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OBSTRUCTIVE LUNG DISEASE AND EXPOSURE TO BURNING BIOMASS FUEL IN THE INDOOR ENVIRONMENT

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Abstract

It is estimated that up to half of the world's population burns biomass fuel (wood, crop residues, animal dung and coal) for indoor uses such as cooking, lighting and heating. As a result, a large proportion of women and children are exposed to high levels of household air pollution (HAP). The short and long term effects of these exposures on the respiratory health of this population are not clearly understood. On May 9–11, 2011 NIH held an international workshop on the "Health Burden of Indoor Air Pollution on Women and Children," in Arlington, VA. To gather information on the knowledge base on this topic and identify research gaps, ahead of the meeting we conducted a literature search using PubMed to identify publications that related to HAP, asthma, and chronic obstructive pulmonary disease (COPD). Abstracts were all analyzed and we report on those considered by the respiratory sub study group at the meeting to be most relevant to the field. Many of the studies published are symptom-based studies (as opposed to objective measures of lung function or clinical examination etc.) and measurement of HAP was not done. Many found some association between indoor exposures to biomass smoke as assessed by stove type (e.g., open fire vs. liquid propane gas) and respiratory symptoms such as wheeze and cough. Among the studies that examined objective measures (e.g. spirometry) as a health outcome, the data supporting an association between biomass smoke exposure and COPD in adult women are fairly robust, but the findings for asthma are mixed. If an association was observed between the exposures and lung function, most data seemed to demonstrate mild to moderate reductions in

lung function, the pathophysiological mechanisms of which need to be investigated. In the end, the group identified a series of scientific gaps and opportunities for research that need to be addressed to better understand the respiratory effects of exposure to indoor burning of the different forms of biomass fuels.

Keywords

Biomass smoke; cooking fuel smoke; Indoor air pollution; COPD; asthma

BACKGROUND and METHODS

The leading environmental cause of death worldwide is household air pollution (HAP), and a major contributor to HAP is use of biomass and coal as fuels for cooking and heating [1]. For example meta-analyses of global epidemiologic studies suggest that HAP from solid fuel use in China is responsible for approximately 420,000 premature deaths annually, more than the approximately 300,000 attributed to urban outdoor air pollution in the country [2]. The combined current rates of smoking and solid-fuel use are predicted to be responsible for 65 million deaths from chronic obstructive pulmonary disease (COPD) between 2003 and 2033 in China [3]. Here the prevalence of COPD was significantly higher in rural residents, elderly patients, smokers, in those with lower body mass index, less education, and poor ventilation in their homes, and occupational exposures to dusts or biomass fuels, and in those with pulmonary problems in childhood and family history of pulmonary diseases[4].

In low- and middle-income countries (LMIC) especially, women and children have the highest exposures to HAP. In order to determine research needs to understand risks of HAP for women and children, the NIH convened a workshop of international experts "Health Burden of Indoor Air Pollution on Women and Children," on May 9–11, 2011 to review the existing evidence base, and to make recommendation to guide future research efforts. Working subgroups were organized thematically (e.g., cardiovascular effects, cancer, ocular injury, etc.) and one group was tasked with evaluating evidence for links between HAP and obstructive lung diseases (COPD and asthma).

In preparation for the international workshop, we conducted a literature search combining terms for exposures: "Indoor air pollution, biomass smoke and cooking fuel smoke" with disease "COPD, asthma and chronic lung disease". More than 1,450 publications were found covering literature from the early 1990s to the current time, and their content and significance were discussed among the participants of the respiratory committee at the meeting. Most of the relevant studies examined were from LMIC, where the widespread use of biomass fuel such as wood, charcoal, crop residues, and dried animal excrements, leads to the release and accumulation in the indoor environment of multiple compounds similar to those present in tobacco smoke [5–9]. In the process of evaluating the publications, we found that there were relatively few papers providing high quality causal evidence for biomass effects in LMIC. For that reason, we also considered some evidence from high income countries and from non-biomass fuel types, where lessons could be drawn about pollutants that generally result from combustion (e.g., nitrogen oxides).

