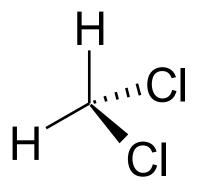


United States Environmental Protection Agency EPA Document# 740-R1-4003 August 2014 Office of Chemical Safety and Pollution Prevention

# **TSCA Work Plan Chemical Risk Assessment**

Methylene Chloride: Paint Stripping Use

CASRN: 75-09-2



August 2014

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#### **External Peer Review**

EPA/OPPT released peer review plan in August of 2012 and draft risk assessment and charge questions for peer review for public comment in January 2013. EPA/OPPT contracted with The Scientific Consulting Group, Inc. (SCG) to convene a panel of ad hoc reviewers to conduct an independent external peer review for the EPA's draft work plan risk assessment for DCM. As an influential scientific product, the draft risk assessment was peer reviewed in accordance with EPA's peer review guidance. The peer review panel performed its functions by web conference and teleconference between September 26 and December 13, 2013. The panel consisted of the following individuals:

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Please visit the EPA/OPPT's Work Plan Chemicals web page for additional information on the DCM's peer review process (<u>http://www.epa.gov/oppt/existingchemicals/pubs/riskassess.html</u>), the public docket (<u>Docket:</u> EPA-HQ-OPPT-2012-0725) for the independent external peer review report and the response to

# **GLOSSARY OF TERMS AND ABBREVIATIONS**

μg	Microgram(s)
μg/m³	Microgram(s) per cubic meter
AC	Acute concentration
ACGIH	American Conference of Governmental Industrial Hygienists
ACH	Air changes per hour
ADC	Average daily concentration
AEGL	Acute exposure guideline level
AEGL-1	Discomfort/non-disabling threshold
AEGL-2	Disability threshold
AEGL-3	Death threshold
APF	Assigned protection factor
AT	Averaging time
atm	Atmosphere(s)
ATSDR	Agency for Toxic Substances and Disease Registry
BAF	Bioaccumulation factor
BCF	Bioconcentration factor
BMD	Benchmark dose
BMD <sub>10</sub>	Benchmark dose at 10% response
BMDL	Benchmark dose, lower confidence limit(s)
BMDL <sub>10</sub>	Benchmark dose, lower confidence limit(s) at 10% response
BMDS	Benchmark Dose Software
BMR	Benchmark response
BOD	Biochemical oxygen demand
BW	Body weight
С	Contaminant concentration
°C	Degree Celsius
CROH	Concentration in the rest of the house
Cal EPA	California Environmental Protection Agency
CASRN	Chemical abstracts service registry number
CBI	Confidential business information
CDC	Centers for Disease Control and Prevention
CCD	Chemical Control Division
CCRIS	Chemical Carcinogenesis Research Information System
CDR	Chemical data report
cm	Centimeter(s)
cm <sup>2</sup>	Square centimeter(s)
cm <sup>3</sup>	Cubic centimeter(s)
CNS	Central nervous system
CO	Carbon monoxide
CO <sub>2</sub>	Carbon dioxide
COHb	Carboxyhemoglobin

CPSC	Consumer Product Safety Commission
СРЭС	•
CYP2E1	Cytochrome P450
-	Cytochrome P450, family 2, subfamily E, polypeptide 1 Developmental and Reproductive Toxicology/Environmental Teratology
DART/ETIC	Information Center
	Dichloromethane
DCM	
DEM	Department of Environmental Management
DIY	Do-it-yourself Deoxyribonucleic acid
DNA DNA SSB	•
DOE	Single stranded DNA-binding protein
DOSH	U.S. Department of Energy Division of Occupation Sofety and Health
E	Division of Occupation Safety and Health Emission rate
E	Initial emission rate
EC	
	European Commission
EC <sub>50</sub>	Effective concentration necessary to produce a 50% response
EC <sub>i,i+1</sub>	Exposure concentration over the time interval <i>i</i> to <i>i</i> +1
EC <sub>scenario1</sub>	Exposure concentration for scenario 1
EC <sub>scenario 2<math>\rightarrow</math>4</sub>	Exposure concentration for scenario 2, 3 or 4
$EC_{scenario 2 \rightarrow 16}$	Exposure concentration for scenario 2 through 16
ECG	Electrocardiogram
ED	Exposure duration
EETD	Economics, Exposure and Technology Division
EF	Exposure frequency
EFH	Exposure Factors Handbook
EPA	U.S. Environmental Protection Agency
EPCRA	Emergency Planning and Community Right-to-Know Act
ERG	Eastern Research Group
EU	European Union
°F	Fraction of time spent in the use zone
	Degrees Fahrenheit
FACE	Fatality Assessment and Control Evaluation
FDA	Food and Drug Administration
ft ft <sup>2</sup>	Foot/feet
ft <sup>3</sup>	Square foot/feet
	Cubic foot/feet
FTIR	Fourier transform infrared
g g/ama²	Gram(s)
g/cm <sup>2</sup>	Gram(s) per square centimeter
g/cm <sup>3</sup>	Grams(s) per cubic centimeter
GENE-TOX	Genetic Toxicology Data Bank
g/ft <sup>2</sup> , g/sq ft	Grams(s) per square foot
g/L	Gram(s) per liter
GLP	Good Laboratory Practices

g/minute	Grams(s) per minute
g/mol	Gram(s) per mole
GC/ECD	Gas chromatography and electron capture detector
GST	Glutathione S-transferase
GST-T1	GST-theta1-1
GWP	Global warming potential
HEC	Human equivalent concentration
HEC99	The HEC for which there is 99% likelihood that a randomly selected
	individual would have an internal dose less than or equal to the internal
	dose of the hazard value
HFC-32	Hydrofluorocarbon-32
HHE	Health hazard evaluation
HQ	Hazard quotient
HPLC	High-performance liquid chromatography
hr(s)	Hour(s)
HSDB	Hazardous Substances Data Bank
HSIA	Halogenated Solvents Industry Alliance, Inc.
HVAC	Heating, ventilation, and air conditioning
IDLH	Immediately dangerous to life and health
IMIS	Integrated Management Information Systems
IPCC	Intergovernmental Panel on Climate Change
IRIS	Integrated Risk Information System
IRTA	Institute for Research and Technical Assistance
IUR	Inhalation unit risk
IURR	Inventory Update Reporting Rule
K (upper-case)	Kelvin
k (lower-case)	first-order rate constant
K <sub>oc</sub>	Soil organic carbon partition coefficient
Kow	Octanol:water partition coefficient
kPa	kilopascal(s)
L	Liter (s)
lb(s)	Pound(s)
LADC	Lifetime average daily concentration
LBL	Lawrence Berkeley Laboratory
LC <sub>50</sub>	Median lethal concentration
LOAEL	Lowest-observed-adverse-effect level
LOEC	Lowest-observed-effect concentration
m	Meter(s)
m <sup>2</sup>	Square meter(s)
m <sup>3</sup>	Cubic meter(s)
M <sub>acute</sub>	Scenario-specific acute exposure modifier
M <sub>chronic</sub>	Scenario-specific chronic exposure modifier
m <sup>3</sup> /hr	Cubic meter(s) per hour Maximum assentable toxicant concentration
MATC	Maximum acceptable toxicant concentration

MCCEM	Multi-Chamber Concentration and Exposure Model
MCL	Maximum contaminant level
mg	Milligram(s)
mg/L	Milligram(s) per liter
mg/m <sup>3</sup>	Milligram(s) per cubic meter
min	Minute(s)
MITI	Ministry of International Trade and Industry
mM	Millimolar
mm Hg	Millimeters of mercury
mL	Milliliter(s)
MMWR	Morbidity and Mortality Weekly Report
MOE	Margin of exposure
MR	Mass released
MRI	Midwest Research Institute
MSDS	Material safety data sheets
MSU	Michigan State University
NAICS	North American Industry Classification System
NAS	National Academies
NESHAP	National Emission Standards for Hazardous Air Pollutants
NHANES	National Health and Nutrition Examination Survey
NIH	National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
NLS	Non-linear least squares
NMP	N-Methylpyrrolidone
NOAEL	No-observed-adverse-effect level
NRC	National Research Council
NTP	National Toxicology Program
OCSPP	Office of Chemical Safety and Pollution Prevention
OECD	Organization for Economic Cooperation and Development
OEHHA	Office of Environmental Health Hazard Assessment
OEM	Original Equipment Manufacturing
OPPT	Office of Pollution Prevention and Toxics
OSHA	Occupational Safety and Health Administration
PMN	Premanufacture Notification Program
РВРК	Physiologically-based pharmacokinetic
PEL	Permissible exposure limit
PFT	Perfluorocarbon tracer
POD	Point of departure
ppb	parts per billion
ppm	Parts per million
psi	Pound per square inch
Q	Compartment ventilation rate or air flow rate in and out of the chamber
RAD	Risk Assessment Division
RCRA	Resource Conservation and Recovery Act

REL	Reference exposure level
RfC	Reference concentration
RIA	Regulatory impact analysis
ROH	Rest of the house
RTECS	Registry of Toxic Effects of Chemical Substances
SCG	The Scientific Consulting Group, Inc.
SDWA	Safe Drinking Water Act
SIC	Standard Industry Classification
SMAC	Spacecraft maximum allowable concentration
SNAP	Significant New Alternatives Policy
SRC	Syracuse Research Corporation
STEL	Short-term exposure limit
sq ft	Square foot (feet)
t	Time
TLV	Threshold limit value
TOXLINE	Toxicology Literature Online
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TSCATS	Toxic Substance Control Act Test Submission Database
TWA	Time-weighted average
UF	Uncertainty factor
UFA	Interspecies uncertainty factor
UFD	Database uncertainty factor
UF <sub>H</sub>	Intraspecies uncertainty factor
UFi	LOAEL-to-NOAEL uncertainty factor
UF <sub>total</sub>	Total uncertainty factor
US or U.S.	United States
UK	United Kingdom
V	Volume
VOC	Volatile organic compound
wt	Weight
WY	Working years
yr	Year(s)

# **EXECUTIVE SUMMARY**

As a part of the Environmental Protection Agency's (EPA) comprehensive approach to enhance the Agency's existing chemicals management, in March 2012 EPA identified a work plan of chemicals for further assessment under the Toxic Substances Control Act (TSCA)<sup>1</sup>. The Agency is performing risk assessments on chemicals in the work plan. If an assessment identifies unacceptable risks to humans or the environment, EPA will pursue risk management. Methylene chloride (also called dichloromethane or DCM) was assessed as part of the work plan.

