollutants (Apte et al. 1989; Cheng et al. 2001; Leaderer 1982; Lionel et al. 1986; Traynor et al. 1983; 1987; 1990; Yamanaka et al. 1979; Yamanaka 1984; Zhou et al. 2000). EF_{co} were, on average, four- to fivefold higher for radiant heaters than for convective heaters; conversely, EF_{NOx} from convective heaters were two- to fourfold higher than from radiant heaters (Table 1), indicative of the hotter combustion temperatures of convective heaters. SO_2 was not influenced greatly by device type, as emissions are primarily dependent on fuel sulfur content.

The grade of kerosene was also shown to affect pollutant emissions from heaters. In two studies investigating the exposure of soldiers to pollutants generated from kerosene heaters in tents, three grades of kerosene—one commonly available (1–K) and two kerosene-based jet fuels—were tested in both convective and radiant heaters. Indoor concentrations of PM_{10} and $PM_{2.5}$ varied up to eightfold, depending on fuel grade (Zhou et al. 2000). A follow-up study reported that emissions of elemental carbon (EC), organic carbon (OC), SO_4^{2-}, NH^{4+}, CO, SO_2, and NO were also influenced (Cheng et al. 2001).

Most of the investigations into the exposure risk of kerosene heaters were performed more than 20 years ago, when use of such heaters was more widespread. This makes current interpretation difficult, since the extent to which those heaters were replaced by improved designs is not known. However, more recent studies (Cheng et al. 2001; Ruiz et al. 2010; Zhou et al. 2000) suggest that improvements in emissions have not been significant. There is strong laboratory- and field-based evidence suggesting that fine PM, NO_2, and SO_2 are generated at rates that could exceed WHO guideline concentrations in households. Given the high combustion temperatures, UFP and PAH formation would also be expected to be high. However this has been confirmed by only a handful of field measurements (Mumford et al. 1991; Ruiz et al. 2010).

Lighting

The few published studies that document pollutant emissions and resulting air quality impacts of kerosene lighting are summarized in Tables 3 and 4. All studies were performed under controlled conditions intended to simulate real-world applications. To date, no study has reported personal

exposure or environmental concentrations from field-based measurements, partly because it is difficult to distinguish the contribution of lighting from that of other common and coemitting household sources, such as cooking or smoking.

TABLE 3

Emission Factors: Lighting

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Note. No studies report SO_2 emissions; NS = not specified. No emissions data found for pressure type lanterns.

^Emission factor estimated using reported emission rates and device specific average fuel consumption rates reported within the same study or by Apple et al. 2010 (simple wick = 14.9 kg h^{-1}, small hurricane = 14.4 kg h^{-1})

[a]Well-mixed room (WMR), well-mixed chamber (WMC), emission collection hood with carbon balance (HCB).

TABLE 4

Microenvironmental PM Concentrations: Lighting

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Note. No data for CO, NO_2, and SO_2 concentrations resulting from kerosene lighting were found. All lighting studies in table were conducted under mock exposure scenarios lasting between 1 and 2 h; thus, values in the table represent average concentrations during lamp usage, not daily averages.

*Study country/region shown when fuel grade is not reported. *N* = number of devices, *n* = tests per device.

[a]Average of steady-state concentrations.

[b]Ranges and values from Apple et al. (2010) estimated from figures.

Household kerosene lighting devices are of three types: “simple wick” lamps, hurricane lamps, and pressure lamps. The “simple wick” (synonyms include kerosene candles, taboodas, tuki) is the cheapest, most basic design, typically constructed locally from recycled metal or glass containers. Fuel is burned via capillary action of a wick, usually made of cloth or rope. The hurricane lantern also uses capillary action to transport fuel, but with a standard rope wick that can be adjusted for height. The flame is shrouded within a glass enclosure and more luminescent than a simple wick lantern, but typically consumes more fuel (simple wick = 14.9 ± 6.6 g fuel/h; small hurricane = 14.4 ± 2.6 g fuel/h; large hurricane = 20.5 ± 6 g fuel/h, as calculated by Apple et al. [2010]). In pressure lamps (primus, mantle lamps), fuel is transferred via pressurization with a manual pump or heat, aerosolized in a “jet”

of fuel, and burned, heating a metal (e.g., thorium) oxide-coated mantle that generates a bright white light. Pressure lamps produce substantial amounts of light, but also have the highest fuel consumption rate (74.1 ± 15.6 g/h) and capital cost, limiting their use in households without electricity.

In a chamber study, total suspended particles (PM_{10}), CO, methane, and total hydrocarbons (THC) emissions from a hurricane lantern were calculated using a mass-balance model. Emission factors of 3.2 and 9 mg PM_{10}/g kerosene under "normal" and "tall" wick settings (Fan et al. 2001), respectively, were similar to those measured previously by Schare et al. (1995) from a similar lantern design (Table 3). EF_{PM} from a simple wick lamp was significantly higher than from a hurricane lamp measured in the same study, generating an average of 8 mg PM_{TSP}/g kerosene under "normal" conditions and 32 mg PM_{TSP}/g with a "high" wick height (Schare et al. 1995). However, since mass concentrations were obtained from an uncalibrated light-scattering monitor, absolute values should be interpreted with caution. Measurements of size-resolved particle count indicated that almost all emitted particles from the hurricane lamp were <0.4 μm and no particles were observed above 1.0 μm in diameter (Fan et al. 2001). Increased wick height was associated with a higher PM count, mostly in particles <0.5 μm. The EF_{CO} was low at 0.52 ± 0.19 mg CO/g kerosene.

Fan and Zhang (2001) then modeled indoor concentrations using emission measurements and the assumption of well-mixed room conditions. Model results indicated that hurricane lamps operated indoors could easily lead to exceedances of U.S. National Air Quality Standards for $PM_{2.5}$. It could be inferred that simple wick lamps may also lead to exceedances, since their emission rates appear to be higher (Schare et al. 1995). From model results, it appears that either lamp design might also elevate indoor $PM_{2.5}$ concentrations above WHO guideline and interim targets. Using lamp oil, often a deodorized kerosene or lighter hydrocarbon blend (e.g., Flash 142 solvent), the hurricane lamp EF_{PM10} decreased by approximately one order of magnitude (0.35 ± 0.06 mg_{PM10}/g) when operated normally, but less so when operated with a high wick (7.3 ± 1.2 mg_{PM10}/g), suggesting that more refined fuel grades (e.g., with fewer aromatics) may significantly decrease emissions from lighting appliances. No measurements for pressure lamps were found in the literature.

Two studies quantified the effects of lighting on indoor air quality, both using mock setups to represent real-world conditions. Simple and hurricane lamps operated at "normal" wick height resulted in steady-state indoor PM concentrations of 2500 ± 900 and 6700 ± 1800 μg/m^3, respectively (Schare et al. 1995). Operated at high wick settings, these levels rose to 5000 and 21,800 μg/m^3, respectively. Size-resolved PM concentrations varying from 0.02 to 1 μm were measured during lamp operation inside a mock roadside kiosk, similar to those used in many African countries. Results indicated that the majority of the particles counted were in the inhalable size range (0.3–10 μm), leading to calculated $PM_{2.5}$ and PM_{10} mass concentrations of 10 to 400 μg/m^3 and 20 to 10,000 μg/m^3, respectively (Apple et al. 2010). $PM_{2.5}$ and PM_{10} mass concentrations were increased with breathing zone elevation, but a marked effect horizontally from the source (same elevation) was not reported. The increase in concentration with elevation may be due to the thermal plume generated by some lamps. Wick height and device type (e.g., simple wick or hurricane) were found to be important determinants of indoor PM concentrations in both studies. To our knowledge, no study has measured SO_2 from lighting appliances. Kerosene fuel is known to contain low levels of sulfur, usually <0.10%, or <0.04% for 1–K grade. These levels of sulfur would be expected to generate low indoor SO_2 emission rates and would not be likely to exceed WHO guideline concentrations. Higher kerosene sulfur content is possible in developing-country settings, however, and fuel mixing (e.g., with diesel) might also increase exposure risk.

