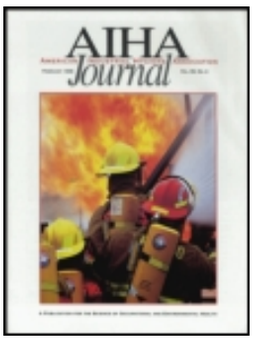

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Inhalation Exposure to Formaldehyde: An Overview of Its Toxicology, Epidemiology, Monitoring, and Control

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Increasing production and use of formaldehyde in consumer products have resulted in widespread recognition of its acute irritant effects at exposure levels below the current occupational health standard [3 parts per million parts of air (ppm)]. Formaldehyde is an allergic (immunologically mediated) skin sensitizer which may also cause or exacerbate respiratory distress in individuals with preexisting or formaldehyde-induced bronchial hyperreactivity. Formaldehyde gas is a very reactive alkylating agent which is mutagenic in several *in vitro* test systems. At exposure levels less than one order of magnitude greater than those often found in human occupational and nonoccupational environments, it induces squamous cell carcinomas in the nasal cavity of rats. Recent reviews suggest that formaldhyde exposure should be treated as though it poses a carcinogenic risk to humans and should be reduced to the lowest feasible level. This report reviews information on the epidemiologic evaluation of health effects which may result from hazardous levels of exposure to formaldehyde. Methods for monitoring exposure are discussed in detail because of considerable diversity in the methods used by state health departments for recognition, evaluation and control of nonoccupational exposures. Current guidelines for the evaluation and control of exposures to formaldehyde gas are suggested.

Introduction

In December, 1980, the Centers for Disease Control, National Institute for Occupational Safety and Health (CDC/NIOSH) and the Occupational Safety and Health Administration (OSHA) issued a joint bulletin alerting employers, employees and health officials to the laboratory evidence for formaldehyde's (HCHO) potential human carcinogenicity. The bulletin recommended that it would be prudent to reduce occupational HCHO exposures to the lowest feasible level by the use of engineering controls and stringent work practices coupled with exposure monitoring and medical surveillance.[1,2]

Before formaldehyde's carcinogenic potential was recognized, CDC/NIOSH had recommended that OSHA set an exposure limit of 1 ppm for any 30-minute sampling period to prevent acute (irritant and sensitizing) health effects.[1,3,4] However, the current OSHA occupational health standard requires that a worker's 8-hour time-weighted average (TWA) breathing zone exposure level be limited to 3 ppm (about 3.6 mg/m^3). Furthermore, a worker's permissible exposure limits include a ceiling concentration of 5 ppm and an acceptable maximum peak above the ceiling concentration of 10 ppm for no more than 30 minutes during an 8-hour shift.[5] The lowest levels at which nasal carcinomas have been observed in two-year animal inhalation studies were at airborne concentrations of 6 ppm.[6-8] In April, 1981, CDC/NIOSH reissued the bulletin on HCHO's potential as an occupational carcinogen, although OSHA no longer supported this recommendation.

In 1978, Denmark adopted quality (design-based) standards in the production of construction materials to achieve a maximum acceptable residential HCHO concentration of 0.1 ppm.[9] In 1981, a set of guidelines for the design of buildings and their ventilation systems was developed to assure adequate indoor air quality among five Nordic countries. The Nordic guidelines call for a designed minimum infiltration rate of 0.5 air changes per hour (ach) throughout the dwelling and for externally vented local exhaust systems (10 liters per second) in the kitchen and bathrooms.[9] In the U.S.A., while typical air-exchange rates are between 0.6 and 1.0 ach for older single-family residential buildings, the air-exchange rate for "energy-efficient" homes and mobile homes is often 0.3 ach or less.[10] Where indoor sources of HCHO are present, measurements of HCHO levels in the latter energy-efficient homes have given indoor-to-outdoor ratios of 6 to 10.[10-12]

Except for ozone,[13] there are no mandatory federal health standards for nonoccupational exposure to indoor air pollutants in the U.S.A.[14] Performance-based, mandatory HCHO exposure standards — for new housing (Minnesota: 0.5 ppm) and for new mobile homes (Wisconsin: 0.4 ppm and California: 0.05 ppm) — were recently proposed by state health agencies; however, these proposals have generally not been implemented because of legal challenge or administrative problems.[14,15]

