DATE: May 28, 2019

TO: US Environmental Protection Agency
Chemical Control Division
Office of Pollution Prevention and Toxics (OPPT)

VIA: *Regulations.gov*

FROM: Dennis Shusterman, MD, MPH
Professor of Clinical Medicine, Emeritus
Division of Occupational & Environmental Medicine
University of California, San Francisco

Robert Harrison, MD, MPH
Clinical Professor of Medicine
Division of Occupational & Environmental Medicine
University of California, San Francisco

Timur Durrani, MD, MPH, MBA
Associate Clinical Professor of Medicine
Division of Occupational & Environmental Medicine
University of California, San Francisco

Veena Singla, PhD
Associate Director
Program on Reproductive Health and the Environment
University of California, San Francisco

Michelle Ruha, MD, FACMT
Banner—University Medical Center Phoenix
President, American College of Medical Toxicology

Paul Wax, MD, FACMT
University of Texas, Southwestern School of Medicine
Executive Director, American College of Medical Toxicology

Re: Methylene Chloride
Commercial Paint and Coating Removal Training, Certification and Limited Access Program
Docket # EPA-HQ-OPPT-2018-0844

The following comments are submitted in response to the *Advance Notice of Proposed Rulemaking for use of methylene chloride-containing paint removal products by commercial entities*, as published in the *Federal Register, Volume 84, Pages 11466-11473.* Affiliations are provided for identification purposes only and do not imply institutional endorsement. We are responding to questions 1, 2 and 6 posed on page 11470, pertaining to the appropriateness of the proposed training and certification program in mitigating the risks of methylene chloride use in paint and coatings removal.

We believe that the Training, Certification and Limited Access Program proposal will fail to address the unreasonable risks posed by the continued use of methylene chloride-containing paint removal products. We further document our reasons for supporting the USEPA's original (January 2017) proposed rule to ban the use of methylene chloride in paint removal products for all but a select few applications.

In addition to presenting our detailed rationale regarding the above-referenced questions, we are attaching two peer-reviewed academic publications for inclusion in the folio:

MacIsaac J, Harrison R, Krishnaswami J, McNary J, Jeffery Suchard MD, Boysen-Osborn M, Cierpich H, Styles L, Shusterman D. Fatalities due to dichloromethane in paint strippers: A continuing problem. *Am J Indust Med* 2013; 56:907-910.

Shusterman D, Quinlan P, Lowengart R, Cone J. Methylene chloride intoxication in a furniture refinisher: A comparison of exposure estimates utilizing workplace air sampling and blood carboxyhemoglobin measurements. *J Occup Med* 1990; 32.451-454.

QUESTIONS ADDRESSED:

1. Is a training, certification, and limited access program an appropriate method for reducing any unreasonable risks that EPA could potentially find to be presented by commercial paint and coating removal with methylene chloride? **NO**

2. Would such a program address any such unreasonable risks such that those risks are no longer unreasonable? **NO**

6. Should EPA consider requirements other than a training, certification, and limited access program for commercial uses of methylene chloride in paint and coating removal? **YES – SEE BELOW**

RATIONALE FOR OPINIONS:

The properties of methylene chloride, both physical and toxicologic, render it a uniquely hazardous substance, capable of rapidly causing neurological impairment, unconsciousness, or death.

Methylene chloride has the lowest boiling point (~40^{o} C) and highest equilibrium vapor pressure (349 – 440 mmHg at 20– 25^{o}C) of any commonly used industrial solvent (Tanaka, 1999; ACGIH, 2001; NTP 2016). Its vapors are heavier than air and tend to accumulate in tanks, mixing vessels, and bathtubs (ACGIH, 2001; NTP 2016). It is readily absorbed by ingestion, inhalation and skin contact (Stuart & Dodd, 1964; DiVincenzo et al., 1971; ACGIH 2005; USEPA, 2011). Once absorbed, it is metabolized – in part – to carbon monoxide, leading to impaired oxygen delivery to vital organs throughout the body (Stewart et al., 1972; DiVincenzo et al., 1981). Its most important acute effect is narcosis, ranging from lightheadedness, nausea and headache to respiratory depression and death (Chester et al., 2012; Fechner et al., 2001; Goulle J-P, et al., 1999; Hall & Rumack, 1990; Leikin et al., 1990; MacIsaac et al., 2013; Manno et al., 1992; Takeshita et al., 2000; Winek et al., 1981). Like other halogenated solvents, methylene chloride can sensitize the myocardium (heart) to arrhythmias (irregular heartbeat) leading to potential "sudden death" (Himmel, 2008; Zhou et al., 2011). According to a recently presented abstract

at the American College of Medical Toxicology, at least 83 US fatalities between 1980 and 2018 could be attributed to methylene chloride intoxication, the vast majority of which were due to paint removal products. Of the total fatalities, 72 (87%) were occupational in origin (Hoang et al., 2019). Additionlly, methylene chloride is classified as a confirmed animal carcinogen by three authoritative bodies, two of which also consider it a probable human carcinogen (NTP 2016; US EPA 2011; IARC 2017).

Routine industrial hygiene controls, including commonly available personal protective equipment (PPE), are easily permeated by methylene chloride.

Permeation of methylene chloride through inadequate personal protective equipment has been documented in occupational case studies – two of which are attached (MacIsaac et al., 2013; Shusterman et al., 1990). In one case study, a 65-year-old male paint factory worker was wearing an organic vapor cartridge respirator when he succumbed to methylene chloride vapors (MacIsaac et al., 2013). In an earlier case report, carboxyhemoglobin (COHb) levels were measured in a 35-year-old male furniture refinisher wearing an organic vapor cartridge respirator, goggles, and neoprene apron, gloves and boots. (COHb can be used as an indicator of methylene chloride exposures; it is created when carbon monoxide replaces oxygen in hemoglobin in the blood, thereby disrupting oxygen transport in the body.) COHb levels in this worker were indistinguishable from levels documented in unprotected volunteer subjects exposed to comparable methylene chloride levels, indicating that the PPE utilized by the worker was ineffective against methylene chloride intrusion. (Shusterman et al., 1990).

The extraordinary precautions necessary to use methylene chloride safely can only be achieved on a reliable basis in well-managed fixed operations, and are impractical in field operations. Observance of Federal OSHA standards requires air monitoring, environmental exposure controls, and personal protective equipment.