Kerosene lighting devices were used in laboratory settings to generate particles (referred commonly in the literature as “soot”) for various analytical purposes. In some cases, particles were analyzed to determine composition, including elemental (EC) and organic carbon (OC). Elemental carbon is sometimes treated as if it were black carbon (BC), despite recognized limitations resulting from differences in the way each are measured (Bond et al. 2005). Both EC and BC have served as indicators for PM originating from diesel sources in cities; however, there are presently few data that distinguish health effects attributed to EC or BC from those of traditional mass-based PM measures (Smith et al. 2009). Using a thermal optical measurement method, EC was found to compose the majority (approximately 80%) of aerosol carbon (Chen et al. 2007) taken from an unspecified type of lamp burning kerosene. In another study, light absorbance by particles from a poorly trimmed kerosene lamp, similar to a hurricane design, was measured using a photoacoustic instrument. Results indicated that emitted particles were highly light-absorbing (single scattering albedo = 0.20 at 532 nm), suggesting a high proportion of black carbon (Arnott et al. 2000). Optical properties of kerosene particles from the lamp were similar to those of diesel soot. These studies provide some insight into the potential characteristics of particles emitted in kerosene-using houses.

One study provided a qualitative indicator of exposure using measurements of particulate loading of alveolar macrophages. Samples from 57 Malawian male and female subjects who reported using various forms of lighting and cooking devices were analyzed with digital image analysis software to quantify the particles within the alveolar macrophages (AM) (Fullerton et al. 2009). Higher loading was associated with reported use of kerosene wick lamps, compared to use of electric lighting. Due to the method used to quantify PM loading, it is likely that investigators were able to count only darker colored particles on the film, making this more of a quantification of black carbon loading. Analysis of variance showed a significant difference across lighting device types—simple-wick lamps were associated with the highest median levels, followed by candles, hurricane lamps, and electricity. Caution is needed in interpreting these results, as no adjustment was made for solid fuel cooking, which was also associated with greater AM particulate loading.

The significant quantities of PM generated by some lighting appliances, coupled with the user–device interaction characteristics (e.g., studying, room illumination), suggest that kerosene lighting could be a significant source of household exposure. Unfortunately, few studies investigated this issue in depth, and only a few pollutants have been characterized. No studies to date have quantified exposure or microenvironmental pollutant concentrations in real-world settings. Proximity is likely to be an important exposure component not captured in area-based measurements. There appear to be large differences in emission and exposure potentials across lamp designs (e.g., wick or pressure) and operator settings, which are likely associated with usage purpose (e.g., room lighting versus close proximity reading). This information could be captured easily with a simple questionnaire accompanying household monitoring sessions. Assessing fuel quality inexpensively would be more challenging, but potentially important.

Cooking

The literature on household cooking in developing countries has focused on solid fuels (e.g., wood, dung, charcoal), as they are the most prevalent primary household fuels. Furthermore, kerosene is often regarded as a “step up the energy ladder” from solid cooking fuels (Smith et al. 1994), and often becomes more prominent as a primary or secondary cooking fuel as countries develop and urbanize. This has been observed, for example, in India, where, kerosene was reported as the primary cooking fuel in 8% of urban households and in <1% of rural households in 2005 (NSSO 2007). However, it is

often used as a backup fuel in urban areas for when LPG is unavailable and in rural areas for when biomass fuel is unavailable (Rao 2012). Nationally representative cooking fuel assessments conducted by Demographic and Health Surveys (http://www.measuredhs.com) show a similar urban/rural usage pattern for other countries: Kenya (2008), 26.9%/1.5%; Nigeria (2008), 51.6/11.3%; Indonesia (2007), 54.6/18.7%; Nepal (2006), 15.8%/1%; Peru (2000), 25.7%/4%; and Honduras (2005), 10.4%/1%.

Unlike studies on heating and lighting, several studies on cooking-related exposure to household air pollution have attempted to account for user behavior and time–activity patterns. With the exception of one study in Mozambique, Africa (Ellegard 1996), all field studies reported here took place in India, although kerosene is used for cooking in many developing countries. There are two main categories of kerosene cooking stoves described in the literature: "wick" stoves, with 6–10 wicks (but as many as 20–30), and "pressure" stoves, which pressurize the kerosene manually or using heat, and burn the aerosolized fuel.

Pollutant emission factors for PM, CO, NO_2, and SO_2 from kerosene cooking appliances are presented in Table 5. Few measurements of kerosene cookstove emissions exist, especially in comparison to solid fuel stoves, and all are from laboratory test cycles, rather than emissions during actual household use (Habib et al. 2008; Smith et al. 2000; Zhang et al. 2000). Table 6 presents measured microenvironmental and personal exposure concentrations of the same pollutants over various exposure sampling durations. PM concentrations in kitchens ranged from 300 to 750 μg/m^3. This variation may be due to variability in room sizes, ventilation, and contributions of outdoor sources, which were substantial in some studies (Raiyani et al. 1993; Saksena et al. 2003). Studies reported in Table 6 employed a variety of particle size cut-points, which adds some complexity when comparing across studies. Size-resolved measures of PM from one study showed that 88% of the PM mass in emissions from kerosene stoves was attributable to particles with aerodynamic diameter <9 μm (Raiyani et al. 1993).

TABLE 5

Emission Factors: Cooking Stoves

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Note. Emission rates could not be estimated due to lack of information on fuel burn rates for stoves reported. Smith et al. (2000) reports using a 10-wick stove, Oahn et al. (2002) report fuel consumption for 8-wick stove at 0.104–0.12 kg h^{-1}, and Habib et al. (2008) report 0.2 kg h^{-1} for an unspecified stove type. All reported studies used a variation of the water boiling test (Bailis et al. 2004). *n*, Number of measurements.

*Study country/region shown when fuel grade is not reported.
[a]Emissions collection hood with carbon balance (HCB).

TABLE 6

Microenvironmental and Personal Exposure Concentrations of Selected Pollutants: Cookstoves

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Note. Values in table represent the arithmetic mean and standard deviation (in parentheses) during cooking. No study reported kerosene grade. NS, not specified. Symbols:

^Original values reported as geometric mean and geometric standard deviation. Presented are estimates of the arithmetic mean and standard deviation assuming a lognormal distribution of concentrations.
*Testing was conducted only during meal preparations.
[a]Converted from ppm assuming 25°C, 1 atm pressure.
[b]Reports concentration ranges only.
[c]All experiments conducted in a single test house.

With the exception of one study (Raiyani et al. 1993), average kitchen CO concentrations were consistently below 5–6 mg/m^3. Since exposure-averaging times are on the order of hours (e.g., cooking events) for all studies reported, it is possible that short-term guideline exposure limits could be exceeded during cooking periods.