On April 2, 1982, the Consumer Product Safety Commission (CPSC) issued a rule to ban all future sales of urea-formaldehyde foam insulation (UFFI) products for homes.[16] The CPSC decision to ban these products was based on

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"unreasonable risks to consumers from the irritation, sensitization and possible carcinogenic effects of HCHO emitted by these products." On April 7,1983, the U.S. Court of Appeals, Fifth Circuit vacated the rule because of inadequacies and uncertainties in CPSC's documentation of the evidence for significant hazards from HCHO released by UFFI.[17] On August 25, 1983, the U.S. Solicitor General decided not to appeal the latter decision.[18] Finally, in an Advance Notice of Proposed Rulemaking on May 23, 1984, the Environmental Protection Agency (EPA) designated for priority review the use of formaldehyde resins in construction materials for mobile and conventional homes.[19]

Publicity surrounding the CPSC rule to ban UFFI,[20,21] the refusal thus far of OSHA and EPA to promulgate a new regulation for HCHO as a potential human carcinogen,[22] and the lack of explicit federal regulatory or research responsibility for indoor, nonoccupational air pollution[10,14,23] have resulted in considerable pressure on public health officials to determine at what level individuals are being exposed and to characterize the risks for acute or chronic health problems resulting from exposures to HCHO.[2,11,12,24-27]

A national survey of state health department activities in 1982 revealed that 29 states (57 percent) had a program or person(s) responsible for evaluating nonoccupational exposures to HCHO.[28] However, these programs varied widely in important characteristics such as sampling and analytical methods, uses of data and recommendations for exposed persons, both affected and unaffected.[28]

The purpose of this review is to provide public health officials and others with current information about the state of knowledge, problems, and research needs in regard to the recognition, evaluation and control of inhalation exposure to HCHO. Several detailed reviews of the inhalation toxicology and epidemiology of HCHO exposure have been published recently.[8,10,11 29,30] Thus, we will clarify some misconceptions in the latter areas and provide more detailed information in the areas of monitoring and control of exposures.

Toxicology

The toxic effects of exposure to HCHO are of three types — irritation, immunologically mediated sensitization, and mutagenicity or carcinogenicity.[6-8,10,11,27,29,30] Acute mucous membrane irritation has been well-documented in experimental and epidemiologic studies.[3,10,11,16] Allergic sensitization of the skin has also been well-documented, while evidence for immunologically mediated sensitization of the respiratory tract remains uncertain.[3,10,11] Following chronic exposure, mutagenic effects have been demonstrated *in vitro* and carcinogenic effects in experimental animals.[6-8,29] Inhalation studies in animals and epidemiologic studies of workers have not resulted in convincing evidence of teratogenic effects by this route of exposure.[8,10]

Formaldhyde is a normal intermediary metabolite which is highly reactive with nucleophilic biological compounds and is essential in the mammalian biosynthesis of purines, thymine, histidine and serine.[29]It is generated endogenously from serine or from N-, O-, and S-methyl compounds. The endogenous levels of HCHO and its principal metabolite, formic acid, are closely controlled by homeostatic mechanisms.[29-34]

Regardless of whether formaldehyde is absorbed following inhalation or produced endogenously, it is rapidly metabolized to formic acid ($t_{1/2}$ = 1.5 minutes), CO_2, and one-carbon-pool metabolites which may be incorporated into proteins or nucleic acids. The latter metabolic processes require folic acid and involve the initial adduction of HCHO to the well-known xenobiotic "scavenger" and coenzyme for HCHO dehydrogenase, glutathione.[29] No significant increases over the average endogenous level of HCHO occurred in the nasal mucosa or blood of rats exposed to carcinogenic levels (6 to 15 ppm) of HCHO gas.[8,29,30]

The questions of whether inhaled HCHO, its metabolites, or the products of its reactions with other environmental chemicals are responsible for its toxic effects and whether the pathogenetic mechanisms are the result of individual- or species-specific biochemical, cytotoxic, or immune-mediated events have not been answered.[8,30]

