Domestic gas appliances and lung disease

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Introductory article

Association of respiratory symptoms and lung function in young adults with use of domestic gas appliances

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Background. There is evidence from some studies that people living in homes with gas stoves and other unvented gas appliances experience more respiratory symptoms than those who use other fuels for cooking and heating, but other studies have found no such association. We have investigated whether the use of gas appliances is associated with an increased risk of respiratory symptoms and whether sensitisation to common environmental allergens modifies any such association.

Methods. A stratified random sample of 15,000 adults aged 20–44 years, living in three towns in East Anglia, UK, were sent a questionnaire on asthma and hayfever. From those who responded, a random sample of 1864 were invited to complete an extended questionnaire that included questions on use of gas appliances, to give blood samples for measurement of total IgE and specific IgE to common allergens, and to undergo tests of respiratory function. 659 women and 500 men agreed to an interview. The association of the use of gas appliances with respiratory symptoms, total IgE, specific IgE, and respiratory function was assessed by logistic and multiple regression models.

Findings. Women who reported they mainly used gas for cooking had an increased risk of several asthma-like symptoms during the past 12 months including wheeze (odds ratio 2.07 [95% CI 1.41–3.05]), waking with shortness of breath (2.32 [1.25–4.34]), and asthma attacks (2.60 [1.20–5.65]). Gas cooking increased the risk of symptoms more in women who were atopic than in non-atopic women but the difference did not reach significance (p>0.05). Women who used a gas stove or had an open gas fire had reduced lung function (forced expiratory volume in 1 s (FEV1)) and increased airways obstruction (FEV1 as a percentage of forced vital capacity) compared with women who did not. These associations were not observed in men.

Interpretation. In East Anglia, the use of gas cooking is significantly associated with subjective and objective markers of respiratory morbidity in women but not in men. Women may be more susceptible than men to the products of gas combustion or they may have greater exposure to high concentrations of these products because they cook more frequently than men. 

The lungs provide the most common site for infections in the United States and other developed countries. Although the mortality rate is low, respiratory infections can have serious consequences for groups with increased susceptibility (asthma, COPD). The prevalence, morbidity, and mortality of asthma appear to be increasing in developed countries, and concern about the cause of this increase has drawn attention to environmental exposures that may be contributing factors.

Susceptibility to respiratory infections is determined by a combination of host and environmental factors. The role of indoor pollution has been increasingly recognised. For certain pollutants, the indoor environment is a greater determinant of human exposure than the outdoor environment. Time-activity diaries show that the average person spends approximately 22 hours a day indoors (92%), the majority of that time at home (16 hours). As emphasis has been placed on energy conservation, ventilation rates in newer structures have been reduced and winter air exchange rates in newer homes can be as low as 0.1–0.3/hour. With lowered exchange rates, the concentration of indoor pollutants is increased. For these reasons, research has been directed towards evaluating an association between respiratory illness and indoor exposures. The introductory article is timely in stimulating further interest in the topic and, in particular, in indoor air pollution associated with gas combustion.
Indoor air pollutants from gas combustion

The predominant sources of indoor air pollution are combustion products from gas appliances and tobacco smoking. Unvented cooking or heating appliances using gas or kerosene produce a complex mixture including water vapour, carbon monoxide, carbon dioxide, nitric oxide, sulphur dioxide, formaldehyde, carbon particles, and sulphate particles. The use of gas appliances leads to concentrations of nitrogen oxides that are frequently higher than those found outdoors - in 10% of homes with gas cooking appliances levels higher than the US National Ambient Air Quality Standard of 100 µg/m³ have been documented.1 The dominant oxide produced is nitric oxide (NO) which, during its atmospheric lifetime, is progressively oxidised to nitrogen dioxide (NO₂); the potential for adverse health effects is attributed to both of these substances.

To date, most of the research has focused on the effects of NO₂, which can cause severe lung injury and even death when encountered in high concentrations as illustrated by “silo filler’s disease”.2 The effect of acute exposure to high levels of NO₂ has been demonstrated in other occupational settings. Apollo astronauts accidentally exposed to NO₂ (250 000 ppb for about four minutes) developed clinical and radiographic evidence of chemical pneumonitis. Measurements of urinary hydroxylysine glycosides indicated possible collagen degradation.3,4 It has become increasingly evident that NO also has significant effects on the respiratory system as a vasodilator, a neurotransmitter, and an inflammatory mediator in the airways.5 While it may have beneficial effects on airway function as a bronchodilator and neurotransmitter of bronchodilator nerves in human airways, NO may also have deleterious effects on the airways by increasing plasma exudation and amplifying the inflammatory response. Pro-inflammatory cytokines and oxidants increase the expression of an inducible form of NO synthase in airway epithelial cells.7 The impact of indoor NO as a combustion product from gas appliances has not been studied.