Only two studies reported SO_2 concentrations in houses. Although average levels during cooking reported by Raiyani et al. (1993) are high (121 μg/m^3), they were only twice the levels found in LPG-using households (65 μg/m^3) and lower than those in households using solid fuels (wood, dung, coal), suggesting a strong contribution of outdoor sources. This trend was seen for other pollutants, including CO, reported in the study. Kandpal et al. (1995) reported kitchen SO_2 concentrations of 48 and 74 μg/m^3 at squatting and standing heights, respectively, during kerosene stove use. To our knowledge, there are no reported measurements of the sulfur content in kerosene fuels used in houses. However, for kerosene with sulfur levels similar to those found in 1–K grade kerosene (<0.04% sulfur by weight), it seems unlikely that kerosene stoves alone may generate concentrations of SO_2 in exceedance of WHO guidelines in most indoor environments.

Simultaneous measurement of personal exposure and microenvironmental pollutant concentrations is uncommon in the literature, but can provide insights into cooking habits and exposure characteristics. In a study by Saksena et al. (2003), average personal exposure to RSP (respirable suspended particles) in Indian households using kerosene was 800–900 μg/m^3, depending on kitchen design and background concentration (Saksena et al. 2003). From the methods, it is clear that RSP is PM_5 in this study. These concentrations were higher than those measured in the kitchen during the same sampling period (750–800 μg/m^3). Furthermore, although wood users experienced twice the average kitchen concentration of PM_5 as kerosene users, average personal exposure concentrations were similar in the two groups. An earlier study in India found the average personal exposure concentration of PM_{10} to be approximately 10% higher than corresponding kitchen concentrations, while the trend was reversed (30–60% lower for personal exposure) with solid fuels (agricultural residues, wood, and biomass) (Smith et al. 1994).

Saksena et al. (2003) suggested three distinguishing characteristics of kerosene users to explain why personal/microenvironment concentration ratios differed from solid fuel users in their study: Kerosene users: (1) cook for longer durations, (2) spend more time in close proximity to the stove, and (3) are more likely to cook indoors.

Measurements in Indian kitchens showed that households using kerosene stoves had higher particle surface area concentrations relative to coal-, LPG-, and biogas-using households; results for wood were not reported (Sahu et al. 2011). These high surface area concentrations were attributed to the majority of particles being in the ultrafine size region, which also resulted in low mass concentrations for kerosene relative to other fuels. Sahu et al. (2011) suggested that this may be a particular characteristic of kerosene stove combustion, with important health implications. These results also provide evidence that most particles emitted by kerosene stoves are less than 2.5 μm. The implication is that the particle size cutoff (e.g., TSP, $PM_{2.5}$, PM_{10}) used in sample collection should exert little effect on the resulting mass concentrations (Table 5).

Several studies measured noncriteria air pollutants from kerosene stoves, PAH and volatile organic compounds (VOC). Laboratory emissions from Thai kerosene stoves identified 17 PAH, of which 11 induced genotoxicity by the Ames test (Oanh et al. 2002). Total PAH emission factors were 67 mg/kg for all 17 measured compounds and 28 mg/kg for genotoxic PAH. The aggregated emission factor for the 17 PAH was similar to that from a wood-stove (66 mg/kg) but less than from sawdust briquettes (260 mg/kg) assessed in the same study. The genotoxic PAH emission factor was greater than for both wood (22 mg/kg) and sawdust briquettes (22 mg/kg). Kerosene has a higher energy density than wood and generally burns more efficiently, requiring less fuel mass to complete the same cooking task and therefore producing a lower mass of total PAH emissions. Simultaneous indoor and outdoor concentration measurements of kerosene-using houses in India showed indoor/outdoor (I/O) ratios for 12 measured PAH as high as 10.5 (naphthalene) (Pandit et al. 2001; Raiyani et al. 1993).

Molar emission ratios for 59 nonmethane hydrocarbons (NMHC) from three laboratory-tested kerosene stove designs (wick, pressure, and gravity) were several fold higher than those from wood or charcoal for several NMHC, including cyclohexane (~10×), heptane (~80×), toluene (~2×), 1,3,5-trimethylbenzene (~91×), and *n*- and *p*-xylene (~6×). The kerosene wick stove generated the highest molar emission ratios for nearly all 59 measured compounds (Zhang et al. 1996). Elevated levels of benzene were measured in a sample of five households with kerosene stoves in Mumbai, India (103.4 $\mu g/m^3$; I/O ratio = 3.3) (Pandit et al. 2001; Srivastava et al. 2000). Six other VOC measured in the same studies, including hexane and toluene, had average I/O ratios >1.

Available measurements of kitchen and personal exposure concentrations suggest that kerosene-fueled stoves elevate indoor respirable PM concentrations above WHO guideline and interim targets, while CO may pose risks under some conditions. Substantial quantities of hydrocarbon species, possibly from uncombusted fuel, may be a differentiating exposure characteristic for kerosene among household fuels (Zhang et al. 1996), but this requires field-based measurement confirmation. The generation of particles with a relatively small size distribution, resulting in a greater surface area for chemical adsorption, may also be a distinguishing characteristic (Sahu et al. 2011). There is also an indication that the shift from solid to liquid fuels may influence cooking behaviors, such that reductions in exposure due to lower emissions are negated by the behavioral changes that increase the exposure concentrations (e.g., proximity, time cooking, indoors with less ventilation) (Saksena et al. 2003; Smith

et al. 1994). That there are a wide variety of kerosene stove types in use, but few available pollutant emissions data for kerosene stoves in general, underlines the need for more detailed assessments that cover the range of kerosene stove models used in developing-country households.

TOXICOLOGY

The use of kerosene and its derivatives in the commercial and government sectors prompted several toxicological reviews and risk assessments related to its use in developed countries. The vast majority of information on kerosene toxicity is from occupational exposures or animal models where kerosene-based jet fuels (kerosene with additives) were used. While there is large body of evidence characterizing the toxicity of fuel vapor and aerosol inhalation and dermal exposure to uncombusted fuel, less is known of the toxicity of the combustion product mixture (although a wealth of information is available on the individual pollutant toxicities). As highlighted in the exposure assessment section, this is complicated by the fact that the nature and concentrations of pollutants emitted may be strongly influenced by the source of combustion (e.g., stoves, lamps, heaters).

Given the existence of comprehensive reviews of the toxicity of kerosene fuel, this section is limited to summarizing their findings, while highlighting other studies more directly related to household appliances—the main focus of this review. Although information on the effects of kerosene-based jet fuels is presented, because of the addition of performance additives, they are not compositionally equivalent to kerosene available to consumers. The extent to which these additives alter the toxicological properties of kerosene is currently unclear (Ritchie et al. 2003; American Petroleum Institute 2010).

Kerosene Fuel Vapor and Aerosols

There is an extensive body of literature on the toxicity of liquid kerosene (uncombusted) and its derivatives due to its use as a motive fuel. Excluding poisonings, the extent to which kerosene aerosol or vapor contributes to daily exposures in households is unclear but contribution could reasonably be assumed to occur at least at a low level. For example, in addition to exposure to vapors released to the room from the fuel appliance or the fuel storage container, it is likely that uncombusted fuel components are present in the pollutant plume or adsorbed to the surface of emitted particles. Kerosene fuel is a mixture of hundreds of chemical compounds, several with known adverse health risks. Although present in varying quantities, depending on fuel source and quality, naphthalene, benzene, *n*-hexane, toluene, BaP, and xylene are among several such chemicals present in residential kerosene fuels. The adverse health effects of individual chemical constituents as vapors were reviewed comprehensively by Ritchie et al. (2003). The majority of available literature on kerosene toxicity focuses on the dermal exposure route, and usually in the occupational context (e.g., aviation industry). Several animal studies investigated the toxicity of inhaled aerosolized kerosene, although this route is often regarded as secondary in occupational settings. Therefore, it has received less attention.