Epidemiology — Acute Effects

There is no population threshold for the acute irritant symptoms associated with inhaling HCHO gas.[8,10,11] The characteristic symptoms are experienced by 30 to 50 percent of the general population at exposures of 0.5 to 1.5 ppm (below the current OSHA standard of 3 ppm). They are nonspecific, transient, exposure-dependent, and generally mild (Table I).[35] However, they may be considerably more severe in individuals who have preexisting or HCHO-induced bronchial hyperreactivity.[10,11,26,36-38] The only available prospective survey of inhalation exposures and symptoms among a group of mobile home dwellers demonstrated a strong positive relationship between the incidence of monthly complaints of acute irritant effects and the logarithm of low-level HCHO exposures.[39] The investigators, however, did not characterize the baseline health status of the participants who may not have been representative for such risk factors as atopy, asthma, cigarette smoking, occupational exposures, and bronchial hyperreactivity.[35-39]

About 30 percent of atopic nonasthmatics (10 million Americans), 5 percent of nonatopics (8.5 million), and virtu-

TABLE I
Acute Adverse Health Effects from Inhalation Exposures to Formaldehyde at Various Concentrations[A]

Ambient Exposure Level (ppm)	Reported Adverse Health Effects[B]
<0.05 - 1.5	50% to 70% of exposed may report no effects
0.05 - 1.	Odor threshold (before accommodation occurs)
0.05 - 2.	Eye irritation
0.10 - 25.	Upper airway (nose and throat) irritation
5. - 30.	Lower airway and pulmonary effects
50. - 100.	Pulmonary edema, inflammation, pneumonia
>100.	Death

[A]Adapted from data summarized in References.[1,10,11]

[B]Even at low levels of exposure, indirect central nervous system or systemic effects (*e.g.*, headache, dizziness and nausea) may accompany the symptoms listed.

ally all asthmatics (9 million) will exhibit bronchial hyperreactivity when given a methacholine inhalation challenge test.[10,11,27,37,38] These 27.5 million people (about 10 to 12 percent of the general population) may represent a pool of hypersusceptibles in relation to the acute and chronic effects of a wide variety of airborne irritants, including HCHO. Among a small group of healthy young adults tested in a National Research Council study, 30 percent reported mild irritation of the eyes, nose, and/ or upper airway and an additional 10 to 20 percent had more marked reactions to brief inhalation exposures to 0.5 to 1.5 ppm of formaldehyde.[27] The long-term consequences of mucous membrane and respiratory tract irritation from prolonged or intermittent exposures to various levels of HCHO in humans are not known.[8,10,11,35]

The onset and/ or exacerbation of asthma or nonspecific bronchial hyperreactivity has been associated with exposures to HCHO and HCHO-containing products in case reports.[3,8,10,11,40-42] Recognition of this problem and its evaluation and control in occupational settings has been limited because susceptible workers may migrate out of exposed jobs, leaving nonsusceptibles at prevalent exposure levels (the "survivor" effect). In nonoccupational settings, isolated case reports appear to have increased, as have the use of consumer products which are sources of HCHO and the energy-conserving practice of "tightening" offices and homes.[10,11,16] Environmental HCHO exposure levels and other factors (e.g., individual susceptibility) which may result in skin sensitization or bronchial hyperreactivity are not well understood. If HCHO-induced asthma or bronchial hyperreactivity develops, even control at the lowest feasible level may not prevent symptoms. More research to characterize the epidemiology, elucidate the etiologic mechanism(s), identify susceptibles, and establish safe exposure levels is clearly needed.[8,10,11]

Epidemiology — Chronic Effects

Conclusions cannot be drawn with sufficient confidence from published mortality studies of occupationally exposed adults as to whether or not HCHO is a human carcinogen.[8,43,44] This is because of numerous limitations in study design and methodology, including lack of reliable and complete information on exposure and outcomes for groups of potentially exposed individuals; insufficient latency time between initial exposures and ascertainment of cases; insufficient sample sizes of exposed adults; inadequate characterization of historical exposures to HCHO and other potential carcinogens; use of mortality data, rather than incident cases, despite significant survival after diagnosis and treatment of nasal carcinoma; inadequate follow-up in the face of "survivor" effects due to job-migration among occupational groups; and, consequently, weak statistical power to detect a true excess of nasal cavity cancer among exposed workers.

