Published in final edited form as:
J Allergy Clin Immunol. 2012 January ; 129(1): 3–13. doi:10.1016/j.jaci.2011.11.021.

Respiratory Health Effects of Air Pollution: Update on Biomass Smoke and Traffic Pollution

Robert J. Laumbach, MD, MPH[1,2] and **Howard M. Kipen, MD, MPH**[1,2]
[1]UMDNJ-Robert Wood Johnson Medical School (RWJMS)
[2]Environmental and Occupational Sciences Institute (EOHSI)

Abstract

Mounting evidence suggests that air pollution contributes to the large global burden of respiratory and allergic diseases including asthma, chronic obstructive pulmonary disease, pneumonia and possibly tuberculosis. Although associations between air pollution and respiratory disease are complex, recent epidemiologic studies have led to an increased recognition of the emerging importance of traffic-related air pollution in both developed and less-developed countries, as well as the continued importance of emissions from domestic fires burning biomass fuels primarily in the less-developed world. Emissions from these sources lead to personal exposures to complex mixtures of air pollutants that change rapidly in space and time due to varying emission rates, distances from source, ventilation rates, and other factors. Although the high degree of variability in personal exposure to pollutants from these sources remains a challenge, newer methods for measuring and modeling these exposures are beginning to unravel complex associations with asthma and other respiratory disease. These studies indicate that air pollution from these sources is a major preventable cause of increased incidence and exacerbation of respiratory disease. Physicians can help to reduce the risk of adverse respiratory effects of exposure to biomass and traffic air pollutants by promoting awareness and supporting individual and community-level interventions.

Keywords

biomass; traffic; COPD; asthma; air pollutants; particulate matter

Introduction

Worldwide increases in rates of asthma and COPD over the past several decades have motivated intensive investigation of the role of environmental factors, including air pollution, in their causation. Recent research also suggests that air pollution contributes to the substantial worldwide burden of disease from acute lower respiratory infections and possibly tuberculosis. While the health effects of air pollution have been an international public health concern since at least the 1950's, recent research has heightened the focus on two broad sources of air pollution: biomass fuels (BMF) and motor vehicles. Understanding

Corresponding Author: Howard M. Kipen, MD, MPH, Professor and Director, UMDNJ-Robert Wood Johnson Medical School, EOHSI – 170 Frelinghuysen Road, Piscataway, NJ 08854, Phone: 732-445-0123 x629, Fax: 732-445-3644, kipen@eohsi.rutgers.edu.

of the health effects of BMF and traffic-related air pollution (TRAP) has lagged behind that of ambient air pollution, at least in part due to challenges in estimating highly-variable individual exposure from these widespread, but very localized, air pollution sources.

Of course, air pollution is only one of many environmental (non-genetic) factors for which a causative role in exacerbation or incidence of complex respiratory diseases has been suggested. Indeed, based on ecological analyses from the International Study of Asthma and Allergies in Childhood (ISAAC)[1], generally less-polluted developed countries (DCs) have much higher rates of asthma than many countries with higher levels of air pollution. However, studies with individual-level analyses that control for potential confounding have demonstrated associations between air pollutants, including TRAP, and asthma exacerbation, as well as possible links to increased asthma incidence. Additional evidence suggests that exposure to TRAP is correlated with the rising rates of allergic respiratory disease.[2] Although tobacco smoke is clearly the dominant cause of COPD worldwide, BMF smoke is now recognized as a major cause of COPD, especially among women in less developed countries (LDCs). Current evidence also indicates that BMF smoke plays a causative role in mortality from lower respiratory infections among children living in homes where BMF is used. The effects of indoor air exposures and individual ambient pollutants on asthma have recently been discussed in this forum.[3] Here, we emphasize the growing body of recent research pertaining to the relationship between respiratory health effects and exposure to TRAP and biomass air pollution. For recent reviews of the respiratory health effects of traffic exposures see Kelly and Fussell (2011)[4] and Salam et al. (2008)[5], and for BMF see Torres-Duque C (2008)[6], Balmes (2010)[7], Fullerton (2008).[8]

Exposures to Biomass and Traffic Pollutants

Exposures to BMF smoke and TRAP are widespread. Domestic fires burning biomass (wood, charcoal, dung, crop residues, and other raw plant materials) for cooking and/or heating remain the most pervasive and important source of exposure to air pollution for much of humanity. About 2.4 billion people live in households in which BMF is the primary cooking and/or heating fuel[9–10], with more than 90% of individuals in rural areas of LDCs using BMF.[9] Exposures are often exacerbated by use of open fires or traditional stove designs that lack flues or hoods to exhaust emissions away from the living area.[7]

While stationary industrial “smokestacks” continue to be a major source of outdoor air pollution from the burning of fossil fuels throughout the world, TRAP from motor vehicles are a growing concern in both DCs and LDCs.[11] Regulation of ambient (widespread, regional) ‘criteria’ pollutants in the US and other DCs has resulted in relatively effective stationary and mobile source controls. However, an increased number of vehicles and vehicle-miles-driven has lessened the impact of vehicle emissions controls. At the same time, heavy industry has moved to LDCs, resulting in a higher relative contribution from mobile sources in DCs, while in LDCs both total stationary and mobile source emissions have been increasing. Most of the worldwide growth in fleets of gasoline and diesel vehicles is occurring in LDCs.[11] As discussed below, in addition to making large contributions to background ambient air pollution, mobile sources may dominate exposures near roadways in urban areas, where a growing proportion of the world’s population lives.

Although one is rooted in longstanding cultural practices and the other arises from modern economic development, emissions from domestic fires and from motor vehicles have similarities in composition, toxicity, and exposure characteristics. Complex aerosol emissions from use of BMF share many components with TRAP and other outdoor PM, including particulate matter, carbon monoxide, nitrogen oxides, and scores of toxic organic compounds, such as formaldehyde, acrolein, and polynuclear aromatic hydrocarbons

(PAHs).[12] However, the physical and chemical characteristics of both BMF smoke and TRAP can vary substantially depending on the type of fuel burned and combustion conditions.[13]

Based on robust epidemiological associations between ambient particulate matter (PM) and respiratory and cardiovascular health effects, much attention has focused on the PM component of BMF and motor vehicle emissions.[14] PM of respirable size is classified by size fractions based on aerodynamic diameter. Ultrafine PM (UFP) with diameters ≤ 0.1 μm (PM0.1) is a major component of emissions near fires and tailpipes, but in seconds to minutes accumulates into somewhat larger "fine PM," or $PM_{2.5}$ with diameters ≤ 2.5 μm, within short distances from the point of release. PM_{10}, with diameters ≤ 10 μm, consists of $PM_{2.5}$ and larger particles of mainly crustal or biological origin, including many aeroallergens. Based on epidemiological and laboratory studies, $PM_{2.5}$ appears to be more potent for respiratory and cardiovascular disease effects compared to PM10.[15] According to the "ultrafine hypothesis," ultrafine PM may be still more toxic due to increased surface area and other characteristics.[16] Although they contribute little to the mass concentration of PM due to their small size, ultrafine particles emitted by combustion dominate the particle number concentrations near these sources.

