Respiratory response to formaldehyde and off-gas of urea formaldehyde foam insulation

J.H. Day,* MD, FRCP[C], FACP
R.E.M. Lees,† MD, MFCM, DPH, FRCP[C]
R.H. Clark,‡ PhD
P.L. Pattee,* BSc, BASc

In 18 subjects, 9 of whom had previously complained of various nonrespiratory adverse effects from the urea formaldehyde foam insulation (UFFI) in their homes, pulmonary function was assessed before and after exposure in a laboratory. On separate occasions formaldehyde, 1 part per million (ppm), and UFFI off-gas yielding a formaldehyde concentration of 1.2 ppm, were delivered to each subject in an environmental chamber for 90 minutes and a fume hood for 30 minutes respectively. None of the measures of pulmonary function used (forced vital capacity, forced expiratory volume in 1 second or maximal midexpiratory flow rate) showed any clinically or statistically significant response to the exposure either immediately after or 8 hours after its beginning. There were no statistically significant differences between the responses of the group that had previously complained of adverse effects and of the group that had not. There was no evidence that either formaldehyde or UFFI off-gas operates as a lower airway allergen or important bronchospastic irritant in this heterogeneous population.

On a réalisé en laboratoire une étude de la fonction pulmonaire avant et après exposition à la formaldéhyde chez 18 sujets, dont 9 avaient rapporté divers effets nocifs extra-respiratoires de la présence de mousse isolante d'urée-formaldéhyde (MIUF) dans leurs maisons. Une première fois, on les a placés pendant 90 minutes dans une chambre d'expérience dont l'air contenait un millionième de formaldéhyde. Une seconde fois, on leur a administré dans une hotte une émanation de MIUF donnant 1,2 millionième de formaldéhyde. Les diverses mesures de la fonction pulmonaire (capacité vitale maximum, volume expiratoire maximum seconde, débit maximum en milieu d'expiration) n'ont donné aucune indication cliniquement ou statistiquement significative d'un effet de ces gaz, ni à la fin de l'expérience ni au bout de 8 heures depuis son début. La réponse des sujets ayant préalablement rapporté des effets nocifs et celle des autres ne diffèrent pas non plus de façon statistiquement significative. Il n'est donc pas démontré dans cette population hétérogène que la formaldéhyde ou les émanations de la MIUF agissent comme un allergène sur les voies aériennes inférieures ou comme un irritant susceptible de provoquer un bronchospasme important.

In several countries there has been concern that the degradation products of urea formaldehyde foam insulation (UFFI), especially formaldehyde, may cause adverse health effects.[1] Formaldehyde is the principal product of the deterioration of UFFI, and it also exists as a free gas within the foam. Canada has banned the use of UFFI,[2] and other countries have set upper limits, usually around 0.1 parts per million (ppm), for formaldehyde concentrations in the air of homes or other buildings.[3]

In its final report the Department of National Health and Welfare's Expert Advisory Committee on Urea Formaldehyde Foam Insulation[4] stated that "the role of formaldehyde as an irritant and potential allergen affecting skin and ocular and nasal mucous membranes as well as the lungs is recognized. . . . Some individuals may become highly responsive to low doses leading to debilitating dermatitis, rhinitis, conjunctivitis and asthma." Labelle and coworkers[5] have suggested that formaldehyde most frequently irritates the mucous membranes because it is highly soluble in water. Exposure to a 0.1 ppm concentration in air has been associated with eye irritation and concentrations of 0.8 to 1 ppm with shortness of breath.[6]

The purpose of the following study was to assess the effects on lower respiratory tract function of formaldehyde and UFFI off-gas. We attempted to replicate a living environment through the use of an environmental chamber.

Methods

Two groups of subjects were studied: nine persons living in homes insulated with UFFI who claimed to be

From *the Division of Allergy and Immunology, Department of Medicine, †the departments of community health and epidemiology and of family medicine, and ‡the Department of Chemical Engineering, Queen's University, Kingston

Reprint requests to: Dr. J.H. Day, Department of Medicine, Queen's University, Kingston, Ont. K7L 2V7

adversely affected by that material (but not with respiratory symptoms) and nine who were either unaffected by the UFFI in their homes or were volunteers living in homes without UFFI. Their respiratory function when exposed to formaldehyde and UFFI off-gas was measured under laboratory conditions and assessed by comparison with baseline values determined after they had been breathing room air for 30 minutes. The subjects were exposed to formaldehyde (1.0 ppm) for 90 minutes in a chamber measuring 8.53 × 2.13 × 1.83 m and to UFFI off-gases for 30 minutes in individual hoods. During the 90-minute exposure to formaldehyde the ambient temperature in the chamber was about 27°C, and the relative humidity was 62.5%. Although the target level for formaldehyde in the UFFI off-gas had been 2.0 ppm, the threshold limit value recommended for occupational exposure,[7] the concentration attained was 1.2 ppm.