A number of mortality studies of workers in exposed job categories are in progress or have been completed; however, all of these studies (Table II) have limited statistical power to to detect a true excess of risk for nasal cancer (up to a 28 percent chance or power of detecting a 2-fold excess in relative risk, RR).[45,46] Nasal cancer is extremely rare in the general population. The annual incidence among males is about 8 per million, according to population-based cancer registry data in Connecticut and Denmark. Thus, the most

TABLE II
Epidemiologic Mortality Studies in Progress for Formaldehyde-Exposed Adults

Principal[A] Scientist	Study[B] Type	Study Group	Time Period	Study Size	Power[C] (%) to Detect Nasal Cancer, RR = 2.0
Blair (NCI)	SMR	Anatomists	1888-1970	2500	13
Blair (NCI)	SMR	HCHO use & production	1950-1966	25 000	28
Walrath[47]	PMR	Embalmers	---	1200 deaths	10
Levine[48]	SMR	Undertakers	1944-1967	1500	9
Matanoski (Johns Hopkins)	SMR	Pathologists	1900-1920	4268	20
Grauman (NCI)	SMR	Medical technologists	1948-1970	15 478	20
Grauman (NCI)	SMR	Histotechnologists	1948-1970	5000	12
Stayner (NIOSH)	SMR	Permanent press garment workers	1959-1978	16 000	15

[A]NCI = National Cancer Institute. Unpublished data, unless otherwise indicated.[46]

[B]SMR = Standardized Mortality Ratio, PMR = Proportional Mortality Ratio.

[C]In making these calculations, it was assumed that alpha = 0.05 (1-tail) and person-years for each study were estimated by the product of the study size times the number of years between the midpoint of the study time period and 1980.[45,46] RR = relative risk.

powerful approach to assessing a link between HCHO exposure and nasal cancer in humans maybe a case-control study.

Given inadequacies in the available data and populations under study, it may not be possible to detect an increased risk for HCHO-induced nasal cancer in exposed humans until some time in the future. Nevertheless, such uncertainties should not compromise prudent public health control measures in the interim.

Evaluating Exposure to Formaldehyde Gas — Environmental Monitoring

Approximately 5.2 billion pounds of HCHO were consumed in 1983 in the manufacture of more than 29 types of products (including printing materials, textiles, brake linings, paint pigments, plastic moldings, cosmetics, fumigants and disinfectants) and in the performance of medical services.[1,2]

About 50 percent of the HCHO produced is used to make HCHO-based resins for adhesives in the manufacture of particleboard, fiberboard and plywood.[1] Since 1970, UFFI has been installed in over one million homes and may now be the most notorious source of nonoccupational exposure to HCHO vapor.[2,16] It is not at all certain, however, that emissions from UFFI, when compared with other sources in occupational and nonoccupational settings, result in the most intense or persistent airborne exposures.[1,10,11]

TABLE III
Summary of Formaldehyde Levels in Selected Surveys: Occupational and Nonoccupational Indoor Environments

Sampling Location	Formaldehyde Levels in Air (ppm)[A]
Occupational[B]	
Fertilizer production	0.2 - 1.9
Dyestuffs	<0.1 - 5.8
Textile manufacture	<0.1 - 1.4
Resins (foundry)	<0.1 - 5.5
Bronze foundry	0.12 - 0.8
Iron foundry	<0.02 - 18.3
Treated paper	0.14 - 0.99
Hospital autopsy room	2.2 - 7.9
Plywood industry	1.0 - 2.5
Urea-formaldehyde foam applicators	<0.08 - 2.4
Nonoccupational[C]	
Mobile day care centers, Denmark	0.24 - 0.55
Complaint mobile homes, Minnesota	N.D. - 3.0
Complaint mobile homes, Washington	N.D. - 3.4
Complaint mobile homes, Wisconsin	N.D. - 3.68
Noncomplaint mobile homes, Wisconsin	
≤3 years old (100 homes)	0.54 ppm (average)
>3 years old (37 homes)	0.19 ppm (average)

[A]All of these data were obtained by using NIOSH recommended sampling and analytical methods.[50-52] However, they do not necessarily represent time-weighted averages for samples collected over an 8-hour sampling period, nor were they collected in a sampling strategy for the determination of industry-wide or national averages of exposure (with the exception of urea-formaldehyde foam applicators).[1,3,56]

[B]See References.[1,56]

[C]See References.[10,11,24,25] N.D. = not detectable.