In contrast to large-scale industrial sources of air pollution, the sources of biomass and traffic emissions tend to be in close proximity to individual "receptors." Biomass emissions occur primarily indoors, where women and children are most highly exposed during cooking and other domestic activities. Exposures are exacerbated by reduced ventilation in homes where biomass in used[7], or under conditions in which vehicle emissions may be concentrated as in urban street canyons or tunnels.[17] Concentrations of TRAP have steep gradients near roadways, with heightened exposure to individuals living, attending school, or working near major roads in urban areas, and return of TRAP to background levels within several hundred meters away from roadways.[18–19] For both BMF emissions and TRAP, time-activity patterns are a critical determinant of exposure. Household members who cook have high peak exposures, as when standing over the fire, as well as high time-averaged exposures to biomass pollutants.[20] Individuals living and working in urban areas may have a substantial part of their daily air polllutant exposure during usually relatively brief commuting times on roadways where TRAP are concentrated.[21–22] Substantial differences in TRAP concentrations and in inhaled doses as a consequence of travel mode, biking vs car vs bus, have been demonstrated, with bicyclists generally having the highest doses and electric bus riders the lowest.[23] Moreover, with distance and time away from sources, both BMF and vehicle emissions undergo complex "aging" processes that include oxidation, other chemical reactions and physical processes that alter exposure and toxic properties in ways that are not fully understood.[24]

The levels of BMF air pollutants measured in homes are typically far higher than ambient air pollutants, but they have received less attention from the international research community. Concentrations of PM and other air pollutants in indoor air during biomass burning can be orders of magnitude higher than levels that occur in ambient air in developed cities.[20] Levels of PM_{10} in homes using BMF often exceed several thousand $\mu g/m^3$ [20], compared to the EPA 24-hr ambient air quality standard of 150 $\mu g/m^3$ PM_{10} and the WHO guideline of 50 $\mu g/m^3$ PM_{10}.[25]. Little data is available on $PM_{2.5}$ and UFP indoors from BMF burning. The near-roadway microenvironment is mainly impacted by freshly-emitted UFPs and gas-phase compounds such as carbon monoxide, nitrogen oxides, and VOC's although resuspended road dust, mainly in the "coarse" mode of the PM_{10} fraction, may be an important exposure.[11]

The uneven distributions of exposure to BMF smoke and TRAP leads to uneven distribution of health risks, and environmental justice considerations at local, regional, national, and global scales.[26] There are age, gender, and socioeconomic differences in who is most exposed and most vulnerable to the health effects of BMF emissions and TRAP.[8] Exposure to BMF smoke is greatest among women and among young children who may be carried on mother's back during cooking activities, or spend more time indoors with mother.[26] BMFs that are least expensive and more affordable for impoverished households also burn less efficiently, increasing pollutant emissions.[8] TRAP exposures are concentrated in areas of greater traffic density, which, at least in the US, tend to be inner city communities of lower socioeconomic status with a higher burden of environmental contamination/impacts.

Epidemiology of Health Effects of BMF and TRAP

Individual exposure assessment has been a major challenge for epidemiological studies of both BMF and TRAP, in contrast to studies of ambient air pollution, in which assigning personal exposure based on central air monitoring data has had demonstrated utility. Few epidemiologic studies of respiratory effects of BMF smoke have measured exposure, relying instead on self-report of fuel use, despite evidence for wide variation in exposure depending on combustion and ventilation conditions and time-activity patterns. More recent studies, including controlled trials of stove interventions, have begun to measure exposure. As described above, the spatial and temporal distributions of urban air pollutants are characterized by significant variability with steep gradients in intensity near sources.[27–28] Thus, the use of land use regression and other techniques for modeling microenvironmental exposures for various particle and gas pollutants has become widespread and is featured in a number of the newer studies of TRAP to be described below. Land-use regression (LUR) uses the monitored levels of the pollutant of interest as the dependent variable and variables such as meteorology, traffic, topography, building shapes and sizes, and other geographic variables as the independent variables in a multivariate regression model.[29] Levels of pollution may then be predicted for any other geographic locations, such as residences or schools, using the parameter estimates derived from the regression model. A limitation is that LUR often captures only one time period, and may miss prior or neonatal exposures, however some studies have overcome this.[30–31] Further details are available in recent reviews.[32–33]

The most firmly established health effects of BMF emissions are acute lower respiratory infections in children and COPD in adults. While studies of ambient pollution effects have repeatedly demonstrated increased cardiovascular and respiratory morbidity and mortality for a variety of outcomes[14], those which have studied TRAP specifically have largely focused on asthma and related phenomena, with some investigations of allergy. The World Health Organization has estimated that BMF smoke exposure is responsible for about 1.5 million premature deaths per year,[34] and a global burden of disease of approximately 2.5% of all healthy life-years lost. Most of this burden of disease is due to respiratory infections, mainly among children less than 5 years of age, and COPD among adult women[35]. Several case-control and cross-sectional studies have evaluated associations between use of biomass fuels and prevalence of asthma with equivocal results among children and women.[36–43] Other studies have found strong associations between BMF smoke and COPD among non-smoking women.[9, 20, 44] Important new studies, discussed below, have strengthened the link between TRAP and asthma incidence in children,[30, 45–47] incidence in adults[48] and severity in adults.[49]