DCM is a volatile organic compound (VOC) that is used as a solvent in a wide range of industrial, commercial and consumer use applications, such as adhesives, paint stripping, pharmaceuticals, metal cleaning, chemical processing, and aerosols. It is the primary ingredient in many paint stripping products. The 2012 Chemical Data Report (CDR) indicated 261.5 million pounds of DCM were produced and imported into the U.S. with industry estimated domestic demand in 2010 of 181 million pounds.

EPA/OPPT identified DCM for further evaluation based on its likely carcinogenic properties in humans, high potential for human exposure as it is widely used in consumer products, and reported releases to the environment. For instance, DCM has been detected in drinking water, indoor environments, ambient air, groundwater and soil.

#### Main Conclusions of this Risk Assessment

This risk assessment identifies cancer risk concerns and short-term and long-term non-cancer risks for workers and "occupational bystanders" (other workers within the facility who are indirectly exposed) from the use of DCM-containing paint strippers.

The assessment also identifies short-term non-cancer risks for consumers and residential bystanders from the use of DCM-containing paint strippers.

#### The Focus of this Risk Assessment

This assessment characterizes human health risks from inhalation exposures to DCM for the paint stripping uses. Other uses were considered during problem formulation, but not selected for further risk analysis. Additional information is provided in the risk assessment regarding the criteria for inclusion and exclusion of uses and the various assumptions in applying these criteria.

The main route of exposure for DCM is believed to be inhalation for the paint stripping uses. EPA/OPPT recognizes that highly volatile compounds such as DCM may also be absorbed

<sup>&</sup>lt;sup>1</sup> <u>http://www.epa.gov/oppt/existingchemicals/pubs/workplans.html</u>

through the skin. However, EPA has neither the data nor the methodology to estimate DCM dermal exposure. Based on the physical-chemical properties of DCM and the scenarios described in this assessment, EPA/OPPT believes that inhalation is the main exposure pathway for this risk assessment. The assessment may underestimate total exposures to DCM during paint stripping due to this assumption.

An assessment of environmental effects is not included in this risk assessment. Based on DCM's moderate persistence, low bioaccumulation, and low hazard for aquatic toxicity, potential environmental impacts are judged to be low for the environmental releases associated to the TSCA uses under the scope of this risk assessment. That judgment should not be misinterpreted as a determination that DCM water and soil contamination is likely low. In fact, DCM has been detected in drinking water, groundwater and soil, and EPA is committed to reducing the presence of DCM in the environment through various regulatory programs (see *section 1.1.2.2* for a summary of EPA's regulatory history on DCM).

#### Human Populations Targeted in This Assessment

EPA/OPPT assessed acute and chronic risks for workers using paint strippers containing DCM. EPA/OPPT assumes that workers would be adults of both sexes (>16 and older, including pregnant workers) based upon occupational work permits, although exposures to younger workers in occupational settings cannot be ruled out. Data sources did not often indicate whether exposure concentrations were for occupational users or bystanders. Therefore, EPA/OPPT assumed that occupational exposures were for a combination of users and bystanders.

EPA/OPPT also examined acute risks for consumer exposures in residential settings. EPA/OPPT assumes that consumers would be adult individuals ( $\geq$ 16 and older; both sexes including pregnant women) that intermittently use DCM for paint stripping projects, although exposures to younger users may be possible in residential settings. Bystanders would be individuals of any age group (e.g., children, adults, the elderly) who are in a nearby area during product application.

In either occupational or consumer setting, EPA/OPPT assumes that direct contact or close proximity to the use would likely provide the highest exposures to DCM (*i.e.*, for a consumer or commercial application with substantial frequency or duration of exposure).

#### Workplace Exposures for Workers Using DCM-Based Paint Strippers

The estimation of occupational exposures to DCM relied upon published air monitoring data for industries that use DCM-based paint strippers. These data and different combinations of days per year of exposure (frequency), years of exposure (working lifetime), and respirator use and effectiveness (assigned protection factors) were used to develop a variety of hypothetical occupational scenarios.

Acute risks were estimated from the 8-hour DCM air concentrations reported in the occupational monitoring data. Chronic risks were based on non-cancer and cancer inhalation exposure estimates calculated for various industries, as expressed as average daily concentration (ADC) or lifetime average daily concentration (LADC), respectively. Table ES-1 summarizes the ranges of DCM exposures estimates for the various occupational scenarios assessed in the risk assessment. These scenarios were developed to account for variations in the use of respirators, exposure frequency, and working years for workers handling DCM-based paint strippers.

Due to a lack of data, ADC and LADC estimates could not be made for the bathtub refinishing sector. However, this sector is discussed in Appendix G since a number of deaths may be attributed to use of DCM-based strippers for refinishing bathtubs.

Table ES 1. Ranges of DCM Occupational Exposure Estimates Used in the Risk Assessment							
Based on Monitoring Data							
	Range for acute 8-hr concentration: Scenario 1→4 (mg/m³)		ADC range: Non-Cancer Effects Following Chronic Exposure		LADC range: Cancer Effects Following Chronic Exposure		
Induction							
Industry			Sconaria	os 1→16	Sconoria	concrise 1 >1C	
				/m³)	Scenarios 1→16 (mg/m³)		
	LOW-END	HIGH-END	Low-end	HIGH-END	Low-end	HIGH-END	
	ESTIMATE	ESTIMATE	ESTIMATE	ESTIMATE	ESTIMATE	ESTIMATE	
Professional Contractors	1.2	2,980	0.07	680	0.04	389	
Automotive Refinishing	1.2	416	0.07	95	0.04	54	
Furniture Refinishing	0.08	2,245	0.005	513	0.003	293	
Art Restoration and	0.08	2,245	0.003	0.5	0.003	0.3	
Conservation	0.04	2	0.003	0.5	0.002	0.5	
Aircraft Paint Stripping	1.7	3,802	0.1	868	0.06	496	
Graffiti Removal	0.4	1,188	0.02	271	0.00	155	
Non-Specific Workplace	0.4	7,000	0.02	1,598	0.01	913	
Settings – Immersion	0.7	7,000	0.04	1,558	0.02	515	
Stripping of Wood							
Non-Specific Workplace	13	1,017	0.7	232	0.4	133	
Settings – Immersion	15	1,017	0.7	252	0.4	135	
Stripping of Wood and							
Metal							
Non-Specific Workplace	5.7	428	0.3	98	0.2	56	
Settings – Unknown							
<b>Note:</b> Airborne concentration conversion factor for DCM is 3.47 mg/m <sup>3</sup> per ppm <u>NIOSH (2011b)</u> .							

#### Consumer Exposures from DCM-Based Paint Strippers

EPA/OPPT used the Multi-Chamber Concentration and Exposure Model (MCCEM) to estimate consumer exposures to DCM-based paint strippers. This modeling approach was selected

because published monitoring data for non-occupational inhalation exposures (i.e., consumer do-it-yourself [DIY]) were limited to those from several chamber studies conducted in the U.S. and Europe. The literature search for this assessment did not identify any published exposure information for exposures to other household members (i.e., bystanders). Of the available chamber studies, only one U.S. study provided sufficient information for the exposure modeling (EPA, 1994a).

The model used a two-zone representation of a house to calculate the DCM exposure levels for consumers and bystanders. The modeling approach integrated assumptions and input parameters such as the chemical emission rate over time, the volumes of the house and the room of use, the air exchange rate and interzonal airflow rate. The model also considered product characteristics, use patterns, and user location during and after the product use.

MCCEM was used to evaluate seven indoor exposure scenarios. The primary distinctions among the scenarios were type of application (i.e., brush vs. spray), location of product application (i.e., workshop for six scenarios, bathroom for one scenario), the mass of DCM emitted, the user's location during the wait period, and the air exchange rate of the rest of the house (ROH) with outdoor air. A sensitivity analysis indicated that these latter three inputs were the most sensitive variables in the modeling within application type.

Of the seven scenarios, two are considered central tendency for both the user and bystander, four had combinations of inputs to estimate upper-end concentrations for the user, and two of the latter also had input combinations to estimate upper-end concentrations for the bystander. The seventh scenario simulated the conditions reported in an occupational exposure case where the worker died due to DCM overexposure while stripping a bathtub (CDC, 2012). The bathroom scenario was included in the consumer exposure assessment to estimate potential exposures to bystanders.

Overall, the estimated inhalation exposure levels for the spray-on scenarios are about 2-fold greater than those reported for the brush-on scenarios. Estimated exposure levels for users of DCM-based paint strippers are higher than those reported for the bystander in the ROH. The estimated exposure levels to bystanders in the bathroom scenario is in the same range as the exposures to bystanders in the workshop scenarios.