Using results provided by the American Petroleum Institute, the U.S. Environmental Protection Agency (EPA) performed a screening-level hazard characterization for kerosene/jet fuel (U.S. EPA 2011). Sponsored chemicals for the assessment included kerosene and hydrosulfurized kerosene, while aviation kerosene blends were considered as “supporting chemicals” due to their compositional similarities to the sponsored chemicals. The assessment concluded that the acute oral and dermal toxicity of kerosene was low, while acute inhalation toxicity posed moderate risks. The assessment cites several studies in support of their conclusions. Repeated dermal exposure to kerosene for 4 wk

resulted in a decreased red blood cell count in male rabbits and increased spleen weights in females, at dosages of 200 mg/kg body weight (bw)/d (lowest dose tested). A no-observed-adverse-effect level (NOAEL) for repeated dermal exposure was determined to be 330 mg/kg-bw/d. For inhalation, no observed effects were seen in rats exposed for 4 wk to 0.024 mg/L, the highest concentration tested. Repeated dermal exposure to hydrosulfurized kerosene in rats lasting from 14 d premating through d 20 of gestation resulted in a NOAEL for reproductive toxicity of 494 mg/kg/d (highest dose tested) in both males and females, although body weight was decreased in males at this exposure level. No indication of maternal or developmental toxicity was observed at 494 mg/kg/d. Inhalation studies on rats found no signs of maternal or developmental toxicity at 364 ppm/d, regarded as the no-observed-adverse-effect concentration (NOAEC).

A review by Ritchie et al. (2003) considered a wider range of toxicological effects resulting from exposure to kerosene-based jet fuels and kerosene (Ritchie et al. 2003). As with the U.S. EPA report, the review was occupationally focused, drawing largely from studies of kerosene-based jet fuels. Repeated occupational inhalation exposures to jet fuel and kerosene were determined to result in changes to brainstem/cerebellar systems and complex neurobehavioral performance capacity. Repeated exposures in humans and animals were associated with hematological changes, including reductions in red and white blood cell counts. Acute or long-term exposure to aerosolized kerosene-based jet fuel was associated with persisting damage to the pulmonary system.

Other studies, not included in the review, provide evidence of acute airway activity effects. Inhalation of aerosolized kerosene (20–35 mg/L air) for 4–20 min was associated with bronchoconstriction, hyperirritability, and indicators of inflammatory response (Casacó et al. 1985a; 1985b; Mesa et al. 1988a; 1988b; Rodriguez de la Vega et al. 1990). Human and animal studies indicated that acute or chronic dermal exposure resulted in damage to the dermal barrier, irritation, and tumorigenesis. Hepatic damage was also found in both animal and human studies, ranging from changes in hepatic metabolism to persisting liver histopathology following repeated kerosene exposures. Based on numerous animal studies, there is evidence that exposure to kerosene-based jet fuels or unmodified kerosene results in immunosuppression. Polycyclic aromatic hydrocarbons are present in all kerosene fuels and were also shown to mediate immunosuppression (Nadeau et al. 2010).

Combustion Products

Both kerosene stoves and lamps can emit substantial quantities of fine PM even during normal operation. Both size and composition play a role in determining the toxicological risk of inhaled particles. In general, the median diameter of particles emitted from combustion is well below 2.5 μm, ensuring the majority will deposit in the deep lung (Apple et al. 2010; Sahu et al. 2011). The influence of combustion source, which may alter the extent to which chemicals are adsorbed to the surface of particles, and hence their toxicity, has been less studied. The toxicity of soot generated from residential kerosene combustion sources has been investigated. Arif et al. (1991; 1993) exposed rats intratracheally to kerosene soot. Single exposures of 5 mg resulted in effects on the respiratory tract, including increased levels of AM and hydrogen peroxide generation (Arif et al. 1993). Similar effects were also observed in rats given a single 0.05-ml dose of kerosene fuel intratracheally. Dogs exposed in a room to kerosene emissions, generated by a stove for 15 min/d for 21 d, showed mild to moderate edema, compensatory emphysema, focal areas of collapse, and pneumonitis. Many of these effects were attributed to oxidative stress and tissue inflammation resulting from the effects of PAH, reactive oxygenated species, and sulfur compounds in kerosene smoke. In addition to pulmonary effects, Rai et al. (1980) also reported a thickening of aortic walls. A similar thickening of aortic walls, as well as

development of aortic plaques and valvular changes, was later observed in guinea pigs exposed to kerosene cookstove emissions after exposure durations similar to those in the study by Rai et al. noted by (Noa et al. 1987). On histopathologic examination, both exposed groups showed changes characteristic of early atherosclerotic lesions, not observed in the control animals. Exposed groups also showed significant elevation in total serum cholesterol and decreases in HDL cholesterol relative to control animals. Unfortunately, neither study reported measurements of pollutant concentrations, but exposure levels were intended to be representative of levels found in household kitchens during cooking events.

Kerosene Coexposures

In animal studies, coexposure to kerosene soot or fuel and asbestos resulted in apparently synergistic alterations of the normal metabolic processes of the lung. Arif et al. (1994) reported that rats given a single intratracheal dose of kerosene soot (5 mg) and chrysotile asbestos (5 mg) showed inhibition of drug-metabolizing enzymes critical in the clearance mechanism of the lung. The joint effect was greater than that measured for exposures to soot or asbestos alone. A follow-up study found that rats given intratracheal doses of either kerosene fuel (5 ml) or soot (5 mg) with asbestos exhibited alterations to biochemical parameters indicative of tissue inflammation and injury to alveolar macrophages (Arif et al. 1997). Both kerosene soot and chrysotile asbestos were found to be genotoxic on an approximately additive scale when hamster embryo fibroblasts were exposed (Lohani et al. 2000). Coexposure effects were also demonstrated with dermal exposures. LaDow et al. (2011) recently found that coexposure to kerosene and BaP for mouse skin increased the uptake of BaP by skin and internal organs. These findings suggest that kerosene aerosol/vapor, and perhaps soot, may modify the risk of other health-damaging pollutants that are coemitted or present from other sources.

Accidental Poisonings

Poisonings from ingestion of kerosene, particularly in children, are unfortunately common in developing countries. The problem is exacerbated by the common practice of insecure storage of small amounts of kerosene in soft-drink bottles without safety closures, often because purchasers of kerosene can only afford to buy a small amount at a time and provide their own containers for suppliers to fill. Kerosene poisoning has been well summarized previously (Tshiamo 2009), and only key points are briefly highlighted here. In addition to inadequate storage and packaging of kerosene, risk factors for kerosene ingestion include age, season, poverty, and living in rural areas. Young children have relatively undeveloped senses of taste and smell and may mistake kerosene for familiar drinks, such as water and some sodas. The summer heat increases consumption of fluids, and kerosene lamps are more common in rural and poor households.

The low viscosity and surface tension of kerosene allow it to be aspirated into the lungs of people who have ingested it, provoking a chemical pneumonitis, which can be fatal if untreated. Fortunately, most kerosene ingestions are nonfatal. Nevertheless, kerosene poisonings make up a significant portion of total poisoning incidents each year, particularly in developing countries. For example, a study of 120 unintentional childhood poisoning cases in Pakistan produced a population-attributable risk of 40% (95% confidence interval [CI] 38–42%) for storage of kerosene and petroleum in soft-drink bottles (Ahmed et al. 2011). Studies emphasize the preventability of such poisonings by a few measures: child-proof caps, avoiding decanting into drink bottles, colorizing the liquid, storage out of reach of children, and education programs (Tshiamo 2009).