Under the Federal OSHA standard (because of methylene chloride's high volatility, toxicity, and ability to quickly saturate organic vapor cartridges), only atmosphere-supplying (supplied-air) respirators with full facepieces, hoods, or helmets are allowed for use above its Permissible Exposure Limit of 25 ppm or Short-Term Exposure Limit (STEL) of 125 ppm. (OSHA 1998; Tanaka et al., 1996). Commonly available air-purifying respirators equipped with organic vapor cartridges, whether negative pressure or positive pressure (powered air-purifying respirators) are NOT allowed by OSHA to be used for worker protection from methylene chloride exposure. Methylene chloride also permeates most common glove materials, including latex, vinyl, nitrile, and neoprene. Either PVA (polyvinyl alcohol) or EVOH/PE (ethylene vinyl alcohol / polyethylene laminate) gloves are necessary to effectively prevent significant skin exposure and percutaneous absorption.

Training and certification, to the extent that it is not circumvented in practice, may provide back-ground knowledge to users/buyers, but will not assure that proper protective equipment will actually be made available when methylene chloride-containing paint removal products are used. To achieve this goal, a program of on-site inspections would be necessary. Furthermore, the intermingling of retail and wholesale sales, with continued availability of methylene chloride-containing paint removal products in small containers, is an invitation for untrained (and unequipped) users to circumvent the intention of the proposed rule.

In light of the above, we urge EPA staff to reconsider current plans to attempt to mitigate the risk of methylene chloride exposure in paint removal products in commercial settings based on a training and certification program (i.e., "administrative controls") alone. Such EPA action would neglect the exposure monitoring and control – as well as worker health surveillance requirements – enshrined in the Code of Federal Regulations (as enforceable by Federal or State OSHA agencies). Further, it does not acknowledge the practical difficulty of providing workers with proper and effective PPE in situations in which ventilation (the effectiveness of which can only be gauged with air monitoring) is inadequate.

In this regard, it is instructive to examine the so-called "Industrial Hygiene Hierarchy of Controls," in descending order of effectiveness:

1. Substitution of safer alternative products
2. Engineering controls
3. Administrative controls
4. Personal protective equipment

The current EPA proposal deals with #3 (administrative controls) only, and by neglecting the other elements leaves workers at a continued unacceptable risk of injury, illness, and death.

We therefore wish to reiterate our view that the EPA's current recommendations are not adequate and that the proposed rule put forward by EPA in January 2017 represented the most rational and effective approach to controlling the unacceptable health risks posed by methylene chloride-containing paint removal products in commercial settings.

REFERENCES CITED

American Conference of Governmental Industrial Hygienists (ACGIH). Dichloromethane: TLV® Documentation. Cincinnati, OH; 2001

American Conference of Governmental Industrial Hygienists (ACGIH). Dichloromethane: BEI® Documentation. Cincinnati, OH; 2005.

Chester D, Rosenman KD, Grimes GR, Fagan K, Castillo DN. Fatal exposure to methylene chloride among bathtub refinishers - United States, 2000-2011. MMWR Morb Mortal Wkly Rep 2012; 61(7):119-22.

DiVincenzo GD, Kaplan CJ. Uptake, metabolism, and elimination of methylene chloride vapor by humans. Toxicol Appl Pharmacol 1981; 59(1):130-40.

Divincenzo GD, Yanno FJ, Astill BD. The gas chromatographic analysis of methylene chloride in breath, blood, and urine. Am Ind Hyg Assoc J 1971; 32(6):387-91.

Fechner G, Ortmann C, Du Chesne A, KohIer H. Fatal intoxication due to excessive dichloromethane inhalation. Forensic Sci Int 2001; 122(1):69-72.

Goulle JP, Lacroix C, Vaz E, Rouvier P, Proust B. Fatal case of dichloromethane poisoning. J Anal Toxicol 1999; 23(5):380-3.

Hall AH, Rumack BH. Methylene chloride exposure in furniture-stripping shops: ventilation and respirator use practices. J Occup Med 1990; 32(1):33-7.

Himmel HM. Mechanisms involved in cardiac sensitization by volatile anesthetics: general applicability to halogenated hydrocarbons? Crit Rev Toxicol 2008; 38(9):773-803.

Hoang A, Fagan K, Harrison R, Shusterman D, Fries D, Singla V. A comprehensive review of consumer and occupational methylene chloride fatalities in the US from 1980-2018 and policy implications. (Discussion Poster) *Presented at:* Annual Scientific Meeting of the American College of Medical Toxicology, San Francisco, CA, April 14, 2019.

International Agency for Research on Cancer (IARC). Dichloromethane. IARC Monogr Eval Carcinog Risks Hum 2017; 110 Pt. 4:177-255.

Leikin JB, Kaufman D, Lipscomb JW, Burda AM, Hryhorczuk DO. Methylene chloride: report of five exposures and two deaths. Am J Emerg Med 1990; 8(6):534-7.

MacIsaac J, Harrison R, Krishnaswami J *et al*. Fatalities due to dichloromethane in paint strippers: a continuing problem. Am J Ind Med 2013; 56(8):907-10.

Manno M, Rugge M, Cocheo V. Double fatal inhalation of dichloromethane. Hum Exp Toxicol 1992; 11(6):540-5.

National Toxicology Program (NTP): Dichloromethane. Report on Carcinogens, 14th Edition, US Department of Health and Human Services, 2016.

Occupational Safety and Health Administration (OSHA), US Dept. of Labor: Methylene Chloride Standard 11/20/1998 (https://www.osha.gov/laws-regs/regulations/standardnumber/1910/1910.1052)

Shusterman D, Quinlan P, Lowengart R, Cone J. Methylene chloride intoxication in a furniture refinisher. A comparison of exposure estimates utilizing workplace air sampling and blood carboxyhemoglobin measurements. J Occup Med 1990; 32(5):451-4.

Stewart RD, Dodd HC. Absorption of carbon tetrachloride, trichloroethylene, tetrachloroethylene, methylene chloride, and 1,1,1-trichloroethane through theh human skin. Am Ind Hyg Assoc J 1964; 25:439-46.

Stewart RD, Fisher TN, Hosko MJ, Peterson JE, Baretta ED, Dodd HC. Carboxyhemoglobin elevation after exposure to dichloromethane. Science 1972; 176(4032):295-6.

Takeshita H, Mogi K, Yasuda T *et al*. Postmortem absorption of dichloromethane: a case study and animal experiments. Int J Legal Med 2000; 114(1-2):96-100.

Tanaka S, Nakano Y, Tsunemori K, Shimada M, Seki Y. A study on the relative breakthough time (RBT) of a respirator cartridge for forty-six kinds of organic solvent vapors. Appl Occup Environ Hyg 1999; 14:691-695.

US Environmental Protection Agency (USEPA): Toxicological Review of Dichloromethane (Methylene Chloride) (CAS No. 75-09-2) In Support of Summary Information on the Integrated Risk Information System (IRIS). Washington, DC: November 2011.