#### Characterization of Hazards and Risks to Human Health

#### DCM's Carcinogenic Hazards and Risks:

DCM is likely to be carcinogenic in humans based on a mutagenic mode of action (EPA, 2011c). EPA/OPPT used the inhalation unit risk (IUR) of  $4 \times 10^{-5}$  per ppm ( $1 \times 10^{-5}$  per mg/m<sup>3</sup>) to estimate excess cancer risks for the occupational scenarios. The IUR is reported in the EPA's Integrated Risk Information System (IRIS) *Toxicological Review of Methylene Chloride* (EPA, 2011c) and is the estimated upper bound excess lifetime cancer risk resulting from continuous exposure to an airborne agent at  $1 \mu g/m^3$  (EPA, 2011c). The IUR for DCM was based on mouse liver and lung tumors reported in a cancer inhalation bioassay (<u>Mennear et al., 1988</u>; <u>NTP, 1986</u>). There is high confidence in the IUR because it was based on the best available dose-response data for liver and lung cancer in mice (<u>EPA, 2011c</u>). Moreover, the mutagenic mode of action was supported by the weight of evidence from multiple *in vivo* and *in vitro* studies (<u>EPA, 2011c</u>).

#### DCM's Non-Carcinogenic Hazards and Risks:

Acute and chronic exposure to DCM is primarily associated with neurological and hepatic effects. The primary target organ of DCM toxicity is the brain. Neurological effects result from either direct narcosis or the formation of carbon monoxide (CO). CO is produced as one of the metabolic byproducts of DCM metabolism, which reversibly binds to hemoglobin as carboxyhemoglobin (COHb). Part of the effect of DCM on the central nervous system (CNS) comes from the accumulation of carboxyhemoglobin (COHb) in the blood, especially during acute/short-term exposures to DCM.

Non-cancer risks associated with acute exposures to DCM (i.e., neurological effects) were evaluated for workers, consumers and residential bystanders using the dose-response information supporting the derivations of the *Spacecraft Maximum Allowable Concentrations* (SMACs)(<u>NRC, 1996</u>), the California *acute reference exposure level* (REL) (<u>OEHHA, 2008</u>), and the *Acute Exposure Guideline Levels* (AEGLs)(<u>NAC, 2008</u>).

EPA/OPPT preferred the SMAC hazard value [or point of departure (POD)] over the California acute REL POD as the health protective acute hazard value used to estimate acute risks for the consumer scenarios. The SMAC POD was based on multiple human observations reporting increased COHb levels after DCM exposure, coupled with the knowledge of what would be considered a no-observable-adverse effect level (NOAEL) based on the extensive CO database (<u>NRC, 1996</u>). However, the California acute REL POD was used to estimate risks for occupational scenarios since an 8-hr SMAC POD was not available for the risk calculations. Although AEGLs are intended for emergency response activities, the AEGL PODs were used in this assessment to evaluate acute risks for discomfort/non-disabling (AEGL-1) and incapacitating (AEGL-2) effects following DCM inhalation exposure.

Non-cancer risks for workers repeatedly exposed to DCM were evaluated using the hazard value of 17.2 mg/m<sup>3</sup> (4.8 ppm) for liver effects (EPA, 2011c). The value was derived in the DCM IRIS assessment by PBPK modeling and expressed as the 1<sup>st</sup> percentile of the distribution of human equivalent concentrations (HEC) i.e. the HEC<sub>99</sub> the concentration at which there is 99% likelihood an individual would have an internal dose less than or equal to the internal dose of hazard was used to protect toxicokinetically sensitive individuals. There is high confidence in the non-cancer hazard value because it was derived from a well-conducted, peer-reviewed animal inhalation study (<u>Nitschke et al., 1988a</u>). Further, the inhalation database contains several studies consistently identifying the liver as the most sensitive non-cancer target organ in rats (EPA, 2011c).

#### Uncertainties of this Risk Assessment

The worker risk assessment has a number of uncertainties. While it is clear that the air monitoring data represent real world exposure levels, EPA/OPPT cannot determine whether these concentrations are representative of actual statistical distributions for exposed workers. Further, EPA/OPPT cannot determine how accurately the hypothetical exposure scenarios reflect occupational exposures based on variations in the use of respiratory protective equipment, effectiveness of a used respirator in providing the protection indicated by its APF, and actual exposure frequencies and working years. The estimates of numbers of workers exposed to DCM-based strippers are uncertain due primarily to the assumed numbers of workers per model plant in the estimation approach.

The consumer exposure assessment is composed of modeled exposure scenarios for which the inputs are based on experimental data, survey information, and a number of assumptions with varying degrees of uncertainty. The results are characterized as either plausible estimates of individual exposure (*e.g.*, central tendency), or possibly greater than the distribution of actual exposures (e.g., bounding).

The extent of the identified uncertainties for estimating occupational or residential exposures is not known. Consequently, under real world conditions, exposure could occur to either higher or lower levels of DCM than those estimated, leading to a potential for under- or over-estimation of actual risks.

There is general high confidence in the hazard database supporting the hazard values used to estimate acute and chronic risks for various health effects associated with DCM inhalation exposure (i.e., neurotoxicity, liver toxicity, and liver and lung cancer). However, there are uncertainties about potential human health concerns for developmental neurotoxicity and immunological effects following exposure to DCM.

#### The Results of this Risk Assessment

Size of the Exposed Population:

- Over 230,000 workers nationwide are directly exposed to DCM from DCM-based strippers. This estimate only accounts for workers performing the paint stripping using DCM and does not include other workers ("occupational bystanders") within the facility who are indirectly exposed.
- No data were available to estimate the number of consumers and residential bystanders exposed to DCM during the use of paint strippers.

#### Cancer Risks Associated With Chronic Exposures to DCM:

- There are cancer risk concerns for workers and occupational bystanders exposed to DCM that are employed at various industries handling DCM-containing paint strippers.
- Many of the occupational scenarios exceed at least one of the target cancer risks of 10<sup>-4</sup>, 10<sup>-5</sup> and 10<sup>-6</sup>.
- The greatest cancer risks occur for workers handling DCM-based paint strippers with no respiratory protection for an extended period of time.

#### Non-Cancer Risks Associated With Chronic Exposures to DCM:

- There are non-cancer risks for liver effects for most workers (including bystanders) using DCM-based paint strippers in relevant industries, with the exception of the art renovation and conservation industry.
- Non-cancer risks occur for most workers (including bystanders) handling DCM-based paint strippers with or without respiratory protection for various exposure scenarios that predominantly reflect variations in exposure conditions (i.e., exposure frequency and working years) in facilities reporting central tendency or high-end DCM air levels. Among all of the occupational scenarios, the greatest risk concern is for workers engaging in long-term use of the product (i.e., 250 days/year for 40 years) with no respiratory protection.
- Non-cancer risks are not reported when workers reduce their exposure to DCM-based strippers by taking all three of the following actions; wearing respiratory protection (i.e., respirator with at least an assigned protection factor of 50), limiting exposure to central tendency exposure conditions (i.e., 125 days/year for 20 years) and working in facilities with low-end DCM air concentrations.

#### Non-Cancer Risks Associated With Acute Exposures to DCM:

- There are acute risks for neurological effects for most workers using DCM-based paint strippers. These risks are present in the presence or absence of respiratory protection.
- There are concerns for incapacitating effects in workers handing DCM-containing paint strippers on an acute/short-term basis with no respiratory protection. These concerns are also present for workers wearing different types of respirators while performing paint stripping in industries with high exposure to DCM.
- There are acute risks for neurological effects for consumers of DCM-based paint strippers at residential settings. Also, bystanders are at risk while staying in the residence when paint strippers are being applied.
- There are concerns for discomfort/non-disabling and incapacitating effects for consumers exposed to DCM while applying the product or staying in the residence after completion of the stripping task. These concerns are also present for residential bystanders in some

scenarios when exposure conditions are at the highest in the rest of the house after completing the paint stripping task.

• Application of DCM-based paint strippers in a bathroom generates unsafe exposure conditions for the user of the product, but not residential bystanders. DCM concentrations may reach levels associated with non-disabling and incapacitating effects for the user applying the product. User relocation to the rest of the house after completing the paint stripping task may also produce non-disabling and incapacitating effects as DCM's internal dose builds up in the body over time.

# **1 BACKGROUND AND SCOPE**

## **1.1 INTRODUCTION**

As a part of EPA's comprehensive approach to enhance the Agency's existing chemicals management, in March 2012 EPA identified a work plan of chemicals for further assessment under the Toxic Substances Control Act (TSCA)<sup>2</sup>. The Agency is performing risk assessments on chemicals in the work plan. If an assessment identifies unacceptable risks to humans or the environment, EPA will pursue risk management. After gathering input from stakeholders, EPA developed criteria used for identifying chemicals for further assessment<sup>3</sup>. The criteria focused on chemicals that meet one or more of the following factors: (1) potentially of concern to children's health (for example, because of reproductive or developmental effects); (2) neurotoxic effects; (3) persistent, bioaccumulative, and toxic (PBT); (3) probable or known carcinogens; (4) used in children's products; or (5) detected in biomonitoring programs. Using this methodology, EPA identified a TSCA Work Plan of chemicals as candidates for risk assessment in the next several years. In the prioritization process, DCM was identified for assessment based on human health hazards and high exposure potential.

The target audience for this risk assessment is primarily EPA risk managers; however, it may also be of interest to the broader risk assessment community as well as U.S. stakeholders that are interested in issues related to DCM, especially when used as a paint stripper. The information presented in the risk assessment may be of assistance to other Federal, State and Local agencies as well as to members of the general public who are interested in the chemical risks of DCM. The risk assessment may also help those interested in reducing risks associated with the use of DCM-based paint strippers.

The initial step in EPA's risk assessment development process includes scoping and problem formulation and is distinct from the initial prioritization exercise. During these steps EPA reviews currently available data and information, including but not limited to, assessments conducted by others (e.g., authorities in other countries), published or readily available reports, and published scientific literature. During scoping and problem formulation the more robust review of the factors influencing initial prioritization may result in refinement – either addition/expansion or removal/contraction – of specific hazard or exposure concerns previously identified in the prioritization methodology.