BURNS AND FIRES

Relative to gasoline or LPG, kerosene has a low vapor pressure (high flashpoint) at ambient conditions, reducing the risk of explosion from volatilization into indoor environments. The viscosity is also low enough that kerosene will easily wick up absorbent materials. Nonetheless, kerosene appliances are responsible for many fires and burns, with a variety of contributing factors. As the problem of kerosene-related fires and burns has been recently reviewed (Peck et al. 2008), only the main features are summarized here.

Both kerosene stoves and lamps have led to major fires and serious, often fatal, burns. Exacerbating the problem, these devices are often used in confined spaces in poor, crowded communities, such as slums, where dwellings are packed together and often made of wood and cardboard. Kerosene stoves are often placed on the floor and easily knocked over, particularly by children, causing kerosene spillage and a rapidly spreading fire. Women, who predominantly do the cooking, often wear loose-fitting flammable clothing. A gust of air may suddenly increase wick flame size, igniting clothing.

Many devices, particularly those with wicks, are poorly constructed and leak. The leakage may ignite. Explosions can result from the mixing of gasoline with kerosene. This can arise, for example, from use of the same container for the two fuels. Even a small amount of gasoline, with its much lower flash-point and higher vapor pressure, mixed with kerosene can lead to kerosene devices exploding. Another common cause of fires and explosions is adding more kerosene fuel to a device when it is lit.

Manufactured pressure lamps suffer from blocking of nozzles by soot. This may lead to attempts to increase flame size by pumping the fuel to higher pressure. Subsequent attempts to clear the nozzle with a pin or wire can cause a sudden high-pressure release of an air–fuel mixture, with resulting explosion.

EPIDEMIOLOGY STUDIES INVOLVING EXPOSURE TO KEROSENE COMBUSTION PRODUCTS

This section examines the epidemiologic studies of household kerosene-burning appliances in relation to adverse health effects. It separately considers studies of cancer etiology and studies of nonmalignant effects. The focus is on studies that reported results for kerosene use as a distinct category. Studies that, for the purposes of data analysis or results presentation, combined kerosene-burning devices with devices using other fuels (e.g., electricity, gas, solid fuel) into one category have been disregarded. Results are summarized in Table 7.

TABLE 7

Summary of Results for Epidemiologic Studies Investigating Kerosene Use as a Possible Risk Factor for Health Effects

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Note. CC, case-control study; CS, cross-sectional study; $FEF_{25–75}$, forced expiratory flow 25–75%; FEV_1 forced expiratory volume in 1 s; FVC, forced vital capacity; NA, not applicable;

NS, not specified; OR, odds ratio; PEFR, peak expiratory flow rate. Symbols:

†Calculated from data in article.
‡Parentheses contain 95% confidence intervals for relative risk estimates (including odds ratios).
#Number of cases/number of controls (for case-control studies).

Cancer

A few studies examined kerosene use as a possible cancer risk factor. The International Agency for Research on Cancer (IARC) concluded that there was inadequate evidence for kerosene as a human carcinogen, and limited evidence for its carcinogenicity in experimental animals (IARC 1989). It was generally not possible to distinguish between any direct effects of kerosene and those of its combustion products. The following is a brief review of the available epidemiologic studies.

Respiratory Cancer

In a survey of 314 Hong Kong families, 36% used kerosene stoves "habitually" (daily use for more than 2 yr) (Leung 1977). Of 44 female lung cancer cases, 40 (91%) were "habitual" kerosene stove users. From the published data, an unadjusted odds ratio (OR) of 17.8 (95% CI 6.2–70) for having a kerosene stove can be calculated. Leung (1977) concluded that female lung cancer was associated with kerosene stove use.

In a case-control study of bronchial cancer in Hong Kong, Chan et al. (1979) examined the relationship of kerosene used for cooking in 189 female cancer cases and 189 female controls from orthopedic wards. The authors reported "no significant difference" between nonsmoking cases and controls for an unadjusted analysis. We calculated unadjusted OR from data presented in Table IX of the paper. For nonsmokers the OR for ever cooking with kerosene was 1.79 (95% CI: 0.96–3.36) and for all women, the OR was 1.51 (95% CI: 0.97–2.35).

Koo et al. (1984) conducted a case-control study in Hong Kong of 200 female lung cancer patients and 200 controls matched by age, housing type, and district. Of the cases, 91.5% had ever used kerosene for cooking, compared with 93.5% of controls. However, cases had cooked with kerosene 2–4 yr longer than controls. OR were elevated only for more than 30 yr of kerosene use. Evidence indicated that data provided minimal evidence for a role of kerosene in lung cancer.

Salivary-Gland Cancer

To identify possible risk factors for salivary-gland cancer, Zheng et al. (1996) conducted a population-based case-control study of 41 cancer cases and 414 controls, in Shanghai, China. Risk factors identified included use of kerosene cooking fuel (OR = 3, 95% CI: 1.4–6.8). This lone study is difficult to interpret. It had few cases and many exposures were examined.

Nonmalignant Effects

Studies are divided into those that examined (1) respiratory symptoms and spirometry values, (2) asthma, (3) respiratory infections, and (4) effects on the eye.

Respiratory Symptoms and/or Spirometry

Azizi and Henry (1990) studied 1600 school children aged 7–12 yr in Kuala Lumpur, Malaysia. In multivariate regression, after adjusting for a number of covariates, including asthma, both wood-burning and kerosene-burning stoves, as well as sharing a bedroom with an adult smoker, were associated with decrements in spirometric parameters. For household kerosene stove use, mean percent predicted values, all statistically significant, were forced vital capacity (FVC), 95.8%; forced expiratory volume in 1 s (FEV_1), 95.7%; forced expiratory flow between 25% and 75% of forced vital capacity ($FEF_{25–75}$), 96.8%; and peak expiratory flow rate (PEFR), 97.2%. The authors concluded that exposure to wood- or kerosene-burning stoves affected lung function adversely. However, inclusion of asthma in the same model may have led to underestimates of the effects, as asthma is on the causal pathway for spirometric results. In a subsequent publication, intended to define normal spirometric parameters for ethnic groups in Malaysia and restricted to 1098 of these children without respiratory symptoms, decrements of 3–8% in lung function parameters were reported for children whose families cooked with kerosene (Azizi et al. 1994). The exclusion of children with respiratory symptoms potentially led to an underestimate of any kerosene-related effect on lung function. Azizi et al. (1991) also reported on respiratory symptoms in these Kuala Lumpur children and found no association between having either wood stoves or kerosene stoves and any of chronic cough or phlegm, persistent wheeze, asthma, or chest illness.

To investigate the relationship between household fuel type and lung function, 3991 women were recruited from villages near Chandigarh, India (Behera et al. 1994). After excluding smokers and women with respiratory symptoms or other concomitant diseases, 3318 women remained. Women were categorized by cooking fuel used: biomass, LPG, kerosene, and “mixed.” For all 4 groups, mean FVC was in the range of 73–77% of expected, although the biomass group had the lowest (73.4%) and kerosene the highest (76.7%). Mean PEFR values were 74–76% of expected for all 4 groups and FEV_1 was in the range of 90–94%, with the biomass group again having the lowest values. It is difficult to draw conclusions from these results, for several reasons. First, there was no unexposed comparison group, as all the women cooked with a combusting fuel. Second, excluding women with respiratory symptoms may have introduced a selection bias, making the groups more similar. Third, data were not adjusted for possible confounding factors. Mitigating the latter concern, all the women came from the lower or lower-middle classes.