RESULTS

Because of the tight connection of biomass fuel indoor burning with domestic daily activities, we found substantial evidence that the population subjected to the highest exposure is children and women [8, 10–16]. When measured, pollutant concentrations were

typically high. For example, the particulate matter released by burning solid biomass fuel reached several milligrams per cubic meter [8, 17–19].

Association of COPD and Household Air Pollution

Several case-control and cross-sectional studies have found a consistent association between biomass burning and respiratory symptoms. Studies showed that subjects exposed to biomass fumes experience chronic bronchitis and chronic airflow obstruction [20–24] and one study found a link between chronic domestic wood smoke inhalation and the development of *cor pulmonale* [25]. A recent publication from Brazil evaluated the correlation of exposure to fine particulate matter (PM) (< 2.5 µm in aerodynamic diameter or PM_{2.5}) emitted from partial combustion of biomass fuel and lung function compared to a group from the same community that was using liquefied petroleum gas. The data showed that exposure to biomass alone was associated with increased prevalence of respiratory symptoms, reduced lung function and development of COPD. Moreover, these effects were associated with the duration and magnitude of exposure, and are exacerbated by tobacco smoke [26]. A recent meta-analysis of 15 epidemiologic studies covering a wide range of countries found that people exposed to biomass smoke had combined odds ratio (OR) of 2.44 for developing COPD as assessed by lung function measurements or symptom-diagnosed chronic bronchitis, and that this was true for both women and men. Moreover cigarette smoking appeared to have a synergistic effect with biomass smoke, increasing the OR for COPD development to 4.39 [27]. Importantly, another observational study showed, retrospectively, that homes where people had undertaken simple ventilation measures had a lower incidence of COPD [28]. Because this retrospective analysis did not measure exposures after changes in ventilation it is not possible to determine the efficacy of specific interventions such as simply adding a chimney vs. more complex modernized bio-energy programs. This needs to be carefully assessed and can have an impact only with coordinated support from governmental and commercial sectors [29].

Association of Asthma and Household Air Pollution

Background—There are several studies that address whether or not indoor cooking methods or certain cooking fuels affect the risk of developing asthma, and separately, whether these exposures can aggravate existing disease. The study designs include survey, cross-sectional and case control. Multiple different definitions of asthma were used across studies and most exposure measures were reported by survey, though a few studies included physical measures of particles and gases. Most studies included either adults or children, while a few included both adults and children. The studies represented here originated from a wide range of countries in various stages of socio-economic developments: China, Iran, India, Guatemala, Nepal, Poland, USA, Great Britain, Australia, and The Netherlands.

Evidence of asthma risk in adults—Studies examining biomass and adult asthma are not uniformly positive and the associations with specific fuel type are inconsistent. A cross sectional study of more than 4,000 adults in rural China, a history of asthma was less common when improved stoves or “traditional biomass” was used for cooking rather than coal, though the values did not reach statistical significance [30]. When specific fuels were examined in multivariate models, there was an increased risk for the development of asthma when coal rather than wood was used. Other surveys in China found an increased risk of asthma diagnosis when coal was used for cooking [31, 32]. Though the first of these two studies only examined men, a survey of 508 adults in Southeastern Kentucky found a significant association with asthma when wood and coal were both used in cooking, but there was no additional risk when just wood or just coal was used [33]. A recent population survey study from India indicates that adult women living in households using biomass and solid fuels have a significantly higher risk of asthma than those living in households using

cleaner fuels (OR: 1.26; 95%CI: 1.06–1.49; $p = .010$), even after controlling for the effects of a number of potentially confounding factors [34].

When other fuels, such as gas, are considered, the findings are mixed. Studies performed in Copenhagen [35], and New York State [36], for example showed no association between asthma and use of gas stoves. A survey that included not only adults, but also children, was conducted in Iran where traditional Persian stoves are used. This study reported that current asthma was associated with bread baking and the use of kerosene or gas as fuels [37]. In a Polish study that only included women over 65 years of age, increased frequency and duration of gas cooking were each associated with asthma diagnosis [38]. Thus, these studies do not provide a clear positive association for specific cooking fuels and risk of asthma in adults.