US Environmental Protection Agency (USEPA): Methylene Chloride and N-Methylpyrrolidone; Regulation of Certain Uses Under TSCA Section 6(a). Federal Register 2017; Vol. 82, No. 12, pp. 7464-7533.

Winek CL, Collom WD, Esposito F. Accidental methylene chloride fatality. Forensic Sci Int 1981; 18(2):165-8.

Zhou Y, Wu HJ, Zhang YH, Sun HY, Wong TM, Li GR. Ionic mechanisms underlying cardiac toxicity of the organochloride solvent trichloromethane. Toxicology 2011; 290(2-3):295-304.

AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 56:907–910 (2013)

Case Report

Fatalities Due to Dichloromethane in Paint Strippers: A Continuing Problem

Julia MacIsaac, MD, MPH,[1] Robert Harrison, MD, MPH,[2] Janani Krishnaswami, MD, MPH,[3] Jennifer McNary, IH,[2] Jeffrey Suchard, MD,[4] Megan Boysen-Osborn, MD,[4] Hank Cierpich, BS,[5] Laura Styles, MPH,[5] and Dennis Shusterman, MD, MPH[2]*

Background *Exposure to dichloromethane (DCM or methylene chloride - CH_2Cl_2) in paint strippers continues to be an avoidable source of morbidity and mortality. DCM has been under regulatory scrutiny by occupational and consumer product agencies since the identification of its carcinogenicity in the mid-1980s.*
Methods *We investigated two independent workplace incidents that resulted in three cases of DCM intoxication from paint stripper use.*
Results *Each incident investigated resulted in a fatality. A third worker suffered obtundation requiring hospitalization and intubation.*
Conclusions *The continued occurrence of fatalities and other serious injuries due to DCM-containing paint strippers in the United States calls for a re-evaluation of existing regulatory strategies.* Am. J. Ind. Med. 56:907–910, 2013. *© 2013 Wiley Periodicals, Inc.*

KEY WORDS: dichloromethane; methylene chloride; paint stripper; fatality; chlorinated solvent; asphyxia; confined space

INTRODUCTION

Inhalation of dichloromethane (DCM) at high concentrations can cause central nervous system and respiratory depression [ATSDR, 2000, 2010]. In addition, DCM is metabolized in the liver to carbon monoxide, a phenomenon that was first reported over 40 years ago [Stewart et al., 1972]. Paint strippers may contain high concentrations of DCM and are a regulated occupational hazard. The US Consumer Product Safety Commission (CPSC) requires that consumer-available DCM-containing strippers display a warning to use in a well-ventilated space. However, the following cases illustrate that warnings alone do not ensure safety, and even personal protective equipment (such as respirators) may fail to protect. The continued occurrence of DCM-related fatalities from paint strippers argues for a more aggressive regulatory approach to protect both workers and consumers.

Additional supporting information may be found in the online version of this article.

[1]Division of Occupational and Environmental Medicine, Department of Medicine, San Francisco General Hospital, University of California, San Francisco, San Francisco, California
[2]California Department of Public Health, Occupational Health Branch, Richmond, California
[3]University of California and Kaiser Permanente Combined Preventive Medicine/Internal Medicine Residency Program, San Francisco, California
[4]Department of Emergency Medicine, University of California, Irvine Medical Center, Orange, California
[5]Public Health Institute, Oakland, California

Conflict of interests: These incidents were investigated by the California Fatality Assessment and Control Evaluation Program (FACE), which is funded by the National Institute for Occupational Safety and Health (NIOSH) and by the California Department of Public Health.

Work was performed at: The California Department of Public Health, Occupational Health Branch, 850 Marina Bay Parkway, Building P, 3rd Floor, Richmond, CA, 94804; Division of Occupational and Environmental Medicine, San Francisco General Hospital, The University of California, San Francisco, 1001 Potrero Ave, Bldg 30, Suite 3500, San Francisco, CA 94110; and at Irvine Department of Emergency Medicine, University of California, Irvine Medical Center, 101 The City Drive South, Orange, CA 92868.

*Correspondence to: Dennis Shusterman, MD, MPH, California Department of Public Health, Occupational Health Branch, 850 Marina Bay Parkway, Building P, 3rd Floor, Richmond, CA 94804-6403. E-mail: dshuster@cdph.ca.gov

Accepted 15 January 2013
DOI 10.1002/ajim.22167. Published online 28 February 2013 in Wiley Online Library (wileyonlinelibrary.com).

CASE PRESENTATIONS

Incident #1

In May 2010, a 24-year-old Hispanic male maintenance worker (Case #1) was stripping a waterproof coating off of a baptismal font located within a small enclosed room in a church. Wearing only gloves for protection, he applied one gallon of paint stripper (Klean-Strip Premium Sprayable Stripper) to the floor of the font. The stripper was purchased at a local hardware store and contained 70–85% DCM, with smaller amounts of methanol, isopropyl alcohol, 2-butoxy-ethanol, and ethanol. Six-and-a-half hours after starting the task, he was found unresponsive, on the floor of the baptismal font. Paramedics were called, but despite resuscitation attempts, the patient expired.

The deceased worker had no reported history of chronic health problems. He did not take any medications, did not smoke, and had no known contributory family history. The autopsy revealed cardiomegaly with 4-chamber dilatation and coronary atherosclerosis with 50% occlusion of the left anterior descending artery. Post-mortem studies revealed a carboxyhemoglobin (COHb) of 10% and a blood DCM level of 37.8 mg/dL. Blood methanol, ethanol, and isopropyl alcohol were undetectable. The cause of death was intoxication by DCM, resulting in hypoxia, dysrhythmia, and death.

Incident #2

In November 2011, a 65-year-old Hispanic male at a paint manufacturing facility, entered an empty paint-mixing tank, through a small opening at the top of the tank. He worked alone brushing on a chemical paint stripper (Jasco Premium Paint and Epoxy Remover; 60–100% DCM, 10–30% methanol, 1–5% Stoddard solvent) to the inside walls of the tank to remove dried paint. He wore an organic vapor cartridge respirator, but no other personal protective equipment. A fan and hose assembly exhausted contaminated air out of the tank; however, it was positioned only half way between the tank opening and the floor of the tank. Two-and-a-half hours after entering the tank, he was found unconscious at the bottom of the tank by a 45-year-old Hispanic male coworker (Case #3). Case #3 entered the tank in an attempted rescue, but was also overcome by the vapors.

