ABSTRACT

Twenty preschool children from the Frank Porter Graham Child Care Center, and their mothers, were monitored for NO_2 exposure in their homes and at the day care center. Subjects were monitored for one week during the summer and winter months using Bendix 8101 Oxides of Nitrogen Analyzers and Palmes passive monitors. In-home stationary Palmes tubes are very accurate compared to chemiluminescent monitors (R^2=0.97). Chemiluminescent monitors and Palmes tubes worn by subjects do not give the same measurement of of personal NO_2 exposure (R^2=0.83), presumably because it is not possible for a stationary monitor to estimate personal exposure as well as Palmes tubes do. Mean winter NO_2 levels in homes with kerosene space heaters varied widely (71.4 ±73.2 ppb), but were more than 2 times the levels found in homes with gas stoves (26.5 ±14.2 ppb) and ten times the levels of homes without combustion sources (4.6 ±3.2 ppb). Clinical, epidemiological, and toxicological evidence suggest that peak short-term levels are more important than long-term averages in determining human health effects. Continuous monitoring data indicate that combustion appliances caused peak NO_2 levels of 100 ppb or more during 9 of the 40 monitoring-weeks in this study. Regression analysis suggests that peak values contributed more significantly to a mother's net exposure than to her child's ($P < 0.05$).

In conclusion Palmes tubes provide an accurate and convenient measure of net personal exposure to NO_2. Average exposure, however, is not the best indicator of indoor NO_2 levels which may have significant health impacts. Increased venting and improved combustion appliance technology provides the most feasible alternative for lowering in-home exposure to NO_2.

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Introduction: Studies Measuring Indoor NO_2 Levels

The Oxides of Nitrogen (NO_X) are formed chiefly during the combustion of fossil fuels, through NO_X are produced whenever combustion above 1350 degrees F occurs (conventional combustion temperatures are greater than 2250 degrees F) (Wark and Warner, 1981). Outdoor sources of NO_X include automobiles, power stations, and emissions by plants manufacturing nitric acid and explosives. Indoor air may be contaminated by NO_X from outdoor sources, but indoor sources such as gas stoves, kerosene space heaters, and tobacco smoke also contribute significantly to NO_X levels. While NO_X are a large family of compounds, only one compound, nitrogen dioxide (NO_2) is considered a threat to human health at levels normally encountered in homes (USEPA, 1982).

Studies measuring indoor levels of NO_2 have suggested an association, in children and asthmatics, between increased respiratory illness (coupled with decreased lung function) and low level exposure to gas stove related pollutants, including NO_2, in the range of 0.1 to 1.0 ppm. Some of these studies have considered the presence of a gas stove synonymous with exposure to NO_2 (Florey C. du V. et al., 1979; Melia, R.J.W. et al., 1977). More recently other studies have measured NO_2 levels using passive samplers, known as Palmes tubes (Palmes, E.D. et al., 1977a and b). These have provided ample evidence establishing higher NO_2 levels in homes with gas compared to homes with electric stoves (Federal Register June 19, 1985). At least 7 studies have reported long-term NO_2 levels between 50 and 300 ug/m^3 in homes with gas stoves and smokers (USEPA, 1982). Spengler et al. (1983b) took week-long NO_2 measurements using Palmes tubes inside and outside of 137 homes in Portage, Wisconsin over a 1 year period, and found NO_2 levels inside the kitchens of 112 homes with gas stoves averaged about 25 ppb (50 ug/m^3) higher than outdoor levels (at room temperature 1 ppb NO_2 = 1.88 ug/m^3 [Lindvall, T., 1985]). Ten

percent of the gas cooking homes had annual average kitchen levels higher than the National Ambient Air Quality Standard of 100 ug/m^3. These researchers also noted a wide range in the variability in personal exposure. Only 5% of the variability could be accounted for by stratification by stove type, whereas up to 60% could be explained by measurements in the home.

Only one large scale study has measured both average and real-time NO_2 levels in homes with combustion sources other than gas stoves or smokers. Leaderer et al. studied acute respiratory illness associated with the use of unvented kerosene space heaters in 333 residences in Connecticut from September 1982 to April 1983. Of these 333 homes 14 were monitored with a chemiluminescent analyzer for a period of between 2 and 7 days. The remaining homes in the study were monitored for 2 week periods using Palmes tubes. Indoor to outdoor ratios of NO_2 concentrations were found to be 0.58 $\pm$0.31 for residences without known sources of NO_2. Two-week average levels of indoor NO_2 exposure were found to be excellent predictors of total personal NO_2 exposure for a small sample of adults.

This study was undertaken to determine NO_2 levels in homes to assess the relationship between exposure to NO_2 and urinary excretion of hydroxyproline, which previous research has suggested is a biological marker of exposure to NO_2 (Yanagisawa et al., 1986). As a part of this larger study this technical report will: 1) assess the reliability and cost effectiveness of Palmes tubes in determining NO_2 levels in the field; 2) determine how precisely personal exposure to NO_2 can be estimated using chemiluminescent monitors and Palmes tubes; and, 3) examine in-home NO_2 exposure in light of epidemiological, clinical, and toxicological evidence suggesting individuals are more sensitive to short-term peak NO_2 levels than long-term average levels. In order to

assess the health effects associated with NO_2 it is first necessary to examine the properties of NO_X and ambient levels found in other monitoring studies.

Physical/Chemical Properties NO_X

Nitrogen dioxide is a highly reactive compound, formed via several pathways; therefore its formation and subsequent health effects cannot be discussed apart from the entire nitrogen oxides family of compounds that serve as potential sources. Typically all NO_X derived from indoor combustion processes are discharged as nitrogen oxide (NO), with the rate of formation rapidly increasing as the temperature increases (Wark and Warner, 1981). Only about 5-10% of total NO_X produced by combustion is NO_2; almost all the remainder is NO. Nitrogen dioxide, NO_2, the primary oxide of concern for human health effects, is mainly formed by the oxidation of NO at lower temperatures. Tables 1 and 2 list the equilibrium constants (K_p) for the following two reactions:

1) $N_2 + O_2 \rightleftharpoons 2NO$

2) $NO + O_2 \rightleftharpoons NO_2 + O$

The K_p for reaction two is large at room temperature, i.e. NO_2 formation is favored at low temperatures, but at higher temperatures it dissociates back into NO and O_2. The K_p for reaction one is small at room temperature, thus little NO is formed; whatever NO is formed is rapidly converted to NO_2. These laboratory kinetic observations are confirmed in studies of the aging of sidestream cigarette smoke, which has relatively higher proportions of NO_2 than mainstream smoke (Lindvall, 1985). To summarize the two major conclusions about indoor NO_X equilibria: 1) the rate of NO_2 formation decreases

Table 1
Equilibrium Constants for the Formation
of Nitric Oxide from Molecular Oxygen and Nitrogen

$$N_2 + O_2 \rightleftharpoons 2NO$$

$$K_p = \frac{[NO]^2}{[N_2]\,[O_2]}$$

Temperature (K)	Temperature (F)	K_p
300	80	10^{-30}
1000	1340	7.5×10^{-9}
1200	1700	2.8×10^{-7}
1500	2240	1.1×10^{-5}
2000	3140	4.0×10^{-4}
2500	4040	3.5×10^{-3}

Table 2
Equilibrium Constants for the Oxidation of
Nitrogen Oxide to Nitrogen Dioxide

$$NO + 1/2\ O_2 \rightleftharpoons NO_2$$

$$K_p = \frac{[NO_2]}{[NO]\,[O_2]^{1/2}}$$

Temperature (K)	Temperature (F)	K_p
300	80	10^{6}
500	440	1.2×10^{2}
1000	1340	1.1×10^{-1}
1500	2240	1.1×10^{-2}
2000	3140	3.5×10^{-3}

From Wark et al., 1981, p. 376-377.

with increasing temperature; and 2) the rate of NO_2 formation increases with increasing NO levels.

Relatively low emissions of NO_x result in formation of more NO_2 than might be expected simply by measuring source exhaust directly or via predictive models based on exhaust column emissions. Outdoors the thermal oxidation reaction $2NO + O_2 \rightleftharpoons 2NO_2$ is too slow to account for any significant fraction of the NO to NO_2 conversion observed in the atmosphere for typical ambient levels of NO, because the rate at which NO is converted to NO_2 through oxidation by oxygen in the air is proportional to the square of the concentration of NO

(USEPA, 1982). Indoor combustion appliance NO_X exhaust has a high concentration of NO; this is important in generating a relatively high proportion of NO_2 (up to 25% of total NO_X) during the initial combustion source plume cooling and dilution, when the concentration of NO is high (USEPA, 1982). In conclusion, indoors the above reaction produce higher than expected levels of NO_2 because it is very sensitive to the high levels of NO produced by combustion appliances.

Other oxides of nitrogen include nitrous oxide (N_2O), unsymmetrical and symmetrical nitrogen trioxide (NO_3), dinitrogen trioxide (N_2O_3), dinitrogen tetroxide (N_2O_4), and nitrogen pentoxide (N_2O_5). Of these only N_2O is present in the outdoor atmosphere in appreciable concentrations (USEPA, 1982). It is not considered a hazard at indoor levels.