BMF and COPD

Given that cigarette smoke is a type of biomass smoke, a causal association between exposure to BMF smoke and COPD would not be surprising. Three recent meta-analyses have evaluated associations between BMF smoke and COPD.[50–52] In a systematic review and meta-analysis of 23 studies, Kurmi et al found that exposure to all types of BMF smoke was consistently associated with COPD with risk more than doubled, with greater risk suggested for woodsmoke compared to other fuels.[50] Hu et al analyzed 15 studies and found that BMF smoke was associated with increased risk of COPD among both women and men, and in both Asian and non-Asian populations.[52] In a meta-analysis of 6 studies that evaluated COPD among women using biomass compared to alternative fuels, Po et al also found a statistically significant pooled estimate of greater than 2-fold increased risk.[51] In another 6 studies that assessed chronic bronchitis, the pooled risk estimate was also greater than 2-fold. However, most studies have lacked direct exposure measurements, none have described a dose-response relationship, and estimated effect sizes have varied widely. This variation may be due to heterogeneity in fuel types and conditions of use, as well as in study design and differences in control of confounders such as exposure to mainstream or second-hand cigarette smoke, occupational exposures, socioeconomic factors, and changes in fuel use over time.[50] In a study of 841 nonsmoking women in Mexico that was notable for objective exposure and outcome measurements, Regalado et al found that peak PM_{10} over 2,600 μg/m3 among those using biomass fuels was related to small, but significant, reductions in FEV1 (81 ml), FVC (122 ml), and FEV1% predicted (4.7) compared to women who cooked with gas.[53] In an accompanying editorial, Jaakola noted that these effects were comparable to the estimates from environmental tobacco smoke exposure in adults.[54] Cigarette smoking rates are relatively low in most LDCs, especially among women.[55] Among women living in rural Turkey, the fraction of COPD attributed to exposures to BMF smoke was 23% after adjusting for possible confounders.[56]

TRAP AND COPD

A number of studies have established that children living in more polluted areas,have reduced lung growth compared to those living in cleaner areas, and that moving from a more polluted to a cleaner area demonstrates improved growth.[57] Similar findings for lung function have been reported in adults, as well as a limited data base of studies documenting an association between ambient air pollutants and objectively defined COPD.[57]

Most recently, a 35 year prospective study of over 57,000 Danes, used individual modeled assignments of traffic pollution and extensive control of confounders, with the end point of first hospital admission for COPD.[58] This outcome was associated with chronic NO_2 exposure (HR=1.08, 1.02–1.14) with a stronger association in asthmatics. This is the first longitudinal study of COPD with hard outcomes in association with modeled TRAP exposures, and seems to confirm the previous findings of cross-sectional studies that TRAP is likely to be a cause of COPD.

TRAP and Asthma Overview

Studies have long shown that asthma can be exacerbated, often measured as visits to emergency rooms, on days with higher levels of ozone and other pollutants.[59] More complex cohort study designs have been required to understand whether or not traffic-related pollutants play a role in the genesis, or causation, of asthma. To date all such investigations in a non-occupational setting have related to chronic, rather than acute, air pollution exposures. More sophisticated designs have been used in recent years and this part of the review will focus on those reported since 2009.

TRAP and Childhood Asthma

Initial studies of air pollution and asthma examined associations in children, looking first at exacerbations and more recently, through cohort studies, at incidence. Initial reports of incidence in children were variable in their results. Investigators interpreted these inconsistencies to result from misclassification of pollution exposure to the individual cases, likely due to reliance on central or regional air monitoring stations that do not reflect urban microgeographies. Subsequent studies using LUR or dispersion modeling produced significant associations in both children. and adults, with refinement of exposure through use of LUR techniques yielding larger and significant associations with chronic pollution exposure.

Two recent studies, one a birth cohort, add substantially to our confidence that TRAP exposure of young childen contributes to the development of asthma.[30, 45] Both used sophisticated exposure assessment in the form of LUR or a related technique to study the association between childhood asthma and TRAP exposure at home and/or school. Increased risk of for childhood asthma incidence demonstrated significant increases of 26% up to 51%, with good control of relevant confounders. Interestingly the birth cohort study did not find corresponding associations of potentially explanatory mechanistic variables such as atopic eczema, allergic sensitization, and bronchial hyperresponsiveness, leaving open questions about pathophysiology and roles of irritancy versus allergy. For the Southern California Children's Study[60] non-freewway pollutants demonstrated a stronger effect than those from freeways, possibly reflecting an effect of frequent acceleration and deceleration on TRAP characteristics.

In a smaller study, Carlsten et al (2011)[31] recruited infants at high familial risk for asthma and examined birth year home exposures to NO, NO_2, black carbon, and $PM_{2.5}$ by land use regression with follow up at 7 years of age. Birth year $PM_{2.5}$ (IQR=4.1ug/m3) was associated with a significantly increased risk of asthma with an OR of 3.1 (1.3–7.4). NO and NO_2 demonstrated similar associations but black carbon did not. This dramatic finding with relatively small exposure magnitude is intriguing but needs replication.

A study of self-reported allergic disease (using the ISAAC questionnaire) and home traffic density based on distance to major roadways, found approximately 1.5 to 3 fold prevalence ratios for heavy traffic density for wheeze, asthma, rhinitis and rhinoconjunctivitis, with no associations for children who slept in air conditioned homes,[46] with obvious important implications for prevention in atopics and others at heightened risk.

Using a cross sectional design and an "enhanced" ISAAC protocol for outcomes,[47] 6683 children in the French Six Cities Study were studied, with exposures based on a 3-year dispersion model for each school address to assign individual school exposures,. Asthma (either past year, or lifetime) was significantly associated with benzene, SO_2, PM_{10}, NOx, and CO. All of those but SO_2 were associated with eczema, and allergic rhinitis with PM_{10},. Sensitization to pollens was associated with benzene and PM_{10}, The findings for benzene and CO are somewhat surprising, and given their presence as constituents of motor vehicle fuel and/or exhaust, uncontrolled confounding may be present.

TRAP and Adult Asthma

The recent data base on asthma and traffic is less robust in adults. The Swiss Cohort Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) is a Swiss population-based cohort of adult lung disease-free non-smokers initiated in 1991 with 11y follow in 2002.[48] Using a dispersion model that included hourly meteorological and emission data on industrial, construction, heating, agricultural and forestry, and traffic emissions, the latter

separated by type of vehicle (truck vs. car), each participant was assigned an exposure to PM_{10}. Outcomes were adjusted for age, gender, baseline atopy, BMI, bronchial reactivity, and maternal allergies. They found a hazard ratio for doctor-diagnosed asthma of 1.30 (1.05–1.61) new cases for a given (1ug/m3 as PM_{10}) change in traffic pollution over the 10 years, more frequent in those with baseline atopy or bronchial hyperreactivity.

Trupin et al looked at the simultaneous impact on FEV1 percent and an asthma severity score of diverse social and physical environmental exposures on adult asthma in 176 subjects. Their final model had an R^2 of 0.30 for FEV1 percent predicted and 0.16 for Severity of Asthma Score. Distance to nearest road was a significant predictor of FEV1 but not Severity of Asthma Score. The importance here is that even when other variables strongly associated with usual clinical management of asthma are accounted for, a role for roadway traffic still persists.