<sup>&</sup>lt;sup>2</sup> <u>http://www.epa.gov/oppt/existingchemicals/pubs/workplans.html</u>

<sup>&</sup>lt;sup>3</sup> <u>http://www.epa.gov/oppt/existingchemicals/pubs/wpmethods.pdf</u>

## **1.2 BACKGROUND**

#### 1.2.1 Rationale for Selecting DCM for Risk Assessment

DCM was identified for assessment based on high human health hazards and exposure potential. The high human health hazard ranking was assigned for potential cancer risks (i.e., likely human carcinogen) and acute<sup>4</sup> and chronic<sup>5</sup> non-cancer effects. DCM is a liquid VOC and its high vapor pressure leads to rapid evaporation, which may pose an inhalation hazard for humans. The high exposure potential ranking was assigned because DCM is widely used with industrial, commercial and consumer user applications and at a relatively high percent content particularly in paint stripping products. DCM is ubiquitously present in the environment with levels detected in drinking water, indoor environments, ambient air, groundwater, and soil (<u>EPA, 2012d</u>).

### **1.2.2 Overview of DCM Uses and Production Volume**

DCM is mainly used as a solvent with a wide range of industrial, commercial, and consumer uses, which include: solvent for vapor degreasing; paint/varnish removers; electronics; resin cleaners; adhesives; tablet coatings; process solvent for cellulose acetate; butyl rubber; cleaning solvent; plastics processing; blowing agent in polyurethane foams; propellant for paint aerosols; refrigerant; heat-transfer fluid; extraction solvent for industrial applications and food; color diluents for foods; and food packaging adhesives (<u>Ash and Ash, 2009</u>). DCM is the primary ingredient in many paint stripping products (<u>Mannsville, 1999</u>).

U.S. demand for DCM in 2006 was estimated at 185 million pounds (lbs) with a projected demand of 181 million lbs for 2010 (HSIA, 2008; ICIS, 2007). The 2012 non-confidential business information (CBI) Chemical Data Reporting (CDR) indicated 261.5 million lbs of DCM that were produced and imported into the U.S. (EPA, 2013). More information on production volumes can be found in Section 2.2.

#### 1.2.3 Overview of Assessments of DCM's Human Health Hazards

Several organizations have developed high quality, peer-reviewed hazard/dose-response assessments documenting the adverse health effects of DCM. These reports indicate that DCM is likely to be carcinogenic to humans and is a liver and neurological toxicant. EPA/OPPT used the human health toxicity information from these reports rather than developing a new hazard/dose-response analysis for DCM.

<sup>&</sup>lt;sup>4</sup> Acute exposure is defined as exposure by the oral, dermal, or inhalation route for 24 hours or less (EPA, 2011b).

<sup>&</sup>lt;sup>5</sup> Chronic exposure is defined as repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans and more than approximately 90 days to 2 years in typically used laboratory animal species) (EPA, 2011b).

For the evaluation of cancer and non-cancer risks following repeated exposure to DCM (i.e., occupational scenarios), EPA/OPPT relied on the cancer and non-cancer dose-response information reported in the *Toxicological Review of Methylene Chloride* recently published by EPA's Integrated Risk Information System (IRIS) (EPA, 2011c).

Non-cancer risks associated with acute residential exposures to DCM were assessed using the dose-response information supporting the derivations of the *Spacecraft Maximum Allowable Concentrations* (SMACs) (NRC, 1996) and the *Acute Exposure Guideline Levels* (AEGLs)(NAC, 2008). The assessment also evaluated acute occupational risks with the California acute reference exposure level (REL) and AEGL hazard values (OEHHA, 2008). The California acute REL, but not the SMAC hazard value, was used to estimate acute occupational risks since an 8-hr SMAC hazard value was not available for the risk calculations.

Refer to Chapter 3 for more information about the hazard/dose-response approach for cancer and non-cancer health endpoints, specifically sections 3.3.1.2 and 3.3.1.3.

## **1.2.4 Overview EPA's Regulatory History of DCM**

DCM has been the subject of various EPA regulatory actions. EPA lists DCM as a toxic (*i.e.,* nonacute) hazardous waste under the Resource Conservation and Recovery Act (RCRA) (Code U080) (EPA, 2012c). DCM is also listed on the Toxics Release Inventory (TRI) pursuant to section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) (EPA, 2014). Moreover, DCM is listed on the TSCA Inventory of Chemical Substances and is subject to reporting under the TSCA CDR rule (EPA, 2011e).

EPA's Office of Air Quality Planning and Standards issued a final rule in January 2008, under the National Emission Standards for Hazardous Air Pollutants (NESHAP) that established national emission standards for using DCM to remove dried paint (*i.e.*, including, but not limited to: paint, enamel, varnish, shellac, and lacquer) from wood, metal, plastic, and other substrates (EPA, 2008). The NESHAP also implemented management practices that minimize DCM emissions.

Additionally, the Safe Drinking Water Act (SDWA) requires EPA to determine the level of contaminants in drinking water at which no adverse health effects are likely to occur. EPA has set an enforceable maximum contaminant level (MCL) for DCM at 0.005 mg/L or 5 ppb (EPA, 2010b).

Please refer to Appendix A for more information about the U.S. regulatory history of DCM, including actions in other U.S. federal agencies and States. Appendix A also provides a brief description of actions in Canada and Europe.

## **1.3 SCOPE OF THE ASSESSMENT**

## **1.3.1 Selection of DCM Uses**

EPA/OPPT focused the assessment on the use of DCM in paint stripping. Uses other than paint stripping are not covered in the risk assessment because EPA/OPPT decided to focus on the use of DCM with the highest potential exposures to both consumers and workers. Table 1-1 lists the primary uses of DCM, indicates whether a use was considered for inclusion in this assessment, and also presents the rationale for why a use was included or excluded from further consideration.

Narrowing of the scope required exclusion of some uses based on comparative judgments relative to paint stripping. These comparative judgments considered potential exposure among the primary uses identified (*e.g.*, percent content relative to potential exposure). In addition, EPA/OPPT has a special interest in small shops and consumer use for this assessment due to the possibility that these shops and consumers may have fewer resources or less expertise and awareness of hazards, exposures, or controls as compared to large shops.

## **1.3.2 Selection of Exposure Pathway**

This risk assessment assumed that DCM is primarily absorbed through the respiratory tract because of DCM's high vapor pressure. EPA/OPPT recognizes that highly volatile compounds such as DCM may also be absorbed through the skin. However, EPA has neither the data nor the methodology to assess DCM dermal exposure. Based on the physical-chemical properties of DCM and the scenarios described in this assessment, EPA/OPPT focuses on inhalation as the main exposure pathway for this risk assessment. This assessment may underestimate total exposures of DCM in paint stripping due to this assumption.

# 1.3.3 Identification of Human Populations Exposed During the Use of DCM-Based Paint Strippers

EPA/OPPT's assessment evaluated the quantitative acute and chronic risk(s) for workers using DCM-based paint strippers. EPA/OPPT has a special interest in exposures to workers employed by "small commercial shops." The shop sizes can vary in most industries that do paint stripping, and this issue is discussed in section 3.1.1.1.

Occupational exposures include possible direct exposures to workers who may use these products at work, in training, or other situations. Data sources did not often indicate whether exposure concentrations were for occupational users or bystanders. Therefore, EPA/OPPT assumed that occupational exposures were for a combination of users and bystanders. We also assumed that workers would be adults of both sexes [≥16 years (yrs) and older], although exposures to younger individuals may be possible in occupational settings.

Use Category	Percent DCM Content	Population Exposed <sup>a</sup>	teria Considered in this Assessment?	
Adhesives	60-100	Small commercial shop workers, consumers [including do-it-yourself (DIYs)]; industrial workers	No – Relatively narrower range of removal applications and likely lower exposure levels compared to paint stripping. Information indicates that many of the adhesive uses are in adhesive removers.	
Paint stripping	25-100	Small commercial shop workers, consumers (including "DIYs"); industrial workers	Yes – Relatively high percent content range, broad range of stripping and removal applications (automotive, furniture, marine, wall paint, similar coating removal).	
Pharmaceuticals	N/A <sup>b</sup>	Industrial workers	No – Industrial use settings which are generally believed to be better controlled and monitored.	
Metal cleaning	15-40	Small commercial shop workers, consumers (including "DIYs"); industrial workers	No – Small market percentage (7 percent) and likely lower exposure levels compared to paint stripping.	
Chemical processing	N/A <sup>b</sup>	Industrial workers	No – Industrial use settings which are generally believed to be better controlled and monitored.	
Aerosols (propellant use)	<25	Small commercial shop workers, consumers (including "DIYs"); industrial workers	No – Relatively low percent content range, small market percentage (5 percent), and likely lower exposure levels compared to paint stripping.	
Polyurethane foam	N/A <sup>b</sup>	Industrial workers	No – Industrial use settings which are generally believed to be better controlled and monitored.	

#### Notes:

<sup>a</sup> For the purposes of this assessment, consumers are defined as non-commercial/non-industrial users of products containing DCM. Commercial workers are defined as persons employed in a commercial enterprise providing salable goods or services. Examples of a commercial enterprise include, but are not limited to, commercial and residential cleaning services, painting companies, carpet installers, commercial and residential repair and refurbishing companies, and automotive painting and repair shops.

<sup>b</sup> For these industrial applications, the percent of DCM content is expected to be at or near 100 percent.

This assessment also examined consumer exposures to DCM-based paint strippers in residential settings. Consumers were adult individuals of both sexes (i.e.,  $\geq$ 16 yrs and older, including pregnant women) using DCM in their homes for paint stripping projects. It is possible that younger users (i.e.,  $\leq$ 16 yrs) would be using the product in residential settings, but this assessment did not look at this age group. EPA/OPPT also evaluated exposures to bystanders, who are individuals of any age (e.g., children, adults, the elderly) that did not use the product, but were indirectly exposed in the home while being nearby during product use.

