The health and social implications of household air pollution and respiratory diseases

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Approximately three billion individuals are exposed to household air pollution (HAP) from the burning of biomass fuels worldwide. Household air pollution is responsible for 2.9 million annual deaths and causes significant health, economic and social consequences, particularly in low- and middle-income countries. Although there is biological plausibility to draw an association between HAP exposure and respiratory diseases, existing evidence is either lacking or conflicting. We abstracted systematic reviews and meta-analyses for summaries available for common respiratory diseases in any age group and performed a literature search to complement these reviews with newly published studies. Based on the literature summarized in this review, HAP exposure has been associated with acute respiratory infections, tuberculosis, asthma, chronic obstructive pulmonary disease, pneumoconiosis, head and neck cancers, and lung cancer. No study, however, has established a causal link between HAP exposure and respiratory disease. Furthermore, few studies have controlled for tobacco smoke exposure and outdoor air pollution. More studies with consistent diagnostic criteria and exposure monitoring are needed to accurately document the association between household air pollution exposure and respiratory disease. Better environmental exposure monitoring is critical to better separate the contributions of household air pollution from that of other exposures, including ambient air pollution and tobacco smoking. Clinicians should be aware that patients with current or past HAP exposure are at increased risk for respiratory diseases or malignancies and may want to consider earlier screening in this population.

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INTRODUCTION

Respiratory diseases are responsible for a significant burden worldwide from direct healthcare costs, significant disability, premature mortality, lost productivity and social consequences. Specifically, chronic respiratory diseases are estimated to result in 92.5 million disability-adjusted life years (DALYs) lost in 2016 worldwide.1 There is limited published data on the health expenditures for respiratory disease outside of the United States (US) and the European Union (EU).2 Furthermore, available statistics grossly underestimate health costs due to widespread underdiagnoses of respiratory disease.3 For the 28 countries in the EU, lung disease is estimated to cost €379.6 billion and results in an annual loss of 5.2 million DALYs, valued at an additional €300 billion.2 In the US, lung diseases cost an estimated $129 billion, with $106 billion of this attributed to chronic obstructive pulmonary disease (COPD), asthma, and pneumonia.3

Individuals in low- and middle-income countries (LMICs) have different exposures, and consequently risk factors, for the development of respiratory diseases as compared to those in higher income countries.4 Household air pollution (HAP) exposure is an important attributable risk factor for both acute and chronic respiratory diseases in LMICs, including acute respiratory infections,4–8 tuberculosis,9,10 asthma,9 COPD,5,11 pneumoconiosis,12 head and neck cancers,13 and lung cancer.14,15 HAP exposure results from the incomplete combustion of biomass fuels (e.g., wood, dung, agricultural crop waste, and coal) during cooking and heating. Almost three billion individuals, 42.2% of the world population, continue to cook with biomass fuels due to inadequate access to clean energy.16 According to the 2016 Global Burden of Disease estimates, HAP was responsible for 2.9 million annual deaths and 81.1 million DALYs lost.1 These estimates show that 26% of HAP deaths were attributed to lower respiratory infections, 5% to tracheal, bronchial and lung cancers, and 23% to COPD.17 Other respiratory diseases were not included in the 2016 study.18 Although HAP exposure affects all members of the household, women often have the highest risk of exposure due to their involvement in the cooking process.4 Children are also often close to their mothers and therefore can be exposed to HAP from a young age.4

Exposure to HAP not only has deleterious health effects, but also has important social consequences. Welfare and labor income losses are estimated at $1.6 trillion and $94 billion, respectively, due to lost productivity and poor health from HAP exposure.18,19 These losses are reflected in the poverty trap, a phenomenon where those who are in poor health, resulting from an environmental exposure such as HAP, cannot work or if they can work, their wages are lower. These individuals then cannot...
and have theorized that some people are more susceptible to respiratory disease. The rate of lung function decline is heterogeneous as some people likely experience periods of rapid decline followed by slower decline. Similar mechanisms may apply in the case of HAP exposure, where noxious particles, such as particulate matter and carbon monoxide, may affect lung development starting in utero.

Although biological plausibility and several observational studies support an association between HAP exposure and respiratory diseases, existing literature is either lacking or conflicting, limiting our ability to make causal inferences. There are few randomized controlled trials evaluating the effect of reducing HAP exposure on respiratory health outcomes. As a result, existing reviews and meta-analyses rely primarily on case-series and observational studies. The goal of this review is to summarize the systematic reviews and meta-analyses available for each respiratory disease then update this evidence summary of available literature since the publication of these reviews.

**RESULTS**

Our primary search yielded 11 eligible systematic reviews, summarized in Table 1. The manuscripts from the secondary search, of which 19 were included in this paper, are summarized in Table 2. Based on our scoping review, HAP exposure may be associated with ALRI, COPD, tuberculosis, pneumoconiosis, head and neck cancer, and lung cancer. All of the systematic reviews included studies that were heterogeneous in methods and results. None of the systematic reviews had an objective measure of HAP exposure, instead exposure was often based on proxies and self-reporting. Furthermore, biomass fuel type was inconsistent between each study. Many of the studies do not separate cooking and heating and some include women only or both men and women.