Interaction of asthma risk by gender—In two studies, analyses were stratified by sex, which demonstrated greater risks for women than for men. A survey in East Anglia found evidence of an interaction by sex, with asthma attacks and use of asthma medications higher in women who cooked with gas stoves, but that risk was not found for men [39]. A similar interaction was found in a cross sectional study from India that examined asthma rates by type of fuel used in cooking [40]. Compared with use of cleaner fuels (e.g. LPG), use of biomass or a mixture of fuels were each associated with asthma in women, and similar findings emerged from another, more recent, study [34]. In men, the risks were lower than for women, and in the case of a mixture of fuels, not statistically significant. Thus, the small number of studies that considered adult gender difference, suggest that the risk of biomass for asthma in adults, if any exist, may be confined to women, among the reasons possibly being type of exposure, its duration, and respiratory system anatomical differences.

Evidence of asthma risk in children—Children may be more vulnerable to the effects of air pollution and several studies have examined the use of fuels and asthma prevalence in children. As with adults, though, the evidence is mixed. A single study in 1,505 children in eastern India showed a strong association of asthma with use of biomass as fuel [41]. Furthermore, this study showed that measured pollutants [CO, CO₂, NO, NO₂, SO₂, O₃ and PM] were higher in the homes using biomass compared with those using LPG. On the other hand, a study performed in 4 Chinese cities of 7,058 children examined lifetime exposure to cooking coal smoke [30] and when categorized as light, moderate or heavy exposure, there was no differential association with asthma. Finally, two recent studies, one in Guatemala and one in Peru, compared cooking methods (open fire vs. improved stoves) or biomass exposure and did not show a significant association with prevalence or severity of asthma symptoms [42, 43].

While some studies in children examined exposure to fuels other than biomass, there was mixed support of an association with asthma. A few studies reported a positive association of gas cooking with childhood asthma [44–46], but several others demonstrated the absence of a significant association [36, 41, 47, 48]. In one of the studies showing a positive association of gas cooking with asthma [45], the NO₂ concentrations in the home were not associated with asthma, which suggests the possible alternative role of an unmeasured confounder. A cross sectional study in Hong Kong compared the effect of cooking with gas in areas with high vs. low outdoor pollution. Frequency of cooking with gas was significantly associated with asthma only in the area with low outdoor pollution, which suggests an important interaction [49]. Thus, there is not a consistent signal to implicate indoor biomass, or other fuels, as a cause for childhood asthma. One study suggested the role of indoor cooking fuel may only be relevant in regions where the outdoor air is relatively cleaner [49].

Evidence for asthma exacerbation in adults—There is a lack of evidence for whether or not indoor biomass use contributes to worse morbidity and exacerbations in adults with asthma. The evidence from five studies is mixed for use of other fuels and exacerbations in adults. In a panel study of 164 adults with asthma in California, gas stove use was significantly associated with nocturnal symptoms, physician or emergency room visits and missing work [50]. Other studies, including a cohort [51], and cross sectional surveys [52, 53] have not shown associations with gas stove use and lung function, symptoms and quality of life. In a study of 100 women with asthma in India, peak flow was shown to be lower after cooking, compared to before cooking, when the fuel of LPG or biomass was used [54]. Taken together, though, the evidence is lacking to demonstrate the impact of biomass and other fuels on exacerbations of asthma in adults.

Evidence for asthma exacerbation in children—While it is not possible to find evidence for the effects of indoor biomass on asthma exacerbations in children, there is consistent evidence for an impact by other types of fuels. For example, a cross sectional study of children under 12 years old with active asthma in Connecticut showed that NO₂ was higher in homes with a gas stove [55]. Furthermore, exposure to gas stoves and higher NO₂ were both associated with more frequent respiratory symptoms. A panel study of 2–6 year old children in Baltimore examined presence of a gas stove and in home NO₂ [56] and found that higher NO₂ concentrations were associated with greater asthma symptoms, including cough and nighttime awakening. This study also demonstrated that higher NO₂ was associated with use of the gas stove. A cross-sectional study of 8–16 year olds in the U.S., examined gas stove use and lung function [57]. In asthmatic girls, use of a gas stove was associated with lower lung function when not using prescription respiratory medication. Among girls who used respiratory medication and among boys, regardless of whether or not they used respiratory medications, there was no association of gas stove use and lower lung function. Thus, overall there is supportive evidence for exacerbation of asthma in children by gas stove use, though there is no direct evidence to implicate indoor biomass fuel burning.