Air flowed into the chamber (Fig. 1) at a rate of 0.47 m^3/s, or 28.3 m^3/min, and thus provided 51 air changes per hour. Formaldehyde was prepared from a solution of formalin in methyl alcohol and was pumped into the airstream at a constant rate. The solution was atmosized and sprayed onto a hot plate, from which it rapidly evaporated. The formaldehyde concentration within the chamber could be changed by varying either the concentration in the solution or the rate of delivery. This concentration was monitored continuously with the chromatropic acid analytic method of the National Institute for Occupational Safety and Health[7] and intermittently with Gastec colorimetric tubes.

Wet- and dry-bulb temperatures were determined frequently throughout the exposure periods with a Wibget meter.

UFFI off-gas was generated in a sealed drum containing about 1 kg of broken-up urea formaldehyde foam that had been dampened with water and allowed to stand overnight at 28°C. Pumping of air into the drum forced the gases emitted from the UFFI into two 4500-L polyethylene balloons, where the concentration of formaldehyde was measured before and during the exposure periods. Individual feeder tubes led from each balloon to the hoods. The balloons exerted a constant pressure as they were forcibly collapsed, thereby ensuring a steady flow of contaminated air to each subject.

Inside each fume hood the corrugated plastic tubing that delivered the UFFI off-gas had been fashioned into a crown with holes cut in it so as to direct the air flow away from the eyes. The mixture of air and gas filled the hood and escaped at shoulder level through an open cape.

To eliminate the possibility that clothing, toiletries or perfumes might produce confounding effects the subjects washed with soap, showered in warm water and donned surgical gowns and caps before entering the chamber or hood. Room air (which actually contained formaldehyde at a concentration of 0.02 ppm) was administered before either of the study gases was introduced.

Lung function was assessed before, immediately after and 8 hours after the beginning of each exposure by measuring the forced vital capacity (FVC), the forced expiratory volume in 1 second (FEV_1) and the forced expiratory flow at 25% to 75% of the vital capacity ($FEF_{25\%-75\%}$). A Collins Apex DS spirometer was used with an Apex 420 data processing unit. Together they produced results with an average systematic difference of 4%. Pulmonary function was tested while the subjects wore a noseclip and were standing. Each participant first received a 10-minute period of training. Spirograms with artefacts caused by coughing, hesitation or premature termination were rejected. All measurements were made in triplicate, and the best effort of each subject was used for analysis.

Each subject kept a log of symptoms during the exposure periods, making entries every 15 minutes. The data recorded by subjects, technicians, nurses and physicians were submitted to the investigators at the end of the day.

All the subjects were challenged with five inhalations of 5 mg/mL of methacholine administered with a dosimeter, and the change in FEV_1 was assessed.

Skin patch testing was carried out with an aqueous solution containing 2% formalin.

The *t*-test for paired observations was used to evaluate changes in pulmonary function before and after exposure, and Wilcoxon rank sums were calculated to determine whether there was any difference between the two groups of subjects. A probability of 5% or less was taken as the level of statistical significance in all the tests.

Results

Complaints of conjunctival and upper respiratory tract irritation were common in both groups of subjects during exposure to formaldehyde and to UFFI off-gas (Table I). Symptoms were particularly noticeable early in each period.

The *t*-test for paired observations showed that the differences in FVC, FEV_1 and $FEF_{25\%-75\%}$ measured before and after each exposure were not statistically significant (Tables II and III). The result of the Wilcoxon rank sum test for 9 × 9 observations gave a value of 66, which was not statistically significant.

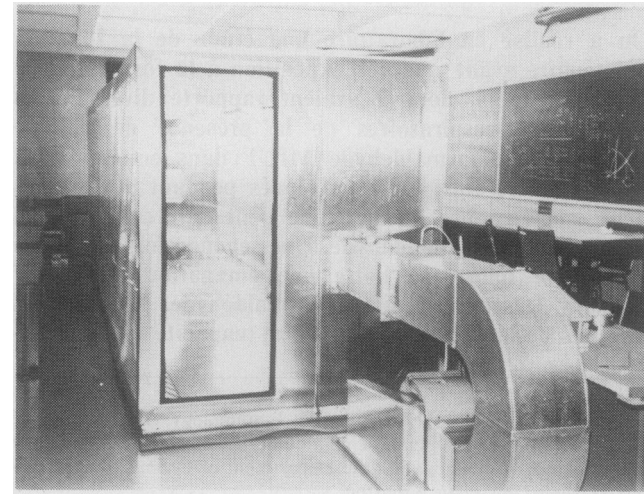

Fig. 1—Environmental chamber used to assess effects on lower respiratory tract function of formaldehyde and off-gas of urea formaldehyde foam insulation.