**Acute respiratory infections**

Acute respiratory infections include both upper respiratory infections (URI) and acute lower respiratory infections (ALRI). Upper respiratory infections are defined as infections of the upper respiratory structure of the aerodigestive tract, including diagnoses such as the common cold and sinusitis. ALRI is an acute infection of the lung from a viral or bacterial cause resulting in inflammation of the lung. ALRI is the leading cause of death in children under 5 years of age and a frequent cause of hospitalization for adults in LMICs. Risk factors include low birth weight, malnutrition, low socioeconomic status, and smoking.
| Authors (Year of publication) | Number of studies included | Meta-analysis (if yes, number of studies) | Exposure | Relevant outcome | Effect size | Bias & heterogeneity |
|-------------------------------|----------------------------|------------------------------------------|----------|-----------------|------------|---------------------|
| Acute respiratory infections (ARI) and acute lower respiratory infections (ALRI) |
| Po et al.\(^5\) (2011) | 8 | Yes (8) | Household combustion of wood, dung, crop residue, or charcoal indoors in non-industrialized or domestic settings | ARI and ALRI in children | ARIs in children exposed to biomass fuel smoke compared to those exposed to cleaner fuel (pooled OR = 3.52, 95% CI 1.94–6.43) | The Begg funnel plot asymmetry and the Egger test indicated publication bias after removing one outlier study. Significant heterogeneity was found among studies and so a random-effect model was used (\(I^2 = 91.3\%\), \(p < 0.001\)) |
| Jary et al.\(^6\) (2016) | 8 | No | Air pollution from indoor burning of any solid fuels (wood, charcoal, animal dung, crop residues, and coal) for household purposes. This included studies that quantified exposure through direct measurement of specific pollutants, questionnaires regarding exposure history, comparison of groups exposed to types of exposure (e.g., different stove types), or before and after an exposure reduction intervention | ALRI in adults including pneumonia, acute bronchitis or bronchiolitis in adults. This included studies that defined the outcome as “acute” or specified duration of less than 14 days, even if infection was not confirmed, assuming that acute respiratory illnesses in the absence of underlying disease would likely be infectious in origin | Two of the studies documented increased risk of ALRI, two documented an unadjusted association, and the remaining four documented no association to ALRI and HAP |
| Misra et al.\(^7\) (2012) | 24 | Yes (9) | Use of solid and biomass fuels defined as (1) availability of measurements of HAP and/or exposure that demonstrate substantive exposure differential, (2) child carried while cooking, and (3) fuel use: unprocessed solid fuels compared to clean(er) fuels such as liquefied petroleum gas and electricity (fuels for comparison need to be specified) | At least one ALRI (pneumonia, emphysema, bronchiolitis, bronchiolitis) reported in children by a caregiver, study personnel or physician, death certificate or verbal autopsy, or detected in nasopharyngeal swab culture or nasopharyngeal aspirate immunofluorescent microscopy | 16 studies reported significant ORs (1.38–6.0) of ALRI exposed to HAP. Meta-analysis of 9 studies found that children exposed to HAP were more likely to have ALRI than those not exposed (pooled OR = 2.51, 95% CI 1.53–4.10) |
| Jackson et al.\(^8\) (2013) | 36 | Yes (36) | Use of biomass fuels for cooking or a description of indoor smoke | Severe ALRI, defined differently depending on study setting: 1) hospital-based study: hospitalization for pneumonia or bronchiolitis in children under five years of age 2) community-based studies: presence of chest indrawing in a child with cough and difficulty breathing with increased respiratory rate for age within the WHO cut off for respiratory rate | HAP exposure increased risk of severe ALRI (pooled OR = 1.6, 95% CI 1.1–2.3) |
| Tuberculosis |
| Kurmi et al.\(^9\) (2014) | 12 | Yes (12) | Smoke from solid fuel burning | Positive association between solid fuel use and TB. Adjusted pooled |
| | | | | The Egger plot indicated no publication bias (\(p = 0.14\)). |
| Authors (Year of publication) | Number of studies included | Meta-analysis (if yes, number of studies) | Exposure | Relevant outcome | Effect size | Bias & heterogeneity |
|-------------------------------|----------------------------|------------------------------------------|---------|----------------|------------|---------------------|
| Lin et. al. (2014)            | 16                         | Yes (15)                                 | Combustion of solid fuel (defined as coal/lignite, charcoal, wood, straw/shrubs/grass, animal dung or crop residues) for cooking and/or heating. Reference group included clean or non-solid fuels (electricity, liquefied petroleum gas, natural gas, biogas, and kerosene) | TB defined by microbiological criteria (sputum smear alcohol-fast bacilli-positive) or doctor-diagnosed active TB | Effect for all types of solid fuel (OR = 1.43, 95% CI 1.07 to 1.91) was greater than for those using kerosene only (OR = 0.70, 95% CI 0.13 to 3.87) and mixed fuel (kerosene and biomass) (OR = 1.30, 95% CI 0.20 to 8.63) | Significant heterogeneity was found between studies ($I^2 = 70.8\%$, $p < 0.001$) |
| Po et al. (2011)              | 9                          | Yes (9)                                  | Household combustion of wood, dung, crop residue, or charcoal indoors in non-industrialized or domestic settings | Asthma in children and women | No significant association with HAP exposure and asthma. Children: (Pooled OR = 0.50, 95% CI 0.12–1.98); Adults: (Pooled OR = 1.34, 95% CI 0.93–1.93) | The Begg funnel plot asymmetry and the Egger test indicated publication bias after removing one outlier study. Both meta-analyses found significant heterogeneity among studies in children ($I^2 = 88.6\%$, $p < 0.001$) and in women ($I^2 = 58.6\%$, $p < 0.05$) and used random effects models |
| Po et al. (2011)              | 12                         | Yes (12)                                 | Household combustion of wood, dung, crop residue, or charcoal indoors in non-industrialized or domestic settings | COPD and chronic bronchitis in women | Exposure to biomass fuel smoke was significantly associated with COPD (OR = 2.40, 95% CI 1.47 to 3.93). Exposure to biomass fuel smoke was significantly associated with chronic bronchitis (OR = 2.52; 95% CI 1.88 to 3.38) | The Begg funnel plot asymmetry and the Egger test indicated publication bias after removing one outlier study. Chronic bronchitis: Borderline, nonsignificant heterogeneity among studies was found ($I^2 = 47.3\%$, $p = 0.09$) but random effects models were still used. Egger test and funnel plot asymmetry suggested publication bias COPD: |
### Table 1 continued

| Authors (Year of publication) | Number of studies included | Meta-analysis (if yes, number of studies) | Exposure | Relevant outcome | Effect size | Bias & heterogeneity |
|------------------------------|-----------------------------|------------------------------------------|----------|-----------------|-------------|---------------------|
| Kurmi et al. 11 (2010)       | 23                          | Yes (23)                                  | Domestic use of solid fuels | COPD was defined according to ATS and/or GOLD criteria, using the spirometry criteria of a forced expiratory volume in 1 s (FEV1)/forced vital capacity (FVC) ratio < 70% or physician diagnosis. Chronic bronchitis was defined according to Medical Research Council (MRC) criteria | Positive associations between the use of solid fuels and COPD (pooled OR = 2.80, 95% CI 1.85 to 4.0) and chronic bronchitis (pooled OR = 2.32, 95% CI 1.92 to 2.80) | Heterogeneity was found among studies ($I^2 = 67.2\%$, $p < 0.001$) and random effects models were used. The Begg funnel plot and Egger test did not show indications of publication bias. For both COPD and chronic bronchitis, heterogeneity between studies was found (lung function defined COPD: $I^2 = 91.8\%$, $p < 0.001$; physician defined COPD: $I^2 = 96.9\%$, $p = 0.17$; CB: $I^2 = 68.9\%$, $p < 0.001$). There was heterogeneity in the pooled risk estimates for COPD and chronic bronchitis across studies, possibly due to variation in exposure between different settings and types of fuels and stoves used. Meta-regression found that COPD studies diagnosed on lung function criteria, year of publication and the year that study was conducted were significant contributors to heterogeneity. No heterogeneity from meta-regression were found for studies of chronic bronchitis |
| Josyula et al. 13 (2015)    | 14                          | Yes (14)                                  | HAP from all solid fuel types (coal, wood and mixed exposures) that were primarily derived from household cooking and/or heating and not from other forms of urban/outdoor air pollution or occupational exposures | Oral cancer, Pharyngeal cancer, Laryngeal cancer, Esophageal cancer, Nasopharyngeal cancer | HAP was associated with oral (OR = 2.44; 95% CI 1.87–3.19); nasopharyngeal (OR = 1.80; 95% CI 1.42–2.29); pharyngeal (OR = 3.56; 95% CI 2.22–5.70) and laryngeal (OR = 2.35; 95% CI 1.72–3.21) cancers. The elevated risk for esophageal cancer (OR = 1.92; 95% CI 0.82–4.49) was non-significant | Funnel plot did not indicate publication bias. Heterogeneity was found for studies of nasopharyngeal cancer ($p = 0.09$). No significant heterogeneity was found for studies of oral ($p = 0.93$); pharyngeal ($p = 0.99$), esophageal ($p = 0.53$) and laryngeal ($p = 0.49$) cancers |
| Kurmi et al. 14 (2012)       | 28                          | Yes (28)                                  | Biomass and solid fuel smoke, coal smoke | Lung cancer | Lung cancer | Lung cancer | Lung cancer | Lung cancer | Lung cancer | Lung cancer | Lung cancer | Lung cancer | Lung cancer |

**Pneumoconiosis**

No systematic reviews have been published.  