Ambient Levels: Outdoor and Indoor NO_X

Outdoor NO_X levels contribute to indoor levels substantially: at least 50% of indoor NO_X comes from outdoor sources—if there is no indoor source (Leaderer, B.P. et al., 1986). Indoor levels of NO_2, the primary oxide of concern for human health effects, are generally lower than outdoor levels. Gas heating and cooking appliances can increase the indoor levels, which may occasionally rise in places to several thousand ug/m^3, depending on the type and age of the house, air exchange, decay rates, and source strength (Sexton, K. et al., 1983). Furthermore convective kerosene heaters produce approximately 3.5 times more NO_2 than the radiant type (Woodring, J.L. et al., 1985). NO_X in mainstream cigarette smoke are mostly present as NO; sidestream smoke raises indoor NO_2 levels because NO is converted to NO_2 via the reaction noted in the last section. One cigarette produces between 160-500 ug NO_X, which may increase indoor levels of NO_2 3-5 ppb (Lindvall, T., 1985).

Background levels of NO_2 have been determined for both urban and rural areas in the U.S. The background concentration of NO_2 in rural areas usually ranges from 0.5 to 10 ug/m^3 (0.3 - 5.3 ppb) (USEPA, 1985). Urban levels vary widely, depending on location and time. Annual means are about 20-90 ug/m^3 (11-48 ppb); for example, the mean annual concentration from 186 urbanized areas in the U.S. during 1977-79 was 55 ug/m^3 (29 ppb) (USEPA, 1985). The effect of outdoor levels on indoor levels was addressed in two separate studies. Sexton et al. developed a model relating total NO_2 exposure to background ambient levels, indoor values, and human activities. Results suggest indoor NO_2 concentrations in private dwellings vary primarily with outdoor levels and type of cooking fuel, but are also affected by factors such as air-exchange rates and strength of indoor sources. An exposure assessment performed from September 1982 to April 1983 by Leaderer and colleagues (1986) found that the indoor to outdoor ratios of NO_2 were 0.58 $\pm$ 0.31 for residences without known sources of NO_2.

In the past data from central monitoring sites have been used to establish air pollution exposures. It is now recognized, however, that people spend a majority of their time away from fixed monitors where pollution concentration may be drastically different (Sexton, K. et al., 1983). Several important exposure studies over the past 10 years have demonstrated that personal exposures to many pollutants show little or no relationship to outdoor measurements obtained at central monitoring sites and that personal exposures are often higher than, though poorly correlated with outdoor exposures (Spengler, J.D. et al., 1983b, 1984). Dockery et al. (1981) showed that personal exposures are functions of outdoor levels as well as stove type; personal exposures of subjects with gas stoves were two times higher than those with electric stoves. Furthermore indoor NO_2 levels in homes with gas stoves

were three times higher than outdoor levels. Leaderer et al. also showed that personal and indoor NO_2 levels for subjects using unvented kerosene heaters were significantly higher than outdoor levels. In summary total personal exposure to NO_2 is dependent on the levels and amount of time spend in each of the many indoor and outdoor "microenvironments" an individual passes through each day (Sexton, K. et al., 1983).

Sources

Of the indoor sources listed previously, NO_X monitoring has identified kerosene heaters as the highest NO_X producers. Kerosene heaters come in two types: convective and radiant. The convective type has an exposed flame and the radiant type has a metal mesh over the wick. Woodring et al. measured emission rates from both types of heaters and found that convective heaters produce approximately 3.6 times more NO_2 than radiant heaters ($p < 0.001$). Comparison of these figures with data on gas ranges show that convective kerosene heaters have greater emission rates. The highest peak levels found in gas stoves were the lowest levels expected from convective kerosene heaters. In-home measures of NO_2 levels by Leaderer et al. (using Palmes tubes) found that levels for homes with a kerosene heater only and those with a gas stove only were similar. Homes with a kerosene heater and a gas stove had average two-week NO_2 levels approximately 2 times those with only one source. These seemingly contradictory results may have been obtained because the measurements from the laboratory studies were taken directly from the exhaust gas column, whereas the in-home levels actually measured resulted from the room temperature kinetic interactions mentioned in Tables 1 and 2. Average levels are also dependent on the duration of use of the combustion appliance; levels in homes reflect different cooking and heating activities.

Literature Review: Health Effects of NO_2

NO is the most prevalent oxide of nitrogen emitted into the ambient air from combustion sources. Concern about possible health effects of virtually all of the types of nitrogen oxide compounds, including NO, has existed for many years. Despite these concerns "and considerable scientific inquiry, there is now little hard data linking specific health effects to any of the oxides of nitrogen, with the notable exception of NO_2" (USEPA, 1982).

NO_2 is a pulmonary irritant, but has a high odor threshold, easily raised by adaptation (Lindvall, T., 1985). Thus NO_2 has insufficient sensory warning properties, especially since the dominant irritation effects of the substances are localized to the deeper airways. NO_2 also causes bronchoconstriction via various mechanisms, resulting in increased airway resistance (Lindvall, T., 1985). The irritant and bronchoconstrictive effects of NO_2 on the respiratory system are the most extensively characterized to date and appear to be of most concern in terms of both acute and long-term health implications (USEPA, 1982).

Acute accidental exposure (less than one hour) to levels of NO_2 greater than 300 ppm causes rapid death from edema; exposure to levels greater than 25 ppm cause bronchitis or bronchial pneumonia (USEPA, 1982). Lower levels (< 5ppm) are associated with a variety of respiratory system effects. Investigations characterizing low level health effects have taken three general approaches, shown below, each approach showing different but related respiratory system effects (USEPA, 1982):

1. Controlled Human Exposure Studies—increased airway resistance and other indications of altered pulmonary function.
2. Human Epidemiology Studies—increased incidence of human respiratory illness, especially in sensitive populations.
3. Animal Toxicology Studies—lung tissue damage and increased susceptibility to respiratory infection.

Collectively, these studies provide evidence indicating that certain human health effects may occur as the result of exposure to NO_2 concentrations approaching or falling within the range of recorded ambient air NO_2 levels: 1 hour peaks of 0.4 to 0.5 ppm in some urban areas, and annual means of 0.11-0.48 ppm in urban areas (USEPA, 1982). EPA scientists have identified three important issues to consider when reviewing the low level health effects literature:

> 1. "Consideration of lowest effective single NO_2 exposure level(s) inducing particular respiratory effects in healthy and sensitive human subjects;
> 2. Assessment of lowest effective exposure levels at which repeated or intermittent NO_2 exposures produce effects in human populations; and
> 3. Consideration of the relative significance of observed effects in terms of understanding the likely impact of ambient NO_2 exposures on human health" (USEPA, 1982).

The following is a brief summary of respiratory system effects observed in the three major study approaches.

Controlled Human Exposure Studies

Controlled clinical studies have generated extensive information about the lowest effective dose levels for the induction of respiratory effects due to a single short-term NO_2 exposure. The mechanism underlying most of the changes in lung function ascribed to such exposure is still unclear, but would appear to be the irritation of smooth muscles or specific receptors (Lindvall, T., 1985).

Exposure at concentrations of 9,400 ug/m^3 (5.0 ppm) NO_2 or above for as little as 15 minutes will increase airway resistance in healthy human adults and impede the normal transport of gases between the blood and lungs (von Nieding et al., 1973). In healthy adult individuals, concentrations of 4,700 ug/m^3 (2.5 ppm) NO_2 for 2 hours have been reported to increase airway resistance significantly without altering arterial oxygen pressure (USEPA,

1982). Single exposures for 15 minutes to NO_2 at concentrations of 3,000 ug/m^3 (1.6 ppm) are also likely to increase airway resistance in healthy adults and individuals with chronic bronchitis but not to interfere with the transport of gases between blood and lungs (USEPA, 1982). Single exposures for times ranging from 15 minutes to 2 hours to NO_2 at concentrations of 2,800 ug/m^3 (1.5 ppm) or below have not been shown to affect respiratory function in healthy individuals or in those with bronchitis (Lindvall, T., 1985).

In its most recent review of technical data on this subject EPA concludes that the more recent controlled human exposure studies present mixed and conflicting results concerning respiratory effects in asthmatic and normals in the range of 0.1 to 4.0 ppm NO_2 (Federal Register June 19, 1985). Whether asthmatic subjects are more sensitive than healthy adults in experiencing NO_2-induced pulmonary function changes remains to be definitively resolved. The results of one controlled human exposure study suggest that some asthmatics may experience chest discomfort, dyspnea, headache, and/or slight nasal discharge following 2 hour exposures to 0.5 ppm NO_2 but did not provide convincing evidence of pulmonary function changes in asthmatics at that NO_2 concentration (Grant, L. 1982). Results by Bauer et al. showed that in asthmatics pulmonary function decrements occurred at 0.3 ppm NO_2. These results were replicated at EPA Health Effects Research Lab in Chapel Hill by Grant (1982)

Studies have been performed on bronchial reactivity induced by NO_2 in which lung function was tested for sensitivity to agents that contract the airways (Lindvall, T., 1985). Orehek et al reported increased bronchial reactivity in asthmatics at fairly low concentrations (190 ug/m^3) during 1-hour exposures. The results of this study support the results by von Nieding et al, who suggested that acute effects on pulmonary acetylcholine sensitivity to NO_2 may take place at very low levels (100 ug/m^3) in combination with a mixture of

sulfur dioxide (300 ug/m^3) and ozone (50 ug/m^3) (USEPA, 1982). These results are inconclusive--the Orehek study mainly because the grouping of the subjects was made after the pulmonary test with no independent criterion for pulmonary sensitivity and in the von Nieding study because the results were inconsistent (Lindvall, T., 1985). These studies, though flawed, suggest a possible exposure-effect association with complex mixtures, such as those emitted by unvented combustion appliances.