Based on high quality studies discussed herein there is an increasingly robust literature that supports a causal relationship between various aspects of TRAP and new onset asthma or worsened asthma in children and adults. These risks need to be both incorporated into both public policy and explored for their role in medical decision making at the individual level.

BMF and asthma

In contrast to the abundance of studies showing exacerbation of asthma from increased exposure to ambient air pollution and TRAP, asthma prevalence has been the main outcome considered in studies of BMF smoke. Using Burden of Obstructive Lung Disease (BOLD) data on self-reported prevalence of asthma among 508 individuals in Southeastern Kentucky, increased odds of reporting current asthma was associated with cooking indoors with wood or coal for more than 6 months of one's life (OR 2.3, CI 1.1–5.0), but not with history of domestic heating with wood or coal (OR 0.8, CI 0.4–1.8).[61] However, a handful of earlier studies had not found compelling evidence of an increased risk of asthma among women or children in households using BMF.[36, 40] Among six studies that examined risk of asthma among women using BMF in rural India, Iran and Turkey, two found a statistically significant increased risk,[62] and two showed increased risks that were not statistically significant.[38, 41] Using national health survey data, Mishra (2003)[62] found that elderly Indian men and women who lived in households using BMF had a higher prevalence of self-reported asthma compared to those who used cleaner fuels (OR=1.59, CI 1.30–1.94). Also in India, Padhi[63] found increased physician-diagnosed asthma and decline in lung function among rural biomass burners. In a recent meta-analysis, Po et al (2011)[51] found that pooled risk estimates did not provide evidence of overall increased risk of asthma in children or women using BMF. Limitations of the available studies include likely exposure misclassification, outcome misclassification, low power, and/or incomplete control for confounding.

Respiratory Infection

The WHO has concluded that exposure to indoor air pollution doubles the risk of pneumonia and other acute lower respiratory infections, and may account for half of the roughly 800,000 annual worldwide deaths in children under 5 years of age attributed to pneumonia.[25, 64] Dherani et al conducted a meta-analysis of 24 studies, and found that exposure to BMF increased the risk of pneumonia by almost 2-fold.[65] In a meta-analysis that included 8 studies, Po et al found a greater than 3 fold increased risk of acute respiratory infection in children.[51] Deaths among children contribute disproportionately to years of life lost in global burden-of-disease calculations. Increased rates of chronic bronchitis and viral infection have been associated with both gaseous and particulate ambient pollutants, although not specifically with TRAP.[4, 66]

Tuberculosis

Greater use of BMFs appears to be correlated with higher rates of TB infection in global geographic regions, but few studies have evaluated associations at the individual level. In a cross-sectional study of a large national sample of Indian households, Mishra 1999 found increased risk of self-reported TB infection with BMF use (OR 2.58, CI 1.98–3.37), but neither active nor passive smoking was measured. In a later well-designed case-control study in India, Shetty et al 2006 found that risk of bacteriologically or radiographically-confirmed TB was not increased after adjustment for smoking, education, income and other possible confounders.[67] Although a recent systematic review concluded that there was not sufficient epidemiological evidence to support an association between BMF and TB infection,[68] and no epidemiological associations between TRAP and TB have been reported, ambient particles and diesel exhaust particles have been shown to impair macrophage function in animal models, suggesting that such associations are biologically plausible.[69–70]

Mechanistic Insight

As discussed above, oxidative stress is a commonly cited mechanism for the relationship between air pollutants, many of them with oxidant constituents, and asthma worsening or onset. Both particles and gases may produce oxidative stress and may act in concert. Polymorphisms of GSTM1, GSTP1, and TNF-alpha are all reported to have associations/interactions with asthma and air pollution, but data are not consistent enough to allow firm causal conclusions. [71–72] Further support for oxidative stress as an explanatory mechanism as to how TRAP exerts effects on intracellular regulation of inflammation and the oxidative stress response comes from an experimental study of air pollution aerosols, including fresh diesel exhaust demonstrating approximately a 10% decease in WBC proteasome activity following 2 hr of aerosol exposure.[73]

Diesel exhaust inhalation is frequently used as a model for acute inhalation of TRAP. Acute exposure to diesel exhaust in a real-world street canyon setting has been shown to significantly reduce pulmonary function in asthmatics (up to 6% decline in FEV1) along with an increase in sputum inflammation as measured by myeloperoxidase[74]. However, experimental exposures to diesel exhaust, despite showing increased airway reactivity in asthmatics, have not elicited evidence of airway inflammation in asthmatic subjects, in surprising contrast to elicitation of inflammatory changes in healthy subjects.[75–77] Diesel exhaust particles have been shown to have adjuvant effects on IgE synthesis in atopics, so that allergen-specific IgE production upregulates by as much as 50-fold with a skew towards a TH2 profile.[78] Diesel exhaust has been shown to acutely produce human bronchial epithelial inflammation characterized by inflammatory cell recruitment, increased expression of vascular endothelial adhesion molecules, cytokines, mitogen-activated protein kinases, and transcription factors. It has been proposed that epithelial damage from diesel exhaust may lead to decreased mucociliary clearance and consequent increased access of allergens to immune cells in the mucosa.[76–77] Another recent study examined diesel exhaust produced under realistic conditions to simulate actual driving conditions and emissions.[79] Evaluating inflammatory markers 6 hours after a 1 hour exposure, they found increased expression of p-selectin ($p=0.036$) and vascular cell adhesion molecule-1 ($p=0.030$) in bronchial mucosal biopsies as well as the novel finding of increased eosinophils in bronchial alveolar lavage ($p=0.017$), not previously seen under idling engine conditions. The implications for diesel potentiation of allergic respiratory disease are substantial, especially in light of previous experimental work.[80]

To improve understanding of biological pathways underlying respiratory and cardiovascular effects, a number of panel or experimental studies have measured biomarkers of oxidative stress and inflammation following exposure to TRAP.[63] Examining respiratory effects associated with studies in commuters, there were modest effects of two hour commuting exposures on peak flow, eNO, and airway resistance.[81] Particle number (PN) doses were associated with decreased maximum mid-expiratory flow (MMEF) and FEV1 6 hours after exposure. PN and soot were associated with decreased MMEF and FEV1 immediately after exposure, and increased FeNO after car and bus but not bicycle trips. PN was also associated with an increase in airway resistance immediately but not 6 hours following exposure. There were no associations of exposures or doses with symptoms. They interpreted these findings in healthy individuals to show modest effects of a 2-hour in-traffic exposure on peak flow, eNO, and airway resistance. Examining inflammation and coagulation as a consequence of TRAP exposure, an accompanying study in the same subjects found no consistent associations in blood cell counts, CRP, IL-6, IL-8, IL-10, and TNFa, aPTT, fibrinogen, Factor VII, vWF, and CC 16 6 hours after the commute.[82] Thus these data do not indicate that short term changes predict the serious long-term consequences seen with chronic exposure.