**Head and neck cancers**

Josyula et al. 13 (2015): HAP from all solid fuel types (coal, wood and mixed exposures) that were primarily derived from household cooking and/or heating and not from other forms of urban/outdoor air pollution or occupational exposures. HAP was associated with oral (OR = 2.44; 95% CI 1.87–3.19); nasopharyngeal (OR = 1.80; 95% CI 1.42–2.29); pharyngeal (OR = 3.56; 95% CI 2.22–5.70) and laryngeal (OR = 2.35; 95% CI 1.72–3.21) cancers. The elevated risk for esophageal cancer (OR = 1.92; 95% CI 0.82–4.49) was non-significant. Funnel plot did not indicate publication bias. Heterogeneity was found for studies of nasopharyngeal cancer ($p = 0.09$). No significant heterogeneity was found for studies of oral ($p = 0.93$); pharyngeal ($p = 0.99$), esophageal ($p = 0.53$) and laryngeal ($p = 0.49$) cancers.

Lung Cancer

Kurmi et al. 14 (2012): Biomass and solid fuel smoke, coal smoke. Coal smoke had a slightly stronger association with lung cancer than biomass smoke but the confidence intervals overlap (Coal smoke: pooled OR 1.82, 95% CI 1.60–2.06; biomass smoke: pooled OR = 1.50, 95% CI 1.17–1.94). The risk of lung cancer was significant across studies ($I^2 = 562.7\%$, $p < 0.001$). No significant heterogeneity was observed in the different strata for HAP exposure studies.
Table 1 continued

| Authors (Year of publication) | Number of studies included | Meta-analysis (if yes, number of studies) | Bias & heterogeneity |
|------------------------------|---------------------------|------------------------------------------|---------------------|
| Jary et al. (2014)           | 16                        | Yes (14)                                 |                     |
| Bruce et al. (2014)          | 14                        | Yes (14)                                 |                     |
| Po et al. (2013)             | 13                        | Yes (13)                                 |                     |
| Po et al. (2012)             | 12                        | Yes (12)                                 |                     |
| Po et al. (2011)             | 11                        | Yes (11)                                 |                     |
| Po et al. (2010)             | 10                        | Yes (10)                                 |                     |

Exposure Relevant outcome Effect size Bias & heterogeneity

| Authors (Year of publication) | Number of studies included | Meta-analysis (if yes, number of studies) | Bias & heterogeneity |
|------------------------------|---------------------------|------------------------------------------|---------------------|
| Jary et al. (2014)           | 16                        | Yes (14)                                 |                     |
| Bruce et al. (2014)          | 14                        | Yes (14)                                 |                     |
| Po et al. (2013)             | 13                        | Yes (13)                                 |                     |
| Po et al. (2012)             | 12                        | Yes (12)                                 |                     |
| Po et al. (2011)             | 11                        | Yes (11)                                 |                     |
| Po et al. (2010)             | 10                        | Yes (10)                                 |                     |

Exposure Relevant outcome Effect size Bias & heterogeneity

| Authors (Year of publication) | Number of studies included | Meta-analysis (if yes, number of studies) | Bias & heterogeneity |
|------------------------------|---------------------------|------------------------------------------|---------------------|
| Jary et al. (2014)           | 16                        | Yes (14)                                 |                     |
| Bruce et al. (2014)          | 14                        | Yes (14)                                 |                     |
| Po et al. (2013)             | 13                        | Yes (13)                                 |                     |
| Po et al. (2012)             | 12                        | Yes (12)                                 |                     |
| Po et al. (2011)             | 11                        | Yes (11)                                 |                     |
| Po et al. (2010)             | 10                        | Yes (10)                                 |                     |

Exposure Relevant outcome Effect size Bias & heterogeneity

| Authors (Year of publication) | Number of studies included | Meta-analysis (if yes, number of studies) | Bias & heterogeneity |
|------------------------------|---------------------------|------------------------------------------|---------------------|
| Jary et al. (2014)           | 16                        | Yes (14)                                 |                     |
| Bruce et al. (2014)          | 14                        | Yes (14)                                 |                     |
| Po et al. (2013)             | 13                        | Yes (13)                                 |                     |
| Po et al. (2012)             | 12                        | Yes (12)                                 |                     |
| Po et al. (2011)             | 11                        | Yes (11)                                 |                     |
| Po et al. (2010)             | 10                        | Yes (10)                                 |                     |

Exposure Relevant outcome Effect size Bias & heterogeneity

| Authors (Year of publication) | Number of studies included | Meta-analysis (if yes, number of studies) | Bias & heterogeneity |
|------------------------------|---------------------------|------------------------------------------|---------------------|
| Jary et al. (2014)           | 16                        | Yes (14)                                 |                     |
| Bruce et al. (2014)          | 14                        | Yes (14)                                 |                     |
| Po et al. (2013)             | 13                        | Yes (13)                                 |                     |
| Po et al. (2012)             | 12                        | Yes (12)                                 |                     |
| Po et al. (2011)             | 11                        | Yes (11)                                 |                     |
| Po et al. (2010)             | 10                        | Yes (10)                                 |                     |

Existing literature has not specifically investigated the link between URI and HAP exposure, instead considering URI in combination with all other acute respiratory infections. A 2011 meta-analysis from Po et al. found that, in eight studies of acute respiratory infection, children were 3.52 times more likely to develop acute respiratory infections when exposed to HAP than those exposed to cleaner fuel or kerosene (95% CI 1.93–6.43). Among adults, the evidence is less clear, and existing studies have included charcoal in the comparison group, which is not a clean fuel, or did not adjust for confounders.

There is no clear consensus on the association between HAP exposure and ALRIs in adults. Jary et al., the only systematic review investigating this relationship, included eight eligible studies. Two of the studies documented an increased risk of ALRI, two documented an unadjusted association, and the remaining four documented no association. A meta-analysis was not performed as the studies were too heterogeneous in methods and results. Since its publication, no other studies have been published that further evaluate the relationship between HAP exposure and acute lower respiratory infections in adults.

The majority of studies investigating the association between HAP exposure and ALRI have focused on children under 5 years of age, as they are thought to be more susceptible to respiratory infections. HAP has been associated with increased risk of childhood ALRI. A systematic review by Misra et al. examined studies investigating the relationship between HAP exposure and ALRI in children under five years of age. Of 24 studies included for review, 16 reported significantly elevated OR, ranging from 1.38 to 6.0, of ALRI in those participants exposed to HAP. Nine studies were included in the meta-analysis and found that children exposed to HAP were 2.51 times more likely to have ALRI than children without exposure (95% CI 1.53–4.10). This review did not have clear criteria for ALRI and included a spectrum of severity.