After the methacholine challenge the mean change in the FEV_1 in the group originally complaining of UFFI-related symptoms was −3.2% (extremes, −13.0% and 7.4%). In the other group the mean change was 0.04% (extremes, −6.0% and 5.0%). The number of subjects reacting to the methacholine with a decrease of 10% or more in the FEV_1 did not differ significantly between the two groups ($\chi^2 = 0.74$ with 1 degree of freedom; $0.2 < p < 0.5$). Patch testing with formalin gave no positive results in either group.

Discussion

Both groups experienced a high rate of eye irritation and moderate rates of nasal congestion and tearing during exposure to the gases. Most of the subjects rapidly became tolerant of eye, nose and throat irritation.

Table I—Frequency of symptoms in two groups of nine subjects each who were exposed to formaldehyde, 1 parts per million (ppm), for 90 minutes

Symptom	No. of subjects		
	Previously complaining of adverse effects	Not previously complaining	Total
Eye irritation	7	8	15
Nasal congestion	3	4	7
Tearing	3	3	6
Throat irritation	2	3	5
Nasal discharge	2	1	3
Cough	0	2	2
Chest tightness	1	0	1

The symptoms were consistent with those found in previous studies of formaldehyde exposure.[3,8] Breysse,[8] for instance, studied 92 people in mobile homes in the United States and recorded complaints of respiratory tract irritation, headache, drowsiness, nausea and nasal irritation from a large number of them. Formaldehyde concentrations in the master bedrooms of their homes ranged from 0.04 to 2.1 ppm. The source of formaldehyde was particle board and not UFFI.

The irritant properties of formaldehyde are accepted as a cause of respiratory symptoms. The role of this substance as an allergen is uncertain. Formaldehyde is known to sensitize skin, though, and this supports the possibility that it may also function as an allergen of the respiratory tract. In that case people allergic to it could be at risk from exposure to low concentrations. Porter,[9] for instance, reported the repeated development of acute respiratory distress in a physician who, in preparing anatomical specimens for demonstration, inhaled formaldehyde. While a chemical pneumonitis might have been responsible, the recurrent nature of the distress raised the possibility that the physician had become sensitized.

Popa and colleagues[10] reported bronchial asthma secondary to occupational exposure to a variety of compounds of low molecular weight, including formalin and urea formaldehyde resin. One patient had a positive response to an inhalation challenge with fumes from urea formaldehyde resin, a response accompanied by eosinophilia. It is possible that this patient had been sensitized to formaldehyde too.

We could find only one instance of lung function studies having been done on people who had been exposed to formaldehyde. In two nurses working in a renal dialysis unit the airways had become reactive in response to formalin vapours,[11,12] though in one this asthmatic response was lost after 8 years.

Table II—Pulmonary function in the 18 subjects before, immediately after and 6½ hours after the end of exposure to formaldehyde, 1 ppm, for 90 minutes

Subjects	Mean ± standard deviation (SD)					
	Forced vital capacity (FVC), L			Forced expiratory volume in 1 sec (FEV_1), L		
	Before	Immediately after	6½ hours after	Before	Immediately after	6½ hours after
Previously complaining of adverse effects	4.32 ± 0.91	4.30 ± 0.91	4.41 ± 0.90	3.31 ± 0.79	3.32 ± 0.81	3.41 ± 0.77
Not previously complaining	4.77 ± 1.26	4.77 ± 1.24	4.73 ± 1.22	3.81 ± 1.00	3.75 ± 1.04	3.71 ± 1.02

Table III—Pulmonary function in the 18 subjects before and immediately after exposure to off-gas of urea formaldehyde foam insulation (formaldehyde concentration in the air, 1.2 ppm) for 30 minutes

Subjects	Mean (± SD)			
	FVC, L		FEV_1, L	
	Before	Immediately after	Before	Immediately after
Previously complaining of adverse effects	4.21 ± 0.81	4.18 ± 0.84	3.29 ± 0.72	3.30 ± 0.72
Not previously complaining	4.67 ± 1.23	4.77 ± 1.24	3.69 ± 0.99	3.77 ± 1.02

A 20% change in the FEV_1 is widely regarded as a positive response to a challenge and is commonly observed when airways are irritable or reactive. In none of our subjects was there a change in pulmonary function of that magnitude. In one subject there was a 12.6% reduction in the FVC and a 15.0% reduction in the FEV_1 immediately after the 90-minute exposure to formaldehyde. However, the fact that there was no change after the 30-minute exposure to UFFI off-gas, which had a slightly greater concentration of formaldehyde, indicates that the first response was not a type I (immediate hypersensitivity) response to formaldehyde. There was also no evidence in any subject of a delayed response, such as is observed in challenges with certain other compounds of low molecular weight.[13,14] There was no response to a single 5-mg methacholine challenge in any of the subjects, indicating that they did not have hyperirritable airways. Thus, we found no evidence that formaldehyde or UFFI off-gas acts on the lower airways as an allergen or bronchospastic irritant.