One hour later, coworkers rescued the two men from the bottom of the tank. Paramedics were called and found Case #2 in asystole. He did not respond to resuscitative efforts and was pronounced dead. Case #2 had a history of diabetes and chronic neuropathic pain; medications were metformin and gabapentin. He had no known allergies and did not smoke. Post-mortem testing revealed a COHb level below the limit of detection (<5%) and a blood DCM level of 220 mg/dL. The lungs and myocardium showed congestion, but no pre-disposing organ system pathology was identified. The cause of death was asphyxia due to inhalation of DCM.

Case #3, also Hispanic, had no past medical history, took no medications, and had no allergies. He had a remote history of tobacco use. Paramedics found Case #3 to have a patent airway, shallow respirations, and a Glasgow Coma Scale (GCS) score of 3. Oxygen saturation was 82% on ambient air, which increased to 100% with bag-valve-mask ventilation and supplemental oxygen. His pulse rate was 100 beats/min, blood pressure 118/68 mm Hg, and he was afebrile. Cardiac monitoring revealed sinus tachycardia.

In the emergency department, Case #3 was combative, GCS 1-4-1 and he was intubated for airway protection. CT scans of the head, cervical spine, abdomen and pelvis, and a chest X-ray revealed no injury. Laboratory tests revealed a respiratory acidosis (pH of 7.32, pCO_2 51 mmHg), a COHb level of 4.0%, and an osmol gap of 17 mOsm/kg (normal < 10 mOsm/kg). The initial serum methanol level was 15.1 mg/dL by gas chromatography analysis (normal < 1.5; Lindinger et al., 1997). Serum ethanol, isopropyl alcohol, acetone, and ethylene glycol were undetectable (a DCM level was not determined). After 4 hr, the patient's mental status improved, he was successfully extubated and admitted to the intensive care unit for observation. His mental status continued to improve; however, his COHb levels continued to rise over the first 24 hr of hospitalization, only returning to normal

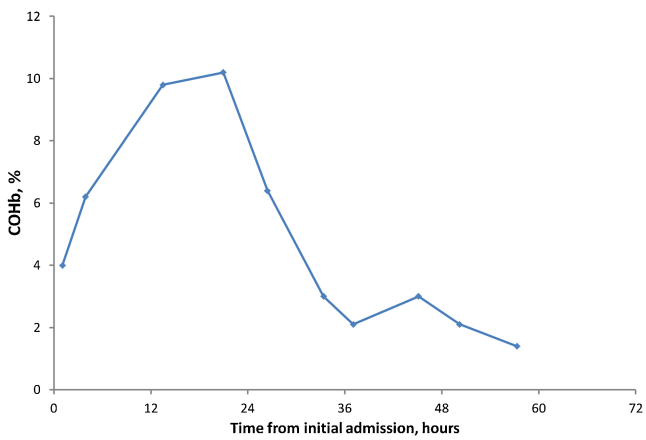

FIGURE 1. Carboxyhemoglobin (COHb) level as a function of time (in hours) from Case #3's initial arrival in the Emergency Department. COHb was 4.0% upon arrival, peaking at 10.2% 24 hr later. The patient was receiving supplemental oxygen throughout this sampling period.

after nearly 60 hr (see Fig. 1). He was discharged home without sequelae on hospital day #4.

DISCUSSION

We report here two occupational incidents: two fatalities and one case of severe obtundation linked to the use of DCM-containing paint strippers. Although the cases presented here involved occupational exposures, both products were consumer-available formulations, one of which was purchased at a local hardware store. DCM-containing paint strippers are sold in U.S. retail stores, placing consumers at risk for similar injury. Consumer deaths due to use of DCM-based strippers have been documented [Stewart and Hake, 1976; Harris County Institute of Forensic Sciences, 2007]. The hazards highlighted herein thus apply to both workers (e.g., painters, furniture or bathtub refinishers) and consumers alike.

Toxicologically, DCM is primarily an inhalational hazard causing CNS and respiratory depression: concentration of 800 ppm can disturb psychomotor performance [Dhillon and Von Burg, 1995], and concentrations of 2,300 ppm are considered to be ''Immediately Dangerous to Life and Health'' [NIOSH, 1994]. The hepatic conversion of DCM to carbon monoxide was discovered serendipitously in the early 1970s [Stewart et al., 1972; Stewart and Hake, 1976]. A rising COHb level in the absence of ongoing chemical exposure is highly suggestive, if not pathognomonic, for DCM toxicity. When methanol is co-present the peak COHb is further delayed [Stewart and Hake, 1976]. This phenomenon was apparent in Case #3. In previously reported DCM intoxications, COHb levels typically rise to 13–16% [Dhillon and Von Burg, 1995], but may reach as high as 50% [Fagin et al., 1980]. DCM-associated COHb elevations (''chemical asphyxia'') may precipitate angina, dysrhythmia and death, especially in patients with underlying cardio-pulmonary disease (as was the case for the deceased worker in Incident #1). In addition, DCM, like other chlorinated solvents, can directly sensitize the myocardium, lowering the threshold for dysrhythmias (National Institute on Drug Abuse, 1977). Each of these mechanisms likely contributed to the morbidity and mortality of the cases presented here. Despite DCM's extreme volatility, simple asphyxia was excluded given that estimated air concentrations of the stripping products were insufficient to create an oxygen deficient atmosphere (see Supplemental Information).

Beyond its acute toxicity, DCM is classified as ''reasonably anticipated to be a human carcinogen'' [NTP, 2011]. Based on carcinogenicity, the Occupational Safety and Health Administration (OSHA, OSHA 1997) set the DCM permissible exposure limit (PEL) to 25 ppm (averaged over 8 hr) and a short-term (15 min) exposure limit to 125 ppm. Even at these levels, there will be an estimated 5–11 excess cases of cancer for every 1,000 people exposed over a working lifetime [OEHHA, 2007]. In the cases presented here, DCM levels were estimated to be as high as 30,000 ppm (see Supplemental Information; Keil, 2009).

DCM-based products used in home settings can also result in high concentrations, exceeding OSHA PELs. For example, in one in-home simulation study of furniture stripping, DCM-concentrations exceeded 2,000 ppm (when used indoors without local exhaust ventilation; Hodgson and Girman, 1987). A separate case report of furniture stripping in a large basement (5,425 cubic feet) with the windows and doors closed led to some of the highest DCM-induced COHb levels ever reported (up to 40%; Langehenning et al., 1976).

Local exhaust ventilation, a supplied air respirator, and protective (e.g., polyvinyl alcohol) gloves should be employed when using DCM. Cartridge respirators are not permitted under OSHA standards since breakthrough times are extremely short, even when DCM concentrations are low. For example, for a DCM concentration of 50 ppm and at high relative humidity (80%), the breakthrough time was as short as 30 min [Moyer and Peterson, 1993]. The fact that Case #2 became obtunded and subsequently died while wearing a cartridge respirator, underscores this point. Only supplied air respirators provide sustained, reliable protection against DCM inhalation.