Unlike previous reviews which focused on ALRI of any severity, a meta-analysis conducted by Jackson et al. aimed to identify risk factors specific to severe ALRI, defined as hospitalization for pneumonia or bronchiolitis, in children under five. In the pooled analysis of five studies in LMICs, the overall odds ratio was 1.6 (95% CI 1.1–2.3), indicating that exposure increased the risk of severe ALRI.

Since these two systematic reviews, three studies found that HAP exposure was associated with higher chances of developing ALRI in children. In addition, one recent randomized controlled trial in Malawi documented no association between use of a cleaner burning biomass stove and decreased pneumonia in children. However, this trial may have suffered from insufficient reduction of exposure. A second randomized controlled trial in Guatemala (the RESPIRE study) demonstrated the importance of misclassification of exposure in understanding the relationship between childhood pneumonia and an intervention to reduce HAP exposure. In this study, the intention-to-treat analysis did not show a relationship between physician-diagnosed pneumonia and the use of the improved biomass stove and chimney when compared with the control (OR = 0.84, 95% CI: 0.63–1.13) whereas the exposure-response analysis uncovered a significant relationship (OR = 0.82, 95% CI 0.70–0.98). This emphasizes the importance of obtaining personal exposure data, as not only do exposure-response analyses aid in the integration of exposure data, but they also help to understand the threshold at which reductions in HAP exposure lead to significant health benefits.

As shown in Fig. 2, an exposure-response curve from Burnett et al., meaningful reductions in ALRI can only be achieved if PM2.5 concentrations are reduced to <35 µg/m3, the World Health Organization intermediate target goals for air quality in the household. This relationship may be applicable for all respiratory diseases discussed in this paper, but thus far has been most studied in childhood ALRI.
Relative to use of electricity for cooking, ALRI was increased in association with any use of biomass stoves (OR = 2.98), kerosene stoves (OR = 1.87, 95% CI: 1.24, 2.83), and gas stoves (OR = 1.62; 95% CI: 1.05, 2.50).

Maternal education and occupation, having one or more family members who smoke indoors, and living in a single-family dwelling or shared home were associated with acute lower respiratory tract infection as defined by the 1995 WHO definition.

Cooking fuel other than LPG was associated significantly with acute lower respiratory tract infection (94.1% vs 7.6%, OR = 26.3, 95% CI: 10.5–65.7; p < 0.0001). Odds ratios of ALRI by survey compared to low polluting fuels: Medium polluting fuels: NFHS-1: OR = 1.39, 95% CI: 1.01–1.92, p < 0.05. NFHS-2: OR = 1.47 95% CI: 1.22–1.75, p < 0.001. NFHS-3: OR = 1.31, 95% CI: 0.92–1.88. High polluting fuels: NFHS-1: OR = 1.48, 95% CI: 1.08–2.03; p < 0.05. NFHS-2: OR = 1.54, 95% CI: 1.33–1.77; p < 0.01. NFHS-3: OR = 1.53 (95% CI: 1.21–1.93; p < 0.001).