Nearly a third of the more than 1.5 million workers who are potentially exposed to HCHO are engaged in health-related work. Occupational exposure levels have been measured in autopsy rooms from 2.2 to 7.9 ppm while levels in nonmedical jobs ranged from less than 0.02 to 18.3 ppm (Table III).[1] Potentially exposed consumers are being offered residential air-monitoring and air-cleaning devices which may not be capable of reliable detecting or eliminating HCHO gas at the levels which may be present in nonoccupational settings.[49] Currently, no single device is capable of detecting or removeing the wide variety of particulates, vapors and gases which might be present and could cause or exacerbate acute (irritant) symptoms similar to those attributable to HCHO in susceptible individuals.[10,11]

Several methods are available for determining the level of HCHO gas in air.[10] Selecting the appropriate sampling and analytical method is critical and must be consistent with the type of environment concerned and the anticipated concentration levels to be sampled. Most of the available methods have been developed by trained professionals who have evaluated them for use in occupational environments.[49-52] These methods may be unsuitable for the relatively low concentrations of HCHO prevalent in nonoccupational environments.[10] In an attempt to improve sensitivity, researchers have modified standard methods.[53,55] However new and modified methods have not been sufficiently evaluated for sensitivity, precision and accuracy, interferences or storage stability, especially under conditions of use by nonprofessionals in residential settings.[49]

The standard impinger collection method (in which room air is drawn or pumped at a calibrated rate through water or a derivatizing agent in solution, and the resulting solution or adduct is then analyzed with a spectrophotometer) is still suitable for short-term area samples in the workplace at a sensitivity (lower limit of detection) of about 0.1 ppm (0.19 mg/m^3) for a 25 liter sample of air.[50] However, this method is not suitable for use by nonprofessionals.

The current NIOSH-recommended adsorbent method has been designed and carefully evaluated to measure personal exposures in occupational settings down to 0.46 ppm (0.55 mg/m^3) for a 12 liter sample of air.[51] However, the latter method may not be capable of reliably detecting low-level occupational and residential exposure levels which are frequently less than 0.5 ppm (0.6 mg/m^3).[10]

Detector tube methods are relatively inexpensive and accessible to the general public (about $150-$170 for a syringe pump and 10 tubes); yet, they are only qualitative tools at best in that they do not have the sensitivity to quantify levels adequately. The recently marketed "passive" (diffusional) monitors or "badges" are accessible, convenient and relatively inexpensive (about $35 per badge, including analysis by the manufacturer). However, when used in residential settings or by nonprofessionals, they may be seriously imprecise.[49]

Most of the published data on HCHO levels in offices, factories, laboratories and mobile homes have been obtained with "active" sampling methods (*i.e.* with impingers or detector tubes). The data in Table III reflect the range of

levels reported in some occupational and nonoccupational environments.[1,9,10,56] In most instances, these data do not depict TWAs for the term of exposure nor do they identify peak excursions. Thus it cannot be determined from these data whether exposures exceeded the current OSHA standard of 3 ppm or the ACGIH TLV of 1 ppm. The data in Table III, however, show that significant occupational and nonoccupational inhalation exposures to HCHO may occur widely.

