EPIDEMIOLOGY SECTION

Connecticut Epidemiologist

STATE OF CONNECTICUT DEPARTMENT OF HEALTH SERVICES 1984
Douglas S. Lloyd, M.D., M.P.H., Commissioner Vol. 3, No 1

HEALTH EFFECTS OF EXPOSURE TO UNVENTED KEROSENE HEATER EMISSIONS

Although official efforts to control air pollution have traditionally focused on outdoor air, it is now apparent that elevated contaminant concentrations are common inside some private homes. Public health concerns regarding indoor air pollution are based on evidence that urban residents typically spend more than 90 percent of their time indoors and that concentrations of some contaminants are higher indoors than outdoors. Energy conservation efforts, such as reducing ventilation rates, increasing insulation, and using supplementary heat sources, all potentially increase levels of air contaminants in the indoor environment.

A problem of particular concern is the increased use of unvented portable kerosene heaters. The popular use of these efficient, low-cost, supplementary heaters in residences has raised health and safety issues in two areas: (1) the potential for fires and asphyxiation with improper use, and (2) the potential for more subtle contamination of the indoor environment under normal use.

The Consumer Product Safety Commission (CPSC) has evaluated the safety of these heaters. They have found that they operate without fire incident or fuel spillage in a laboratory setting as long as the following operating specifications are met:

- The heater is filled with good quality pure kerosene (1K).
- The heater mantle (burner chamber) is clean and properly seated.
- The heater's flame is adjusted according to manufacturer's directions.
- Spilled fuel is not allowed to remain in the drip tray under the air tube.
- Proper clearance to combustible materials is maintained.
- Flammable fabrics are kept out of contact with the heater surfaces.
- There is no tracer flame and no fuel leakage on tipover.
- Level condition is maintained when transporting cartridge tank heaters.
- The wick is properly installed and is kept clean.
- The heater's tank is not overfilled.
- The heater is not knocked over.

CPSC laboratory studies have demonstrated that variation from these criteria may result in fuel overflow, spillage, or unstable heater conditions which predispose to fire.

There is a second concern about the use of unvented kerosene heaters. These heaters, as the name implies, are unvented - meaning that they operate without the aid of a chimney or flue-type vent. The combustion by-products generated are vented into the residence's indoor air. These by-products include the following gases which are known to have detrimental effects on persons exposed to them in sufficient concentrations: carbon monoxide (CO), carbon dioxide (CO_2), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2).

CARBON MONOXIDE

Carbon monoxide (CO) interferes with the blood's ability to carry oxygen to tissues. Inhaled CO binds to the hemoglobin of red blood cells reducing its availability to oxygen. The degree of interference is expressed as percent carboxyhemoglobin (COHb) or the percentage of the blood's hemoglobin bound up by carbon monoxide. Healthy non-smoking males have COHb levels of 0.4-0.7%, while tobacco smokers can have COHb saturations from 4-20% with a mean for one-pack-per-day consumers of 5-6%.(1) Death due to lack of oxygen to vital organs results from COHb levels of 60-80%(2).

The threshold for adverse effects from elevated COHb levels varies according to the health status of the individual. Exercising adults with angina pectoris are sensitive to COHb concentrations as low as 1 percent.(3) On the other hand, healthy young men on a submarine did not have headache, one of the earliest symptoms of toxicity, until their COHb levels exceeded 8-10%. Of great concern is the unwitting exposure of the fetus (in utero) to CO as a result of both environmental pollution and maternal smoking. Data indicates that the maternal to fetal carboxyhemoglobin ratio varies around one (0.7-1.8) and the concentration of fetal COHb has been measured as high as 7.6%.(4)

Recent unvented kerosene heater emission cham-

ber studies have raised questions about the levels of CO emitted from these devices. The Environmental Protection Agency (EPA) outdoor limit of CO for an eight hour average is 9 ppm not to exceed 35 ppm for one hour. Traynor et al. reported CO levels from radiant heaters which exceeded the EPA 8-hr average but were below its 1-hr standard.(5) Leaderer et al. concluded that concentrations of CO may be elevated to dangerously high levels if a radiant kerosene heater is used in a small room with a moderate ventilation rate.(6)