Human Epidemiology Studies

Epidemiological studies of the effects of community air pollution are difficult to do well since there are almost always complex mixtures of pollutants in the air. Usually the most that can be demonstrated from such studies is a "close association between health effects and ambient concentrations of a given mixture of pollutants or subfractions of the mixture" (USEPA, 1982). Epidemiological studies of air pollution effects are also often hampered by difficulty in defining actual exposures of study populations. Due to differences in monitoring techniques and analysis, but also because few of these studies have adequately considered indoor exposure levels, outdoor studies have produced conflicting results. Although outdoor studies are too numerous to mention in full, one series of studies of local interest has been widely cited, criticized, and its data analyzed and re-analyzed to an extent that it should be mentioned here.

Studies in Chattanooga, Tennessee, from 1968 to 1973 related ambient air levels on the order of 40 to 90 ppb mean NO_2 (mostly from a local TNT plant) to respiratory disease incidences in the general populus (USEPA, 1982). Initial findings indicated that 6-month average ambient NO_2 levels (using the Jacob-Hochheiser method, which was later found to be erratic) were related to

decreased lung function and increased respiratory disease incidence. No fewer than 5 subsequent analyses have been performed on the same data, most using more sophisticated statistical techniques and additional questionnaire data. Results of these subsequent studies have not provided a clear picture since, for instance, Harrington and Krupnick found that "over the range of pollution values experienced, more illness is associated with low pollution values than with high ones (Harrington and Krupnick, 1985)." EPA has concluded that:

> "The Chattanooga studies provide limited quantitative evidence of an association between elevated long-term NO_2 exposures and the occurrence of increased incidence of acute respiratory disease and lung function impairment. The findings of these studies are not inconsistent with the hypothesis that NO_2 in a complex mix with other pollutants in the ambient air adversely affects lung function and contributes to excess respiratory illness in children" (Federal Register June 19, 1985).

Other outdoor studies of NO_2 concentrations in the range of 0.02 to 0.51 ppm among adults found no differences in pulmonary function tests although complex mixtures of other pollutants were not controlled for (USEPA, 1982). A study performed in Japan did report a statistically significant correlation between one-hour concentrations of NO_2 in the range of 0.01 to 0.19 ppm and decreased respiratory function of school children (Kagawa and Toyama, 1975). However, at typical one-hour mean NO_2 concentrations temperature was more strongly associated with decreased lung function than any of the pollutants measured in the study (NO_2, NO, O_3, SO_2, and suspended particulates) (Lindvall, T., 1985).

While these ambient air studies indicate a correlation between NO_2 and respiratory problems, particularly with younger children, and do demonstrate that peak exposures are most likely more important than average exposure, they are qualitative in nature because they do not measure indoor or personal exposure levels directly. Since people generally spend 70-90% of their time indoors, indoor studies are of interest, not only to establish more exactly the

dose-response relationship with respect to susceptible populations, but also to show the temporal nature of the dose-response relationship.

Indoor epidemiological studies by British (Melia, R.J.W. et al., 1977, 1978, 1982) and Harvard (Speizer, F.E. et al., 1980; Dockery et al., 1981; Ware, J.H. et al., 1984; Berkley C.S. et al., 1986) investigators provide some support for the hypothesis that children are at special risk for NO_2-induced increases in acute respiratory illnesses. These studies investigated possible decrements in lung function and/or increased respiratory symptoms and illness rates among children living in homes using gas stoves for cooking in comparison to children from homes with electric ranges (USEPA, 1982). Gas cooking can produce considerable indoor NO_2 levels, and several studies substantiate that higher NO_2 levels accumulate in homes using gas stoves in comparison to NO_2 levels found in homes with electric stoves. In 1978 Melia et al. reported that average NO_2 concentrations in 2 homes over a 96-hour test period, during which stoves were in use for 8.5 to 10 hours, were 136 ug/m^3 (72 ppb) when gas was burned, and 18 ug/m^3 (10 ppb) in 2 homes where electricity was used. Other studies, including those by Goldstein et al. and Spengler et al. (1983b) confirm that the levels of NO_2 in gas stove homes are higher than those in homes using electric stoves. The initial publication by Melia et al. (1977) reported a weak association between increased respiratory illness in school children and residence in homes using gas stoves. This study, however, failed to adjust for tobacco smoking in the home. Later publications by Melia et al. (1978, 1982), which corrected for the number of smokers in the home, found weak associations between gas cooking and respiratory illness in children in urban areas but not in rural areas (USEPA, 1982).

In the U.S. Speizer et al. (1984), in the first of a series of studies, reported that 6 to 10 year old children from homes with gas stoves had lower lung capacity and a greater history of respiratory illness before two years of age. Florey et al. (1979) found no relationship between NO_2 concentrations in either bedrooms or kitchen and the results of lung function tests on school-age children. However, the prevalence of respiratory illness was found to increase with the NO_2 levels in the bedrooms of children in homes with gas stoves. In a population of rural, non-smoking, adult women in the Netherlands Fischer et al. (1985) found no significant association between NO_2 exposure and pulmonary function decline. Most recently Berkley et al. (1986) reported an association between exposure to sidestream cigarette smoke or gas stove emissions and pulmonary function level and growth rate in a large sample of children 6-10 years old. The data from this report provide some evidence for an association between gas stove exposure and pulmonary function level, especially at younger ages, but no evidence for an effect of gas stove exposure on growth rate.

The inconsistency of results obtained so far is most clearly shown in the case of Speizer et al (USEPA, 1982). When they reanalyzed the same data that they had previously reported on with additional cohorts from the same population and a more detailed assessment of socioeconomic status, one of the findings, a statistically significant increased incidence of respiratory illness before age 2, became less statistically significant (Ware, J.H. et al., 1984). This study also demonstrated that, in children annually tested, with respect to the rate of change in lung function, no cumulative effect of gas-stove exposure was revealed. Nonetheless this study does confirm small but statistically significant decreases in lung function in school age children, although there is some evidence that parental education levels may confound this relationship. EPA and the authors of the study concur in stating "that a

better understanding of the health significance of indoor pollutants such as NO_2 may require more defined measurements of personal exposure (Federal Register June 19, 1985)."

Animal Studies

Clinical and epidemiological studies have suggested, in a qualitative sense, an association, in children and asthmatics, between increased respiratory illness and low level exposure to gas stove related pollutants such as NO_2 in the ranges of 0.1 to 1 ppm (USEPA, 1982). Two types of animal experiments have shed light on the nature of the physiologic damage and NO_2 dose response curves:

1. Experiments measuring alterations in pulmonary function. Biochemical parameters of exposure have been detected as low as 752 ug/m^3 (0.4 ppm).
2. Experiments inducing susceptibility to pathogenic bacteria. One study has shown an excess in mortality (after bacterial challenge) following a 3 hour exposure to NO_2 at concentrations as low as 1 ppm (Lindvall, T., 1985).

While NO_2 studies have been performed at levels above 10 ppm, EPA has judged that this concentration is the maximum at which animal studies provide relevant data to estimating the human health effects of ambient or near ambient concentrations of NO_2 (USEPA, 1982). No studies so far have shown that NO_2 is a carcinogen, even at high concentrations.

An unusual aspect of the toxicity of NO_2 is a delay between exposure and observed health effects (USEPA, 1982). The nature of this temporal sequence is important for understanding the toxicity of NO_2 and has important implications for the effects of both short-term and long-term exposures to this air pollutant. This trend has been observed in many species despite the differences in NO_2 sensitivity among these species and the many different endpoints of toxicity (USEPA, 1982).

NO_2 water soluble and can be absorbed in the mucous lining of the nasopharyngeal cavity where it converts to nitrous and nitric acid (Lindvall, T., 1985). Few data examining respiratory tract uptake and transformation have been published, but it seems that about 50-60% of incoming NO_2 is retained in animals (USEPA, 1982). Radiotracer studies in monkeys have shown that $^{13}NO_2$ is fairly uniformly distributed in the lung and retained for a prolonged period (USEPA, 1982). A significant fraction of NO_2 is not removed in the upper airways, and this penetrates deep within the lung to produce its toxic effects. Because of its high reactivity, the predominant response observed on the inhalation of NO_2 is direct injury to the tissues of the lung, possibly resulting in cell death. "The magnitude and site of the injury resulting from NO_2 exposure will depend upon the concentration of NO_2 which was inhaled; therefore, the absolute degree of response will depend upon both the rate and magnitude of respiration and the NO_2 concentration (USEPA, 1982)." Morphologically, the damage caused in animal pulmonary tissues by prolonged exposure to nitrogen dioxide at fairly high concentrations (600-2000 ug/m^3 or above) is inflammatory; signs of bronchitis are typical findings in rodents (Lindvall, T., 1985). Changes in lung morphology and emphysema-like effects occur at higher exposure levels, with resultant decrements in lung function.

The most obvious effect of exposure to NO_2 is a change in pulmonary function. In rats, increased respiratory rates have been observed at 1500 ug/m^3 (0.75 ppm) after long-term exposure (Lindvall, T., 1985). The same effect has been found in other animals at higher levels (non-human primates, guinea pigs, cats), although the sensitivity between species varies.