Pollution Intervention studies

Beijing Olympics Intervention Studies

Natural (or politically organized) changes in the environment are viewed by researchers as great opportunities to study the effects on human health of greater than usual degrees of independent variable (pollution) change. This has been applied to the effects of sudden or dramatic changes in air pollution.[83–84] When these changes are anticipated, detailed clinical studies can be designed.[85] One such example was the Beijing Olympics of 2008. (see Figure 1) One study that came out of this examined visits for outpatient treatment of asthma at a Beijing Hospital.[86] During the Olympics, the Chinese government endeavored to reduce air pollution by substantial amounts.[87] While somewhat sparse in clinical detail, they reported a reduction from 12.5 visits per day to 7.3 visits per day, a 41.6% reduction during the Olympic Games.

Also based on the Beijing Olympics, Lin et al (2011)[88] measured serial FeNO as a function of ambient black carbon, a marker of diesel exhaust, in 36 fourth grade Beijing children, before, during, and after the 2008 Beijing Olympics.[88] FeNO was significantly lower during the Olympic period, and increased 16.6% (14.1%–19.2%) per interquartile range increase in BC, particularly in the first hours after exposure, suggesting rapid changes in inflammation. Asthmatics were not significantly different from healthy children.

Intervention Studies for BMF

Romieu (2009)[89] randomized an improved stove (Patsari stove) among 668 households in central Mexico where open wood-burning fires were used for cooking. The stoves had been shown in previous studies to reduce indoor air pollution levels by 70%.[90] At one-year follow-up, among the 50% of households still using the stove, there was a significant reduction in respiratory symptoms and a significantly lower decline in FEV1.[89] Using a randomized controlled trial, Smith et al found that an improved cooking stove halved average exposure to carbon monoxide, but did not significantly reduce physician-diagnosed pneumonia among infants in Guatemala.[91] However, there was a significant reduction in severe pneumonia, and a 50% reduction in exposure was significantly associated with a lower rate of diagnosis of pneumonia. Other randomized controlled trials of improved stoves are underway, but results are yet to be published (Bruce 2007).[92]

Clinical Guidance

Reducing the impact of BMF smoke and TRAP on respiratory health will require both public policy and the actions of individual patients. Consensus standards recognize the importance of air pollutants in the prevention and management of asthma and COPD, and have recommended that clinicians counsel patients to become aware of, and avoid, exposures to air pollution (GOLD and NHLBI Expert Panel 3 report on asthma). Interventions at the individual level may include recommendations by clinicians that patients avoid exercising or cycling near busy roadways to reduce exposure toTRAP, and to improve ventilation in homes were BMF are used. Public policy can encourage or mandate engineering solutions that drastically reduce emissions from cook stoves and vehicles, but adoption of new technologies can be slowed by lack of awareness of health risks, traditional cultural practices, and economic costs.[8] In some respects public health and regulatory approaches to traffic emissions may be considered the low hanging fruit for opportunities toward health improvement on a societal scale. “Experiments” such as the Beijing Olympics have demonstrated how such changes may have health implications.

Abbreviations

TRAP	Traffic-related air pollution
DC	Developed countries
LDC	Less developed countries
PM	Particulate Matter
BMF	Biomass fuel
LUR	Land use regression
GST	G;utathione S-Transferase
FeNO	Fractional exhaled Nitric Oxide

Acknowledgments

This work was supported in part from NIEHS P30 ES05022 and USEPA “STAR Grant RD 83457901. Photo credit to Jicheng Gong, M.S., UMDNJ Graduate School of Biomedical Sciences.

References

1. Beasley R. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. The Lancet. 1998; 351(9111):1225–1232.
2. D'Amato G, Cecchi L, D'Amato M, Liccardi G. Urban Air Pollution and Climate Change as Environmental Risk Factors of Respiratory Allergy: An Update. J Invest Allerg Clin. 2010; 20(2): 95–102.
3. Peden D, Reed CE. Environmental and occupational allergies. J Allerg Clinl Immunol. 2010; 125 Suppl 2(2):S150–S160.
4. Kelly FJ, Fussell JC. Air pollution and airway disease. Clin Exp Allergy. 2011 Epub ahead of print.
5. Salam MT, Islam T, Gilliland FD. Recent evidence for adverse effects of residential proximity to traffic sources on asthma. Curr Opin Pulm Med. 2008; 14(1):3–8. [PubMed: 18043269]
6. Torres-Duque C, Maldonado D, Perez-Padilla R, Ezzati M, Viegi G. Forum of International Respiratory Studies Task Force on Health Effects of Biomass E. Biomass fuels and respiratory diseases: a review of the evidence. Proc Am Thorac Soc. 2008; 5(5):577–590. [PubMed: 18625750]
7. Balmes JR. When smoke gets in your lungs. Proc Am Thorac Soc. 2010; 7(2):98–101. [PubMed: 20427578]