Finally, O'Sullivan reported a case of CO poisoning in a 21-day-old infant exposed to a kerosene heater. The infant repeatedly developed profound lethargy (once with vomiting) on being left in a room heated with a kerosene heater. After his most severe episode, his acute COHb level was calculated to be approximately 15%. In a healthy adult this level might cause minor symptoms (headache); however, in an infant, with a greater metabolic rate and oxygen demand, the immediate consequences were more severe.(7)

CARBON DIOXIDE

Carbon dioxide (CO_2) is the major by-product of any combustion process. Since it is a normal part of our atmosphere (.03%), there is no EPA ambient air quality standard. Nevertheless, exposure to abnormal concentrations of CO_2 can produce harmful physiological effects.

When inhaled in elevated concentrations, CO_2 may act to produce acidosis, mild narcotic effects, stimulation of the respiratory center, or asphyxiation depending on the concentration present and the duration of exposure. Acidosis and adrenal cortical exhaustion may occur as a result of prolonged continuous exposure to an atmosphere containing 10,000-20,000 ppm (1-2%)*.(2)

At 30,000 ppm, CO_2 is weakly narcotic giving rise to reduced acuity of hearing and increased blood pressure and pulse. At this level subjective symptoms may occur depending on O_2 concentration. Submarine personnel exposed continuously to 30,000 ppm were only slightly affected provided the oxygen content of the air was maintained at normal concentrations. When the oxygen content was reduced to 15-17%, the crew complained of ill effects.

At 50,000 ppm (5%), stimulation of the respiratory center is produced. Signs of intoxication occur after a 30-minute exposure at this level. Exposure at 7-10% produces unconsciousness within a few minutes.(8) Deaths have been reported from asphyxiation in workers exposed to higher concentrations.

Indoor CO_2 levels generally run from 300 to 600 ppm (.03-.06%). The recent Consumer Union (CU) kerosene emission study cited CO_2 levels approaching 10,000 ppm which were twice the Occupational Safety and Health Administration (OSHA) standard and four times the American Society of Heating, Refrigerating & Air Conditioning Engineers (ASHRAE) standard.(9) Subsequent studies seem to support CU findings.(5,6)

NITROGEN DIOXIDE

Nitrogen oxides (NO_2, NO) are formed when organic fuels are burned at high temperatures. Probably the most harmful to human health, even at low levels, is nitrogen dioxide (NO_2). Its normal atmospheric concentration is less than 0.05 ppm (one year average) but may range in some cities to as high as 0.6 ppm (eight hour average).

* (% x 10,000 = ppm)

In humans, the major acute effects, mild mucous membrane irritations, begin to occur at concentrations of 10-20 ppm. Exposure to concentrations greater than 200 ppm may result in pulmonary edema and death.(10) Chronic effects have also been reported, especially from long exposures at low concentrations. Animal studies in rats showed lung injury with 4 hour/day, 5 day/week exposure to 8 ppm. Monkeys exposed to 5 ppm for two months had lowered resistance to infection. A report of Russian workers exposed to NO_2 levels of approximately 2.8 ppm for 3 to 5 years associated "probable chronic bronchitis" and emphysema with that exposure.(11)

NO_2 is also a major by-product of natural gas and propane combustion in gas stoves. In a British study, schoolchildren living in homes with gas stoves had reduced respiratory function and an increased incidence of upper respiratory infections compared with children living in homes without gas stoves.(12) In a similar American study of 9,280 school children in six cities, the incidence of respiratory illness among children below age two was about 20% higher in homes with gas than in those with electricity.(13)

Two studies of kerosene heater emissions conducted in environmental chambers revealed NO_2 levels in excess of EPA's permissible annual average of 0.05 ppm. In addition, preliminary results of an unpublished study conducted by the John B. Pierce Foundation Laboratory indicate that "homes with one kerosene heater and no gas stove have NO_2 levels approximately four to five times the levels of residences with no kersosene heater or gas stove."(14)

SULFUR DIOXIDE

Sulfur dioxide (SO_2) is formed when sulfur-containing fuels, such as kerosene, are burned. Its normal atmospheric concentration is negligible, less than 0.1 ppm. However, in some cities where large quantities of fossil fuels are used, levels may rise to as high as 0.2-0.8 ppm (24 hour average).