Increased susceptibility to airway infection after exposure to NO_2 has been demonstrated in a number of animal experiments. Following exposure to airborne bacteria combined with a 2 hour exposure to NO_2 at concentrations of 6,600

ug/m^3 (3.5 ppm) or above, the mortality among exposed mice, hamsters, and squirrel monkeys was higher than among control animals (Lindvall, T., 1985). Mortality due to exogenous infectious agents is influenced more by the concentration of NO_2 than the exposure. The EPA NO_x Criteria Document (1982) points out that this observation is consistent with the hypothetical temporal sequence of injury. It is thought that pulmonary damage occurs rapidly on exposure to NO_2, but the functional effects of pulmonary damage may be observed much later, depending upon the extent of damage and the system which has been used to measure the damage: biochemical endpoints or susceptibility to airborne infections (USEPA, 1982). The hypothesized temporal sequence of injury and repair for short-term (less than 8 hours) one-time exposures begins with an essentially instantaneous chemical reaction of NO_2 with the tissues of the respiratory tract. Susceptibility to microorganisms begins to rise almost immediately, and biochemical indicators of injury peak about 18 hours after exposure; cell death peaks at 24 hours, and replacement of dead and injured cells and biochemical indicators of repair reach their maximum at 48 hours (USEPA, 1982). Injury caused by NO_2 concentrations as low as 4,700 ug/m^3 (2.5 ppm) may result in excess cell mortality from a single exposure of only 3 hours (Gardner D. et al., 1977). The injury of a 3 hour exposure appears to be repaired within 24 to 36 hours after exposure, but not by 18 hours (USEPA, 1982). The increased susceptibility to airway infections caused by exposure to NO_2 might be due to an effect of the gas on the lung macrophages, the mucocilliary clearance mechanism, or the immune system (Lindvall, T., 1985).

Long-term exposures to NO_2 also result in major alterations of lung morphology (USEPA, 1982). These studies, however, are difficult to interpret because of the fine gradation and slow development of response once the initial phase of replacement of cells susceptible to NO_2 has passed. The development

of an emphysema-like disease in experimental animals requires considerable time as seen in studies of rats and mice.

Health Effects: Summary

In conclusion the most commonly observed human health effect due to NO_2 exposure is increased airway resistance mediated by the stimulation of local receptors or other reflex mechanisms not necessarily specific to NO_2 (Lindvall, T., 1985). A number of experiments have shown that challenge with NO_2 increases bronchial sensitivity. This leads to the general impression that organisms might be more sensitive to short-term exposures of high concentrations of NO_2 than to long-term exposures to low concentrations (Lindvall, T., 1985). This view is supported by studies on animals, such as those by Gardner et al. on the influence of exposure mode on the toxicity of NO_2 in combination with bacteria. Gardner found that when a constant concentration*time level was employed, a short-term exposure to a high concentration produced a greater effect than exposure to a lower concentration administered over a longer period. With this in mind and an 0.05 ppm annual mean exposure standard to justify EPA (1982) concluded that "estimates of repeated short-term peak concentrations of NO_2 possibly associated with increased respiratory illness in homes with gas stoves are not markedly below the general range of the lowest (0.5 to 1.0 ppm) intermittent exposure concentrations found to cause increased susceptibility to respiratory infections in animal infectivity model studies." In order to attribute health effects to NO_2 exposure more hard data on levels actually encountered by individuals is needed.

To put these conclusions in perspective, ambient air NO_2 monitoring results indicate that peak 1-hour NO_2 rarely exceeded 0.4 to 0.5 ppm (USEPA, 1982). Long-term annual average NO_2 concentrations exceeding 0.05 ppm, the EPA annual

standard, were only found in a few scattered locations, mostly in Southern California and Chicago (USEPA, 1982).

Materials and Methods

Study Population. The study population were volunteers from among the families with children at the Frank Porter Graham Child Development Center (FPG) and data were collected between February 1986 and July 1987. Twenty preschool children from FPG and their mothers were monitored for NO_2 exposure in their homes and at the day care center during a seven day period. The children range in age from 6 months to 4 years old. Volunteers were recruited

Table 3
Potential NO_2 Sources in Study Homes

	Unvented Sources		No. of	Vented	
Family	Space Heater	Gas Stove	Smokers	Heating	Comments
Kerosene Space Heaters					
1	Kerosene	None	0	Oil	Moved*
2	Kerosene	Propane	0	Propane	
3	Kerosene	None	1	Electric	
4	Kerosene	None	0	Electric	
5	Kerosene	None	1	Elec. Baseboard	Moved*
Gas Stoves					
6	None	Natural Gas	1	Gas	
7	None	Propane	1	Propane	
8	None	Natural Gas	1	Natural Gas	
9	None	Natural Gas	0	Natural Gas	
10	None	Propane	0	Propane	Gas Dryer
Smokers					
11	None	None	2	Kerosene	
12	None	None	2	Natural Gas	
13	None	None	1	Natural Gas	
14	None	None	2	Elec. Heat Pump	
15	None	None	1	Natural Gas	
Non-Smokers					
16	None	None	0	Elec. Heat Pump	
17	None	None	0	Elec. Heat Pump	
18	None	None	0	Elec. Baseboard	
19	None	None	0	Natural Gas	
20	None	None	0	Elec. Heat Pump	

*-Family moved to a new residence between the first and second monitorings

from homes in 3 potential exposure categories, and a control group: 1) kerosene space heater users; 2) gas stove users; 3) tobacco smokers without unvented combustion appliances; and 4) non-tobacco smokers without unvented combustion appliances. There were twenty families were included in the study; two of the families in the kerosene exposure category and 3 from the gas stove exposure category were also smokers. One of the families in the kerosene exposure category were also gas stove users. A complete description of each family's exposure to combustion appliances, tobacco smoke, and their central heat source is listed in Table 3.

NO_2 Monitoring Techniques

Most studies of personal exposure to NO_2 rely almost exclusively on passive monitors known as Palmes tubes. These are considered less reliable than the conventional "gold standard," chemiluminescent monitors, but are far more convenient for measuring personal exposures. In this study both monitoring techniques were used: Palmes tubes and continuous chemiluminescent Bendix 8081 Oxides of Nitrogen Analyzers (Bendix instruments subsidiary was purchased and is now operated by: Combustion Engineering Incorporated, U.S. 219 North, Lewisburg, West Virginia 24901).

Continuous Monitors. Continuous chemiluminescent monitors, which are EPA's reference method for measuring NO_2, provide both peak short term levels and average concentration over any period of time. They do not measure NO_2 directly. Rather, they obtain NO concentrations by measuring the luminescence produced by high energy NO_2 during the gas phase reaction between NO and ozone. The instrument has two sampling phases; in the NO_X phase it diverts a portion of the sample air to a reduction catalyst that converts NO_2 to NO. NO originally present in the sample remains unchanged. The second phase, or NO channel, is not converted. The two samples are then analyzed by the

luminescence chamber. The instrument interprets the measured concentrations as $NO_X=(NO + NO_2)$; thus the sample NO_2 concentration is determined by the difference between NO_X and NO phases of sampling (Bendix, 1977).

The Bendix monitors used in this study are sensitive in the range of 5 to 500 ppb NO_2. They sample 150 cubic centimeters of air per minute using Thomas vacuum pumps (purchased from Southern Fluid Power, Lake Wylie South Carolina, 29710) and the inlets were fitted with one eighth inch diameter teflon tubing. The monitors were calibrated using a Bendix 8861 "suitcase" calibrator and nitric oxide span gas purchased from Airco Industrial Gases (Research Triangle Park, North Carolina) and certified by EPA-Research Triangle Park.

Passive Monitors. The Palmes tube is a passive NO_2 or NO_X monitor that provides an average exposure level over a week or month. Palmes tubes have typically been used for one week exposure periods, both indoors and outdoors, and for personal monitoring. The principle of operation is based on Fick's First Law of Diffusion (Palmes, E.D. et al., 1977a):

$$J= -D\ dC/dL$$

Where:

J = amount of NO_2 passing through a unit area per unit time= moles/cm^2-sec
D = diffusion coefficient = cm^2/sec
C = concentration = moles/cm^3
L = diffusion path length = cm

The right hand side of the equation is negative since flux is in the direction of decreasing concentration, i.e. as L increases C decreases (Palmes, E.D. et al., 1977a)

When the above equation is used to calculate the quantity of NO_2 transported by molecular diffusion through the tube in a given time, it must be modified to include the cross-sectional area of the tube, A, and the time for which the flux persisted, t. The total equation becomes (Palmes, E.D. et al.,

1977a):

$$Q = JAt = -D\ C/L\ At$$

Where:
Q = Quantity of NO_2 transferred = moles
A = cross-sectional area of the tube = cm^2
t = time = seconds

The quantity of gas transferred, therefore, depends on the three constants, D, A, and L, and the two variables, C (concentration) and t (time).

The sampler depends on the transfer of NO_2 by diffusion to a triethanolamine (TEA) coated collector at the sealed end of a tube; the open end of the tube is exposed to the test environment:

> "The quantity of gas transferred by diffusion from the environment through an orifice of known dimensions into a chamber maintained at substantially zero concentration by a suitable collecting medium [TEA] can be used as the basis for calculating average concentrations during the time the sampler is in the environment (Palmes, E.D. et al., 1977a)."

TEA was selected as the absorber instead of other reagents for several reasons: it captures NO_2 very efficiently, can easily be coated on to solid materials, and the TEA-NO_2 complex is very stable. The minimum sampling period is dependent upon expected NO_2 concentrations; the detection limit of the Palmes tube is 600 ppb*hrs (Majahad, A.M., 1986).

Palmes (1977b) reports a 97%-102% efficiency (in lab studies) for the sampler and a precision of ±3% as compared to the chemiluminescent analyzer. These devices have also been compared with standard chemiluminescent NO_2 monitors by Warren Spring Laboratory, Lawrence Berkeley Laboratories, and the U.S. National Bureau of Standards. "Accuracy has been shown to be better than ±10%. Replicate samples indicated an absolute difference of 10%. Hence, these NO_2 devices are accurate to within 10% and have a precision of 10%" (Dockery et al., 1981). In a study by Goldstein and co-workers two measurements of NO_2 levels were obtained in each of 507 kitchens. The difference between the pairs of readings was less than 10 ppb in 63% of cases and less than 20 ppb in 83% of

the cases. The lower limit of sensitivity of 600 ppb*hours makes these devices suitable for week-long integrated measurements at ambient levels (Majahad, A.M., 1986). Palmes tubes are inexpensive, unobtrusive, and have been used to obtain average indoor NO_2 levels in a number of large scale studies (Spengler, J.D. et al., 1983b,1984; Melia, R.J.W. et al., 1978,1979).