Household Air Pollution, Asthma, COPD, and exacerbations summary

To date, there is moderately strong and consistent evidence to support associations between domestic use of solid biomass fuels (wood, crop residues, animal dung and coal) and the development of COPD. Studies have shown links between these indoor exposures with the diagnosis of COPD as well as symptoms of the disease. However, whether indoor air pollution is associated with COPD progression or worse morbidity including exacerbations among patients with COPD remains unclear. On the other hand, studies provide conflicting evidence of an association between cooking fuels and methods, and development of asthma whether in children or adults [15, 26]. With regard to asthma exacerbations, conflicting results have been reported for biomass in children and adults. Gas stoves, though, have been shown to cause asthma exacerbations in pediatric populations.

Recommendations for Future Research: Gaps and Scientific Opportunities

The expert group considered not only amount, but also the quality of the available evidence. Given the great importance of potential links between indoor biomass use and obstructive lung disease, we concluded that additional research is needed, especially utilizing enhanced methodologies. Future studies considerations should include:

- Large cohort studies (longitudinal) with sufficient sample size and follow-up time to establish potentially causative relationships between HAP and obstructive lung disease.

- Use of direct measures of pollutant exposures, ideally at the level of the individual study subject as questionnaires may not bear strong reliability.

Studies that examine the long-term effects of *in utero* exposures to determine the effect of HAP on early life origin of disease.

Surveillance for asthma and COPD prevalence and incidence to assess whether disease patterns are changing over time and geographic region in association with variations of fuel use.

Use of objective measures of disease (such as spirometry) to complement reports of symptoms.

Longitudinal assessments including spirometry to demonstrate whether or not any observed changes are variable, reversible or progress to lung diseases.

Development of biomarkers of exposure that could make large scale studies more feasible to conduct.

Collection of robust data on potential confounding factors and effect modifiers
Attention to inclusion of most relevant and vulnerable populations, including women and children.

Field intervention trials that demonstrate that modification of exposures can prevent or reverse disease.

References

1. WHO, GLOBAL HEALTH RISKS:Mortality and burden of disease attributable to selected major risks. 2009.
2. Zhang JJ, Smith KR. Household air pollution from coal and biomass fuels in China: measurements, health impacts, and interventions. *Environ Health Perspect.* 2007; 115(6):848–855. [PubMed: 17589590]
3. Lin HH, et al. Effects of smoking and solid-fuel use on COPD, lung cancer, and tuberculosis in China: a time-based, multiple risk factor, modelling study. *Lancet.* 2008; 372(9648):1473–1483. [PubMed: 18835640]
4. Zhong N, et al. Prevalence of chronic obstructive pulmonary disease in China: a large, population-based survey. *Am J Respir Crit Care Med.* 2007; 176(8):753–760. [PubMed: 17575095]
5. Smith, KR. Biofuels, air pollution, and health: A Global Review. Plenum Press; New York: 1987.
6. Smith KR. National burden of disease in India from indoor air pollution. *Proc Natl Acad Sci U S A.* 2000; 97(24):13286–13293. [PubMed: 11087870]
7. Fullerton DG, Bruce N, Gordon SB. Indoor air pollution from biomass fuel smoke is a major health concern in the developing world. *Trans R Soc Trop Med Hyg.* 2008; 102(9):843–851. [PubMed: 18639310]
8. Salvi S, Barnes PJ. Is exposure to biomass smoke the biggest risk factor for COPD globally? *Chest.* 2010; 138(1):3–6. [PubMed: 20605806]
9. Kurmi OP, Lam KB, Ayres JG. Indoor air pollution and the lung in low and medium income countries. *Eur Respir J.* 2012
10. Rinne ST, et al. Relationship of pulmonary function among women and children to indoor air pollution from biomass use in rural Ecuador. *Respir Med.* 2006; 100(7):1208–1215. [PubMed: 16318916]
11. Saha A, et al. Pulmonary function and fuel use: a population survey. *Respir Res.* 2005; 6:127. [PubMed: 16255784]
12. Ellegard A. Cooking fuel smoke and respiratory symptoms among women in low-income areas in Maputo. *Environ Health Perspect.* 1996; 104(9):980–985. [PubMed: 8899378]
13. Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest.* 1991; 100(2):385–388. [PubMed: 18641111]
14. Behera D, Jindal SK, Malhotra HS. Ventilatory function in nonsmoking rural Indian women using different cooking fuels. *Respiration.* 1994; 61(2):89–92. [PubMed: 8008994]