These incidents suggest that current regulatory standards are insufficient to protect workers and consumers from both acute and chronic DCM toxicity. The CPSC requires that DCM-containing strippers display a warning on the label advising use in a well-ventilated space [CPSC, 1986]. However, a study in which consumers were interviewed regarding their use of DCM-based paint strippers, found that warnings on the label are not adequate to guide safe use or protect against high exposures [Riley et al., 2001]. In 2009, the European Union (EU) concluded that warnings were insufficient, resulting in acute fatalities to workers and consumers. The EU subsequently banned the sale of paint strippers containing DCM to consumers and to professionals working in the field, and restricted sales to fixed commercial/industrial operations with appropriate local exhaust ventilation [European Parliament, 2009].

The EU restrictions are feasible since safe and effective alternatives to DCM-based stripping methods exist. Thermal or mechanical paint-removal methods may obviate the need for a chemical stripper altogether. Less-toxic alternatives such as benzyl alcohol-based paint strippers have been shown to be effective, economical substitutes [Morris and Wolf, 2006]. Successful strategies resulting in either DCM-elimination or reduction have been reported in several industrial settings in the United States [Roelofs and Ellenbecker, 2003].

CONCLUSIONS

We report on two deaths and one serious injury related to the use of DCM-containing paint strippers. Despite more than two decades of regulatory attention, DCM use in paint strippers has evaded effective hazard control, resulting in continued preventable morbidity and mortality. Like our counterparts across the Atlantic, regulatory agencies in the United States should consider approaches to require the use of safer, less-toxic alternatives to DCM in paint strippers.

The cases in this report were investigated under the California Fatality Control and Evaluation Program. This program has an institutional review board exemption from human subjects approval based on public health authority to investigate causes of work-related deaths. Due to this exemption, there is no requirement to obtain written permission from the cases or their next of kin and to report them at the level of detail at which they are described herein.

ACKNOWLEDGMENTS

The preparation of this manuscript was not funded. The authors wish to acknowledge Lauren Joe, MPH, and Timur Durrani, MD, MPH, for their assistance in identifying DCM-containing products and accessing poison control center data, respectively.

REFERENCES

ATSDR. 2000. Toxicological profile for methylene chloride. Atlanta, GA: US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Available at: http://www.atsdr.cdc.gov/toxprofiles/tp14.pdf (Accessed April 9, 2012).

ATSDR. 2010. Addendum to the toxicological profile for methylene chloride. Atlanta, GA: US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Available at: http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=1161&tid=42 (Accessed March 15, 2012).

California Environmental Protection Agency; Office of Environmental Health Hazard Assessment Reproductive and Cancer Hazard Assessment Branch. 2007. Occupational health hazard risk assessment project for California: Identification of chemicals of concern, possible risk assessment methods, and examples of health protective occupational air concentrations. Oakland, CA: California Environmental Protection Agency. Available at: http://www.cdph.ca.gov/programs/hesis/Documents/riskreport.pdf (Accessed November 14, 2012).

Consumer Product Safety Commission. 1986. Press release: CPSC to begin rulemaking process on methylene chloride. Washington, DC: Consumer Product Safety Commission. Available at: http://www.cpsc.gov/cpscpub/prerel/prhtml86/86012.html (Accessed April 10, 2012).

Dhillon S, Von Burg R. 1995. Toxicology update: Methylene chloride. J Appl Toxicol 15:329–335.

European Parliament. 2009. European Parliament press release: Dichloromethane to be banned in paint-strippers. Ref.: 20090113IPR46095. Brussels: European Parliament. Available at: http://www.europarl.europa.eu/sides/getDoc.do?language=EN&type=IM-PRESS&reference=20090113IPR46095 (Accessed April 10, 2012).

Fagin J, Bradley J, Williams D. 1980. Carbon monoxide poisoning secondary to inhaling methylene chloride. BMJ 281:1461.

Harris County Institute of Forensic Sciences. 2007. Investigator report ML07-3849. Houston, TX: Harris County Institute of Forensic Sciences.

Hodgson AT, Girman JR. 1987. Exposure to methylene chloride from controlled use of a paint remover in residences. Berkeley CA: Indoor Environment Program, Lawrence Berkeley Laboratory, University of California, Publication No. LBL-23078. Presented at the 80th Annual Meeting of the Air Pollution Control Association: New York.

Keil CB. 2009. Well-mixed box model. In: Keil CB, Simmons CE, Anthony TR, editors. Mathematical models for estimating occupational exposure to chemicals. 2nd edition. Fairfax, VA: American Industrial Hygiene Association, pp. 23–31.

Langehenning PL, Seeler RA, Berman E. 1976. Paint removers and carboxyhemoglobin. NEJM 295:1137.

Lindinger W, Taucher J, Jordan A, Hansel A, Vogel W. 1997. Endogenous production of methanol after the consumption of fruit. Alcohol Clin Exp Res 21:939–943.

Morris M, Wolf K. 2006. Methylene chloride consumer product paint strippers: low-VOC, low toxicity alternatives. Glendale, CA. Institute for Research and Technical Assistance (IRTA). Report prepared for: California Environmental Protection Agency Department of Toxic Substance Control. Sacramento, CA. Available at: http://www.irta.us/PaintStrippers06.pdf (Accessed April 1, 2012).

Moyer ES, Peterson JA. 1993. Organic vapor (OV) respirator cartridge and canister testing against methylene chloride. Appl Occup Environ Hyg 8:553–563.

National Institute on Drug Abuse. 1977. Review of inhalants. Euphoria to dysfunction. Sharp CW, Brehm ML, editors. Rockville, MD (DHHS Research Monograph Series No. 15).

NIOSH. 1994. Documentation for immediately dangerous to life and health concentrations (IDLHs). Cincinnati, OH: US Pepartment of Health and Human Services, CDC, National Institute for Occupational Safety and Health. Available at: http://www.cdc.gov/niosh/idlh/75092.html (Accessed April 5, 2012).

NTP. 2011. Report on carcinogens. 12th edition. Research Triangle Park, NC: US Department of Health, Human Services, Public Health Service, National Toxicology Program. Available at: http://ntp.niehs.nih.gov/ntp/roc/twelfth/roc12.pdf (Accessed April 6, 2012).

Occupational Safety and Health Administration. 1997. Occupational safety and health standards: methylene chloride. 29 CFR 1910.1052. Washington, DC: US Department of Labor, Occupational Safety and Health Administration. Available at: http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=standards&p_id=10094 (Accessed March 17, 2012).