Like any other sampling system, Palmes tubes have limitations. Results are reported as average concentrations for the period sampled: they give no information about peak exposure levels, and acute levels are most likely more important in terms of human health effects (Lindvall, T., 1985). Furthermore, diffusion theory predicts only a 1.7% change in the sampling rate with a 10 degree C change in temperature at 21 degrees C (Girman, J.R. et al.). TEA, however, has a liquid-solid phase transition at 21 degrees C. Girman et al. have found that the collection efficiency of the sampler is dependent on temperature, with the collection efficiency decreasing 15%, from 96% to 82%, when the temperature decreased from 27 to 15 degrees C. This change in the capture efficiency would under-report actual values, thus false positives (high readings) would be less likely at the temperatures found in this study. The cause of the variation in collection efficiency with phase is unknown. If it is due to the kinetics of absorption, the possibility exists that the sampling rate is concentration dependent at low temperatures. Girman reports an 87% collection efficiency at 21 degrees C, lower than the efficiencies of 92-95% reported by Palmes (1977a) and Woebkenberg (1982).

The Palmes tubes used in this study were purchased from the Harvard School of Public Health Six City Air Pollution Health Study Group (Harvard School of Public Health, Cambridge Massachusetts) and developed by Anthony M. Majahad, the Quality Assurance/Quality Control Officer. Lots of 100 Palmes tubes were purchased, exposed, and returned to Harvard for analysis within six months of

initial acquisition date, which is within the shelf life limit for this sampler. Results are obtained by measuring the absorbance of the diazo compound of NO_2 and NEDA in the presence of a color reagent (NEDA=N-[1-Napthyl]-ethylene diamine dihydrochloride) (Majahad, A.M., 1986). The samples are measured at 540 nm and converted to nanomoles of nitrite based on the forced regression coefficient of a standard calibration curve. At least 5% of the tubes in each lot were field or laboratory blanks.

Exposure Assessment Protocol. Homes were monitored for one week during the heating season (November-April) and another during the summer months (May-October). On the first day of the monitored week (usually Sunday) the chemiluminescent monitor was installed and questionnaire data collected to indicate heating, cooking, and smoking status to document possible NO_2 sources. The inlet of the chemiluminescent monitor was placed in a location to sample air from the evening activity room of the child. One Palmes tube was attached approximately five inches from the end of the inlet tube for the chemiluminescent monitor. At the same time individual Palmes tubes to be worn by the child and mother were opened. Parents were also instructed on record keeping on source use and the heating system was checked to confirm its type and ventilation. A second chemiluminescent monitor recorded NO_2 levels in the child's classroom at school. On the second day of the week the in-home chemiluminescent monitors were calibrated (NO span = 400 ppb) using the Bendix 8861 suitcase calibrator. Continuous monitors were recalibrated before they were unplugged at the end of the monitored week. The in-school continuous monitors were calibrated weekly. Thus for each subject there is a continuous record of exposure at home and school as well as an estimate of average exposure from the Palmes diffusion sampler.

Data Analysis. For each participating family five different measures of NO_2 exposure, or response variables, were obtained. Palmes tubes results were reported as ppb*hours. A Textronics digitizing tablet (courtesy of the UNC Department of Geography) was used to compute the area under the curve from the chemiluminescent monitor strip charts (c.f. Figure 1). The area under the curve values obtained from the digitizer were converted to mean ppb NO_2 per week using the Lotus spreadsheet package to divide total NO_2 by elapsed time monitored. The five response variables measuring personal and in-home NO_2 levels (in ppb) are described below:

1) CONH7 - seven day in-home chemiluminescent monitoring value.
2) HSCON - a combination of home and school chemiluminescent monitoring values. This response variable is the weighted average of NO_2 levels at school and at home. The assumption used in creating HSCON is that the child is at home when not in school. This value, then, is the closest approximation of a child's personal exposure using a chemiluminescent monitor.
3) PH7D - Palmes tube in-home value. This tube was attached to the chemiluminescent monitor inlet, to provide an indication of Palmes tube reliability.
4) PKID - Palmes tube worn by child.
5) PMOM - Palmes tube worn by the child's mother.

HSCON, PKID, and PMOM are individual exposure averages whereas CONH7 and PH7D are average levels obtained in a subject's primary microenvironment. Two Palmes tubes were lost over the course of the study, both by mothers, hence PMOM is short one observation in each of the monitoring seasons.

Statistical analysis was done using PC-SAS (SAS Institute Incorporated, Box 8000, Cary, North Carolina). Paired mean T-tests were performed using Proc Means; regression analysis was performed using Proc GLM.

Continuous Monitor Data Quality Assurance. Chemiluminescent monitor calibration data were checked for error. No continuous monitoring data was used if the monitor calibration changed more than 10% over the course of a week; most calibrations changed less than 3% over the course of a week.

The monitors used in this study were tested for a low level response to elevated relative humidity, which could possibly lead to overestimation of NO_2 levels. This phenomena has been observed in Bendix 8101 Oxides of Nitrogen Analyzers, but is apparently monitor specific (personal communication, Dave Gimble, Environmental Monitoring Systems Incorporated). Monitors were zeroed using dry air (relative humidity < 30 %) from the Bendix 8861 calibrator and then sampled humidified air (relative humidity > 72 %) for as little as 3 and as many as 7 hours. Five replicate experiments were performed on four monitors used in this study. The amount of change in the zero was recorded from the strip chart recorders as a positive or negative response (ppb) to the increased relative humidity.

Results

Reliability of Palmes tubes verses chemiluminescent monitoring results was assessed using univariate regression analysis. Table 4 lists the correlation coefficients for comparisons of chemiluminescent verses fixed Palmes tubes, fixed Palmes tubes verses personal Palmes tubes, and two personal Palmes tubes. The regression of PH7D on CONH7 gives the equation PH7D = 2.8ppb + 0.94CONH7 (F=1121; d.f. 2,19; P >F 0.001) with an R^2 of 0.97. This indicates a very high degree of precision between continuous monitors and Palmes tubes. The regression of PKID on HSCON yields the equation: PKID = 6.0 + 0.64HSCON (F=192.3, 1,39 d.f., P > F 0.0001) with an R^2 of 0.83. These data indicate that though chemiluminescent monitors provide a close approximation of personal exposure, Palmes tubes are more precise indicators of the variability in personal exposure. The variability between measures of the home microenvironment and personal exposure is due, most likely, to to an individual spending more time away from the fixed sampler's microenvironment, as can be seen in the lower R^2 for the summer monitoring season.

Table 4
Univariate Regression Comparing Personal and Microenvironment NO_2 Levels
(All regression models are significant at Prob > F 0.0001, except as noted)

Model (Y=X)	Overall R^2	R^2 Summer	R^2 Winter
PH7D = CONH7	0.97	0.91	0.97
HSCON = CONH7	0.99	0.96	0.99
PKID = CONH7	0.81	0.48	0.81
PMOM = CONH7	0.90	0.79	0.90
PKID = HSCON	0.83	0.48	0.83
PMOM = PH7D	0.88	0.74	0.87
PKID = PH7D	0.85	0.45	0.85
PKID = PMOM	0.69	0.40*	0.65

*significant at Prob > F 0.0002

One of the initial aims in this study study was to see if personal exposure levels differed significantly from week long in-home levels. Table 5 and Figure 1 data show overall and stratified (summer and winter) mean NO_2 levels for the five response variables. The annual mean HSCON level is 3.9 ppb lower than PH7D ($T=-3.43$, $P > |T|=0.001$); all the other means are not significantly different at the 0.05 confidence level. Figure 1 is a graphic representation of some of the data in Table 5. Mean winter measures of personal exposure are two to three times the corresponding summer levels. Winter values, however, have much larger standard deviations, indicating a wide range of source use. The paired difference T test for the stratified data set indicates that the summer NO_2 levels reported by HSCON were, on the average, 2 ppb less than those reported by PKID ($T=-2.18$, 19 d.f., $P > |T|=0.042$). This indicates that, during the summer, HSCON is not as good an indicator of a child's exposure to NO_2 as PKID is.