15. Po JY, 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]
16. Kodgule R, Salvi S. Exposure to biomass smoke as a cause for airway disease in women and children. *Curr Opin Allergy Clin Immunol*. 2012; 12(1):82–90. [PubMed: 22157154]
17. Albalak R, Frisancho AR, Keeler GJ. Domestic biomass fuel combustion and chronic bronchitis in two rural Bolivian villages. *Thorax*. 1999; 54(11):1004–1008. [PubMed: 10525559]
18. Regalado J, 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]
19. Liu S, et al. Biomass fuels are the probable risk factor for chronic obstructive pulmonary disease in rural South China. *Thorax*. 2007; 62(10):889–897. [PubMed: 17483137]
20. Pandey MR. Domestic smoke pollution and chronic bronchitis in a rural community of the Hill Region of Nepal. *Thorax*. 1984; 39(5):337–339. [PubMed: 6740536]
21. Menezes AM, Victora CG, Rigatto M. Prevalence and risk factors for chronic bronchitis in Pelotas, RS, Brazil: a population-based study. *Thorax*. 1994; 49(12):1217–1221. [PubMed: 7878555]
22. Dennis RJ, et al. Woodsmoke exposure and risk for obstructive airways disease among women. *Chest*. 1996; 109(1):115–119. [PubMed: 8549171]
23. Orozco-Levi M, et al. Wood smoke exposure and risk of chronic obstructive pulmonary disease. *Eur Respir J*. 2006; 27(3):542–546. [PubMed: 16507854]
24. Akhtar T, et al. Chronic bronchitis in women using solid biomass fuel in rural Peshawar, Pakistan. *Chest*. 2007; 132(5):1472–1475. [PubMed: 17646238]
25. Sandoval J, et al. Pulmonary arterial hypertension and cor pulmonale associated with chronic domestic woodsmoke inhalation. *Chest*. 1993; 103(1):12–20. [PubMed: 8417864]
26. da Silva LF, et al. Impaired lung function in individuals chronically exposed to biomass combustion. *Environ Res*. 2012; 112:111–117. [PubMed: 22136759]
27. Hu G, et al. Risk of COPD from exposure to biomass smoke: a metaanalysis. *Chest*. 2010; 138(1):20–31. [PubMed: 20139228]
28. Chapman RS, et al. Improvement in household stoves and risk of chronic obstructive pulmonary disease in Xuanwei, China: retrospective cohort study. *BMJ*. 2005; 331(7524):1050. [PubMed: 16234255]
29. Romieu I, et al. 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]
30. Qian Z, et al. Factor analysis of household factors: are they associated with respiratory conditions in Chinese children? *Int J Epidemiol*. 2004; 33(3):582–588. [PubMed: 15166210]
31. Qian Z, et al. Respiratory responses to diverse indoor combustion air pollution sources. *Indoor Air*. 2007; 17(2):135–142. [PubMed: 17391236]
32. Wilson D, et al. Respiratory symptoms among residents of a heavy-industry province in China: prevalence and risk factors. *Respir Med*. 2008; 102(11):1536–144. [PubMed: 18684604]
33. Barry AC, 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]
34. Agrawal S. Effect of Indoor Air Pollution from Biomass and Solid Fuel Combustion on Prevalence of Self-Reported Asthma among Adult Men and Women in India: Findings from a Nationwide Large-Scale Cross-Sectional Survey. *J Asthma*. 2012
35. Hersoug LG, et al. Indoor exposure to environmental cigarette smoke, but not other inhaled particulates associates with respiratory symptoms and diminished lung function in adults. *Respirology*. 2010; 15(6):993–1000. [PubMed: 20456673]
36. Nguyen T, et al. The National Asthma Survey--New York State: association of the home environment with current asthma status. *Public Health Rep*. 2010; 125(6):877–887. [PubMed: 21121233]
37. Golshan M, Faghihi M, Marandi MM. Indoor women jobs and pulmonary risks in rural areas of Isfahan, Iran, 2000. *Respir Med*. 2002; 96(6):382–388. [PubMed: 12117036]