Behera et al. (1998) also carried out spirometry on 200 school children in Chandigarh. The children were divided into four categories, based on cooking fuel used at home: biomass, LPG, kerosene, and mixed fuels. Predicted (normal) values were available only for PEFR. For boys, PEFR values as percent predicted were lower for kerosene (67.6%) and biomass (67.3%) than for LPG (75.2%) and mixed fuels (72.6%). For girls, PEFR was highest for kerosene (72.3%), relative to biomass (67.4%),

LPG (70.3%), and mixed fuels (68.3%). As children with respiratory illnesses were excluded from the participant group, this study is hard to interpret. No potential confounding factors were fully taken into account.

In Lucknow, India, Awasthi et al. (1996) carried out a cross-sectional survey involving 650 children less than 5 yr of age. The outcome measure was observation on the day of the interview of one or more of the following without the presence of exanthematous rash: runny nose, cough, sore throat, breathlessness, and stridor or wheeze. Cooking fuel exposures were the fuel(s) used by the family in the last week. In a logistic regression model the only fuel that was associated with symptoms was dung cakes (OR = 2.69, 95% CI: 1.37–5.31). Coal (OR = 0.61), wood (OR = 0.96), and kerosene (OR = 0.87) were not associated with symptoms.

A study of the association between winter respiratory symptoms and home heating sources was carried out in 890 infants, aged 3–5 mo, born in Connecticut and Virginia hospitals in 1993–1996 (Triche et al. 2002). Mothers recorded wheeze and cough in their children. Kerosene heater use was associated with about a 7% elevation in episodes of cough for each 8–h increase in use, but there was no rise in total days of cough. An increase in gas heater use by 8 h/d was associated with a 25% rise in days of wheeze and a 28% elevation in episodes of wheeze; increase in woodstove use of 8 h/d was associated with a 10% rise in days with cough. This study did not include air pollution measurements.

A later study of respiratory symptoms and heating focused on the mothers of these children (n = 888), and included indoor air pollution measurements (Triche et al. 2005). After controlling for various factors, each hour-per-day increase in kerosene heater use was associated with an elevation in wheezing (RR = 1.06; 95% CI: 1.01–1.11). Each 10-ppb rise in SO_2, produced only by the kerosene heaters, was associated with increased wheezing (RR = 1.57; 1.10–2.26) and chest tightness (RR = 1.32; 1.01–1.71). Median SO_2 concentrations associated with kerosene heater use and with no use were 6.4 ppb and 0.2 ppb, respectively. Elevated NO_2 was associated mainly with gas space heater use (median concentrations, 54.8 and 12.5 ppb for use and no use, respectively) and less strongly with kerosene heater use (17.7 and 11.5 ppb, respectively). When NO_2 concentration was dichotomized at 80 ppb, associations were found for chest tightness (RR = 1.94; 95% CI: 0.98–3.85) and wheezing (RR = 4.00; 95% CI: 1.45–11.0). This study provides some evidence that kerosene appliance use, and possibly gas appliance use, is associated with respiratory symptoms. However, a later study of these women (nonsmokers only) that examined variability of peak expiratory flow rates in relation to self-reported use of supplementary heating sources in winter found no marked association with any source, including kerosene heaters (Beckett et al. 2006).

Mallol et al. (2008) studied self-reported wheezing in children from a low-income population in Santiago, Chile. Two random samples (100 each) of children aged 13–14 yr were selected according to whether or not they reported wheezing in the past 12 mo. The unadjusted OR for kerosene used for heating or cooking at home was 1.3 (0.7–2.5). Wood and gas were combined in the reference group. No adjusted results were presented.

To identify risk factors for wheeze in children in the first year of life in low-resourced countries, Bueso et al. (2010) surveyed parents of 1827 children in 2 communities in Honduras and El Salvador. Results from the two countries combined showed some evidence of an association with kerosene for cooking, compared to electricity, for recurrent wheeze (three or more episodes) (OR = 2.78, 95% CI: 0.94–8.25).

In the Niger-Delta region of Nigeria, Mustapha et al. (2011) carried out a cross-sectional study of respiratory symptoms in relation to sources of outdoor and indoor air pollution sources in children aged 7–14 yr. Compared with gas cooking, nonsignificant positive associations with phlegm production were found for cooking with wood or coal (OR = 2.99, 95% CI: 0.88–10.18) and for kerosene cooking (OR = 2.83, 95% CI: 0.85–9.44). In general, the magnitudes of associations with night cough, asthma diagnosis (ever), and rhinitis (ever), although weaker than for phlegm production, were similar for kerosene and coal.

In summary, these studies are hard to interpret in a coherent way. Interpretation is complicated by the range of symptoms studied, the different ages of the populations, and the variations in reference fuel categories. However, there is some indication of an association of kerosene use with wheeze and cough, and reduction in spirometric values.

Asthma and Allergic Diseases

In a study in Richmond, VA, Cooper and Alberti (1984) showed that use of kerosene heaters in homes might lead to levels of SO_2 that would be expected to produce bronchospasm in some people with asthma. Further study of adverse health effects of kerosene heater emissions was recommended in asthmatics, as well as long-term effects in both sensitive and normal persons.

To investigate indoor air pollution effects on asthma, a case-control study was conducted with 158 children, aged 1–60 mo, hospitalized with asthma for the first time in Kuala Lumpur, Malaysia (Azizi et al. 1995). Controls were 201 children of the same age hospitalized in the same 24 h for nonrespiratory reasons. Although sharing a bedroom with a smoker and a mosquito coil used at least three nights per week were both associated with asthma, neither kerosene- nor wood-burning stoves in the household appeared to be risk factors. The unadjusted OR for kerosene stoves was 0.9 (95% CI: 0.5–1.6) and for woodstoves was 1.4 (0.6–3.6).

A case-control study involving 77 asthma cases and 77 controls was conducted among children aged 9–11 yr in Nairobi, Kenya (Mohamed et al. 1995). Cases and controls were drawn from a cross-sectional study. The use of kerosene as a cooking fuel was less frequent in the families of cases (26%) than in control families (29%), and indoor cooking fuels were not associated with asthma.

A study of Kenyan schoolchildren investigated why higher rates of exercise-induced bronchospasm (EIB), a common feature of asthma, were reported for children living in urban areas compared to rural areas (Ng'ang'a et al. 1998). Children (N = 1071) aged <12 yr at schools in Nairobi (urban) and in Muranga district (rural) underwent an exercise challenge test. A questionnaire was administered to parents/guardians. Kerosene was used for cooking by 37% of rural households and 81% of urban households. The OR for kerosene use was 1.17 (95% CI: 0.74–1.84) when a broad range of covariates was included in the model. Unfortunately, household lighting type was not included in models. It is likely that many of the rural households used kerosene lamps, and electric lighting may have been more common in urban areas. If so, this may have attenuated any true relationship with asthma.

A cross-sectional study in Jimma district, Ethiopia, was prompted by evidence that switching from biomass to fossil fuels was potentially associated with increased allergic diseases, including asthma (Venn et al. 2001). The study recruited 9844 people, an estimated 95% of the eligible population. Data were collected by questionnaire. Allergen skin sensitization testing was conducted in a sub-sample of 2372 people. Biomass fuels were used for cooking by 99% of the study population. However, "modern fuels" (gas, electricity, and kerosene) were used concurrently by 10%, with only 34 (<1%) reporting

exclusive use. The majority of kerosene stoves were the wick type. For kerosene, after controlling for age, gender, socioeconomic status, and identified confounders, the odds ratio for allergen skin sensitization was 1.95 (95% CI 1.02–3.73). Kerosene use was also associated with all allergic symptom outcomes (wheeze in past year: adjusted OR 1.55 [CI 1.01–2.38]; rhinitis, 2.57 [1.76–3.75]; eczema ever, 2.99 [1.78–5.04]; and eczema in past year, 2.22 [1.08–4.57]). For allergen skin sensitization, the adjusted OR for gas use was 3.20 (95% CI 1.62–6.32), but there were no associations between gas or electricity use and reported symptoms. Evidence The authors hypothesized that exposure to combustion pollutants from refined fossil fuels may have played a major role in the emergence of allergic diseases in the developing and developed world. Data on lighting were not reported.