Table 5
Mean NO_2 Levels in Monitored Homes (ppb)

	CONH7	PH7D	HSCON	PKID	PMOM
Mean ±S.D.	17.7 ±33.9	19.5 ±32.6	16.6 ±29.6	16.6 ±20.7	16.3 ±19.8
Range	2.0-173.9	1.9-152.1	2-139.6	1.4-98.9	2.6-106.2
n =	40	40	40	40	38

Mean NO_2 Levels by Season (ppb)

SUMMER	CONH7	PH7D	HSCON	PKID	PMOM
Mean ±S.D.	7.7 ±4.9	10.0 ±8.7	7.3 ±4.3	8.7 ±5.6	9.3 ±4.5
Range	2.0-20.4	1.0-32.2	2.2-18.0	1.4-19.7	2.6-18.9
n =	20	20	20	20	19
WINTER					
Mean ±S.D.	27.7 ±45.0	29.3 ±42.5	25.8 ±39.0	23.9 ±26.4	23.2 ±25.4
Range	2.1-173.9	1.2-152.1	4.0-139.6	1.9-98.9	4.2-106.2
n =	20	20	20	20	19

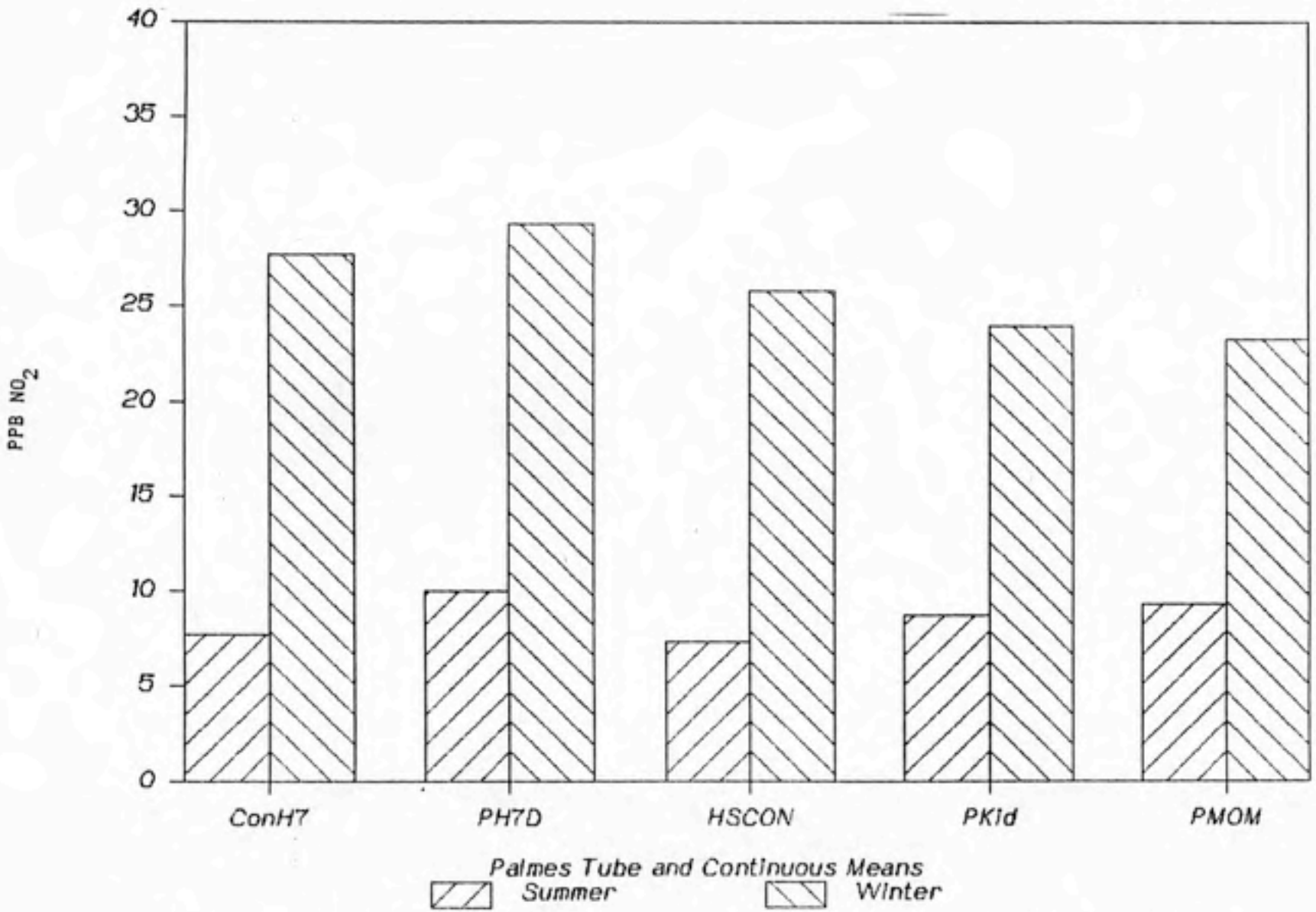

Figure 1. Bar Chart of Mean NO_2 Levels (ppb) by season for the Palmes tube data (*PH7D, KID, PMOM*) and continuous Monitors (*CONH7, HSCON*).

Table 6 lists summer and winter mean NO_2 levels for the 4 exposure groups defined in Table 1. During the summer gas stoves increase indoor levels most significantly. Multivariate regression of personal exposure levels on source

Table 6
Summer and Winter Mean NO_2 Levels (ppb) for the 4 Exposure Categories

Exposure	CONH7	PH7D	HSCON	PKID	PMOM
			SUMMER		
Kerosene	7.4 ±7.3	9.3 ±10.7	7.3 ±5.7	8.2 ±3.7	8.8 ±5.8
n=	5	5	5	5	5
Gas Stove	11.5 ±4.7	17.1 ±10.7	10.9 ±5.0	10.2 ±6.2	13.5 ±4.2
n=	5	5	5	5	5
Smoker	7.1 ±3.0	8.7 ±4.0	6.7 ±2.5	10.2 ±6.2	8.3 ±2.6
n=	5	5	5	5	5
Controls	4.7 ±2.6	4.9 ±6.2	4.4 ±1.8	6.3 ±4.6	6.0 ±2.7
n=	5	5	5	5	4
			WINTER		
Kerosene	70.0 ±81.2	71.4 ±73.2	63.3 ±69.8	50.6 ±41.6	44.1 ±41.1
n=	5	5	5	5	5
Gas Stove	22.4 ±15.0	26.5 ±14.2	20.1 ±12.3	19.3 ±17.8	23.8 ±17.0
n=	5	5	5	5	5
Smoker	12.0 ±12.2	14.7 ±14.6	12.2 ±11.7	16.3 ±13.2	16.9 ±14.4
n=	5	5	5	5	4
Controls	6.4 ±3.6	4.6 ±3.2	7.7 ±3.0	9.5 ±3.5	6.8 ±1.0
n=	5	5	5	5	5

variables indicates that, during the summer, gas stoves and parental smoking are most closely associated with increased personal NO_2 exposure as measured by PKID ($T=2.33$, $P > |T|$ 0.032 for stoves; $T=2.55$, $P > |T|$ 0.02 for smoking). Summer time mean NO_2 personal exposure levels are not significantly different for any of the four exposure groups ($P > 0.05$). During the winter months kerosene space heater increase the indoor NO_2 levels significantly. Mean

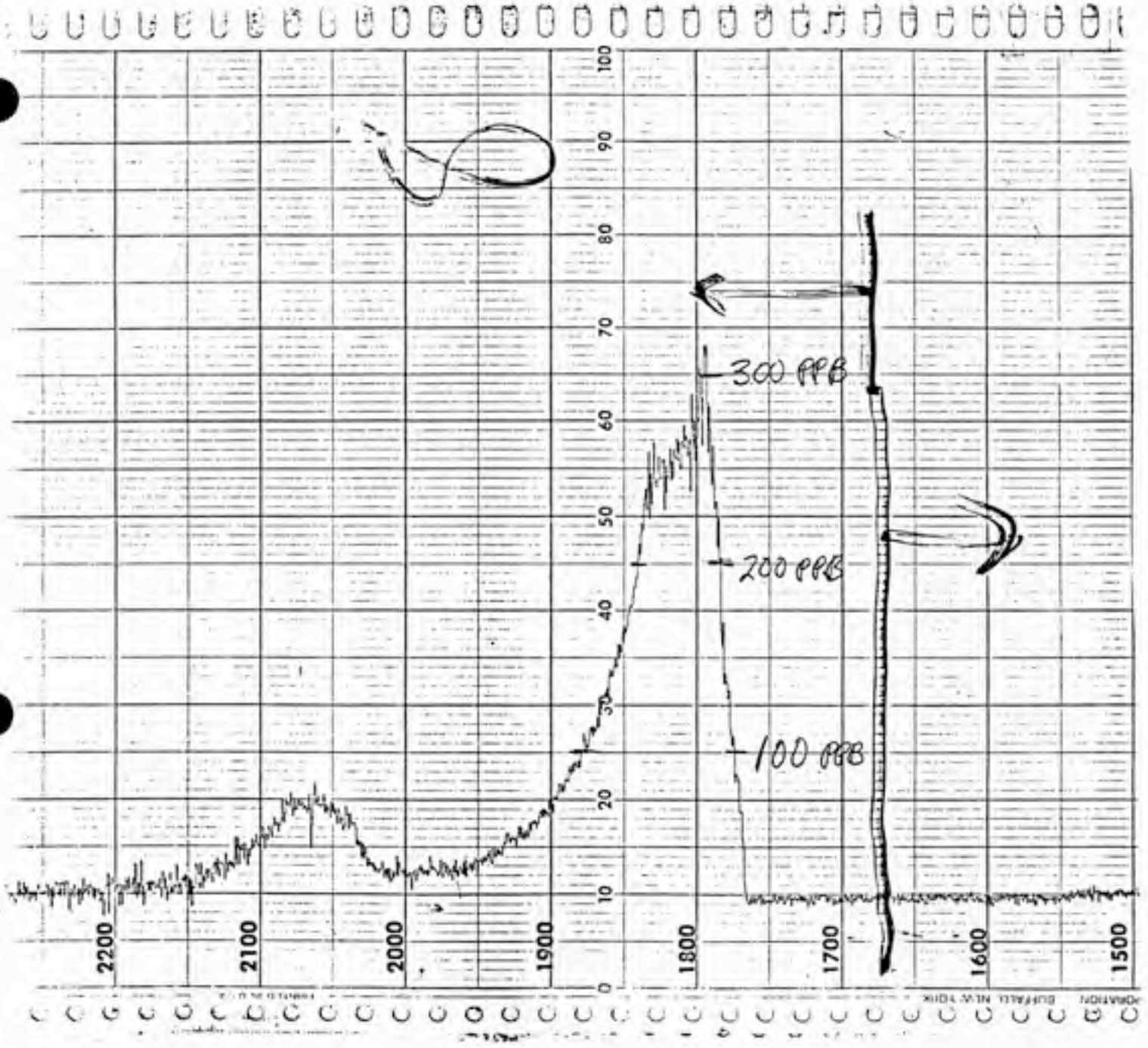

Figure 2. Chemiluminescent monitor strip chart trace of NO_2 levels during kerosene heater use. One percent on the Y axis is equal to 4 ppb NO_2; chart zero is at 5% of the Y scale. The X axis units are hours.