8. Fullerton DG, Bruce N, Gordon SB. Indoor air pollution from biomass fuel smoke is a major health concern in the developing world. Trans Royal Soc Trop Med Hygiene. 2008; 102(9):843–851.
9. Smith, KR.; Mehta, S.; Maeusezahl-Feuz, M. Indoor air-pollution from solid fuel use. In: Ezzatti, M.; Lopez, AD.; Rodgers, A.; Murray, CJL., editors. Comparative Quantification of Health Risks: Global and regional burden of diseases attributable to selected major risk factors. Geneva, Switzerland: World Health Organization; 2004. p. 1435-1493.
10. Reddy, A., editor. Prospects and challenges. New York: United Nations Development Programme; 1996. Energy after Rio.
11. Health Effects Institute. Traffic-related air pollution: A critical review of the literature on emissions, exposure and health effects. 2010 Special Report #17.
12. Zhang JJ, Smith KR. Household air pollution from coal and biomass fuels in China: measurements, health impacts, and interventions. Environ Health Perspect. 2007 Jun; 115(6):848–855. [PubMed: 17589590]
13. Naeher LP, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, Koenig JQ, et al. Woodsmoke health effects: a review. Inhal Toxicol. 2007; 19(1):67–106. [PubMed: 17127644]
14. Brook RD. Cardiovascular effects of air pollution. Clin Sci. 2008; 115:175–187. [PubMed: 18691154]
15. Brook RD, Rajagopalan S, Pope I, Arden C, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease An update to the scientific statement from the American Heart Association. Circulation. 2010; 121:2331–2378. [PubMed: 20458016]
16. Utell MJ, Frampton MW. Acute Health effects of ambient air pollution: the utlrafine hypothesis. J Aerosol Med. 2000; 13(4)
17. Zhou Y, Levy JI. The impact of urban street canyons on population exposure to traffic-related primary pollutants. Atmos Environ. 2008; 42(13):3087–3098.
18. Zhu Y, Hinds WC, Shen S, Sioutas C. Seasonal Trends of Concentration and Size Distribution of Ultrafine Particles Near Major Highways in Los Angeles. Aerosol Sci Technol. 2004; 38:5–13.
19. Gilbert NL, Woodhouse S, Stieb DM, Brook JR. Ambient nitrogen dioxide and distance from a major highway. Sci Total Environ. 2003; 312(1–3):43–46. [PubMed: 12873397]
20. Ezzati M, Kammen DM. The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs. Environ Health Perspect. 2002; 110(11): 1057–1068. [PubMed: 12417475]
21. Fruin SA, Winer AM, Rodes CE. Black carbon concentrations in California vehicles and estimation of in-vehicle diesel exhaust particulate matter exposures. Atmos Environ. 2004; 38(25): 4123–4133.
22. Zhu Y, Fung DC, Kennedy N, Hinds WC, Eiguren-Fernandez A. Measurements of ultrafine particles and other vehicular pollutants inside a mobile exposure system on Los Angeles freeways. J Air Waste Manag Assoc. 2008; 58(3):424–434. [PubMed: 18376645]
23. Zuurbier M, Hoek G, van den Hazel P, Brunekreef B. Minute ventilation of cyclists, car and bus passengers: an experimental study. Environmental Health: A Global Access Science Source. 2009; 8:48. [PubMed: 19860870]
24. US Environmental Protection Agency. Air Quality Criteria for Particulate Matter. Washington DC: EPA; 2004. [updated 2004; cited]; Available from: http://www.epa.gov/EPA-AIR/2004/October/Day-29/a24232.htm
25. Organization, WH. Centre, WM., editor. Air quality and health. 2011. WHO Fact Sheet No 313
26. Smith KR. Fuel Combustion, Air-Pollution Exposure, and Health - the Situation in Developing-Countries. Annu Rev Energy Environ. 1993; 18:529–566.
27. Briggs DJ, de Hoogh C, Gulliver J, Wills J, Elliott P, Kingham S, et al. A regression-based method for mapping traffic-related air pollution: application and testing in four contrasting urban environments. Sci Total Environ. 2000; 253(1–3):151–167. [PubMed: 10843339]
28. Brauer M, Hoek G, van Vliet P, Meliefste K, Fischer P, Gehring U, et al. Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems. Epidemiol. 2003; 14(2):228–239.
29. Gilliland F, Avol E, Kinney P, Jerrett M, Dvonch T, Lurmann F, et al. Air Pollution Exposure Assessment for Epidemiologic Studies of Pregnant Women and Children: Lessons Learned from

the Centers for Childrenâ€™s Environmental Health and Disease Prevention Research. Environ Health Perspect. 2005; 113(10):1447. [PubMed: 16203261]

30. Gehring U, Wilga AH, Brauer M, Fischer P, deJongste JC, Kerkhof M, et al. Traffic-related air pollution and the develoment of asthma and allergies during the first 8 years of life. Am J Resp Crit Care Med. 2010; 181:596–603. [PubMed: 19965811]
31. Carlsten C, Dybuncio A, Becker A, Chan-Yeung M, Brauer M. Traffic-related air pollution and incident asthma in a high-risk birth cohort. Occup Environ Med. 2011; 68(4):291–295. [PubMed: 20952556]
32. Jerrett M, Burnett RT, Ma R, Pope CA III, Krewski D, Newbold KB, et al. Spatial Analysis of Air Pollution and Mortality in Los Angeles. Epidemiol. 2005; 16(6):727–736.
33. Ryan PH, Lemasters GK, Biswas P, Levin L, Hu S, Lindsey M, et al. A comparison of proximity and land use regression traffic exposure models and wheezing in infants. Environ Health Perspect. 2007; 115(2):278–284. [PubMed: 17384778]
34. Pruss-Ustun A, Bonjour S, Corvalan C. The impact of the environment on health by country: a meta-synthesis. Environ Health. 2008; 7:7. [PubMed: 18298819]
35. Rehfuess E, Corvalan C, Neira M. Indoor air pollution: 4000 deaths a day must no longer be ignored. Bull World Health Organ. 2006; 84(7):508. [PubMed: 16878216]
36. Fagbule D, Ekanem EE. Some environmental risk factors for childhood asthma: a case-control study. Ann Trop Paed. 1994; 14(1):15–19.
37. Behera D, Sood P, Singh S. Passive smoking, domestic fuels and lung function in north Indian children. Indian J Chest Dis Allied Sci. 1998; 40(2):89–98. [PubMed: 9775566]
38. Behera D, Chakrabarti T, Khanduja KL. Effect of exposure to domestic cooking fuels on bronchial asthma. Indian J Chest Dis Allied Sci. 2001; 43(1):27–31. [PubMed: 11370503]
39. Noorhassim I, Rampal KG, Hashim JH. The relationship between prevalence of asthma and environmental factors in rural households. Med J Malaysia. 1995; 50(3):263–267. [PubMed: 8926906]
40. Melsom T, Brinch L, Hessen JO, Schei MA, Kolstrup N, Jacobsen BK, et al. Asthma and indoor environment in Nepal. Thorax. 2001; 56(6):477–481. [PubMed: 11359965]
41. Golshan M, Faghihi M, Marandi MM. Indoor women jobs and pulmonary risks in rural areas of Isfahan, Iran, 2000. Resp Med. 2002; 96(6):382–388.
42. Qureshi KA. Domestic smoke pollution and prevalence of chronic bronchitis/asthma in a rural area of Kashmir. Indian J Chest Dis Allied Sci. 1994; 36(2):61–72. [PubMed: 7851950]
43. Uzun K, Ozbay B, Ceylan E, Gencer M, Zehir I. Prevalence of chronic bronchitis-asthma symptoms in biomass fuel exposed females. Environ Health Prev Med. 2003; 8:13–17. [PubMed: 21432110]
44. Orozco-Levi M, Garcia-Aymerich J, Villar J, Ramirez-Sarmiento A, Anto JM, Gea J. Wood smoke exposure and risk of chronic obstructive pulmonary disease. Eur Respir J. 2006; 27(3):542–546. [PubMed: 16507854]
45. McConnell R, Islam T, Shankardass K, Jerrett M, Lurmann F, Gilliland F, et al. Childhood incident asthma and traffic-related air pollution at home and school. Environ Health Perspect. 2010; 118(7):1021–1026. [PubMed: 20371422]
46. Zuraimi MS, Tham K-W, Chew F-T, Ooi P-L, Koh D. Home air-conditioning, traffic exposure, and asthma and allergic symptoms among preschool children. Ped Allergy Immunol. 2011; 22(1pt2):e112–e118.
47. Penard-Morand C, Raherison C, Charpin D, Kopferschmitt C, Lavaud F, Caillaud D, et al. Long-term exposure to close-proximity air pollution and asthma and allergies in urban children. Eur Respir J. 2010; 36:33–40. [PubMed: 20075054]
48. Kunzli N, Bridevaux P-O, Liu L-JS, Garcia-Esteban R, Schindler C, Gerbase MW, et al. Traffic-related air pollution correlates with adult-onset asthma among never-smokers. Thorax. 2009; 64:664–670. [PubMed: 19359271]
49. Trupin L, Balmes JR, Chen H, Eisner MD, Hammond SK, Katz PP, et al. An integrated model of environmental factors in adult asthma lung function and disease severity: a cross-sectional study. Environ Health. 2010; 9:24. [PubMed: 20487557]