Determinants of NO₂ exposure

The indoor air concentration of NO₂ and other pollutants depends on the indoor source and on dispersion, conversion to other compounds, and removal by ventilation. Indoor levels are also influenced by outdoor concentrations and building characteristics. Personal exposure is influenced by time-activity patterns, the amount of time spent indoors, within the home, in various rooms, and in activities that increase exposure. Infants spend most of their time sleeping, so the bedroom environment is particularly important. For young women, who are traditionally responsible for most household cooking, peak exposures occur during cooking. Persons cooking with a gas stove can be exposed to levels of pollutants two orders of magnitude higher (>1000 ppb) than the average room concentration.8 The personal health effects are then influenced by host factors including age, sex, coexisting state of health, physiological state (exercising versus resting during exposure), previous exposure history, and personal susceptibility. These pollutants may also interact with other substances in the indoor environment such as allergens, other gases, passive and active smoking. The health effects in an individual are likely to be the result of a complex interaction between all of these factors (fig 1).

Figure 1 Framework for exposure assessment.

Toxicology of NO₂ exposure

The mechanism of NO₂ toxicity is related to oxidant injury. NO₂ is a strong oxidiser that initiates lipid peroxidation in cells which, in turn, results in cell damage or death. The toxicology of NO₂ has been studied and the work has been summarised in several reviews.9 In brief, exposure to NO₂ has, in animal models, multiple effects on the respiratory system. Long term exposure to high concentrations (>1000 ppb) can result in permanent damage to the epithelium in the centriacinar region of the lung and emphysematous changes. Exposure to NO₂ can affect the defence mechanisms of the lung and increase susceptibility to infection. Some studies have documented alterations in the function of ciliated cells that line the airways and of alveolar macrophages (reduced mobility, phagocytic activity and killing capacity), while others investigating infectivity in animal models have shown increased susceptibility to and mortality from experimental infections after exposure to NO₂.10 These studies have limitations because the level of exposure was 1–2 orders of magnitude higher than is typically found in indoor environments.

Epidemiological evidence

Early work that triggered interest in the effects of gas appliances came from Melia et al11 who studied a cohort of primary school children in England and Scotland. Controlling for social class, family size, and other factors, they reported a higher prevalence of respiratory symptoms among children from homes with a gas cooker than from homes with electric cookers.
Many studies examining the health effects of gas appliances and exposure to NO2 have subsequently been published. Initial studies focused on children as those believed to be at increased risk of exposure. The results have not yielded a consistent picture of an association between gas appliances, NO2, and respiratory health. Some studies have shown a small but significant effect, while others have shown no effect or a non-significant association. Fewer studies have been performed in adults, but they too have given conflicting results. Studies of indoor exposure offer only modest support for the hypothesis that exposure to NO2 can lead to increased frequency of respiratory illnesses and/or symptoms. Methodological limitations associated with such studies – low statistical power, exposure misclassification, confounding or effect modification by other pollutants, and insensitivity of health outcomes – could explain the inability to obtain definitive conclusions.13-15

To address the concern that low statistical power was responsible for the lack of consistent findings, in 1992 Hasselblad et al performed a meta-analysis.17 They made several assumptions, adjustments, and acknowledgments in combining the studies. Firstly, the end point being measured was similar in all studies. Secondly, the NO2 exposure levels differed among studies and were indirectly assessed in some. A standard increase of 30 μg/m3 (15 ppb) was used. This was the average increase in background NO2 exposure for homes with gas appliances over those without.18 All studies were used to estimate the effect of an increase of 30 μg/m3 (15 ppb) even if they had a different exposure range. Thirdly, each study controlled for key covariates. The results were combined using four different methods with similar results and the combined analysis yielded an estimated risk ratio of 1.18 (95% CI 1.08 to 1.29; fig 2). Thus, the combined results suggest an increase in odds of respiratory illness of about 18% in children exposed to an additional 15 ppb NO2 for extended periods.