The pneumonia incidence rate in the intervention group was 158. (95% CI: 14.89–16.63) per 100 child-years and in the control group 15.58 (95% CI: 14.7–16.5) per 100 child-years, with an intervention versus...
| Study design | Sample size | Study population | Exposure | Outcome | Adjustment for confounders | Effect size |
|--------------|-------------|------------------|----------|---------|---------------------------|-------------|
| **Table 2 continued** | | | | | | |
| Smith, K. R. et al. (2011) | Randomized controlled trial. Intervention: locally developed chimney stove. Control: wood fire use for cooking. | 265 children in intervention, 253 in control | Pregnant women or children < 4 months of age in households using an open fire for cooking in an enclosed kitchen, in the San Marcos region of the Guatemalan highlands | Personal 48 h carbon monoxide measurements obtained with diffusion tubes as indicators for wood smoke exposure | Physician-diagnosed pneumonia: not defined, stated as without use of a chest radiograph. Secondary outcomes: fieldworker-assessed pneumonia (all and severe) and seven other conditions of physician-diagnosed pneumonia | Not described |
| **Tuberculosis** | | | | | | |
| Rabbani et al. (2017) | Case-control | Total of 356 women (178 cases and 178 controls) | Large secondary care hospital in Pakistan. Cases: Non-smoking 20 to 65-year-old women with pulmonary TB; Controls: Age and area of residence matched women suffering from other diseases | Self-reported type of kitchen (ventilated vs non-ventilated), age at which cooking was started, average daily cooking time, current and past use of specific types of cooking fuels (biomass which included wood, crop residues and animal dung or cleaner fuels which included natural gas and LPG) | New pulmonary TB cases diagnosed by physician through sputum smear for acid-fast bacilli or chest radiograph | Household monthly income and second hand tobacco smoke |
| Jubulis et al. (2014) | Case-control | 60 cases, 118 controls | Recruited from a large tertiary care hospital in Pune, India. Cases: Children less than 5 years of age with confirmed/probable TB; Parent or guardian self-reported tobacco smoke exposure, primary cooking fuel used. No | TB cases were defined according to the WHO and India’s Revised National Tuberculosis Control Program guidelines as confirmed or probable TB | Age, sex, school attendance, household TB exposure, household food insecurity and vitamin D deficiency | Exposure to IAP was independently associated with TB (OR = 2.67, 95% CI 1.02–6.97) |
| Authors/ Year of publication | Study design | Sample size | Study population | Exposure | Outcome | Adjustment for confounders | Effect size |
|-------------------------------|-------------|-------------|------------------|----------|---------|---------------------------|------------|
| **Asthma**                    |             |             |                  |          |         |                           |            |
| Oluwole et al. (2017)         | Cross-sectional survey | 1,690 children | Children aged 6-21 years attending primary and secondary schools in Ibadan, Nigeria. | Child's parent or guardians report of household cooking fuel type: biomass (cow dung/animal residue, firewood, charcoal) or no biomass (LPG, electricity) | Asthma symptoms were defined according to ISAAC definition | Age, sex, maternal level of education, tobacco exposure, indoor environmental characteristics, indoor pet exposure | Biomass fuel was associated with increased odds of asthma symptoms: adjusted odds ratios were 1.38 (95% CI: 1.05–1.80) for nocturnal cough, 1.26 (95% CI: 1.00–1.61) for current wheeze, and 1.33 (95% CI: 1.05–1.69) for report of any asthma-related symptoms |
| Oluwole et al. (2017)         | Cross-sectional survey | 1,690 children | Children aged 6-21 years attending primary and secondary schools in Ibadan, Nigeria. | Parent or guardian household cooking fuel type: biomass (cow dung/animal residue, firewood, charcoal) or no biomass (LPG, electricity) | Asthma symptoms were defined according to ISAAC definitions | Age, sex, maternal level of education, tobacco exposure, indoor environmental characteristics, indoor exposure to pets, BMI | In adjusted analyses, biomass fuel use was associated with increased odds of severe symptoms of asthma (OR = 2.37, 95% CI: 1.16–4.84), but not with possible asthma (OR = 1.22, 95% CI: 0.95–1.56) |
| Kumar et al. (2017)           | Cross-sectional survey | 204,568 individuals of all ages | Indian Human Development Survey II, a nation-wide survey conducted across India | Self-reported type of fuel used (clean only or other), cooking stove type. Quantification of use was not reported | Self-reported previous diagnosis of asthma or cough with shortness of breath | Sex, age, marital status, completed years of schooling, tobacco smoking, chewing tobacco, alcohol use, vegetarian, nutritional status, wealth quantile, religion, caste, place of residence | The odds of reporting asthma were higher for individuals living in households using unclean fuels (OR = 1.21, 95% CI 1.08–1.34) |
| Gonzalez-Garcia et al. (2015) | Cross-sectional survey | 5,539 adults of all ages | Adults of both genders older than 40 years of age in urban areas of five Colombian cities | Self-reported history of using wood for cooking | Wheezing: affirmative answer to the question “Have you ever had two or more attacks of wheezes causing you to feel short of breath?” Asthma: wheezing definition plus a post-bronchodilator FEV1/FVC ratio less than 70% of predicted | City of residence, sex, BMI, education, respiratory disease before 16 years old, first-degree relative with asthma, occupational gases or fumes exposure, occupational dust or particles exposure | History of wood smoke exposure for cooking was associated with wheezing (OR = 1.24, 95% CI: 1.02–1.50, p = 0.033) but not with asthma (OR = 1.07, 95% CI: 0.87–1.32, p = 0.50) |
| Gaviola et al. (2016)         | Cross-sectional survey | 4,325 participants, of whom 2,953 had complete questionnaires and spirometry | Adults aged 35 years of age and older in four sites in Peru | Self-reported daily use of biomass fuels | Adults were defined as having asthma if they met any of the following three criteria: (1) physician- | Age, sex, height, living at high altitude, smoking, BMI, hypertension, family | Current daily exposure to biomass fuel smoke (OR = 1.18, 95% CI 0.70 |
| Table 2 continued |
|-------------------|
| **Authors/ Year of publication** | **Study design** | **Sample size** | **Study population** | **Exposure** | **Outcome** | **Adjustment for confounders** | **Effect size** |
| COPD Miele et al. (2016) | Cross-sectional | 4,325 participants, of whom 2,947 met eligibility criteria based on completion of data | Adults aged 35 years and older in four sites in Peru | Self-reported daily use of biomass fuels. | Chronic bronchitis: having self-reported phlegm production (or both cough and phlegm production) for at least three months each year in two consecutive years. COPD: post-bronchodilator FEV1/FVC less than the lower limit of normal for a given age, sex, and height | Age, sex, history of asthma, socioeconomic status, urbanization, and history of asthma, socioeconomic status, urbanization to 1.91) was not associated with asthma for the cases was 35.13 ± 55.86 h in a year and for the controls was 28.2 ± 40.09 h in a year; | Daily biomass fuel use was associated with chronic bronchitis (Prevalence Ratio = 2.00, 95% CI: 1.30–3.07, p < 0.01). |
| Amal et al. (2018) | Cross-sectional | 18,554 subjects across 25 international sites | Adults aged 40 years or older from low-, middle-, and high-income countries | Self-reported use of solid fuels was defined based on whether the participant had used an open fire with charcoal, coal, wood, crop residues, or dung as the primary means of cooking or heating the house or water for > 6 months in their lifetime. Self-reported exposure levels assessed | Airflow obstruction: a post-bronchodilator FEV1/FVC less than the lower limit of normal (LLN), based on reference equations for white individuals from the third U.S. National Health and Nutrition Examination Survey | Age, sex, BMI, pack-years of smoking, cumulative years of exposure to dust in the workplace | There was no association between airflow obstruction and use of solid fuels for cooking or heating (OR for men = 1.20, 95% CI: 0.94–1.53; OR for women = 0.88, 95% CI: 0.67–1.15) |
| Siddhardtan et al. (2018) | Cross-sectional | 12,396 participants | Adults aged 35–95 in six countries in Latin America, Sub-Saharan Africa, and Southeast Asia | Household air pollution exposure was defined as self-reported use of biomass materials as the primary fuel source in the home | COPD: postbronchodilator FEV1/FVC z-score less than or equal to 21.64 SDs of the Global Lung Function Initiative mixed ethnic reference population | Age, sex, daily cigarette smoking, body mass index, post-treatment pulmonary tuberculosis, and secondary education | Participants with household air pollution exposure were 41% more likely to have COPD (95% CI: 1.18–1.68) than those without the exposure, and 13.3% (95% CI: 6.40–20.6%) of COPD prevalence may be caused by household air pollution exposure |
| Pneumoconiosis Singh et al. (2015) | Case-control | 30 cases, 53 controls | Cases: patients aged 20–85 years with acanthosis on bronchoscopy recruited from SMS Hospital in Jaipur, India. Controls: patients | Self-reported hours of biomass exposure | Acanthosis: black pigmentation of the mucosal lining of the tracheobronchial tree on bronchoscopy | Not described | Biomass exposure for the cases was 35.13 ± 55.86 h in a year and for the controls was 28.2 ± 40.09 h in a year. |
| Authors/ Year of publication | Study design | Sample size | Study population | Exposure | Outcome | Adjustment for confounders | Effect size |
|-----------------------------|-------------|-------------|------------------|----------|---------|--------------------------|------------|
| Pilaniya et al.78 (2017)     | Case-control| 60 female participants. Based on bronchoscopy findings, participants were divided into three groups: Group 1: patients with bronchial anthracofibrosis, Group 2: patients with only anthracotic pigmentation without narrowing/distortion, and Group 3: patients with a normal tracheobronchial tree | Clinical, radiologic and electrocardiographic evidence of Pulmonary arterial hypertension and cor pulmonale, and the antecedent of at least 10 years of domestic woodsmoke exposure, non-smokers, no known lung disease | Self-reported mean exposure to biomass fuels | Observations of the patients, findings on high resolution chest computerized tomography, arterial blood gases | No adjustment | this was not statistically significant (p > 0.05). ORs not reported |
| Sandoval et al.79 (1993)     | Case-series  | 30 patients at an outpatient clinic in Mexico City, Mexico who lived in the countryside | 30 patients over 2 years who presented to clinic with (1) clinical and radiological diagnosis of COPD and/or interstitial lung disease, (2) antecedent long-standing domestic biomass exposure, (3) non-smokers (4) no chronic lung disease, (5) living in a rural area | Self-reported mean exposure to biomass fuels | No adjustment | | 14 of 22 patients who underwent bronchoscopy with direct visualization were found to have anthracosis. 5 out of 5 patients who underwent open lung biopsy had anthracotic pigment deposition |
| Ozbay et al.80 (2001)        | Case-series  | 30 consecutive patients | matched according to age, gender and smoking habits, without black patches on bronchoscopy | Self-reported hours of biomass fuel smoke exposure, number of years of cooking, and exposure index (average number of hours of exposure per day multiplied by the number of years of cooking) | Bronchial anthracofibrosis: (1) long-standing history of biomass fuel smoke exposure, (2) on HRCT, the occurrence of multifocal narrowing of involved bronchus when present and (3) visual confirmation on fiberoptic bronchoscopy of (a) bluish-black mucosal pigmentation, along with (b) narrowed/distorted bronchus | No adjustment described | Mean biomass exposure was 3.96 (2–10) hours per week for a mean of 37±10 years. PaO₂ (mmHg) 54.4±11.4, PaCO₂ 45 ±8.9, 76% had increased lung volumes or diffuse emphysema, 76% had reticulonodular pattern and/or thickening of interlobular septa, 40% had ground glass appearance, 30% had in a honeycombing-lobe or segment |
Tuberculosis