When indicated, exposure should be monitored with an appropriately designed sampling strategy[4] and personal sampling devices. Whatever sampling method is chosen, it should be critically evaluated for compatibility with the sampling strategy as well as for adequate sensitivity and precision. The specificity of the analytical method should be considered, including the potential for interference by phenols and other chemicals which may be present in the indoor environment.[5,50,51]

Strict adherence to the manufacturer's directions for use or the NIOSH-recommended sampling and analytical procedure is essential to obtaining valid results. If analytical results are to be properly interpreted, the following information must be gathered and recorded:

1. The basis for conducting environmental monitoring (e.g., the occurrence of relevant acute or chronic health complaints, the introduction of new products or processes which are potential sources of HCHO or the evaluation of control measures).
2. Date and time of sampling; characteristics of room ventilation (*e.g.*, air exchange rate per occupant, use of natural or mechanical ventilation); room temperature, barometric pressure, and relative humidity; and presence of potential sources of HCHO or other potentially hazardous chemicals (*e.g.*, from cigarette smoke; gas-burning range; wood-, kerosene-, or coal-burning stove; construction and insulation materials; carpets and furnishing; deodorants; household chemicals; and industrial processes).
3. The type of sampling method used, the length of sampling time, flow rate and volume of air sampled, location and number of samples taken, possible interferences, the manner in which samples were stored and handled before analysis, modifications (if any) made to the recommended sampling and analytical procedures, and the limit of detection for the analytical method.
4. Information similar to that outlined above (in 3.) for comparison samples (personal, area and outdoor), obtained concurrently from neighboring or nearby noncomplainant environments, and for blank samples.
5. Information about the methods and results of previous (historical) air sampling for HCHO and other contaminants — for comparison with present (prevalent) levels and for assessment of the efficacy of control measures.
6. Results, including an indication of the reliability and precision of the method at the levels of exposure reported (*e.g.*, by reporting the 95 percent confidence limits) and an interpretation by reference to current evaluation criteria for occupational or nonoccupational settings.[1,3,8,10]
7. If epidemiologic or health effects evaluations are being conducted, the demographic characteristics and health status of the exposed occupants and a suitable comparison group, specified by the use of a standardized questionnaire which includes queries regarding medical risk factors, occupational as well as lifestyle exposures, chronic symptoms, and the temporal and spatial occurrence and nature of acute complaints.[57]

Evaluating Exposures to Formaldehyde Gas — Biological Monitoring

Highly sensitive methods are available for measuring HCHO[33] and formate[34,58] in biological fluids and tissues. However, the level of formate in human blood or urine may vary widely from individual to individual, as well as within an individual on an hourly or daily basis as a result of genetic diversity, differences in diet and exercise, and other factors apart from exposures to HCHO gas from cigarette smoke and other sources. Also, the limits of detection and quantitation of these methods for formate may not permit recognition of increments from low-level inhalation exposures to HCHO.[59]

Determination of formic acid in blood and urine may be useful in clinical monitoring of a patient who has been seriously poisoned by **ingestion** of formaldehyde.[31,32,58] However, the relationship between environmental or even occupational **inhalation** exposures to HCHO and formate concentration in human tissues has not been adequately studied. In efforts to document inhalation exposure to formaldehyde, numerous physicians have referred their patients for blood and urine analyses of formaldehyde and formate levels. At the current state of knowledge, blood and urine HCHO and formate levels can not be utilized for biological monitoring of inhalation exposure to HCHO.

Control of Exposures to Formaldehyde Gas

The "tightening" of air exchange systems in buildings and mobile homes for energy conservation may result in increases in the concentration, or prolongation of exposure by recirculation, of potentially hazardous indoor pollutants.[10,60-62] These contaminants, including HCHO, may arise from outside the building; from the fabric of the building, including furnishings, carpets, construction materials and insulation; and from activities within the building, such as industrial processes, cigarette smoking (0.38 mg of HCHO is inhaled per pack of cigarettes in mainstream smoke; sidestream levels are not well characterized), and gas-burning stoves (HCHO emission rates of 15-25 mg/h have been reported).[10,11]

One or more of the following classical industrial hygiene methods may be applicable for the control of exposures in occupational settings. In residential settings where potentially hazardous exposures have been documented, the technical and economic feasibility as well as the relative effectiveness of these control measures have not been adequately evaluated, although they would presumably be useful:[1,3,10,16]

1. Eliminate the source or substitute an alternative material, product or process with a lower potential health risk.
2. Seal or enclose the source
3. Use mechanical and externally vented ventilation systems (and appropriate air-cleaning devices, if necessary) to achieve sufficient air exchange with fresh air.
4. Limit access or the total time of exposure to the contaminated environment.
5. Use appropriate personal protective equipment as an interim measure or during renovation, removal or maintenance work.
6. Educate potentially exposed workers and consumers regarding appropriate work or use practices and the above control methods.