50. Kurmi OP, Semple S, Simkhada P, Smith WC, Ayres JG, Kurmi OP, et al. COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis. Thorax. 2010; 65(3):221–228. [PubMed: 20335290]
51. Po JYT, FitzGerald JM, Carlsten C. Respiratory disease associated with solid biomass fuel exposure in rural women and children: systematic review and meta-analysis. Thorax. 2011; 66(3): 232–239. [PubMed: 21248322]
52. Hu G, Zhou Y, Tian J, Yao W, Li J, Li B, et al. Risk of COPD from exposure to biomass smoke: a metaanalysis. Chest. 2010; 138(1):20–31. [PubMed: 20139228]
53. Regalado J, Perez-Padilla R, Sansores R, Paramo Ramirez JI, Brauer M, Pare P, et al. The effect of biomass burning on respiratory symptoms and lung function in rural Mexican women. Am J Respir Crit Care Med. 2006; 174(8):901–905. [PubMed: 16799080]
54. Jaakkola MS, Jaakkola JJK. Biomass fuels and health: the gap between global relevance and research activity. Am J Respir Crit Care Med. 2006; 174(8):851–852. [PubMed: 17021357]
55. Mackay, J.; Eriksen, MP. The Tobacco Atlas [1 atlas (128 p)]. Geneva: World Health Organization; 2002. World Health Organization, cartographer^cartographers.
56. Ekici A, Ekici M, Kurtipek E, Akin A, Arslan M, Kara T, et al. Obstructive airway diseases in women exposed to biomass smoke. Environ Res. 2005; 99(1):93–98. [PubMed: 16053933]
57. Eisner MD, Anthonisen N, Coultas D, Kuenzli N, Perez-Padilla R, Postma D, et al. An Official American Thoracic Society Public Policy Statement: Novel Risk Factors and the Global Burden of Chronic Obstructive Pulmonary Disease. Am J Respir Crit Care Med. 2010; 182(5):693–718. [PubMed: 20802169]
58. Andersen ZJ, Hvidberg M, Jensen SS, Ketzel M, Loft S, Sorensen M, et al. Chronic Obstructive Pulmonary Disease and Long-Term Exposure to Traffic-related Air Pollution: A Cohort Study. Am J Respir Crit Care Med. 2011; 183(4):455–461. [PubMed: 20870755]
59. Weisel CP, Cody RP, Lioy PJ. Relationship between summertime ambient ozone levels and emergency department visits for asthma in central New Jersey. Environ Health Perspect. 1995; 103 Suppl 2:97–102. [PubMed: 7614954]
60. McConnell R, Islam T, Shankardass K, Jerrett M, Lurmann F, Gilliland F, et al. Childhood incident asthma and traffic-related air pollution at home and school. Environ Health Perspect. 2010; 118(7):1021–1026. [PubMed: 20371422]
61. Barry AC, Mannino DM, Hopenhayn C, Bush H, Barry AC, Mannino DM, et al. Exposure to indoor biomass fuel pollutants and asthma prevalence in Southeastern Kentucky: results from the Burden of Lung Disease (BOLD) study. J Asthma. 2010; 47(7):735–741. [PubMed: 20716015]
62. Mishra V. Effect of indoor air pollution from biomass combustion on prevalence of asthma in the elderly. Environ Health Perspect. 2003; 111(1):71–78. [PubMed: 12515681]
63. Laumbach RJ, Kipen HM. Acute effects of motor vehicle traffic-related air pollution exposures on measures of oxidative stress in human airways. Ann N Y Acad Sci. 2010; 1203:107–112. [PubMed: 20716291]
64. O'Brien KL, Wolfson LJ, Watt JP, Henkle E, Deloria-Knoll M, McCall N, et al. Burden of disease caused by Streptococcus pneumoniae in children younger than 5 years: global estimates. Lancet. 2009; 374(9693):893–902. [PubMed: 19748398]
65. Dherani M, Pope D, Mascarenhas M, Smith KR, Weber M, Bruce N. Indoor air pollution from unprocessed solid fuel use and pneumonia risk in children aged under five years: a systematic review and meta-analysis. Bull World Health Organ. 2008; 86(5):390–398. [PubMed: 18545742]
66. Cienciwicki J, Jaspers I. Air pollution and respiratory viral infection. Inhal Toxicol. 2007; 19(14): 1135–1146. [PubMed: 17987465]
67. Shetty N, Shemko M, Vaz M, D'Souza G. An epidemiological evaluation of risk factors for tuberculosis in South India: a matched case control study. Int J Tuberc Lung Dis. 2006; 10(1):80–86. [PubMed: 16466042]
68. Slama K, Chiang CY, Hinderaker SG, Bruce N, Vedal S, Enarson DA. Indoor solid fuel combustion and tuberculosis: is there an association? Int J Tuberc Lung Dis. 2010; 14(1):6–14. [PubMed: 20003689]