EPA/OPPT used the DCM air concentrations from the occupational exposure assessment to evaluate the acute and chronic human health risks associated with the use of DCM-based paint strippers. For consumer exposures, EPA/OPPT only evaluated the human health risks to acute exposures to DCM. The focus on acute exposures was based on the assumption that DCM is not expected to significantly build up in the body between exposure events. DCM's plasma half-life is estimated to be 40 minutes after inhalation exposure (DiVincenzo et al., 1972). Moreover, EPA/OPPT assumed that consumers would not generally strip paint on a regular basis in their residences allowing sufficient time between exposures to clear DCM and its metabolites from the body.

## 1.3.4 Why Environmental Risks Were Not Evaluated For DCM-Based Paint Strippers

EPA/OPPT did not assess the risks of environmental effects related to the use of DCM in paint stripping products. This decision is supported by DCM's environmental fate and aquatic toxicity data (Section 2.3).

Due to its volatility, DCM does not significantly partition to solid phases. Therefore, releases of DCM to the environment are likely to evaporate to the atmosphere, or if released to soil, migrate to groundwater. This substance has been shown to biodegrade over a range of rates and environmental conditions and is considered to be moderately persistent in the environment. Measured bioconcentration factors for DCM suggest its bioconcentration potential is low.

The aquatic toxicity of DCM for fish, aquatic invertebrates, and aquatic plants is low based on the OPPT criteria described in the *TSCA Work Plan Chemicals Methods Document* (EPA, 2012d) and the *Classification Criteria for Environmental Toxicity and Fate of Industrial Chemicals* (EPA, 1992a). For these reasons, this assessment focused on human receptors rather than ecological receptors.

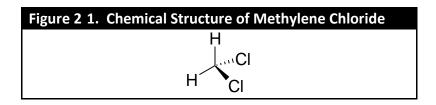
Appendix B contains a summary of the aquatic toxicity studies considered in the evaluation of environmental hazards of DCM.

# **2 SOURCES AND FATE**

Chapter 2 discusses the physical and chemical properties of DCM, sources related to its production and uses, and its fate in the environment. The contents of this chapter supported EPA/OPPT's decision to not evaluate environmental risks in this assessment.

## 2.1 PHYSICAL AND CHEMICAL PROPERTIES

The chemical structure for DCM is shown in Figure 2-1.



DCM is a volatile (vapor pressure = 351.8 mmHg at 25°C), colorless liquid with a chloroform-like, sweet odor (<u>OSHA, 2012b</u>). DCM has a low boiling point (39.7°C) and is moderately water soluble (13.7 g/L at 20°C), but more dense than water (1.33 g/cm<sup>3</sup> at 20°C). DCM is used as a substitute for other solvents because it is non-flammable and non-explosive. DCM also is not readily oxidizable (<u>ECB, 2000</u>; <u>Lide, 2001</u>; <u>O'Neil, 2001</u>). Table 2-1 shows the common physical-chemical properties of DCM.

Molecular formula	lecular formula CH <sub>2</sub> Cl <sub>2</sub>					
Molecular weight	84.93					
Physical form	Colorless liquid; sweet, pleasant odor resembling chloroform					
Melting point	-95°C					
Boiling point	39.7°C					
Vapor pressure	351.8 mmHg at 25°C					
Log Kow	5.3 ("slow stirring" method); 5.9 at 25°C (measured; OECD 117 <sup>b</sup> )					
Water solubility	13.7 g/L at 20°C					
Density	1.33 g/cm <sup>3</sup> at 20°C					
Flash point	none					
Notes:						
<sup>a</sup> Information obtained fro	m ( <u>ECB, 2000</u> )					
<ul> <li><sup>b</sup> OECD Test Number 117: F (HPLC) Method</li> </ul>	Partition Coefficient (n-octanol/water), High Performance Liquid Chromatography					

## 2.2 DCM PRODUCTION AND USES

DCM is mainly used as a solvent, at concentrations ranging from 20 to 100 percent, and is the primary ingredient in many paint stripping products (<u>Mannsville, 1999</u>). It is a quick acting and inexpensive solvent with a wide range of industrial, commercial, and consumer uses, which include: solvent for vapor degreasing; paint/varnish removers; electronics; resin cleaners; adhesives; tablet coatings; process solvent for cellulose acetate; butyl rubber; cleaning solvent; plastics processing; blowing agent in polyurethane foams; propellant for paint aerosols; refrigerant; heat-transfer fluid; extraction solvent for industrial applications and food; color diluents for foods; and food packaging adhesives (<u>Ash and Ash, 2009</u>).

DCM also has several minor uses, especially as an extraction solvent for spice oleoresins and hops, and for the removal of caffeine from coffee. It is approved as an extraction solvent for these uses by the FDA, although most decaffeinators no longer use DCM due to concerns over residuals.

## 2.2.1 Market Trends and Uses

Use of DCM as a solvent in a number of sectors has been declining steadily since the mid-1980s due to increasing government regulation (*i.e.,* both federal and state), and environmental, consumer, and worker exposure concerns (EPA, 1994d, 2006c, 2011c; ICIS, 2007). These regulations include:

- a lower 8-hr time-weighted average (TWA) OSHA PEL of 25 ppm took effect in 1997;
- warning labeling requirements required by CPSC on all products containing more than 1 percent of DCM took effect in 1988 (<u>CPSC, 1987</u>);
- listing of DCM as a potential carcinogen by the National Institute of Occupational Safety and Health (NIOSH);
- new OSHA standards requiring facilities using DCM to use vapor control equipment by 2000 (ICIS, 2007).

U.S. consumption of DCM declined from a high of approximately 540 million lbs in the mid-1980s to approximately 181 million lbs currently (ICIS, 2007). In 1984, there were four domestic producers of DCM selling around 501 million lbs. In 2000, there were three domestic manufacturers with five DCM plants in the U.S. (Cal EPA, 2000). Currently, there are only two manufacturers in the U.S. with a total of three production plants in operation (EPA, 2013). These companies are the Dow Chemical Company (one facility) and Occidental Chemical Corporation (*i.e.*, two facilities) (EPA, 2013).

U.S. demand for DCM in 2006 was estimated at 185 million lbs by industry sources with a projected demand of 181 million lbs for 2010 (HSIA, 2008; ICIS, 2007). The 2012 non-confidential business information (CBI) CDR indicated 261.5 million lbs of DCM that were produced and imported into the U.S. (EPA, 2013). DCM imports were estimated at 20 million lbs in 2006 (ICIS, 2007). Thus, the production volume of DCM makes up 80 to 96 percent of the

market share depending on the high or low estimates of total production and imports. In terms of environmental releases, 292 facilities reported a total of 4.8 million lbs of on- and off-site disposal or other releases of DCM based on the EPA's 2010 TRI (EPA, 2011d).

Table 2-2 presents DCM market trends by use. Based on current estimations, use of DCM is expected to increase in only one category, DCM as feedstock in the production of a refrigerant, hydrofluorcarbon-32 (HFC-32) (Mannsville, 1999).

Table 2 2. DCM M		
DCM Use	Use Trend	Background
Paint stripper	Decreasing	<ul> <li>OSHA's 1997 reduced PEL resulted in new equipment costs (especially for small shops), which led to a reduction of DCM use as a paint stripper <sup>a</sup></li> <li>CPSC warning labels on consumer DIY products has also resulted in less furniture refinishing use <sup>b</sup></li> <li>The aircraft industry has replaced DCM paint stripping on commercial and military planes with non-chemical stripping processes because new technology in chemical processing has resulted in less of a need for DCM <sup>c</sup></li> <li>Use of substitutes like high-boiling ketones, glycol ethers, and N-methylpyrrolidone (NMP) has been increasing <sup>d</sup></li> </ul>
Metal cleaner and degreaser	Decreasing	<ul> <li>Lower OSHA PEL resulted in reduced DCM use <sup>a</sup></li> </ul>
Aerosol products	Decreasing	<ul> <li>CPSC labeling requirements have led most aerosol manufacturers to eliminate DCM use, but it is still somewhat used <sup>b</sup></li> </ul>
Foam adhesives	Decreasing	<ul> <li>EPA's 2007 Flexible Polyurethane Foam Production and Fabrication National Emission Standards for Hazardous Air Pollutants (NESHAP) required a reduction in use of DCM <sup>e</sup></li> <li>Lower OSHA PEL has steered many foam manufacturers into using non-DCM adhesives due to the cost of compliance with the PEL <sup>a</sup></li> </ul>
Feedstock in production of refrigerant HFC-32	Increasing	<ul> <li>Expected to grow because HFC-32 is an EPA Significant New Alternatives Policy (SNAP) replacement chemical for HFC-22<sup>e</sup></li> <li>Fluorocarbon production accounts for less than 10 percent of DCM use <sup>f</sup></li> </ul>
Sources: <sup>a</sup> <u>OSHA (2010)</u> <sup>b</sup> <u>CPSC (1992)</u> <sup>c</sup> <u>Pauli (1996)</u> <sup>d</sup> <u>Mannsville (1999)</u> <sup>e</sup> <u>HSIA (2010)</u> <sup>f</sup> <u>ICIS (2007)</u>		

Table 2-3 presents the major and minor uses of DCM, as well as the potential benefits of using DCM in different industries.