Establishing occupational health standards for workplace exposures to potential human carcinogens and adjusting these standards for application to the nonindustrial environment of the general public present numerous ethical, technical and policymaking dilemmas requiring consistent application of well-established principles of scientific risk assessment.[1,20,22,63] Adjustment of industrial health standards, to provide health-based criteria applicable to the general public, is based on consideration of such factors as (1) the potential for continuous exposure rather than exposure limited to 40 hours per week; (2) the potential for greater susceptibility among the general public due to greater variation in age and health status; and (3) the potential benefits of periodic routine exposure and health monitoring, among certain workers, which is not available to the general public. On the basis of current data and international guidelines concerning the control of acute health effects, a performance-based standard for indoor levels of HCHO has been proposed at the level of 0.1 ppm (about 120 $\mu g/m^3$).[64]

Discussion and Recommendations

Healthful indoor climate and air quality and the energy-efficient design of heating, ventilation and air-conditioning buildings and conveyances are not necessarily mutually exclusive goals for our technological society.[10,60-62,64]

At the current state of knowledge, we are able to derive theoretical models and obtain valid and reliable measurements of indoor air pollution at low (nonindustrial) concentrations.[10,62] However, we are only beginning to apply epidemiologic methods to quantify the acute and chronic health risks of exposures to such common nonindustrial indoor air pollutants as asbestos, radon decay products, tobacco smoke, combustion products from heating and cooling devices, microorganisms, pesticides and HCHO.[10,60,61,65-67]

Based on currently available toxicologic data and the absence of definitive epidemiologic studies regarding HCHO's chronic effects on humans, the following public health approaches seem prudent:

1. When persons with relevant **acute** symptoms related in time and place to their home or workplace seek medical attention, but exposures have not been evaluated, a detailed occupational and environmental health history should be obtained.[35,68] If appropriate, an evaluation of the variety and level of potentially hazardous exposures in the occupational or nonoccupational environment may be requested from a qualified industrial hygienist, from the state or local health department or from the NIOSH regional industrial hygiene consultant.[28,69,70] Biological monitoring of HCHO or formate levels in blood or urine is not indicated.[59] The collection and analysis of HCHO gas samples by passive monitors and detector tube methods should be discouraged, especially in the hands of nonprofessionals, in favor of currently recommended NIOSH methods for occupational settings[49-52] and more sensitive methods for nonoccupational settings.[53-55] When persons with potentially HCHO-related **chronic** diseases[40,44,71] seek medical attention, evaluation of the etiological relationship will require a detailed history and, when appropriate, epidemiologic assessment.[35,44,68,70]
2. When no adverse relevant acute symptoms have been recognized, but persons are concerned about the level and duration of potential inhalation exposures to HCHO — an evaluation of the environment may still be appropriate, since only about 30 to 50 percent of the general population would be expected to experience adverse acute symptoms when exposed to low levels of HCHO (0.5 to 1.5 ppm).[10,11,27]
3. Although at present there are no mandatory nonoccupational exposure standards for HCHO in the U.S.A., when elevated exposures to HCHO gas (in relation to background environmental levels and occupational standards) have been documented by valid and reliable methods, and particularly when acute symptoms are plausibly related to HCHO exposure — exposures should be reduced to the lowest feasible level by the use of control measures such as those listed above.
4. Reconsideration of the evaluation criteria for safe inhalation exposure to HCHO gas would be appropriate in view of recent data regarding its potential for acute and chronic injury at concentrations below the current 3 ppm OSHA standard,[1,8,10,19,41]
5. Specific responsibility for research, standard-setting and enforcement of health-based criteria for indoor air quality and climate needs to be assigned,[14] since urban dwellers spend as much as 90 percent of their time indoors where the health impact, levels and control of exposures to a wide variety of airborne contaminants have not been adequately evaluated.[10,60,61]

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