The major acute effect of SO_2 exposure on humans is mucous membrane irritation and bronchoconstriction. The degree of bronchoconstriction is related to dose and the underlying sensitivity of the subject. Even in low concentrations, SO_2 can impair breathing. Mild asthmatics exposed to as little as 1 ppm of sulfur dioxide experience breathing problems within 10 minutes; asthma attacks -- coughing, wheezing, shortness of breath -- occur at 5 ppm.(9) Furthermore, EPA studies have shown that children in different cities had an incidence of croup and asthmatic symptoms that was directly proportionate to the average ambient SO_2 concentrations in the local area. Symptoms persisted for many months after people moved to an area of lower exposure.(10)

Unvented kerosene heaters are designed to burn kerosene. However, kerosene comes in different grades, only one of which, 1K, a low sulfur content fuel (.04% by weight), is appropriate for kerosene heaters. Environmental chamber studies conducted by Consumer Union and by the John B. Pierce Foundation confirm that kerosene heaters using low-sulfur kerosene (1K) produce sulfur dioxide levels between 0.8 and 1.6 ppm. These levels greatly exceed the EPA 24-hour air quality standard for sulfur dioxide of 0.14 ppm.(9) Even with lower sulfur fuels, kerosene heaters may serve as a potential source of SO_2. In an unpublished study of homes with and without kerosene heaters in Connecticut, sulfur dioxide was observed only in homes which used kerosene heaters, and levels increased with the number of heaters.(14)

In conclusion, increased pollutant levels have been associated with consumer use of kerosene heaters. Use of the heater in a closed room, es-

pecially in a house with a low air-exchange rate, use of a maladjusted or improperly maintained heater, and/or use of kerosene with a high sulfur content can be associated with serious health effects.

Physicians should be aware of the possible health consequences of kerosene heater use. The medical history should include questions about exposure to such heaters when patients present with symptoms consistent with exposure to high levels of CO, CO_2, NO_2, or SO_2 in cold-weather seasons.

People with chronic respiratory and heart disease, asthma, emphysema, and allergies, as well as pregnant women, small children, and the elderly should be advised to avoid exposure to kerosene heater emissions.

(Laurie Gokey of the Toxic Hazards Section served as consultant for this article.)

REFERENCES

1. American Conference of Governmental Industrial Hygienists. Carbon monoxide. In Documentation of the threshold limit values 4th ed. Cincinnati, Ohio: American Conference of Governmental Industrial Hygenists 1980; 72-73.

2. Centers for Disease Control. Carbon monoxide intoxication: preventable environmental health hazard. MMWR 1982; 31: 529.

3. Spengler J, Sexton K. Indoor air pollution: public health perspective. Science 1983; 221: 9-17.

4. Jaeger R. Carbon monoxide in houses and vehicles. Bull N York Acad of Med 1981; 57: 860-872.

5. Traynor G, Allen J, Apte M, Girman J, Hollowell C. Pollutant emissions from portable kerosene-fired space heaters. Environ Sc & Tech 1983; 17: 369-371.

6. Leaderer B. Air pollutant emissions from kerosene space heaters. Science 1982; 218: 1113-1115.

7. O'Sullivan B. Carbon monoxide poisoning in an infant exposed to a kerosene heater. J Pediatrics 1983; 103: 249-251.

8. American Conference of Governmental Industrial Hygienists. Carbon dioxide. In Documentation of the threshold limit values 4th ed. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists 1980, 69-70.

9. Are kerosene heaters safe? Consumer Reports 1982: 499-507.

10. Meyer B. Indoor Air Quality. Reading, Mass: Addison - Wesley Publishing Company, 1983.

11. American Conference of Governmental Industrial Hygienists. Nitrogen dioxide. In Documentation of the threshold limit values 4th ed. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists 1980, 305-306.

12. Melia R, Du V. Florey C, Altman D, Swan A. Association between gas cooking and respiratory disease in children. Brit Med J 1977; 2: 149-152.

13. Speizer F, Ferris B, Bishop Y, Spengler J. Respiratory disease rates and pulmonary function in children associated with NO exposure. Am Rev Resp Dis. 1980; 121: 3-10.