Table 2 3. Major and Minor Uses of DCM				
Minor Uses <sup>a</sup>	Overall Benefits			
<ul> <li>Extraction solvent for oils, waxes, fats, spices, and hops</li> <li>Tablet coating for pharmaceuticals</li> </ul>	<ul> <li>Low flammability<sup>b</sup></li> <li>Non-corrosive to many substrates<sup>b</sup></li> <li>Strong solvency properties<sup>b</sup></li> <li>No flash point under normal use conditions and can be used to reduce the flammability of other substances<sup>c</sup></li> <li>Lower costs</li> </ul>			
(2008); IAQUK (2014)				
	<ul> <li>Minor Uses<sup>a</sup></li> <li>Extraction solvent for oils, waxes, fats, spices, and hops</li> <li>Tablet coating for pharmaceuticals</li> </ul>			

As recently as the 1980s, approximately 50 percent of the total DCM market was made up of paint stripping products (<u>Mannsville, 1999</u>). Industry sources stated 40 percent of the domestic DCM market was made up of paint strippers in 2006 (<u>HSIA, 2008</u>). However, the most recent industry figures indicate paint stripping products now only make up 25 percent of the domestic market for DCM (Table 2-4) (<u>ICIS, 2007</u>). These figures coupled with an overall decline in the demand for DCM suggest manufacturers may be substituting other solvents for DCM in their paint stripping products. Because the data are recent, EPA/OPPT cannot determine at this point if this is a real trend.

The estimates for DCM by use are shown in Table 2-4. The percentages of DCM use by application type are based on production volume for use in domestic products. While DCM use in adhesives is a larger market share than paint stripping, the narrower range of removal applications and likely lower exposure levels compared to paint stripping resulted in adhesive use not being selected as a focal point for this assessment.

Major Uses	Percent of DCM Consumed in End Products
Adhesives	37
Paint stripping	25
Pharmaceuticals	10
Metal cleaning	7
Chemical processing	7
Aerosols	5
Polyurethane foam blowing	5
Miscellaneous	4

#### 2.2.1.1 Consumer Uses

DCM has a number of TSCA consumer uses. Table 2-5 presents the major consumer uses of DCM.

Paint strippersPaint thinnersaPaint removers and strippersAerosol applicationsAerosol paintsc	<ul> <li>Varnish removers<sup>b</sup></li> <li>Graffiti removers<sup>b</sup></li> </ul>
Aerosol applications  • Aerosol paints <sup>c</sup>	
<ul> <li>Automotive products<sup>c</sup></li> <li>Spray shoe polish<sup>a</sup></li> </ul>	<ul> <li>Rust removers<sup>a</sup></li> <li>Primers<sup>a</sup></li> </ul>
Cleaners/protectors <ul> <li>Water repellant/protectors<sup>a</sup></li> <li>Spot removers<sup>a</sup></li> <li>Wood floor and panel cleane</li> <li>Specialized electronic cleane (for TV, VCR, razor, <i>etc.</i>)<sup>a</sup></li> </ul>	
Adhesives  Contact cement <sup>a</sup> Super glues <sup>a</sup> Spray adhesives <sup>a</sup>	<ul> <li>Adhesive removers (general purpose, tile and wallpaper)<sup>a</sup></li> </ul>
Miscellaneous <ul> <li>Silicone lubricants</li> <li>(excluding automotive)<sup>a</sup></li> <li>Outdoor water repellants<sup>a</sup></li> </ul>	Gasket removers <sup>a</sup>

The 2012 CDR data indicated that DCM is used in the following commercial/consumer use categories: paints and coatings, adhesives and sealants, and "other" (EPA, 2013)(Appendix C).

The National Institutes of Health (NIH) Household Products Database currently lists 50 products containing DCM, in concentrations ranging from one to 100 percent. The products are divided

almost evenly between aerosol and liquid formulations (with one in granular form)(<u>DHHS</u>, <u>2012</u>).

DCM uses addressed by other agencies (*i.e.*, non-TSCA uses) have changed over time. For instance, the FDA banned DCM as an ingredient from all cosmetic products in 1989 (FDA, 1989) after it was used as an ingredient in aerosol cosmetic products (e.g., hairsprays) in concentrations ranging from 10 to 25 percent DCM.

# 2.2.1.2 Paint Stripping Applications

DCM is considered the best chemical stripper that is effective on the widest range of cured coatings from the widest variety of substrates (<u>Mannsville, 1999</u>). It is characterized this way because it can be used on almost any substrate, is very inexpensive, works quickly, and typically only requires one application to remove all the necessary paint or coating. The major applications for DCM-based paint strippers include use on Original Equipment Manufacturing (OEM), field maintenance stripping, and home improvement and repair. Most of these users purchase paint stripper from a formulator who mixes the DCM with other chemicals to achieve the desired product (<u>SRRP, 1992</u>). For industrial use, paint strippers are typically 70 to 90 percent DCM by weight. Household paint strippers for consumer use are typically 60 to 80 percent DCM (<u>EPA, 1993b; see Appendix D</u>).

Several studies have been conducted to evaluate the extent of DCM use in paint stripping. In 2008, EPA estimated that a total of 39,000 establishments performed surface coating operations, including paint stripping, motor vehicle, mobile equipment, and miscellaneous activities. Specifically, EPA estimated that about 3,000 of these facilities were paint stripping shops. Of these 3,000 facilities, 2,000 facilities used paint strippers containing  $\leq$  2,000 lbs of DCM, while 1,000 facilities used products containing > 2,000 lbs of the chemical (EPA, 2008).

# 2.3 SUMMARY OF ENVIRONMENTAL FATE

Knowledge of the environmental fate (transport and transformation) of a compound is important to understanding its potential impact on specific environmental media (*e.g.*, water, sediment, soil) and exposures to target organisms of concern.

DCM is volatile and does not significantly partition to solid phases. Therefore, releases of DCM to the environment are likely to evaporate to the atmosphere, or if released to soil, migrate to groundwater. DCM has a global warming potential (GWP) of 8.7 relative to carbon dioxide and thus can act as a greenhouse gas.

DCM has been shown to biodegrade over a range of rates and conditions and is considered to be moderately persistent in the environment. Measured bioconcentration factors for DCM suggest its bioconcentration potential is low. Appendix E has additional information about the environmental fate of DCM.

# **3 HUMAN HEALTH RISK ASSESSMENT**

# 3.1 OCCUPATIONAL EXPOSURE ASSESSMENT FOR THE USE OF DCM IN PAINT STRIPPING

Section 3.1.1 summarizes the approach and methodology used for estimating occupational inhalation exposures to DCM for the use of DCM-based paint strippers. Section 3.1.1.3 lists the occupational exposure estimates for the highest exposed worker population. Additional information is found in Appendices F and G.

Appendix F describes the industries that may use DCM-based paint strippers, worker activities, processes, numbers of sites, and numbers of exposed workers. Appendix G provides details about the air concentrations and associated worker Average Daily Concentrations (ADCs) and Lifetime Average Daily Concentrations (LADCs) presented in this section.

# 3.1.1 Approach and Methodology for Estimating Occupational Exposures

#### 3.1.1.1 Identification of Relevant Industries

Because a variety of industries include paint stripping among their business activities, EPA/OPPT made the effort to determine and characterize these industries, with a special interest in small commercial shops. EPA/OPPT's interest in small shops for this assessment is due to the possibility that these shops may have fewer resources or less expertise and awareness of hazards, exposures, or controls as compared to large shops.

There is no standard or universal definition for the term "small shop". The various meanings of this term can depend upon the industry sector (e.g., metal finishing, furniture repair, foam production, chemical manufacturing) or governmental jurisdiction (e.g. OSHA, EPA, other countries). For the purpose of risk assessment of work plan chemicals, EPA/OPPT generally refers to entities, businesses, operators, plants, sites, facilities, or shops interchangeably and considers a number of factors to categorize these as small. The factors that have been usually considered include revenue, capacity, throughput, production, use rate of materials, or number of employees. Further characterization to determine which factors best distinguish small shops for all the various industries that perform paint stripping would require more research.

EPA/OPPT reviewed the published literature and evaluated the 2007 North American Industry Classification System (NAICS) codes to determine industries that likely include paint stripping activities (see Appendix F, Table F-1).

The following industries were identified:

- Professional contractors;
- Bathtub refinishing;
- Automotive refinishing;
- Furniture refinishing;
- Art restoration and conservation;
- Aircraft paint stripping;
- Ship paint stripping; and
- Graffiti removal

By identifying these industries, EPA/OPPT identified corresponding worker subpopulations that may be exposed to DCM due to the use of these paint strippers. Appendix F details the industries identified, processes and worker activities that may contribute to workplace exposures. Section 3.1.1.2 and Appendix F provide the estimated number of workers exposed nationwide and average numbers of employees per facility for these industries.

#### 3.1.1.2 Estimation of Potential Workplace Exposures for Paint Stripping Facilities

**Workplace exposures based on monitoring data:** EPA/OPPT used air concentration data and estimates found in literature sources to serve as exposure concentrations for occupational inhalation exposures to DCM. These air concentrations were used to estimate the exposure levels for workers exposed to DCM as a result of the use of DCM-based paint strippers.

EPA/OPPT did not find enough monitoring data to determine complete statistical distributions of actual exposure concentrations for the exposed population of workers in each of the industries. Ideally, EPA/OPPT would like to know 50<sup>th</sup> and 95<sup>th</sup> percentiles for each population, which are considered to be the most important parts of complete statistical exposure distributions. The air concentration means and midpoints (means are preferred over midpoints) served as substitutes for 50<sup>th</sup> percentiles, and high ends of ranges served as substitutes for 95<sup>th</sup> percentiles.

Data sources often did not indicate whether monitored exposure concentrations were for occupational users or bystanders. Therefore, EPA/OPPT assumed that these exposure concentrations were for a combination of users and bystanders. Some bystanders may have lower exposures than users, especially when they are further away from the source of exposure.

Additionally, inhalation exposure data from OSHA and state health inspections were obtained from the OSHA's Integrated Management Information System (IMIS) database. However, OSHA IMIS data were not used to estimate workplace exposures, except where noted, because of the high degree of uncertainty and questionable relevancy of these data to stripping with DCM-containing products. Refer to Appendix G for a detailed discussion of the OSHA IMIS data.