Riley DM, Fischhoff B, Small MJ, Fischbeck P. 2001. Evaluating the effectiveness of risk-reduction strategies for consumer chemical products. Risk Anal 21:357–369.

Roelofs CR, Ellenbecker MJ. 2003. Source reduction for prevention of methylene chloride hazards: Cases from four industrial sectors. Environ Health 2:9.

Stewart RD, Fisher TN, Hosko MJ, Peterson JE, Baretta ED, Dodd HC. 1972. Carboxyhemoglobin elevation after exposure to dichloromethane. Science 176:295–296.

Stewart RD, Hake CL. 1976. Paint-remover hazard. JAMA 235:398–401.

Brief Communication

Methylene Chloride Intoxication in a Furniture Refinisher

A Comparison of Exposure Estimates Utilizing Workplace Air Sampling and Blood Carboxyhemoglobin Measurements

Dennis Shusterman, MD, MPH; Patricia Quinlan, MPH, CIH; Ruth Lowengart, MD, MSOM; and James Cone, MD, MPH

A 35-year-old furniture refinisher came to the occupational medicine clinic with complaints of upper respiratory irritation, fatigue, and lightheadedness occurring on a daily basis after using a methylene chloride-containing paint stripper. Determinations of blood carboxyhemoglobin (COHb) on three occasions showed an apparently linear elevation of COHb as a function of hours worked on the day of sampling. COHb levels predicted from spot industrial hygiene measurements were in close concordance with those observed in the patient, indicating the potential usefulness of COHb monitoring in estimating airborne exposure levels.

Methylene chloride (or dichloromethane) is an organic solvent that has found wide use as a degreaser, paint remover, aerosol propellant, and a blowing agent for polyurethane foams, and as a solvent in food processing, photographic film production, and plastics manufacturing.[1]

Discovery of its unusual metabolic fate—conversion to carbon monoxide in vivo— has earned the compound a special place in the solvent toxicology literature.[2-6] Demonstration of oncogenicity in experimental animals has occasioned a reconsideration of exposure limits, with emphasis upon stricter controls.[7,8] In some workplaces, conditions prevail in which controls are inadequate to prevent even acute toxicity, much less long-term exposure risks.

Case Presentation

A 35-year-old male furniture refinisher was referred to the occupational medicine clinic for evaluation of intermittent eye and throat irritation, nasal discharge, nonproductive cough, fatigue, and lightheadedness that he had been suffering for 15 months. He denied headache or chest pain at the time of initial evaluation. He had worked full-time in his present job for approximately 2 1/2 years, applying an 80% methylene chloride, 20% methanol stripping solution to furniture with a brush and pump assembly. Other tasks included dipping furniture in tanks containing the stripper and water-blasting residual stripper and finish from furniture.

The patient wore rubber boots, a waterproof apron, neoprene gloves, goggles, and a half-mask organic vapor cartridge respirator. According to the patient, he could detect solvent smells within a few days of a respirator cartridge change, but described being limited to cartridge changes approximately once every 10 days because of the expense involved. While water-blasting furniture, the patient frequently removed his respirator

From the Occupational Health Clinic, University of California at San Francisco General Hospital, San Francisco, CA 94110 (Dr Shusterman, Ms Quinlan, Dr Cone [Chief]) and the Contra Costa County Occupational Medicine Program, Martinez, CA 94553 and the University of California, San Francisco (Dr Lowengart).

Address correspondence to: Hazard Evaluation Section, California Department of Health Services, 2151 Berkeley Way, Room 517, Berkeley, CA 94704 (Dr Shusterman).

0096-1736/90/3205-451$02.00/0

because the mist increased the effort of breathing through it. A slot ventilation system had reportedly been connected around the periphery of the furniture-scrubbing tables in the past, but when these tables had been relocated about 1 1/2 years earlier, this system was left disconnected. Area ventilation was provided only by air movement through a large sliding door, in the front of the shop, that was normally kept open.

The patient noticed symptoms at work, with improvement on weekends and when on vacation. Symptoms were worse when using old respirator cartridges, on hot days, or with extensive use of the dip tanks. Symptoms of lightheadedness tended to peak in the afternoons and resolve in the evenings, whereas upper respiratory irritation frequently persisted through the work week. The patient was a nonsmoker, having quit 3 years earlier after smoking one quarter of a pack per day for 5 years. He consumed one to two six-packs of beer each weekend, but none during the work week. He took no regular medications and had no history of hemolytic anemia or other blood dyscrasias. There was no history of allergies or hay fever, and review of his nonoccupational exposures revealed no pets, passive smoking, excessive dust, or use of forced-air heating or woodstoves. His occasional hobby was oil painting (once or twice a month), at which times he kept windows open for ventilation.

Physical examination revealed slight injection of the tonsillar pillars and nasal mucosa, with no conjunctival injection. Results of cardiac and pulmonary examinations were normal. Examination of the abdomen revealed a nontender and non-nodular liver edge two fingerbreadths below the costal margin; the liver span was not clearly percussible. The patient behaved normally during a mental status examination, which included alertness, affect, orientation, short-term memory, and calculation. Results of a limited neurological examination (cranial nerves, deep tendon reflexes, sensation, Romberg, and gait) were within normal limits. Review of the results of a chemistry panel obtained 2 months before examination was remarkable only for an aspartate aminotransferase of 44 Karmen units/mL (normal, <40 Karmen units/mL), with normal alanine aminotransferase, alkaline phosphatase, and bilirubin levels.

A presumptive diagnosis was made of upper respiratory irritation and transient symptoms of central nervous system depression secondary to methylene chloride exposure. Note was also made of an isolated, marginally elevated liver function test and possible hepatomegaly.

A carboxyhemoglobin (COHb) level was obtained 3 hours after completing half of an 8-hour workday; the result was later reported as 7.5% (less than 2% is normal for nonsmokers).[9] The patient was instructed to discontinue his weekend alcohol consumption and recreational oil painting, and a repeat liver function panel and COHb were scheduled for 1 month later.

An industrial hygiene inspection of the patient's work site identified the use of uncovered buckets for recycling the stripping solution, a nonoperative slot ventilation system with no overhead ventilation, the draining of paint sludges within the work area, and the performance of water-blasting in an unenclosed portion of the work area. Air sampling ("spot" samples using detector tubes (National Draeger, Inc, Pittsburgh, Pa). in the employee's breathing zone) showed methylene chloride levels of 350 ppm in the production area during manual stripping (the patient's primary activity), and 100 ppm in the break room. A review of the clinic's industrial hygiene records revealed that a previous inspection of the same workplace 4 years earlier had demonstrated similar methylene chloride levels, despite the fact that the slot ventilation system had been working at the time. Recommendations made at that time regarding the installation of a general ventilation system had not been heeded.