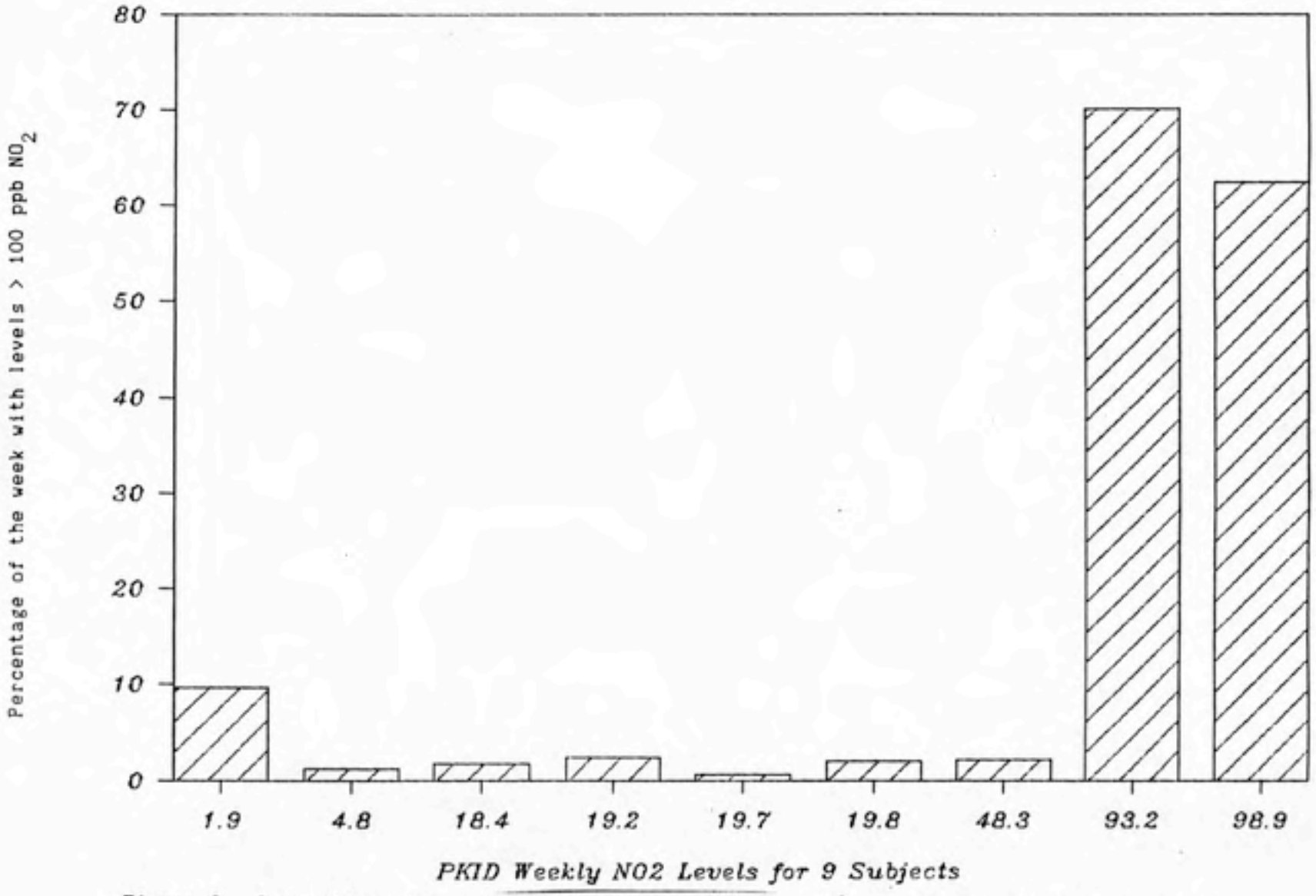

Figure 3. Comparison of continuous and Palmes tube personal monitoring data: 9 subject's exposure to NO_2 levels greater than 100 ppb. This bar chart compares personal exposure, as measured by *PKID*, and peak exposure levels, expressed as a percentage (ratio of elapsed time > 100 ppb NO_2/total elapsed time monitored *100) of the monitored week with levels greater than or equal to 100 ppb .

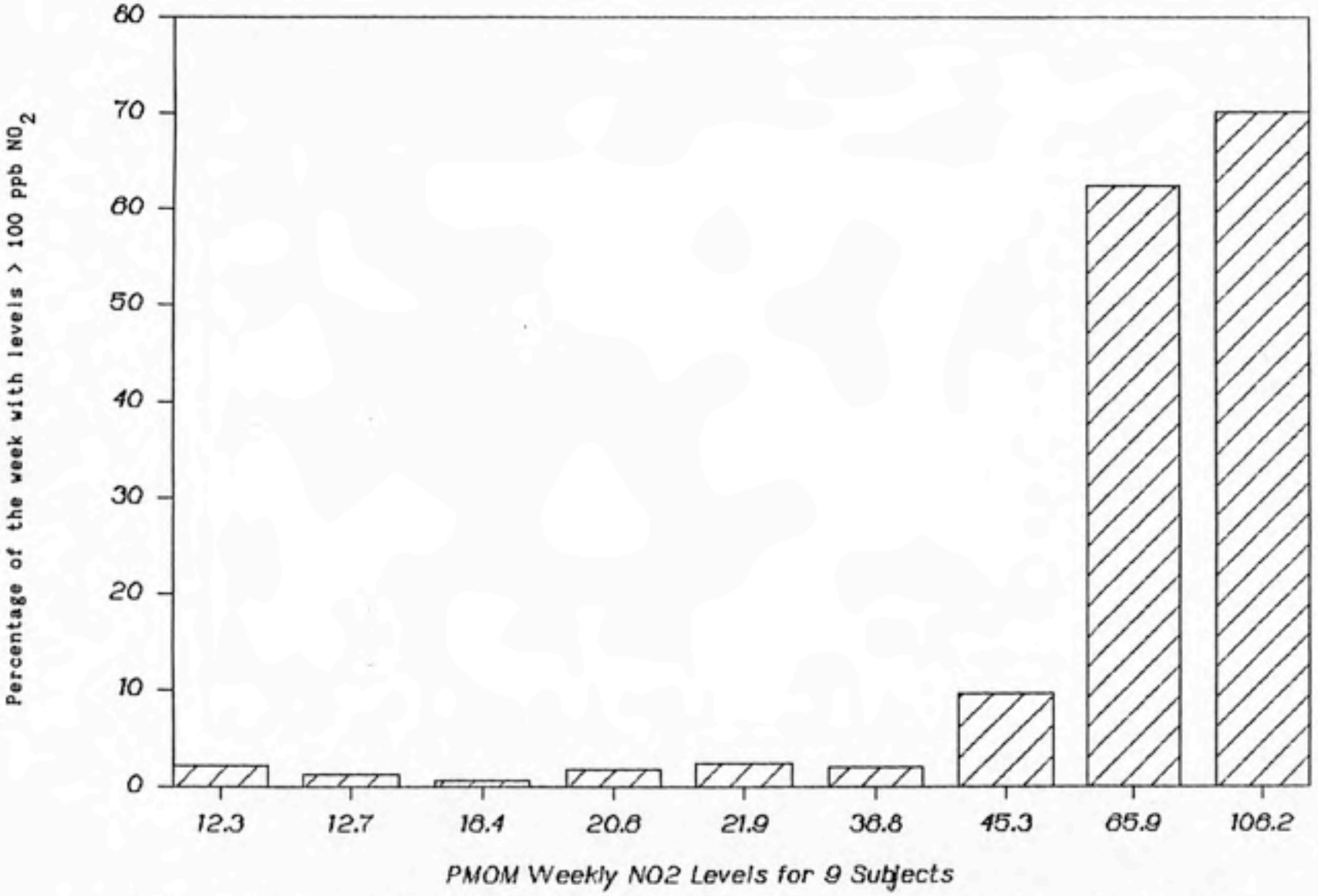

Figure 4. Comparison of continuous and Palmes tube personal monitoring data: 9 subject's exposure to NO_2 levels greater than 100 ppb. This bar chart compares personal exposure, as measured by *PMOM*, and peak exposure levels, expressed as a percentage (ratio of elapsed time > 100 ppb NO_2/total elapsed time monitored *100) of the monitored week with levels greater than or equal to 100 ppb.

levels in the kerosene heater homes are, on the average, more than two times the levels in gas stove homes and ten times the mean NO_2 levels of the control group homes. Personal exposure levels follow this same pattern, and multivariate regression using the three source variables indicate that stoves and kerosene heaters explain 63% of the variability in personal NO_2 levels ($P > 0.05$). In the winter time parental smoking is not significantly associated with a child's personal NO_2 exposure, but this is most likely due to the much stronger gas stove and kerosene heater source variables, since previous research (Leaderer, B.P. et al., 1986) and the summer season monitoring results from this study indicate that smoking is associated with a small but statistically significant 3-5 ppb increase in indoor levels.

Figure 2 is a photocopy of a continuous monitor strip chart from a home where a kerosene monitor was being used. NO_2 levels approached 300 ppb and stayed above 100 ppb for more than an hour. This suggests that mean values, as measured with Palmes tubes, do not reflect exposure levels actually existing while sources are in use.