**Workplace exposure scenarios evaluated in this assessment:** Workers performing DCM-based paint stripping might or might not use a respirator and may be exposed to DCM at different exposure frequencies (days per year) or working years. Thus, EPA/OPPT assessed acute risks for 4 occupational scenarios and chronic risks for 16 occupational scenarios based on 8-hr time-weighted average (TWA) exposure concentrations and different variations in exposure conditions. These scenarios were constructed within each industry evaluated in the assessment.

To estimate acute exposure, EPA/OPPT defined 4 scenarios to reflect a combination of the following (Table 3-1):

- No use of a respirator (APF = zero);
- Use of a respirator with an APF of 10, 25, or 50, which would reduce the personal breathing concentration by 10-, 25- or 50-fold (i.e., 0.1, 0.04, 0.02), respectively.

Acute Scenario	Respirator APF <sup>a</sup>	8-hr TWA Concentration Multiplier <sup>b</sup>	Scenario Description					
1	0	1 No re						
2	<b>2</b> 10 0.1 Respirator							
3	<b>3</b> 25 0.04 Respirator APF 2							
4	50	0.02	Respirator APF 50					
<ul> <li>Notes:         <ul> <li>APF= assigned protection factor. APFs of 10, 25 or 50 mean that the respirator reduced the personal breathing concentration by 10-, 25- or 50-fold (i.e., 0.1, 0.04, 0.02).</li> <li>As indicated in equation 3-2, these multipliers are applied to the 8-hr time-weighted average (TWA) acute exposure concentrations.</li> </ul> </li> </ul>								

To estimate chronic exposure, EPA/OPPT defined 16 scenarios to reflect a combination of the following (Table 3-2):

- No use of a respirator (APF = zero)<sup>6</sup>;
- Use of a respirator with an APF of 10, 25, or 50;
- An exposure frequency (EF) of the assumed Scenario 1 value of 250 days per year or half of the assumed Scenario 1 value (the midpoint between the assumed Scenario 1 value and zero: 125 days per year); and
- Exposed working years (WY) of the assumed Scenario 1 value of 40 years or half of the assumed Scenario 1 value (the midpoint between the assumed Scenario 1 value and zero: 20 years).

The multipliers in Tables 3-1 and 3-2 were used to adjust the exposure estimates of acute and chronic Scenario 1, respectively, to obtain the exposure estimates for the other exposure scenarios. Additional information is presented below about the estimation approach to calculate the acute and chronic exposure estimates.

<sup>&</sup>lt;sup>6</sup> APF assumptions are the same for both acute and chronic scenarios.

Table 3 2. Chronic Occupational Exposure Scenarios for the Use of DCM Based PaintStrippers								
Chronic Scenario	Respirator APF <sup>a</sup>	Exposure Frequency (EF) (days/yr)	Working Years (WY) (years)	ADC/LADC Multiplier <sup>b</sup>	Scenario Description			
1	0	250	40	1	No respirator, high ends of ranges for EF and WY			
2	10	250	40	0.1	Respirator APF 10, high ends of ranges for EF and WY			
3	25	250	40	0.04	Respirator APF 25, high ends of ranges for EF and WY			
4	50	250	40	0.02	Respirator APF 50, high ends of ranges for EF and WY			
5/9	0	250/ 125	20/ 40	0.5	No respirator, one midpoint and one high end of range for EF and WY			
6 / 10	10	250/ 125	20/ 40	0.05	Respirator APF 10, one midpoint and one high end of range for EF and WY			
7/11	25	250/ 125	20/ 40	0.02	Respirator APF 25, one midpoint and one high end of range for EF and WY			
8 / 12	50	250/ 125	20/ 40	0.01	Respirator APF 50, one midpoint and one high end of range for EF and WY			
13	0	125	20	0.25	No respirator, midpoints of ranges for EF and WY			
14	10	125	20	0.025	Respirator APF 10, midpoints of ranges for EF and WY			
15	25	125	20	0.01	Respirator APF 25, midpoints of ranges for EF and WY			
16	50	125	20	0.005	Respirator APF 50, midpoints of ranges for EF and WY			

Notes:

<sup>a</sup> APF= assigned protection factor. APFs of 10, 25 or 50 mean that the respirator reduced the personal breathing concentration by 10-, 25- or 50-fold, respectively.

<sup>b</sup> As indicated in equation 3-4, these multipliers are applied to the chronic average daily concentrations (ADCs) and lifetime average daily concentrations (LADCs).

EPA/OPPT evaluated scenarios both with and without respirator use and a range of respirator APFs because no data were found about the overall prevalence of the use of respirators to reduce DCM exposures and it was not possible to estimate the numbers of workers who have reduced exposures due to the use of respirators (as described by the data and information sources presented in Appendices F and G).

Likewise, EPA/OPPT made assumptions about the exposure frequencies and working years because data were not found to characterize these parameters. Thus, EPA/OPPT evaluated occupational risks by developing hypothetical scenarios under varying exposure conditions (i.e., use of respirators with different respiratory protection factors, and different exposure frequencies and working years).

**Approach for calculating acute and chronic workplace exposures:** To facilitate the exposure calculations for the occupational scenarios, EPA/OPPT first estimated the acute and chronic exposure estimates for Scenario 1 (highest exposure group). Equations are described below.

The exposure estimates for Acute Scenarios 2 to 4 and Chronic Scenarios 2 to 16 were obtained by adjusting scenario 1 (highest exposure group) with various multipliers (Tables 3-1 and 3-2 for acute and chronic, respectively). The acute multipliers reflected the numerical reduction in exposure levels when respirators were used. The chronic multipliers reflected the numerical reduction in exposure levels when respirators were used and/or other EF and WY values were used. Although 16 chronic scenarios were possible, scenarios 5 through 8 and 9 through 12 resulted in the same multiplier regardless of whether the scenario used an EF of 250 days/yr and a WY of 20 yrs, or an EF of 125 days/yr and a WY of 40 years.

#### Acute occupational exposure estimates

For single (acute) workplace exposure estimates, the DCM single (acute) exposure concentration was set to the 8-hour TWA air concentration in mg/m<sup>3</sup> reported for the various relevant industries. EPA/OPPT assumed that some workers could be rotating tasks and not necessarily using DCM-based paint strippers on a daily basis. This type of exposure was characterized as acute in this assessment as the worker would clear DCM and its metabolites before the next encounter with the DCM-containing paint stripper.

Equation 3-1 was used to estimate the single (acute) exposure estimates for acute scenario 1 (EPA, 2009).

EC scenario 
$$1 = C$$
 (Equation 3-1)

where:

- EC scenario 1
- exposure concentration for a single 8-hr exposure to DCM (mg/m<sup>3</sup>) for scenario 1
- С
- contaminant concentration in air for relevant industry (central tendency, low- or high-end 8-hr TWA in mg/m<sup>3</sup> from Appendix G, Table G-2 or G-5);

Equation 3-2 was used to calculate the acute exposure estimates for scenarios 2 through 4.

EC scenario 
$$2 \rightarrow 4 = EC$$
 scenario  $1 \times M$  acute (Equation 3-2)

where:

EC scenario $2 \rightarrow 4$	=	exposure concentration for a single 8-hr exposure to DCM
		(mg/m <sup>3</sup> ) for acute scenarios 2, 3, or 4;
EC scenario 1	=	single (acute) exposure concentration for relevant industry (8-hr
		TWA in mg/m <sup>3</sup> from Appendix G, Table G-2 or G-5);
M acute	=	Scenario-specific acute exposure multiplier (unit less) for relevant industry (see Table 3-1)

Acute exposure estimates for scenario 1 are presented in Table 3-3. Acute exposure estimates for scenarios 2 through 4 were integrated into the risk calculations by applying the scenario-specific multipliers. Thus, separate tables listing the acute exposure estimates for scenarios 2 through 4 are not provided in this section, but are available in a supplemental Excel spreadsheet documenting the risk calculations for this assessment (*DCM Exposure and Risk Estimates\_081114.xlsx*).

#### Chronic occupational exposure estimates

The worker exposure estimates for the non-cancer and cancer risk calculations were estimated as ADCs and LADCs, respectively. Both ADC and LADC calculations for Scenario 1 were based on the 8-hr TWA air concentration in mg/m<sup>3</sup> reported for the various relevant industries (Appendix G, Table G-5). EPA/OPPT assumed that the worker would be doing paint stripping activities during the entire 8-hr work shift on a daily basis. Equation 3-3 was used to estimate the chronic ADCs and LADCs for Scenario 1 (EPA, 2009).

$$EC_{\text{scenario 1}} = \frac{C \times ED \times EF \times WY}{AT} \qquad \text{(Equation 3-3)}$$

where:

EC scenario 1	=	exposure concentration (mg/m <sup>3</sup> ) for Scenario 1 = ADC for chronic non-
		cancer risks or LADC for chronic cancer risks for Scenario 1;
С	=	contaminant concentration in air for relevant industry (central tendency,
		low- or high-end 8-hr TWA in mg/m <sup>3</sup> from Appendix G, Table G-2);
ED	=	exposure duration (hrs/day) = 8 hrs/day;
EF	=	exposure frequency (days/yr) = 250 days/yr for high-end of range
		for both ADC and LADC calculations;

WY	=	working years per lifetime (yrs) = 40 yrs for high end of range
		for both ADC and LADC calculations; and
AT	=	averaging time (years × 365 days/years × 24 hrs/day) = 40 yrs for high
		end of range for ADC calculations; 70 yrs for LADC calculations, which is
		used to match the years used to calculate EPA's cancer inhalation unit
		risk (IUR).

Equation 3-4 was used to estimate the chronic ADCs and LADCs for scenarios 2 through 16.