38. Jedrychowski W, et al. Effects of Domestic Gas Cooking and Passive Smoking on Chronic Respiratory Symptoms and Asthma in Elderly Women. *Int J Occup Environ Health*. 1995; 1(1): 16–20. [PubMed: 9990152]
39. Jarvis D, et al. Association of respiratory symptoms and lung function in young adults with use of domestic gas appliances. *Lancet*. 1996; 347(8999):426–431. [PubMed: 8618483]
40. 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]
41. Padhi BK, Padhy PK. Domestic fuels, indoor air pollution, and children's health. *Ann N Y Acad Sci*. 2008; 1140:209–17. [PubMed: 18991919]
42. Schei MA, et al. Childhood asthma and indoor woodsmoke from cooking in Guatemala. *J Expo Anal Environ Epidemiol*. 2004; 14(Suppl 1):S110–S117. [PubMed: 15118752]
43. Robinson CL, et al. Effect of urbanisation on asthma, allergy and airways inflammation in a developing country setting. *Thorax*. 2011; 66(12):1051–1057. [PubMed: 21730351]
44. Dekker C, et al. Childhood asthma and the indoor environment. *Chest*. 1991; 100(4):922–926. [PubMed: 1914606]
45. Garrett MH, et al. Respiratory symptoms in children and indoor exposure to nitrogen dioxide and gas stoves. *Am J Respir Crit Care Med*. 1998; 158(3):891–895. [PubMed: 9731022]
46. Volkmer RE, et al. The prevalence of respiratory symptoms in South Australian preschool children. II. Factors associated with indoor air quality. *J Paediatr Child Health*. 1995; 31(2):116–120. [PubMed: 7794611]
47. Willers SM, et al. Gas cooking, kitchen ventilation, and asthma, allergic symptoms and sensitization in young children--the PIAMA study. *Allergy*. 2006; 61(5):563–568. [PubMed: 16629785]
48. Zacharasiewicz A, et al. Indoor factors and their association to respiratory symptoms suggestive of asthma in Austrian children aged 6–9 years. *Wien Klin Wochenschr*. 1999; 111(21):882–886. [PubMed: 10599150]
49. Leung R, et al. Indoor environment of residential homes in Hong Kong--relevance to asthma and allergic disease. *Clin Exp Allergy*. 1998; 28(5):585–590. [PubMed: 9645595]
50. Ostro BD, et al. Indoor air pollution and asthma. Results from a panel study. *Am J Respir Crit Care Med*. 1994; 149(6):1400–1406. [PubMed: 8004290]
51. Eisner MD, et al. Exposure to indoor combustion and adult asthma outcomes: environmental tobacco smoke, gas stoves, and woodsmoke. *Thorax*. 2002; 57(11):973–978. [PubMed: 12403881]
52. Eisner MD, Blanc PD. Gas stove use and respiratory health among adults with asthma in NHANES III. *Occup Environ Med*. 2003; 60(10):759–764. [PubMed: 14504364]
53. Rage E, et al. Air pollution and asthma severity in adults. *Occup Environ Med*. 2009; 66(3):182–188. [PubMed: 19017701]
54. 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]
55. Belanger K, et al. Association of indoor nitrogen dioxide exposure with respiratory symptoms in children with asthma. *Am J Respir Crit Care Med*. 2006; 173(3):297–303. [PubMed: 16254270]
56. Hansel NN, et al. A longitudinal study of indoor nitrogen dioxide levels and respiratory symptoms in inner-city children with asthma. *Environ Health Perspect*. 2008; 116(10):1428–1432. [PubMed: 18941590]
57. Chapman RS, Hadden WC, Perlin SA. Influences of asthma and household environment on lung function in children and adolescents: the third national health and nutrition examination survey. *Am J Epidemiol*. 2003; 158(2):175–189. [PubMed: 12851231]