Figures 3 and 4 investigate the relationship between average exposure, as measured by Palmes tubes, and peak exposure level data collected using chemiluminescent monitors. In 40 weeks of in-home monitoring there were 9 weeks with at least one peak of 100 ppb or greater, and 7 of these were during the winter monitoring season. 100 ppb NO_2 was chosen as a reference point because that is the lowest concentration at which human exposure research suggest adverse effects in sensitive individuals (USEPA, 1982). Regression of PMOM on percentage of the week with levels > 100 ppb indicates that PMOM (R^2=0.85) reflects source use more closely than PKID. This would indicate that mothers spend more time in and around combustion sources than their children.

Discussion

Palmes Tube Reliability and Personal Monitoring

NO_2 measurements taken in the same static location with both chemiluminescent NO_2 analyzers and Palmes tubes gave very similar results. Hence it appears that Palmes tubes provide a reliable measure of in-home NO_2 levels. However, chemiluminescent monitors and Palmes tubes do not give the same measurement of personal NO_2 exposure, presumably because it is not possible for stationary monitors to estimate personal exposure as well as Palmes tubes. Palmes tube personal monitoring provides the best measure of net exposure for individuals, especially during the summer months when people spend more time with windows open, outdoors, and/or away from combustion sources. It is also clear from the data in Tables 4 and 5 that monitoring a mother is not a substitute for monitoring her child. Total NO_2 exposure is more easily predicted with in-home monitoring of than by monitoring a family member: the correlation between PKID and PMOM (R^2=0.69, 0.4 during the summer) is not as strong as the correlation between PKID or PMOM and any of the in-home data values (PH7D, CONH7). Apparently during the winter the home microenvironment provides a majority of an individual's exposure to NO_2. This supports earlier research showing that net personal exposure is determined by exposure in the series of different microenvironments an individual passes through each day (Dockery et al., 1981).

Thus Palmes tubes provide a convenient, reliable, and cost effective measure of mean NO_2 exposure and microenvironment levels. Chemiluminescent monitoring equipment acquisition, operation, and maintenance costs run well into the tens of thousands of dollars, depending on the duration and scope of the study. Palmes tubes cost $7.50 apiece to purchase and analyze, which translates to less than $2000 over the course of this study.

Exposure Assessment

The most important question in this study is the relationship between peak levels and net exposure. The data in Tables 5 and 6 clearly show that winter time combustion appliance use provides a majority of an individual's annual net exposure: winter NO_2 averages are 15-50 ppb higher than homes without combustion sources. These results are consistent with results of Spengler et al. (1983) and Leaderer et al. (1986) who used Palmes tubes to assess indoor levels, but not personal exposure.

Figures 2 and 3 show the relationship between peak NO_2 levels greater than 100 ppb, as measured in-home by the continuous monitors, and net dose as measured by an individual's Palmes tube. With only 9 weeks of data with peaks greater than 100 ppb (7 of them occurred during the winter monitoring season, with) it is difficult to make substantive conclusions concerning peak exposure levels and their relation to average personal exposure levels. The data in Figure 2 are inconsistent, since one individual with very low net exposure (1.9 ppb/week) lived in a home where peak levels were >100 ppb almost 10% of the week (most of these in-home peaks occurred when the child was not at school). One can only conclude that this child spent a majority of his time away from combustion sources. The regression of either PKID or PMOM on the percentage of the week with levels greater than 100 ppb shows a fairly strong correlation between PMOM and peak levels >100 ppb ($F=39.6$, $P > F$ 0.0004), $R^2 = 0.85$). This same relationship is not nearly as strong for PKID, indicating that, in this study, children spent less time near combustion sources than their mothers. This means that though higher average exposure and source information could be used to predict the number of peak exposure greater than 100 ppb NO_2 on a weekly or annual basis, it is no substitute for direct personal real-time

monitoring. Whether these levels translate into specific health effects cannot be said for certain, since the relationship between exposure and effect is not well defined. The pending analysis of urinary hydroxyproline levels from this population could provide some insight into the physiological relationship between lung damage and peak levels, and perhaps help clarify the exposure effect relationship.

Sources of Bias

There are two main sources of bias in this study. The first is self protection. This would bias the results by lowering NO_2 exposure, but does not seem significant in this case since NO_2 levels are still above the Palmes threshold level. Moreover the levels found are consistent with other monitoring studies (Dockery et al., 1981; Spengler, J.D. et al., 1983b).

The most important avoidable error in this project is the effect of relative humidity on the performance of Bendix Chemiluminescent monitors. In outdoor monitoring studies by EPA's CHAMP program these monitors were shown to over report NO_2 in some cases when calibrated with dry air (relative humidity less than 30%) and sampled air at a different, usually higher, relative humidity (personal communication, Dave Gimble, Environmental Monitoring Systems Incorporated). Furthermore the humidity effect is monitor specific: in some over reporting of levels occurs, in others under reporting. I performed several replicate experiments on the 5 monitors used in this study to test the humidity effect on the output readings at the lower end of the scale (where the majority of the readings in this study occurred). I found that each instrument varied slightly, but that only one instrument over reported levels significantly: 4 ppb (1 % of scale). This effect was not consistent at 4 ppb and may have been due in part to zero drift. Moreover since 2 of the machines

displayed slight negative effects (< 2 ppb) the overall humidity effect seems to introduce an acceptable level of error into this study.

Recommendations

Methods of Reducing NO_2 Exposure

In light of these results and concern by the scientific community about the overall effects of indoor air pollution, efforts to reduce NO_2 exposure seem justified. Most people spend 75% of their time indoor and susceptible populations spend virtually all their time indoors; therefore actual exposures are often characterized more accurately by indoor measurements than outdoor measures (Sexton, 1987). The relationship between NO_2 and specific health endpoints is, however, not conclusive. The most that can be said is that NO_2 is a potential and highly plausible health problem and that peak exposures, such as those found in this study, should be reduced, especially for sensitive populations. How to implement legislative or regulatory solutions in an essentially unregulated field is an open question at this time. No overall governmental strategy exists to provide a coordinated, well-managed preventative approach to reduce emissions or reduce existing levels (Sexton, 1987).

Research into control of gaseous indoor pollutants such as NO_2 has taken two courses: control systems and dilution by fresh air (Moschandreas, 1987). Neither seems particularly promising at this point. Control technologies, such as integrated systems employing desiccant beds that absorb water vapor, NO_2 and other pollutants, are feasible but expensive for homes (Moschandreas, 1987). Dilution will not necessarily lead to acceptable pollutant levels because emission rates of combustion appliances vary widely. Moschandreas (1987) points out that though control devices exist, they are subject to over

estimation of actual performance. Though it is clear that people want clear indoor air consumer interest is driven almost exclusively by publicity. Moreover the public is not willing to pay more than $ 150 per year for cleaner indoor air (Moschandreas, 1987). Therefore control devices most be inexpensive and virtually maintenance free. Thus to meet long term goals of decreased exposure prevention seems the best strategy.

The preventative approach employs source removal, redirection, and improvement (Moschandreas, 1987). The simplest preventative action, source removal, has garnished considerable support in recent years, as witnessed by the increasing attempts to adopt legislation to prevent smoking in public buildings. Smoking, however, is the smallest contributor it indoor NO_2 levels. Source redirection, such as venting of gas stoves, removes as much as 75% of contaminants from an unvented gas top burner (Revzan, K.L., 1984). Source improvement research continues—improved two stage kerosene heaters emit less CO and NO_2 (approximately 50% less) than models currently in use, and electronic ignition on unvented gas appliances eliminates as many as three pilot lights and thus prevents elevated base line levels indoors (Moschandreas, 1987).

Policy Implications

Despite increasing awareness and concern associated with indoor air pollution no collective effort has been made to deal with the problem. Existing laws, such as NAAQS, apply only to the outdoor environment. Several agencies are currently involved in studies (DOE, EPA, HUD, and the Consumer Product Safety Commission, which provided some of the money for this study), but no one agency has the authority, responsibility, or seeming interest to address the issue. GAO's report to Congress on indoor air pollution recommended that EPA, through modification of the Clean Air Act, be given jurisdiction, because of its previous experience in air pollution (Sexton, K. 1987). The major problem with regulating indoor levels is the sheer magnitude of the enforcement of any standards, especially standard's that would clearly affect aspects of life-style such as smoking and cooking habits. Another regulatory option includes imposing requirement on the manufacturers via the Consumer Product Safety Commission and establishing standards for builder and designer under the auspices of the American Society for Heating, Refrigeration, and Air Conditioning Engineers. In the absence of a single organization committed to overseeing the problem, increased public awareness is important.

Common consensus indicates that indoor air pollution is a potentially serious problem. The average week long winter time levels found in 2 of the 20 study homes were well above EPA's annual mean of 100 ug/m^3 (50 ppb). EPA has no short term standard for NO_2; WHO (1977) has recommended a short-term standard of 170 ppb (320 ug/m^3 as a 1 hour average). This limit was exceeded numerous times in the 2 homes mentioned above. Enforcement of any such standard would, however, be difficult, and depend on the situation and "publicness" of the building (Spengler, 1983a). Eventually there may be added

pressure due to public demand and industry's refusal to accept full worker liability claiming in-home exposure may create a more susceptible individual.

Since short-term exposures are critical, effort should be focused on identifying peak concentrations and protecting the population at risk. Policy makers ultimately must balance energy conservation measures against the costs of deteriorating indoor air quality. If intervention is necessary policy alternatives such as economic incentives, educational efforts, and definition of rights and liabilities is necessary (Spengler, J.D., 1983a).

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