The employer agreed to request an industrial hygiene consultation and to make respirator cartridges available for frequent changes.

One month after the initial visit, the patient reported a slight improvement in his symptoms of sore throat, cough, and dizziness after partial modification of the work practices, including covering solvent reservoirs between use, at least weekly changes of the respirator cartridges, and consistent use of a respirator during water-blasting. The ventilation system, however, remained unchanged. Weekend beer-drinking and oil painting had been curtailed since the last visit, and repeat liver function test results were within normal limits. A follow-up COHb level test was scheduled, but was delayed several times because of the patient's concern with lost work time.

Two months later, the patient reported that he had been unable to obtain replacement respirator cartridges for more than 3 weeks and had begun to experience headaches at work. The repeat carboxyhemoglobin level at that time was 10.4% (approximately 40 minutes after completing 6.5 hours of work). Symptoms persisted into the following working day, and the patient was placed on temporary total disability pending reduction of methylene chloride exposures. The employer was informed of the decision and was again urged to effect rapid abatement of the workplace hazard, in accordance with the industrial hygiene recommendations of his consultant. After 3 days off work, the patient reported nearly complete resolution of symptoms, and a repeat physical examination showed normal results. A baseline COHb level obtained at that time was 1.1%, well within the normal range. Over the subsequent year the patient has been unemployed, his employment having been terminated by the employer, ostensibly for reasons unrelated to health. His symptoms have completely resolved.

Discussion

This patient's symptoms of intermittent eye and throat irritation, lethargy, and (eventually) headaches were consistent with repeated episodes of acute methylene chloride intoxication. The patient's single, marginally elevated liver function test results (and possible hepatomegaly), on the other hand, were difficult to ascribe specifically to his methylene chloride exposure,

given his weekend ethanol consumption, occasional oil painting, and the borderline nature of the findings. There were no clinical indications of cardiovascular compromise or chronic neurotoxicity, as evidenced by the absence of chest pain, neurological abnormalities, or mental status changes.

The formation of carboxyhemoglobin from CO generated in vivo from the metabolism of methylene chloride can be used to estimate methylene chloride exposure levels. Such estimates presume that there are no important confounding exposures (eg, to vehicular exhaust or cigarette smoke) that also raise COHb.[10] Methylene chloride is well absorbed during both inhalation and cutaneous exposure.[11,12] Approximately 70% of an inhaled dose is retained; of this retained fraction, about 5% is excreted unchanged in alveolar air and 25% to 34% is eliminated from the lungs as CO. The remainder (up to 70% of the absorbed dose) is metabolized to carbon dioxide.[11] The conversion to CO occurs via a saturable hepatic microsomal pathway, although saturation probably does not occur at exposure levels below 350 to 500 ppm.[13] Several investigators have examined the kinetics of methylene chloride conversion to carbon monoxide, under either controlled conditions or working conditions.[2-6,11,13,14] In these experiments, COHb levels typically plateau for a period of 2 to 3 hours post-exposure before beginning to drop, presumably reflecting mobilization of solvent from fat stores and continued conversion to carbon monoxide.[6] The subsequent half-time of COHb clearance is approximately 13 hours—3 times longer than is observed in CO intoxication alone.[4,10]

This plateau and prolonged elimination phase of methylene chloride-derived COHb extends the critical period for biological monitoring. This is fortuitous in the present case, because the two post-exposure COHb levels were obtained after differing unexposed intervals. After initially concluding that the upward trend in COHb levels in this patient represented a worsening of the industrial hygiene situation, we realized that the difference might be related to duration of exposure. The three COHb determinations were subsequently graphed as a function of hours of work exposure (Fig. 1), with the data points being consistent with a linear relationship between the two variables ($r = .997$; slope = 1.45% per hour). Based upon extrapolation from these data, it was estimated that an 8-hour exposure in this workplace would be expected to produce a COHb level of 12.7%, assuming continued linearity with exposure time.

Figure 2, by comparison, represents the COHb levels achieved in two separate studies of 7.5-hour controlled exposures of sedentary human subjects at various methylene chloride levels. In these studies, a high degree of linearity is apparent between exposure concentration and COHb level ($r = .997$).[14,15] Extrapolating from the highest exposure level in these experiments (250 ppm) to the measured concentration in the production area of the furniture-stripping shop described here (350 ppm), one would predict a COHb level of 11.3% at the end of 7.5 hours (or 12.1% after 8 hours, because COHb levels rose linearly with time in these experiments). The

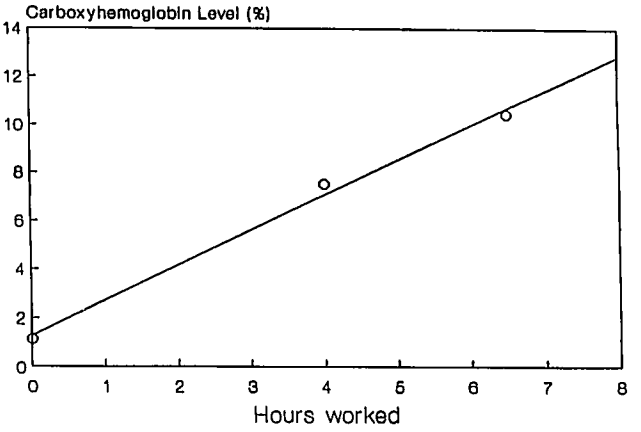

Fig. 1. Patient's carboxyhemoglobin level *v* hours of work before sampling.

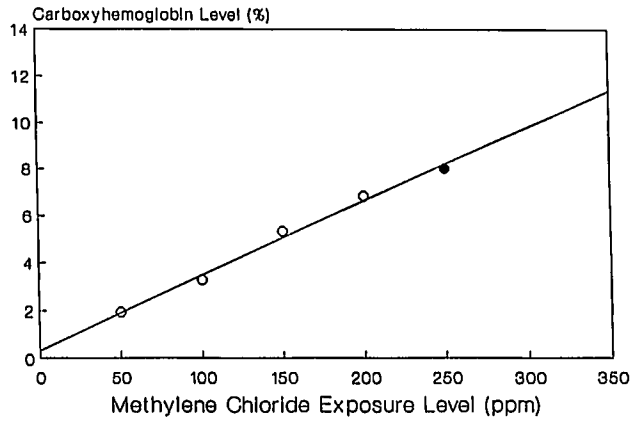

Fig. 2. Carboxyhemoglobin levels after 7.5-hour controlled exposures to methylene chloride at various concentrations. Data are from DiVicenzo and Kaplan[11] (open circles) and Hake et al[14] (closed circle).