As a follow-up to the Venn et al. (2001) study, 7155 children aged 1–4 yr living in Jimma and surrounding rural regions in Ethiopia were recruited (Dagoye et al. 2004). During the last year, the prevalence of wheeze in the urban area was 4.4% and 2% in the rural area. In the urban area, the OR for wheeze and daily household use of kerosene was 3.36 (95% CI: 1.77–6.36), with a rising trend in risk for increasing kerosene use. Few rural households reported use of kerosene for cooking, so it was not possible to examine this separately. Again, data on lighting were not reported.

In Isfahan, Iran, a study of respiratory illness in 561 females aged 1 mo to 81 yr (mean age 27.6 yr) was carried out to identify risk factors (Golshan et al. 2002). The adult women in the study were mostly housewives and there was extensive use of gas, kerosene, and wood fuels for cooking and heating. For current asthma, defined as a reported history of dyspnea attacks associated with wheezy breathing during the last 12 mo, the adjusted OR for kerosene was 62.4 (95% CI: 7.5–520). For asthma ever, the OR for kerosene use was 5.01 (95% CI: 1.45–17.32), with the corresponding result for wood fuel being 1.08 (1.01–1.27).

In summary, evidence for an association between kerosene and asthma is inconsistent, with the strongest evidence of an association coming from studies in Ethiopia and in Iran. The high OR for current asthma in the Iranian study is anomalous. A possible explanation is that the kerosene stoves used in that area are portable and are reportedly taken into living rooms for heating, especially when it is cold.

Respiratory Infections

Acute lower respiratory infections (ALRI) were investigated in 633 infants (<1 yr) in two slums in Delhi, India (Sharma et al. 1998). Approximately equal numbers of participants reported use of wood and kerosene for cooking, and all were monitored for 9 wk in winter. Expressed as ALRI cases per 100 wk of observation, the rates for Kusumpur Pahari were 6.3 and 5.9 for the wood and kerosene groups, respectively, while corresponding rates for Kathputly were 1.6 and 2.9. Families using kerosene were generally of a higher socioeconomic status than those using wood. The study shows little difference in ALRI rates between the wood and kerosene groups. However, if the results of other studies showing biomass use to be associated with increased ALRI risk (Torres-Duque et al. 2008) are applicable here, then cooking with kerosene may confer little advantage over cooking with biomass, at least for ALRI in infants. At least one study (Saksena et al. 2003) suggested that kerosene users tend to cook more frequently indoors, which may counteract the benefits of reduced emissions from kerosene, compared to biomass cooking. This result needs to be confirmed in other settings.

Savitha et al. (2007) carried out a case-control study of 104 ALRI cases and 104 controls among children aged 1 mo to 5 yr in Mysore, India. Controls were healthy siblings of the case children. Consistent with the established association with ALRI, 93% of cases and 30% of controls used

firewood for cooking fuel. The results for kerosene were more discrepant: 5% of cases and 25% of controls used the fuel for cooking (unadjusted OR = 0.15, exact 95% CI: 0.04–0.43), whereas 37% of cases and 3% of controls used kerosene for lighting (unadjusted OR = 19.4, exact 95% CI: 5.7–101). Only three variables were retained in a logistic regression model—partial immunization, overcrowding, and malnutrition. This result is puzzling and raises questions about the modeling procedure, for which virtually no information was provided. Nonetheless, the unadjusted results for kerosene lighting are striking and would be difficult to explain purely by confounding.

In Pokhara, Nepal, associations between pulmonary tuberculosis (TB) and the use of biomass and kerosene fuels were investigated in a hospital-based case-control study (Pokhrel et al. 2010). Cases (n = 125) were women, ranging in age from 20 to 65 yr, with a confirmed TB diagnosis. Age-matched controls (n = 250) were female patients without TB. Compared with using a gas fuel stove, the adjusted OR for using a biomass-fuel stove was 1.21 (95% CI: 0.48–3.05), and 3.36 for use of a kerosene-fuel stove (95% CI: 1.01–11.22). The OR for use of biomass fuel for heating was 3.45 (95% CI: 1.44–8.27) and for use of kerosene lamps for lighting was 9.43 (95% CI: 1.45–61.32). Complicating interpretation of the Nepal study, a similar study in Chandigarh, India, obtained an OR of 3.14 (95% CI: 1.15–8.56) for biomass cooking and 0.49 (95% CI: 0.21–1.20) for cooking with kerosene, both relative to use of liquefied petroleum gas as the reference fuel (Lakshmi et al. 2010). However, an interaction term in the model for the combination of kerosene cooking and having a smoker in the family produced an OR of 2.58 (95% CI: 0.80–8.32). This study did not report data on lighting type.

The few studies on infection are insufficient for any conclusions to be confidently drawn. In particular, the inconsistent results of the Nepal and Chandigarh TB studies underline the need for further studies of this issue.

Effects on the Eye

Results from a case-control study in Nepal (206 cases, 203 controls), intended to investigate whether biomass cooking fuel use was associated with cataract, provided some suggestion of a possible association between kerosene lamp use and cataract (Pokhrel et al. 2005). The adjusted OR for kerosene lamp use was 1.37 (0.81–2.32). This appears to be the only study to have investigated this association, albeit secondarily to the main hypothesis. An association is plausible, as a number of studies found evidence of an association between cooking with solid fuel and cataract (Pokhrel et al. 2005). However, further investigation is needed.

DISCUSSION AND CONCLUSIONS

Compared to household gas fuels in developed countries and solid fuels—biomass and coal—in developing countries, there have been few studies of the health and other impacts of household kerosene use. Well-documented kerosene hazards are poisonings, fires, and explosions. Less investigated are the risks of exposures to kerosene combustion products. Some kerosene appliances emit substantial amounts of fine PM, as well as CO, NO_x, and SO_2. There is wide variability in pollutant emissions and exposures by use (lighting, cooking, or heating) and device type within each use category. Fuel quality and device settings add further variation.

The greenhouse gas implications of household kerosene have not been explored in depth. However, they could be substantial for some appliances, given their high prevalence in many developing countries. A single fuel-based lantern used 4 h/d was estimated to emit more than 100 kg of CO_2 per year, corresponding to 190 million tonnes annually by all fuel-based lighting in houses without

electricity (Mills 2005). It has been suggested, based on anecdotal observation, that black carbon, a component of aerosols and important short-lived forcer that contributes to climate warming, may also be substantial in lamp emissions (DGDA 2010). In that regard, measurements from laboratory-based studies employing kerosene lamps for aerosol generation suggest that emitted PM is composed of particles that are highly light absorbing; however, it is likely that these experiments do not accurately reflect household lamps or usage practices.

Kerosene lamps may seem fairly innocuous, since the fuel is consumed relatively slowly compared to either cooking or heating use. However, the level of human exposure will also be determined by how the user interacts with the device, particularly proximity and duration. For example, since generated light from a simple wick lamp is only useable over a short distance, a person may be in close proximity to the lamp for several hours at a time (e.g., a child studying). On the other hand, a convective heater may be kept relatively distant from the user, but in the same room. The influence of user factors on exposure appears to have been little documented.