69. Zhou H, Kobzik L. Effect of concentrated ambient particles on macrophage phagocytosis and killing of Streptococcus pneumoniae. Am J Respir Cell Mol Biol. 2007; 36(4):460–465. [PubMed: 17079778]
70. Yang HM, Antonini JM, Barger MW, Butterworth L, Roberts BR, Ma JK, et al. Diesel exhaust particles suppress macrophage function and slow the pulmonary clearance of Listeria monocytogenes in rats. Environ Health Perspect. 2001; 109(5):515–521. [PubMed: 11401764]
71. London SJ. Gene-Air pollution interactions in asthma. Proc Am Thorac Soc. 2007; 4:217–220. [PubMed: 17607002]
72. Romieu I, Moreno-Macias H, London SJ. Gene by environment interaction and ambient air pollution. Proc Am Thorac Soc. 2010; 7(2):116–122. [PubMed: 20427582]
73. Kipen HM, Gandhi S, Rich DQ, Ohman-Strickland P, Laumbach R, Fan Z-H, et al. Acute decreases in proteasome pathway activity after inhalation of fresh diesel exhaust or secondary organic aerosol. Environ Health Perspect. 2011; 119(5):658–663. [PubMed: 21163722]
74. McCreanor J, Cullinan P, Nieuwenhuijsen MJ, Stewart-Evans J, Malliarou E, Jarup L, et al. Respiratory effects of exposure to diesel traffic in persons with asthma. New England Journal of Medicine. 2007; 357(23):2348–2358. [PubMed: 18057337]
75. Stenfors N, Nordenhall C, Salvi SS, Mudway I, Soderberg M, Blomberg A, et al. Different airway inflammatory responses in asthmatic and healthy humans exposed to diesel. Eur Respir J. 2004; 23(1):82–86. [PubMed: 14738236]
76. Nordenhall C, J P, Ledin M-C, Levin J-O, TS EA. Diesel exhaust enhances airway responsiveness in asthmatic subjects. Eur Resp J. 2001; 17:909–915.
77. Behndig AF, Larsson N, Brown JL, et al. Proinflammatory doses of diesel exhaust in healthy subjects fail to elicit equivalent or augmented airway inflammation in subjects with asthma. Thorax. 2011; 66(1):12–19. [PubMed: 20837873]
78. Diaz-Sanchez D. The role of diesel exhaust particles and their associated polyaromatic hydrocarbons in the induction of allergic airway disease. Allergy. 1997; 52 Suppl(38):52–56. [PubMed: 9208060]
79. Sehlstedt M, Behndig AF, Boman C, Blomberg A, Sandström T, Pourazar J. Airway inflammatory response to diesel exhaust generated at urban cycle running conditions. Inhalation Toxicol. 2010; 22(14):1144–1150.
80. Diaz-Sanchez D, AR D, H T, Saxon A. Diesel Exhaust Particles Induce Local IgE Production In Vivo and Alter the Pattern of IgE Messenger RNA Isoforms. 1994; 94:1417–1425.
81. Zuurbier M, Hoek G, Oldenwening M, Meliefste K, van den Hazel P, Brunekreef B. Respiratory Effects of Commuters' Exposure to Air Pollution in Traffic. Epidemiol. 2011; 22(2):219–227.
82. Zuurbier M, Hoek G, Oldenwening M, Meliefste K, Krop E, van den Hazel P, et al. In traffic air pollution exposure and CC16, blood coagulation, and inflammation markers in healthy adults. Environ Health Perspect. 2011; 119(10):1384–1389. [PubMed: 21665568]
83. Clancy L, Goodman P. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. Lancet. 2002; 360:1210–1214. [PubMed: 12401247]
84. Peel JL, Klein M, Flanders WD, Mulholland JA, Tolbert PE. Impact of improved air quality during the 1996 Summer Olympic Games in Atlanta on multiple cardiovascular and respiratory outcomes. Res Rep Health Eff Inst. 2010 ApR.(148):3–23. discussion 5–33. [PubMed: 20575278]
85. Kipen HRD, Huang W, Zhu T, Wang G, Hu M, Lu S, Ohman-Strickland P, Zhu P, Wang Y, Zhang J. Measurement of inflammation and oxidative stress following drastic changes in air pollution during the Beijing Olympics: a panel study approach. Ann N Y Acad Sci. 2010; 1203:160–167. [PubMed: 20716299]
86. Li Y, Wang W, Kan H, Xu X, Chen B. Air quality and outpatient visits for asthma in adults during the 2008 Summer Olympic Games in Beijing. Sci Total Environ. 2010; 408(5):1226–1227. [PubMed: 19959207]
87. Cai H, Xie S. Traffic-related air pollution modeling during the 2008 Beijing Olympic Games: The effects of an odd-even day traffic restriction scheme. Sci Total Environ. 2011; 409(10):1935–1948. [PubMed: 21353290]

88. Lin, W.; Zhu, T.; Huang, W.; Hu, M.; Brunekreff, B.; Zhang, Y., et al. Acute respiratory inflammation in children and black carbon in ambient air before and during the 2008 Beijing Olympics. Environ Health Perspect. 2011. (available at http://dx.doi.org/) Online 3 June 2011

89. Romieu I, Riojas-Rodriguez H, Marron-Mares AT, Schilmann A, Perez-Padilla R, Masera O. Improved biomass stove intervention in rural Mexico: impact on the respiratory health of women. Am J Respir Crit Care Med. 2009; 180(7):649–656. [PubMed: 19556519]

90. Armendariz-Arnez C, Edwards RD, Johnson M, Zuk M, Rojas L, Jimenez RD, et al. Reduction in personal exposures to particulate matter and carbon monoxide as a result of the installation of a Patsari improved cook stove in Michoacan Mexico. Indoor Air. 2008; 18(2):93–105. [PubMed: 18333989]

91. Smith KR, McCracken JP, Weber MW, Hubbard A, Jenny A, Thompson LM, et al. Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial. Lancet. 2011; 378(9804):1717–1726. [PubMed: 22078686]

92. Bruce N, Weber M, Arana B, Diaz A, Jenny A, Thompson L, et al. Pneumonia case-finding in the RESPIRE Guatemala indoor air pollution trial: standardizing methods for resource-poor settings. Bull World Health Organ. 2007; 85(7):535–544. [PubMed: 17768502]

Figure 1.
Cooking with wood biomass fuel in Nigeria.

2A

2B

Figure 2.

A and B Two photographs in Beijing taken from the same vantage point and time of day. Figure 2A was taken in early June of 2008, while the one on Figure 2B was taken in mid-July during the height of the Olympic reductions in heavy industry, power generation, construction, truck traffic, and a 50% reduction in automobile traffic.