Tuberculosis (TB) is a communicable infectious disease caused by the bacillus Mycobacterium tuberculosis and is spread by inhalation of the bacteria into the lungs. This is a disease primarily affecting those in LMICs, where 95% of TB deaths occur.10 Annually 250,000 children and 1.7 million adults die from TB.90 Risk factors include HIV, living in poverty, poor nutrition, and smoking.26,50,51

There is no clear consensus on whether there is a direct link between HAP exposure and TB in adults. Given the low incidence of TB disease in single-site studies, this association has been difficult to evaluate.10 Two systematic reviews from 2014, Kurmi et al.9 and Lin et al.10 reached opposing conclusions on the association between TB and HAP exposure. Kurmi et al. identified 12 peer-reviewed studies that evaluated active TB, controlled for smoking and reported adjusted risk estimates.9 The adjusted pooled OR was 1.43 (95% CI: 1.07–1.91) for all 12 studies and 1.26 (95% CI: 0.95–1.68) when studies with physician-diagnosed TB were removed. This analysis concluded that an individual exposed to HAP has a 43% increased risk of having active TB compared to those using clean fuels. Lin et al. identified 15 studies that included adjusted risk estimates, of which 10 were case-control studies and 5 were cross-sectional studies.10 The pooled OR from case-control studies was 1.17 (95% CI: 0.83–1.65) and 1.62 (95% CI: 0.89–2.93) for the cross-sectional studies. This systematic review concluded that there was no strong evidence for a positive association between HAP exposure and TB. In fact, Lin et al. questions the conclusion drawn in Kurmi et al. since they calculated a pooled OR using a fixed-effects model which may not be appropriate given the heterogeneity of the studies. Conversely, Lin et al. used the random-effects model to pool across heterogeneous studies.

Since these systematic reviews in 2014, few studies have been published evaluating the association between HAP exposure and TB. One case-control study among 178 women in rural Pakistan found a three-fold increase (OR: 3.0 95% CI: 1.1–4.9) in TB risk among current biomass fuel users compared to non-biomass users.52

There is also sparse literature evaluating the relationship between HAP exposure and TB in children. Only two studies could be identified that exclusively looked at HAP exposure and reported adjusted risk estimates: Ramachandran et al. and Jubis et al. yielded ORs of 6.9 (95% CI: 2.5–18.9)53 and 7.2 (95% CI: 1.4–44.5)54 respectively. Both of these studies suggest that HAP exposure increases the risk of TB in children. Since that review, there have not been any significant studies published evaluating the association between HAP exposure and TB. Future population-based studies are in progress but results have yet to be published.55

Asthma

Asthma is a non-communicable respiratory disease that is caused by chronic inflammation of the airways and results in wheezing, chest tightness, and cough.56 Asthma may develop as an allergic disorder, and a large proportion of asthma cases have sensitization to aeroallergens.57 For the purpose of population-based studies, there is no clear definition of asthma, and studies use epidemiological definitions that include self-reported symptoms of wheezing in the past 12 months, physician reported wheezing or bronchodilator responsiveness.56 In 2015, approximately 400,000 people died of asthma worldwide, though asthma is considered severely under-diagnosed.58 Many risk factors are thought to be involved in the development of asthma, however, thus far smoking and occupational allergen exposure are the most clear risk factors.59 Interestingly, asthma is more prevalent in higher income countries and more urban areas.56

There is not a clear consensus on whether there is a direct link between HAP exposure and asthma in children or adults. Po et al. performed meta-analyses of four studies on asthma in children and five studies on asthma in adults, and did not find a significant association with HAP exposure (children: OR = 0.50, 95% CI 0.12–1.98; adults: OR = 1.34, 95% CI 0.93–1.93).5 Since the publication of that review, the studies published do not show conclusive results on the relationship between HAP exposure and asthma. Many are contradictory, with inconsistent settings and exposure definitions.60-64

Chronic obstructive pulmonary disease (COPD)

Chronic obstructive pulmonary disease (COPD) is an adult disease characterized by irreversible airflow limitation due to a mixture of small airways disease and parenchymal destruction.65 The definition of COPD includes chronic bronchitis, defined by persistent daily phlegm for three months each year for at least 2 years,66 and emphysema, the destruction of the alveoli.34 In 2015, chronic obstructive pulmonary disease (COPD) caused 3.2 million deaths worldwide (95% CI 3.1–3.3 million).67 The World Health Organization ranks COPD as the fourth leading cause of mortality worldwide, 90% of which was in LMICs.68 Known risk factors of COPD include cigarette smoking, ambient pollution, genetics, poor socioeconomic status, and past history of TB.34

Exposure to HAP has been shown to be associated with COPD and this has been explored in multiple systematic reviews. After the definition of COPD recently changed to encompass chronic bronchitis and emphysema, prior studies looked at each disease separately.34 Two systematic reviews published months apart found that those exposed to HAP were more likely to develop chronic bronchitis and COPD.6,51 The most recent meta-analysis, by Smith et al., yielded a pooled OR of 1.94 for COPD (95% CI 1.62–2.33).69

Since these systematic reviews, several newer publications have investigated the association between COPD and HAP exposure. CRONICAS, a population-based study in Peru, documented that daily biomass fuel use for cooking was associated with COPD (prevalence ratio [PR] = 2.22, 95% CI 1.02–4.81)70 and chronic bronchitis (PR = 2.00, 95% CI 1.30–3.07).71 A recent publication, from the Burden of Obstructive Lung Disease Initiative (BOLD) investigators, questions previous literature as their analysis of post-bronchodilator spirometry measurements from 18,554 adults...
found no association between the use of solid fuels and airflow obstruction. These results, however, may be skewed since these data include high-income settings with little to no biomass fuel use. Another population-based study of 12,396 adults from 13 resource-poor settings documented that those with HAP exposure were 41% more likely to have COPD (OR = 1.41, 95% CI 1.18–1.68) than those without the exposure. This study is the first one to calculate population attributable risk factor and found that 13.5% (6.4%-20.6%) of COPD prevalence may due to HAP exposure.

Pneumoconiosis

Pneumoconiosis is an inflammatory lung disease that results in parenchymal scarring and nodularity and can eventually lead to fibrosis. Bronchial anthracofibrosis (BAF) is type of pneumoconiosis defined by black pigmented lesions along the bronchial mucosa with bronchial narrowing. This is diagnosed exclusively by bronchoscopic evaluation, therefore, limiting the diagnosis in LMICs as bronchoscopy is not widely available. Patients with bronchial anthracofibrosis suffer from dyspnea, cough, and hemoptysis. There are no systematic reviews or meta-analyses that evaluate the potential association between HAP exposure and bronchial anthracofibrosis. To attempt to shed light on this issue, Gupta et al. performed an extensive literature search to evaluate the association between HAP exposure and bronchial anthracofibrosis. From 17 studies and 6 case series, 1320 patients were identified with bronchoscopically confirmed BAF. The review suggested that HAP exposure might be a risk factor for bronchial anthracofibrosis, particularly in non-smoking women in rural areas. After that review's publication, a 2015 case series study in India found that 30 consecutive participants exposed to HAP over a 13-month period were found to have black patches on their bronchial walls. They were matched with controls without black pigmentation. Compared to controls, cases were less likely to be exposed to HAP, although this was not statistically significant (OR = 0.57; 95% CI 0.19–1.74). Furthermore, a study in India in 2017 looked at 60 non-smoking females with respiratory symptoms and exposure to HAP. This study found that 40% of women with respiratory symptoms and exposure to HAP had bronchial anthracofibrosis diagnosed by imaging and fiberoptic bronchoscopy.