It would appear that an individual's tolerance of formaldehyde in the air is determined by the subjective perception of the inconvenience or discomfort caused by eye and upper airway irritation. The symptoms were recorded with almost equal frequency in the two groups of subjects, those who had originally complained of adverse effects and those who had not. The frequency and subjectively reported severity of the symptoms of irritation were also unrelated to changes in FVC, FEV_1 or $FEF_{25\%-75\%}$.

The variability of the constituents of UFFI off-gas leaves questions about the effects of long-term exposure unanswered. Some 50 other compounds have been isolated from UFFI under different conditions; they include acetaldehyde, acryline, ammonia, benzaldehyde, benzene, cresol, methylnaphthalene and phenol (C.J. Shirtliffe, UFFI group, National Research Council: personal communication, 1982). Formaldehyde has been used as an indicator of the amount of UFFI off-gas produced, and if some other component released at a different rate also acts on the respiratory tract the results of a challenge with UFFI off-gas could differ from study to study. By concentrating on formaldehyde, investigators may be overlooking other aspects of UFFI deterioration.

The rate at which UFFI deteriorates within the walls of insulated homes depends on such factors as ventilation, temperature and humidity. Under optimal conditions a sample of UFFI may lose up to 40% of its weight in 5 hours (C.J. Shirtliffe: personal communication, 1982). In this study large quantities of formaldehyde were generated very rapidly; presumably the other compounds were generated in proportion. Since the gas was filtered, though, the particulates that could have enhanced the effect of the formaldehyde were removed.[15]

Conclusion

Neither the formaldehyde derived from formalin nor

the UFFI off-gas used in this study produced a clinically or statistically significant change in pulmonary function, even in subjects who complained that the UFFI in their homes had had adverse effects on their health. There was no indication of an allergic response. The concentrations of formaldehyde were similar to levels reported to cause symptoms of irritation, but the duration of exposure was limited: tests of this kind should probably be carried out over a more extended period.

References

1. The health hazards of formaldehyde [E]. *Lancet* 1981; 1: 926–927

2. Campbell JS, Harrison JR: Current status of urea-formaldehyde foam insulation. *Can Med Assoc J* 1981; 125: 329–331

3. Dudney CS, Walsh PJ: *Report of the Ad Hoc Task Force on Indoor Air Pollution*, Oak Ridge National Laboratory, Oak Ridge, Tenn, 1981: 22

4. Expert Advisory Committee on Urea Formaldehyde Foam Insulation: *Final Report*, Dept of National Health and Welfare, Ottawa, 1981

5. Labelle CW, Long JE, Christofano EE: Synergistic effects of aerosols. *Ind Health* 1982; 20: 297–304

6. National Research Council–National Academy of Sciences, Committee on Toxicology: *Formaldehyde: an Assessment of its Health Effects*, National Academy of Sciences, Washington, 1980

7. National Institute for Occupational Safety and Health: *Manual of Analytical Methods*, 2nd ed, vol 1, Dept of Health, Education, and Welfare, Cincinnati, Ohio, 1977: 1–9

8. Breysse PA: Formaldehyde in mobile and conventional homes. *Environ Health Safety News* 1977; 25: 1–20

9. Porter JAH: Acute respiratory distress following formalin inhalation [C]. *Lancet* 1975; 2: 603–604

10. Popa V, Teculescu D, Stanescu D et al: Bronchial asthma and asthmatic bronchitis determined by simple chemicals. *Dis Chest* 1969; 56: 395–404

11. Hendrick DJ, Lane DJ: Occupational formalin asthma. *Br J Ind Med* 1977; 34: 11–18

12. Hendrick DJ, Rando RJ, Lane DJ et al: Formaldehyde asthma: challenge exposure levels and fate after five years. *JOM* 1982; 24: 893–897

13. O'Brien IM, Newman-Taylor AJ, Burge PS et al: Toluene di-isocyanate-induced asthma. II. Inhalation challenge tests and bronchial reactivity studies. *Clin Allergy* 1979; 9: 7–15

14. Pepys J: Occupational asthma: review of present clinical and immunologic status. *J Allergy Clin Immunol* 1980; 66: 179–185

15. Frigas E, Filley WV, Reed CE: Asthma induced by dust from urea-formaldehyde foam insulating material. *Chest* 1981; 79: 706–707