Accurate assessment of exposure is central to any epidemiological study. Studies of indoor air pollution, and of NO2 in particular, are prone to random misclassification of exposure. Misclassification of health outcome is also of concern. Lung function can be measured reliably with standard procedures in adult and older children, yet many studies use the incidence or severity of acute respiratory illness as the health outcome, not objective quantification of functional impairment. There is no standard protocol for classification of respiratory illness, and classification is largely dependent on the physician making the diagnosis.

Samet et al published results from a large prospective study subsequent to the meta-analysis.19-20 A cohort of 1205 infants was followed prospectively from birth to 18 months. Symptom diaries were used to identify outcome. NO2 concentrations in three rooms of each home were monitored with a Palmes diffusion tube for 14 day periods. There were no significant differences in the incidence or duration of respiratory illness, as reported by symptom diary, between children in homes with gas cookers and those with electric stoves. There was no consistent dose-response relationship between respiratory symptoms in the past 12 months (table 1). Of interest is the finding that these effects differed between the sexes with an increased risk being seen only in women. Thus, in women exposed to gas cooking there was a trend towards increased respiratory symptoms in the past 12 months.
A second important finding is that a cook’s presence of a vented fan does not guarantee it is used appropriately. In homes where cooking was observed to take place for three minutes or more, only 10.8% of the households were thought to use a cooking range vent properly. This finding is not inconsistent with the hypothesis of the association of gas appliances and respiratory symptoms in women who use a gas stove for cooking.

Jarvis et al attempted to look at this question by a stratified analysis. Among women who used gas cookers, housewives and unemployed women did not have an increased risk compared with women who were employed or were students, but we do not know if the latter used a gas stove and/or oven less often than the former. Housewives and unemployed women who had an open gas fire for room heating had a reduction in the concentration of NO₂ in homes with a gas cooking stove. The hypothesis of increased exposure (presence of a stove) may be constitutional — for example, hormonal differences. Both animal and human studies show that the hypothesised role of increased exposure (presence of a stove) may be constitutional — for example, hormonal differences.

Several published studies have examined controlled exposure to NO₂ of both normal and asthmatic subjects. Some subjects exhibit significant increases in bronchial responsiveness after NO₂ challenge but the results have been inconsistent and the response has not been related clearly to dose. There is evidence in the literature to suggest that inhalation of NO₂ at concentrations encountered in the home environment can potentiate specific bronchial responsiveness of asthmatic patients with mild asthma to inhaled antigen (D. pteronyssinus).27 Jarvis et al examined the interaction of atopic status with the association between domestic gas appliances and respiratory health and found that atopic women were more vulnerable than non-atopic women. These results support the effect modification by sex may be constitutional — for example, hormonal differences.

Symptoms among atopic women compared with non-atopic women. These results suggest effect modification of the association of gas appliances and respiratory symptoms by both sex and atopic status. There are two possible explanations for the sex differences. Traditionally, women do most of the cooking and their increased risk may be secondary to increased exposure. Harlos12 studied the variation in NO₂ concentrations in homes with a gas cooking stove. The kitchen area was the most complex room in the house, and the kitchen area was the most complex room in the house. Harlos found occasional high concentration peaks near 1000-1500 ppb.
Individual susceptibility to NO2 exposure may be modified by constitutional characteristics, the population attributable risk fraction (PAR%), which is a function not only of the prevalence of exposure to nitrogen dioxide. This effect can also be modified by constitutional factors and other exposures. Despite these effects, the relative risk but also of the prevalence of a particular exposure in the population, appears to be large (26±43%) for the effect of domestic gas appliances on respiratory symptoms. The OR of nitrogen dioxide on respiratory health is highly dependent on time-activity patterns, and percentages of only modest degree, the PAR% of exposure indoor air pollution. Part I: Assessment of indoor nitrogen dioxide with respiratory symptoms and assessment may explain the inconsistent results published in the literature. In addition, individual exposure is highly dependent on time-activity patterns, and personal health effects can also be modified by constitutional factors and other exposures. Despite these limitations, the existing evidence suggests that there may be a modest effect of exposure to gas appliances on respiratory health. In future research we need to identify the pattern of exposure and the subgroups associated with increased risk.

Conclusions

The difficulty of studying indoor pollutants such as NO2 due to inherent problems of accurate exposure assessment may explain the inconsistent results published in the literature. In addition, individual exposure is highly dependent on time-activity patterns, and personal health effects can also be modified by constitutional factors and other exposures. Despite these limitations, the existing evidence suggests that there may be a modest effect of exposure to gas appliances on respiratory health. In future research we need to identify the pattern of exposure and the subgroups associated with increased risk.

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