Although the evidence indicates that indoor concentrations of pollutants from kerosene cookstoves are often less than from biomass combustion stoves, user behaviors associated with kerosene stoves may differ from biomass cooking, influencing the exposures relative to emissions. Specifically, women and their children may spend longer in the kitchen when cooking with kerosene, possibly because kerosene emissions are less visibly smoky than biomass emissions (Saksena et al. 2003). Increased exposure duration was postulated to be one explanation why, in one study, kerosene cooking was a stronger risk factor for TB than biomass cooking (Pokhrel et al. 2010).

These user-related factors emphasize the need for personal exposure monitoring in at least some studies. The vast majority of epidemiologic studies, to date, have either relied on questionnaires for reported usage or employed microenvironmental pollutant monitoring. No information on kerosene combustion-product-specific biomarkers of exposure was found in the search, but such a biomarker, if one could be found, would be helpful in distinguishing exposure to kerosene emissions from emissions from other household fuels. Similarly, no information on exposures to actual uncombusted kerosene components in the household situation was found, although it is likely that such exposures occur, either from slow emissions from appliances during periods of nonuse or from volatilization during usage. These uncombusted products might have some utility for the development of biomarkers.

When kerosene use has been examined as an exposure, it is usually in the larger context of household fuel use, particularly solid fuel use. In part, this may be because of the favorable way that kerosene is often viewed. Some international agencies have treated kerosene as a “clean” fuel (UNDP/ESMAP 2003). Among researchers, some have grouped it with LPG, natural gas, and electricity for data analysis purposes (Melsom et al. 2001); others have treated it as a “polluting fuel” and grouped it with well-established sources of indoor air pollution, such as coal and biomass (Gharaibeh 1996; Wichmann et al. 2006). This conflicting interpretation may help to account for apparently inconsistent research results—influenced by whether kerosene is combined with the reference (“clean”) fuels in the data analysis or whether it is combined with the fuels under investigation, often biomass. Depending on the actual health impacts of kerosene combustion emissions, such arbitrary decisions may lead to underestimates or overestimates of effects. For the purposes of this review, studies where kerosene was combined with other fuels were disregarded. This led to the discarding of a substantial number of publications that might otherwise have been useful.

The combined studies that evaluated kerosene cooking provide some evidence that emissions may impair lung function, promote asthma, and increase infectious illness and cataract risks. However, studies are few, study designs and quality are varied, and results are inconsistent, limiting conclusions that can be drawn. Similarly, for kerosene cooking emissions as a cause of cancer, the epidemiologic evidence is limited and there have been no new relevant studies. The few studies of respiratory cancer are inconclusive and there is only one study of salivary cancer. However, since kerosene combustion produces known carcinogens, including PAH and formaldehyde, it is likely that kerosene combustion products are at least weakly carcinogenic, even though the epidemiologic studies are presently insufficient to show this. Relevant to this, on June 12, 2012, IARC announced that diesel engine exhaust had been reclassified as carcinogenic to humans (Group 1), based on sufficient evidence that exposure is associated with an increased risk of lung cancer (http://press.iarc.fr/pr213_E.pdf). Diesel fuel is the portion of crude oil that distills within the temperature range 200–370°C, which substantially overlaps with the distillation range for kerosene (145–300°C). Similarities between kerosene particles from a lamp and diesel soot were demonstrated (Arnott et al. 2000).

With the exception of two studies in Nepal (Pokhrel et al. 2005; 2010), one of which found a strong association with TB risk, and one study in India (Savitha et al. 2007), which found a strong, unadjusted association with ALRI, published investigations of whether kerosene used in lamps may cause health effects are virtually nonexistent. Kerosene wick lamps are common in some developing countries, and are one of the primary lighting sources for the 1.3 billion people who still lack access to electricity (IEA 2011).

Animal toxicology studies of kerosene and its combustion products, to provide mechanistic information and biological plausibility for findings of epidemiology studies, are also in short supply. More such studies need to be conducted.

Several points to guide future epidemiologic investigations emerge from our review:

1. Kerosene-fueled appliances should not be combined with appliances using other fuels for the purposes of data analysis or results presentation.
2. Information that differentiates between device types, user settings, and applications should be presented (e.g., wick stove vs. pressure stove, simple-wick vs. hurricane lamp, use for general room lighting vs. use for reading).
3. Desirably, to facilitate understanding of the relationships between combustion, exposure, and health impact, stove and lamp usage should be monitored objectively. For example, monitoring temperature fluctuations of cooking devices with inexpensive temperature loggers has been shown to be a reliable tool for monitoring stove usage (Ruiz-Mercado et al. 2011).
4. For the most accurate air pollutant exposure estimates that best account for the influence of user-device interactions, studies should employ personal exposure monitoring. 5. Although objective measures of stove use and air pollution monitoring are valuable, not all study budgets can accommodate them. In those circumstances, much can be achieved by simply including a few additional questions in questionnaires to inquire about kerosene use for cooking, heating, and lighting. Most of what is known about the health effects of biomass-burning stoves has been obtained using a questionnaire-based approach, rather than from monitoring pollutant concentrations.

The use of kerosene for cooking and lighting continues to be widespread in many developing countries. One driver of this has been the controversial use of government subsidies to secure kerosene availability, mainly for cooking, by poor populations (Koshala et al. 1999; Pitt 1985; Rao 2012; Shenoy 2010). Kerosene is viewed as a step up the energy ladder from solid fuels, and for cooking can provide benefits to poor households in terms of convenience and time savings. In areas where electricity is unaffordable, unavailable, or unreliable, kerosene is often the primary lighting fuel. There are some indications, however, that it may have some health consequences, not only because of poisonings and fires, but also because of exposure to emitted pollutants. Given the widespread use of kerosene lamps and stoves, these exposure sources should be much more extensively investigated.

Assessing the exposure benefits of energy source/device changes should be supported by careful field-based monitoring and evaluation. Better evidence about health impacts is also important, not only for judging the appropriateness of kerosene promotion, but also for cost–benefit evaluations of alternative lighting and cooking solutions for poor households. For example, photovoltaic household and community-level lighting programs, including those combined with village electrification, are underway in a number of countries (DGDA 2010; Palit et al. 2011). However, these are primarily driven by the desire to improve access to lighting, rather than from concerns about toxicity of emissions, for which evidence is presently sparse. National and international efforts to promote advanced combustion biomass cookstoves with low emissions are also underway, including the National Biomass Cookstove Initiative of India (Venkataraman et al. 2011) and the Global Alliance for Clean Cookstoves (Smith 2010). Finally, there is pressure in some countries to reduce subsidies for LPG, a substantially cleaner burning fuel than kerosene (Government of India 2010). On the other hand, in other countries, rollback of kerosene subsidies in favor of LPG was suggested and even implemented (Budya et al. 2011). The costs and benefits of these efforts would need to be reevaluated if kerosene use were shown to present significant health risks.

In conclusion, considering the widespread use in the developing world of kerosene as a household fuel, the scarcity of adequate epidemiologic and exposure investigations, the potential for harm suggested by some of the few relevant studies that do exist, and the implications for national energy policies, researchers are urged to consider collection of data on household kerosene use, for cooking, heating, or lighting, in their household surveys and studies of health in developing countries. As noted, much can be achieved by simple extension of questionnaires. Given the potential risks of kerosene, policymakers may consider alternatives to kerosene subsidies, such as shifting support to cleaner technologies for lighting and cooking.

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