While pneumoconioses such as bronchial anthracofibrosis can result in pulmonary fibrosis, there is no consensus that HAP exposure is associated with pneumoconiosis with higher risk of progression to fibrotic lung disease. Currently, no systematic reviews or meta-analyses have evaluated this potential association, but two case series exist. The first examined 30 Mexican rural women who had evidence of pulmonary hypertension and participants were exposed for an average of 59.1 years. Twenty-two patients underwent bronchoscopy and 14 had antracotic plaques present on visual examination. Transbronchial biopsy from 14 patients showed fibrosis. Pathology from open lung biopsies in 5 patients showed fibrosis with antracotic deposits. In another case series of 30 women who were exposed to HAP over an average of 37 years and had a diagnosis of COPD received a high resolution computed tomography which consistently showed evidence of fibrosis. Two patients had open lung biopsies of which one had pathology showing end-stage fibrosis.

Head and neck cancer

Head and neck cancers encompass cancers of the lip, oral cavity, oropharynx, larynx, and nasopharynx and the associated structures in the regions of the head and neck. This group of malignancies is the ninth most common globally. In LMICs, this type of cancer is often caught in the late stages and has a high mortality rate. Tobacco consumption (smoked and smokeless), chewing areca nut, alcohol, and HPV infection have been associated with head and neck cancers.

Lung cancer

Lung cancer is the most common cause of cancer death worldwide with 1.59 million estimated deaths in 2012. Lung cancer is associated with smoking and more commonly found in high-income countries where smoking is prevalent. There is a rise in incidence of lung cancer in LMICs as tobacco smoking is increasing in popularity, particularly among men. Although lung cancer screening programs have been widely implemented in the US, they are less common in resource-poor settings because treatment options are not as widely available. Beyond smoking, known risk factors for lung cancer include environmental pollutants such as radon and asbestos, as well as chronic inflammation from pneumonia or TB.

Lung cancer has been highly associated with HAP exposure in females. There is not a demonstrated association in males, likely due to reduced time spent cooking. In 2012, Kurmi et al. performed a systematic review and meta-analysis of 28 studies evaluating HAP exposure on development of all types of lung cancer. The pooled analysis found a higher likelihood of developing lung cancer in women (OR = 1.81, 95% CI 1.54–2.12) but not in men (OR = 1.16, 95% CI: 0.79–1.69). This analysis controlled for tobacco smoking. Among fuel types, the fuel with the highest association with lung cancer was coal (OR = 1.82, 95% CI 1.60–2.06). The highest OR was among women in China who use coal for cooking. This meta-analysis may underestimate the impact of HAP on lung cancer risk as the studies selected did not have clean fuel controls. Subsequent to this meta-analysis, Bruce et al. found that among trials using clean fuels as a comparison group, the OR for lung cancer was 1.21 (95% CI 1.05–1.39) for men and 1.95 (95% CI 1.16–3.27) for women. There have not been any subsequently published manuscripts investigating this relationship that met our inclusion criteria.

DISCUSSION

Based on the literature summarized in this review, HAP exposure is associated with ALRI, COPD, tuberculosis, pneumoconiosis, head and neck cancer, and lung cancer. However, there has not been a causal link established between HAP exposure and respiratory disease. The Bradford-Hill criteria of causation allow for assessing causal evidence relating to environmental exposures and disease. An assessment of these criteria in the context of HAP and respiratory diseases is described in Table 3. Future studies should seek to strengthen consistency in outcome and exposure definitions, establish biological gradients through dose-response relationships, and strengthen experimental evidence through randomized controlled trials that implement interventions that adequately reduce HAP exposure.

Clinicians should be aware of the increased risk of respiratory diseases and malignancies of the aerodigestive tract in patients who are actively being exposed to HAP or have been exposed at any point in their lives including in utero exposure. When evaluating respiratory symptoms of HAP exposed patients, clinicians should keep in mind that patients may not reach their

Josyula et al. performed a meta-analysis investigating the relationship between HAP exposure and head and neck cancers. The results from three studies that adjusted for smoking indicated that HAP exposure is associated with a 2.56-fold increase in the risk of oral cancer (95% CI 1.80–3.64). Six studies yielded a pooled OR of 1.8 (95% CI 1.42–2.29) for nasopharyngeal cancer, although none of the individual studies controlled for smoking. Four studies reported smoking-adjusted OR of 3.56 (95% CI 2.22–5.70) for pharyngeal cancer. Five studies yielded smoking-adjusted OR of 2.35 (95% CI 1.72–3.21) for laryngeal cancer. Since this review, there have not been any newly published work disputing the association of HAP and head and neck cancers.
Table 3. Assessment of Hill's criteria of causation about the association between HAP exposure and respiratory disease