12.1% figure corresponds closely to the above-cited 8-hour estimate of 12.7%, based upon measurements of the patient described.

The degree of concordance between predicted and measured COHb levels is remarkable, particularly given various potential complicating factors in this analysis. For example, because the patient was wearing an organic vapor cartridge respirator, one would expect some diminution of exposure compared with ambient measurements. However, this source of error is likely to be small, given the fact that organic vapor cartridges become saturated and breakthrough occurs within 30 minutes of exposure to methylene chloride at concentrations of 500 ppm.[15] On the other hand, the fact that methylene chloride is not only well absorbed through the skin but also penetrates most glove materials (including neoprene) may have compensated for any deficit in presumed inhalation exposure.[15] Likewise, the fact that the patient was involved in vigorous physical activity would have raised his methylene chloride uptake relative to the sedentary exposures applicable to the experimental studies cited above.[16] Finally, the exposure estimates in this case were based on "spot" samples which, although consistent over more than one sampling date, were not true time-weighted averages.

The in vivo conversion of methylene chloride to CO constitutes an important component of its short-term health risks.[10] The acute health effects of CO are well known, with headaches and lightheadedness typically reported in the 10% to 20% COHb range, and decreased perceptual motor performance, dyspnea on exertion, nausea, obtundation, coma, convulsions, and death occurring at progressively higher levels.[17] At the opposite end of the spectrum, COHb levels as low as 2% have been associated with decreased exercise tolerance in coronary artery disease patients exposed to CO alone.[18] Such levels would have been expected in the current workplace after only 1 hour of exposure. The American Conference of Governmental Industrial Hygienists' 8-hour time-weighted average Threshold Limit Value for methylene chloride has been set at 50 ppm; exposure at this concentration produces COHb levels of about 1.9% in experimental subjects.[11,19] By comparison, the federal Occupational Safety and Health Administration's 8-hour permissible exposure level (PEL) at the time of this writing is 10 times higher—500 ppm—although the standard is currently being reviewed by that agency.[20] Eight-hour exposure to methylene chloride at its current 500 ppm PEL would be expected to produce COHb levels in excess of 12%—about twice those produced by exposure to carbon monoxide itself at its 8-hour PEL of 50 ppm.[21,22]

This case illustrates the correlation between measured COHb and duration of methylene chloride exposure in a worker who was symptomatic because of his exposure. Because workplace air monitoring data may not always be available to the clinician, COHb measurements may be considered as a preliminary means of estimating airborne exposures to methylene chloride as well as acute risk from in vivo CO formation. It is likely that the workplace characterized in this case report is representative of many that merit intensive corrective action to reduce methylene chloride exposures.

Acknowledgments

We acknowledge the technical and editorial assistance of George Alexeeff, PhD, Chief, Air Toxicology and Epidemiology Section; and Jon Rosenberg, MD, Chief, Hazard Evaluation Section and Information Service, California Department of Health Services.

References

1. Anonymous. Facts and figures for the chemical industry. *Chem Engr News.*1989;67:41.

2. Steward RD, Fisher TN, Hosko MJ, et al. Carboxyhemoglobin elevation after exposure to dichloromethane. *Science.* 1972;176:295–296.

3. Stewart RD, Fisher TN, Hosko MJ, et al. Experimental human exposure to methylene chloride. *Arch Environ Health.* 1972;25:342–348.

4. Ratney RS, Wegman DH, Elkins HB. In vivo conversion of methylene chloride to carbon monoxide. *Arch Environ Health.* 1974;28:223–226.

5. DiVincenzo CD, Hamilton ML. Fate and disposition of [^{14}C] methylene chloride in the rat. *Toxicol Appl Pharmacol.* 1975;32:385–393.

6. Stewart RD, Hake CL. Paint-remover hazard. *JAMA.* 1976;235:398–401.

7. National Toxicology Program. *Toxicology and Carcinogenesis Studies of Dichloromethane (Methylene Chloride).* Research Triangle Park, NC: National Institute of Health, National Toxicology Program; January, 1986.

8. International Agency for Research on Cancer (IARC). Dichloromethane. *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans.* 1986;41:43–85.

9. Sunderman FW, Sunderman EW, Jr. *Laboratory Diagnosis of Diseases Caused by Toxic Agents.* St Louis, MO: Warren H. Green; 1970:283.

10. Monster AC. Biological monitoring of chlorinated hydrocarbon solvents. *J Occup Med.* 1986;28:583–588.

11. DiVincenzo GD, Kaplan CJ. Uptake, metabolism and elimination of methylene chloride vapor by humans. *Toxicol Appl Pharmacol.* 1981;59:130–140.

12. Stewart RD, Dodd HC. Absorption of carbon tetrachloride, trichloroethylene, tetrachloroethylene, methylene chloride, and 1,1,1-trichloroethane through the human skin. *Am Ind Hyg Assoc J.* 1964;25:439–446.

13. Stewart RD, Forster HV, Hake CL, et al. *Human Responses to Controlled Exposures of Methylene Chloride Vapor.* Report No. NIOSH-MCOW-ENVM-MC-73–7. Milwaukee, Wis: The Medical College of Wisconsin;1973.

14. Hake CL, Stewart A, Wu A, et al. Carboxyhemoglobin levels of humans exposed to methylene chloride. *Toxicol Appl Pharmacol.* 1975;33:145–146. Abstract.

15. Occupational Safety and Health Administration. *Guideline for Controlling Exposure to Methylene Chloride.* Washington, DC:US Department of Labor, publication 8–1.2A; Aug 11, 1986.

16. DiVincenzo GD, Kaplan CJ. Effect of exercise or smoking on the uptake, metabolism, and excretion of methylene chloride vapor. *Toxicol Appl Pharmacol.* 1981;59:141–148.

17. Stewart RD. The effect of carbon monoxide on humans. *Annu Rev Pharmacol.* 1975;15:409–423.

18. Allred EN, Bleecker ER, Chaitman BR, et al. Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary disease. *N Engl J Med.* 1989;321:1426–1432

19. *Documentation of Threshold Limit Values and Biological Exposure Indices: Methylene Chloride.* 5th ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists; 1986.

20. US Department of Labor, Occupational Safety and Health Administration. Air Contaminants—Permissible Exposure Limits. Title 29, Code of Federal Regulations, Part 1910.1000; 1989.

21. Health Assessment Document for Dichloromethane (Methylene Chloride). Washington, DC: US Environmental Protection Agency. USEPA publication EPA/600/8–82/004F; February, 1985.

22. *Documentation of Threshold Limit Values and Biological Exposure Indices: Carbon Monoxide.* 5th ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists; 1986.