14. Leaderer B. unpublished data.

Confirmed AIDS Cases in Connecticut (as of January 24, 1984)

Mean Age	39
Sex: Males	35
Females	3
Race: White	23
Black	10
Hispanic	5
Total Cases	38
Dead	21

By Risk Group	
Homosexual/Bisexual	25
IV Drug Use	6
Hemophiliac	2
Haitian	2
Other	3
TOTAL	38

By Type of Infection	
KS no PCP	5
PCP no KS	24
PCP and KS	3
Other OI	6
TOTAL	38

By County of Residence	
Fairfield	16
New Haven	14
Middlesex	1
Hartford	6
Litchfield	1
TOTAL	38

REPORTED MORBIDITY - DECEMBER, 1983

	AMEBIASIS	BOTULISM	BRUCELLOSIS	ENCEPHALITIS (TOTAL)	Primary	Post	FOODBORNE OUTBREAKS	GONORRHEA	HEPATITIS A	HEPATITIS B	HEPATITIS NON A NON B	HEPATITIS UNSPECIFIED	LEGIONELLOSIS	LEPROSY	MALARIA	MEASLES	MENINGITIS (All Types)	Aseptic	Hemophilus influenzae	Meningococcal	Other	MUMPS	PERTUSSIS	PSITTACOSIS	RABIES IN ANIMALS	REYE'S SYNDROME	ROCKY MT. SPOTTED FEVER	RUBELLA	SALMONELLA	SHIGELLA	SYPHILIS	TUBERCULOSIS (TOTAL)	Pulmonary	Other	TYPHOID FEVER
TOTAL DEC. 1983*	12	0	1	2	2	0	3	982	3	8	3	1	7	0	0	0	41	16	10	11	4	3	0	0	0	1	0	0	68	3	14	23	16	7	1
CUMULATIVE 1983*	43	0	1	26	26	0	15	9889	85	406	57	19	38	1	12	9	342	164	55	66	57	22	7	2	6	1	3	2	1074	186	175	194	151	43	4
CUMULATIVE 1982	44	1	3	32	27	5	25	8795	118	569	33	50	55	2	18	6	262	83	55	57	67	29	5	3	6	2	3	6	908	955	153	155	116	39	2

*Subject to change when final report is submitted to the Centers for Disease Control

STATE TO DISCONTINUE PROVISION OF GAMMA GLOBULIN

For many years, the State of Connecticut Department of Health Services has provided immune globulin (IG) at no cost to health care providers for the prophylaxis of individuals exposed to cases of hepatitis A and for treatment of individuals with documented agammaglobulinemia.

This service was first provided because of limited availability of this biologic and its prohibitive cost. At the present time, there is no problem acquiring IG through normal sources. The cost of the biologic has also become minimal. Our recent experience indicates that little gamma globulin is given by the state for prophylaxis of hepatitis A. Over 90% of the IG presently distributed is for use in the treatment of individuals with agammaglobulinemia. It is no longer cost effective for the department to provide these biologics.

Therefore, effective February 15, 1984, the State of Connecticut Department of Health Services will no longer purchase, maintain, distribute or reimburse for gamma globulin in the state of Connecticut. For those individuals currently on IG maintenance programs, gamma globulin will be provided through February, 1984. Arrangements have been made with the Department of Income Maintenance (DIM) to reimburse providers for IG for individuals with agammaglobulinemia who are eligible under their criteria. All new requests for reimbursement for patients with agammaglobulinemia should be directed to DIM (Dr. Claire Callan). Third party providers have also been notified that the state will no longer be providing IG for this purpose. It is our understanding that they also will reimburse for IG only when used therapeutically for agammaglobulinemia.

Gamma globulin is available through both commercial and hospital pharmacies in the state of Connecticut. If there are any questions regarding this policy, please feel free to contact Mr. Charles Alexander, Immunization Program at 566-4141 or Ms. Patricia J. Checko, Epidemiology Section at 566-5058.

Ellen E. Jones, M.D., P.M.R.
Patricia J. Checko, M.P.H., Editor
Leonard Gilmartin, Coordinator, Public Health Education Section

EPIDEMIOLOGY SECTION
PREVENTABLE DISEASES DIVISION
State of Connecticut Department of Health Services
150 Washington Street
Hartford, CT 06106