| Criteria                      | Assessment                                                                                                                                                                                                 |
|-------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Strength of association       | As outlined in this review, strong and significant associations have been documented between HAP exposure and ALRI, COPD, TB, pneumoconiosis, head and neck cancer, and lung cancer.                                      |
| Consistency across populations| Consistency and reproducibility are lacking in the evidence presented in this paper due to heterogeneity between studies and inconsistent case and exposure definitions. Currently available studies are not easily amenable to meta-analysis due to lack of consistent definitions or diagnostic criteria for respiratory disease, instead relying on caregiver- or self-reported symptoms which lack diagnostic and etiological specificity. Exposure was also inconsistently defined and often not quantifiable. |
| Specificity                   | Since HAP exposure is linked to a wide range of respiratory diseases, specificity is no longer a widely accepted and used criteria.                                                                           |
| Temporality                   | Temporality has been shown through prospective cohort studies that have documented HAP exposure to precede respiratory diseases. There is still a need for randomized trials to lower PM$_{2.5}$ to the World Health Organization standard (<35 µg/m$^3$) and document if HAP reduction leads to an improvement in respiratory outcomes. |
| Biological Gradient (dose-response) | Many studies have failed to collect longitudinal exposure data to characterize the dose-response of HAP exposure to respiratory outcomes. However, evidence is available for a dose-response relationship between ALRI and HAP exposure. (Fig. 2) |
| Biological Plausibility       | Strong evidence for biological plausibility exists linking noxious chemicals and particles in HAP to inflammation. Particulate matter, for example, has been hypothesized to stimulate an inflammatory response in airway macrophages and respiratory epithelium leading to tissue damage that can result in respiratory illnesses in susceptible individuals. Petroleum gas (LPG), may prevent disease, there have been no published results from large-scale randomized controlled trials investigating this hypothesis. |
| Coherence with natural history, animal studies | This scoping review has found evidence of higher risk of respiratory disease in LMICs where individuals have higher exposure to biomass smoke. Animal studies have also documented the harmful effects of HAP exposure. |
| Experiment                    | Experimental or intervention-based epidemiologic evidence for HAP exposure and respiratory disease is thus far limited. Several studies and trials have been conducted with the goal to lower HAP by using more efficient biomass-burning cookstoves; however, it has become clear that reductions achievable by this approach fall short and fail to meet the World Health Organization intermediate target goals for air quality in the household (PM$_{2.5}$ ≤ 35 µg/m$^3$). While it is intuitive that a switch to clean energy, such as liquefied petroleum gas (LPG), may prevent disease, there have been no published results from large-scale randomized controlled trials investigating this hypothesis. |
| Analogy                       | There is clear evidence from similar pollutants, such as cigarette smoke and outdoor air pollution maximal lung function if exposed early in life and may be more susceptible to the development of chronic respiratory diseases. As ALRI is one of the leading causes of death in children under 5 years of age, clinicians should be diligent in rapidly evaluating these children for pneumonia to provide antibiotics as quickly as possible. Although screening for all respiratory diseases and malignancies may not be possible in LMICs, when patients immigrate to developed countries clinicians need to be aware of this prior exposure and the effects on respiratory health when considering risk factors for implementing recommended screening guidelines. For example, although a patient may not have smoked cigarettes and would not qualify for lung cancer screening based on the current screening guidelines technically, biomass exposure was not considered specifically in these guidelines and may be substituted for smoking in calculating patient risk and need for screening. Better designed studies with a focus on characterizing exposure-disease relationship are needed to provide stronger recommendations. Several studies and trials have been conducted with the goal to lower HAP by using more efficient biomass-burning cookstoves; however, it has become clear that achievable reductions fall short and fail to meet the World Health Organization intermediate target goals for air quality in the household (<35 µg/m$^3$). As a result, scientists and policy-makers alike agree that more efficient biomass-burning cookstoves are unlikely to result in health benefits. While there is evidence for clean energy, such as liquefied petroleum gas (LPG), to prevent disease, there have been no published results from large-scale randomized controlled trials investigating this association. However, two ongoing LPG stove trials plan to fill this gap in the literature. If these trials can document health benefits associated with switching to LPG, further economic and implementation evaluations will be needed to understand if scaling up LPG interventions would be a valuable investment. There are some limitations to our scoping review. First, time and manpower constraints limited our capacity to perform a full systematic review of original research articles as part of our secondary search. However, our primary goal was not to conduct a systematic review but instead to summarize current existing evidence for primary care physicians which we could accomplish with the approach presented here. Second, while we selected several chronic respiratory diseases to evaluate that have been linked to tobacco smoke exposure, we may have inadvertently not included some that may also be associated with HAP exposure. Several limitations arose based on the available literature included in this review. For instance, disease definitions vary greatly limiting comparability between studies. Additionally, HAP was inconsistently measured and was rarely quantified to show an exposure-response relationship. Many of the articles included did not consistently control for important confounders, such as tobacco smoke exposure in homes or outdoor air pollution levels. Lastly, there were varying levels of heterogeneity and publication bias among studies included in systematic reviews. While there is a relationship between HAP exposure and many respiratory disease outcomes, better evidence in the form of randomized controlled trials reducing household air pollution are needed to strengthen this association. Further studies are needed to determine the best ways to screen for chronic respiratory diseases resulting from exposure to HAP, and identify adequate treatments. Moreover, clinicians should be aware that patients...
Search strategy and study selection
We searched EMBASE, PubMed, and SCOPUS for systematic reviews and meta-analyses, reported the findings of these evaluations, and summarized the remaining literature since publication of each review. The search for systematic reviews was conducted by two informationists at the Johns Hopkins University Welch Library. We searched for common acute or chronic respiratory diseases (using the terms “acute respiratory disease” or “acute lower respiratory infection” or “pneumonia,” “tuberculosis” or “TB,” “asthma,” “chronic obstructive pulmonary disease,” “COPD,” “chronic bronchitis” or “emphysema,” “pneumonia-coniosis” or “pulmonary fibrosis,” “head and neck cancer,” “lung cancer”), each in combination with the terms “household air pollution,” “biomass,” or “indoor air pollution.” From the search terms provided to the informationists, a reference list of systematic reviews was provided to the authors. Selection of reviews for inclusion was undertaken by two authors (SS and DG). The literature search for systematic reviews occurred for reviews published before September 15, 2017. Based on these criteria, 63 systematic reviews were identified and 11 were included for this manuscript. We present Inclusion and exclusion criteria for systematic reviews in Table 4.

The secondary literature search was performed for manuscripts after the publication of the chosen systematic review/s for each disease up until February 1, 2018. We searched PubMed and EMBASE for original research published subsequent to each of these reviews, using the same search terms as the primary search (“acute respiratory disease” or “acute lower respiratory infection” or “pneumonia,” “tuberculosis” or “TB,” “asthma,” “chronic obstructive pulmonary disease,” “COPD,” “chronic bronchitis” or “emphysema,” “pneumonia-coniosis” or “pulmonary fibrosis,” “head and neck cancer,” “lung cancer”), each in combination with the terms “household air pollution,” “biomass,” or “indoor air pollution.” We searched each selected systematic review from the primary search in PubMed and reviewed each manuscript that cited each systematic review in PubMed Central. Hand searching was performed by examining the reference lists for relevant articles. Inclusion criteria for the secondary search of primary articles were similar: exposure to HAP caused by biomass fuels, all age, and conducted in a LMIC.

Data abstraction and quality assessment
Each systematic review was evaluated by S.S. and D.G. and the most current systematic reviews that met our criteria were selected and mutually agreed upon by both authors. Abstracted statistics for each disease were confirmed by S.S., D.G., C.R., and M.C. All systematic reviews and meta-analyses included met Preferred Reporting Items of Systematic reviews and Meta-Analysis (PRISMA) standards. Each paper published subsequent to the last systematic review was evaluated by the authors and met the same criteria for inclusion as the systematic reviews.

DATA AVAILABILITY
All included papers are published; no primary data are presented in this paper. As such, data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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AUTHOR CONTRIBUTIONS
S.S. and D.G. designed and implemented the literature search in collaboration with W. C. S.S., and D.G. performed screening and data extraction. The initial draft manuscript was written by S.S., D.G., C.R., and W.C. All authors (S.S., D.G., C.R., M.C., G.G., B.K., R.W., and W.C.) provided feedback and agreed with the final version. S.S. and D.G. are co-first authors.

ADDITIONAL INFORMATION
Competing interests: The authors declare no competing interests.

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Table 4. Inclusion and exclusion criteria for systematic reviews

| Inclusion | Exclusion |
|-----------|-----------|
| Exposure to household air pollution (HAP) caused by biomass fuels | Non-domestic exposures |
| Occurred in a low and middle-income country | English translation unavailable |
| Systematic review of the literature and/or meta-analysis | Non-peer reviewed sources |
| PRISMA standards met | All ages |

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