# $EC \text{ scenario } 2 \rightarrow 16 = EC \text{ scenario } 1 \times M \text{ chronic}$ (Equation 3-4)

where:

EC scenario $2 \rightarrow 16$	=	exposure concentration for chronic exposure concentration (ADC or LADC) to DCM (mg/m <sup>3</sup> ) for chronic scenarios 2 through 16
EC scenario 1	=	chronic exposure concentration (ADC or LADC) for relevant industry, chronic scenario 1 (in mg/m <sup>3</sup> from Table 3-3);
${\sf M}$ chronic	=	scenario-specific ADC/LADC chronic multiplier for relevant industry (see Table 3-2)

Non-cancer and cancer exposure estimates (i.e., ADC and LADC, respectively) for scenario 1 are presented in Table 3-3. The estimates for scenarios 2 through 16 were integrated into the risk calculations by applying the scenario-specific ADC/LADC multipliers. Thus, separate tables listing the chronic exposure estimates for scenarios 2 through 16 are not provided in this section, but are available in a supplemental Excel spreadsheet documenting the risk calculations for this assessment (*DCM Exposure and Risk Estimates\_081114.xlsx*).

**Numbers of exposed workers and shop sizes:** Knowing the sizes of exposed populations provides perspective on the prevalence of the health effects. Thus, EPA/OPPT estimated the current total number of workers in the potentially exposed populations.

EPA/OPPT found limited data on numbers of workers exposed to DCM in shops that use DCMbased paint strippers. EPA/OPPT relied on an estimation approach to estimate the total number of exposed workers from the technical support document for the National Emission Standards for Hazardous Air Pollutants (NESHAP) Paint Stripping Operations at Area Sources proposed rule (EPA, 2007).

Based on the NESHAP data and analyses, EPA/OPPT estimates that over 230,000 workers nationwide are directly exposed to DCM from DCM-based paint strippers. This estimate only accounts for workers performing the paint stripping using DCM and does not include other workers ("occupational bystanders") within the facility who are indirectly exposed. EPA/OPPT cannot estimate the numbers of workers exposed in each of the individual industries that may use DCM-based strippers. EPA/OPPT also cannot estimate the numbers of workers exposed in small shops. Appendix E details the literature search, data found, and assumptions for worker population exposed nationwide.

EPA/OPPT estimated the average number of employees per facility which can be a factor in determining shop sizes. These estimates were derived by combining the facility and population data obtained from the U.S. Census data, as described in Appendix F. The average number of employees for the identified industries based on U.S. Census data were the following:

- Professional contractors (likely to include Bathtub refinishing): 5 workers/facility;
- Automotive refinishing: 6 workers/facility;
- Furniture refinishing: 3 workers/facility;
- Art restoration and conservation (not estimated);
- Aircraft paint stripping: 320 workers/facility (for aircraft manufacturing only);
- Ship paint stripping: 100 workers/facility; and
- Graffiti removal: 8 workers/facility.

These averages give some perspective on shop size but are simple generalizations.

#### **3.1.1.3 Summary of Occupational DCM Exposure Estimates**

Table 3-3 shows the DCM air concentrations used in this assessment for estimating acute and chronic risks for the highest exposed worker scenario group (Scenario 1) within each industry. The statistical issues of these estimates are briefly discussed in section 3.5.1.

Acute and chronic DCM exposure estimates for Acute Scenarios 2 through 4 and Chronic Scenarios 2 through 16 were integrated into the risk calculations by applying multipliers to Scenario 1. Separate tables listing the acute and chronic exposure estimates are not provided in this section, but can be found in the supplemental Excel spreadsheet - *DCM Exposure and Risk Estimates\_081114.xlsx*. Also, Table ES-1 provides a summary of the ranges of acute, ADC and LADC estimates for the various occupational scenarios.

Table 3 3. DCM Acute and Chronic Exposure Concentrations (ADCs and LADCs) for WorkersScenario 1Highest Exposed Scenario Group													
Industry / Activity	Time Range of Studies		ACUTE EXPOSURE ESTIMATES Single 8-hr Concentration (mg/m <sup>3</sup> ) <sup>a</sup> Mean High Midpoint Low			CHRONIC EXPOSURE ESTIMATES USED IN THE NON-CANCER RISK ESTIMATES ADC (mg/m <sup>3</sup> ) <sup>b</sup> Mean High Midpoint Low			CHRONIC EXPOSURE ESTIMATES USED IN THE CANCER RISK ESTIMATES LADC (mg/m <sup>3</sup> ) <sup>b</sup> Mean High Midpoint Low				
Professional Contractors	1981- 2004		2,980	1,520	60		680	347	14		389	198	7.8
Bathtub Refinishing		-				-			-	-			
Automotive Refinishing	2003	253	416	253	90	58	95	58	21	33	54	33	12
Furniture Refinishing	1989- 2007	499	2,245 (1,266) c	1,125	4.0	114	513 (289) c	257	0.9	65	293 (165) د	147	0.5
Art Restoration and Conservation	2005	2.0			0.5				0.3				
Aircraft Paint Stripping	1977- 2006	1	3,802	1,944	86	1	868	444	20	-	496	254	11
Ship Paint Stripping	1980								-	1			
Graffiti Removal	1993	260	1,188	603	18	59	271	138	4.1	34	155	79	2.3
Non-Specific Workplace Settings - Immersion Stripping of Wood	1980- 1994		7,000	3,518	35		1,598	803	8.0		913	459	4.6

Table 3 3. DCM Acute and Chronic Exposure Concentrations (ADCs and LADCs) for WorkersScenario 1Highest Exposed Scenario Group													
Industry / Activity	Time Range of Studies		ACUTE EXPOSURE ESTIMATES Single 8-hr Concentration (mg/m <sup>3</sup> ) <sup>a</sup>			CHRONIC EXPOSURE ESTIMATES USED IN THE NON-CANCER RISK ESTIMATES ADC (mg/m <sup>3</sup> ) <sup>b</sup>			CHRONIC EXPOSURE ESTIMATES USED IN THE CANCER RISK ESTIMATES LADC (mg/m <sup>3</sup> ) <sup>b</sup>				
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	1980		1,017	825	633		232	188	145		133	108	83
Non-Specific Workplace Settings - Immersion Stripping of Metal													
Non-Specific Workplace Settings – Unknown	1997- 2004	357	428	357	285	81	98	81	65	47	56	47	37

Notes:

Sources are reported in Table G-2 and discussed in section G-3.

<sup>a</sup> Calculated acute single 8-hr concentrations are only estimated from 8-hr TWA exposures; see Equation 3-1. Airborne concentration conversion factor for DCM is 3.47 mg/m<sup>3</sup> per ppm (<u>NIOSH, 2011b</u>).

<sup>b</sup> Calculated ADCs and LADCs are only calculated from 8-hr TWA exposures; see Equation 3-3.

<sup>c</sup> The values in parentheses are the 95<sup>th</sup> percentiles of the calculated acute single 8-hr concentrations and the calculated ADCs and LADCs.

-- Indicates no data found.

#### 3.1.1.4 Worker Exposure Limits for DCM

Both regulatory and non-regulatory worker exposure limits have been established for DCM by OSHA, NIOSH, and the American Conference of Government Industrial Hygienists (ACGIH). EPA/OPPT analysis showed that the OSHA permissible exposure limit (PEL) and Action Level values were exceeded for some industries using DCM-based strippers when the OSHA values were compared to the air concentrations.

Table 3-4 provides a summary of the current occupational exposure values established by OSHA, NIOSH, and ACGIH. Appendix F presents additional background on processes, respiratory protection, facilities and worker populations.

OSHA's amended regulatory occupational exposure limits for DCM were effective April 10, 1997. The amendments included reducing the PEL, reducing and changing the averaging time of the short-term exposure limit (STEL), adding an Action Level, and removing the ceiling limit (<u>OSHA, 1997a</u>). See Appendix G, section G-2-3, for more details.

Table 3 4. Occupational Exposure Limits for DCM <sup>a</sup>							
Source	Limit Type	Exposure Limit					
	PEL (8-hr TWA) <sup>b</sup>	25 ppm <sup>c</sup>					
OSHA PEL	STEL (15-minute TWA)	125 ppm					
	Action Level (8-hr TWA)	12.5 ppm					
	IDLH <sup>d</sup>	2,300 ppm					
NIOSH exposure limits	REL <sup>e</sup>	Са					
ACGIH TLV <sup>f</sup>	8-hr TWA	50 ppm					

Notes:

<sup>a</sup> Source: OSHA (1997a)

- <sup>b</sup> PEL= Permissible exposure limit ; TWA= Time-weighted average
- <sup>c</sup> Airborne concentration conversion factor for DCM is 3.47 mg/m<sup>3</sup> per ppm (<u>NIOSH, 2011b</u>).
- <sup>d</sup> IDLH = Immediately dangerous to life and health. IDLH values are based on effects that might occur from a 30-minute exposure.
- REL = Recommended Exposure Limit. The REL notation "Ca" is for a potential occupational carcinogen. The NIOSH Pocket Guide website has detailed policy recommendations for chemicals with "Ca" notations (<u>NIOSH, 2011a</u>).

<sup>f</sup> TLV = Threshold limit value

# 3.2 CONSUMER EXPOSURE ASSESSMENT FOR THE USE OF DCM IN PAINT STRIPPING

Section 3.2 summarizes the modeling approach used for estimating consumer inhalation exposures to DCM for the use of DCM-based paint strippers. The consumer modeling is discussed in greater detail in Appendix H.

# **3.2.1 Approach and Methodology for Estimating Consumer Exposures**

EPA/OPPT used the Multi-Chamber Concentration and Exposure Model (MCCEM) to estimate consumer exposures to DCM-based paint strippers. This modeling approach was selected because published monitoring data for non-occupational inhalation exposures (i.e., consumer do-it-yourself [DIY]) were limited to those from several chamber studies conducted in the U.S. and Europe. The literature search for this assessment did not identify any published exposure information for exposures to other household members (i.e., bystanders). Of the available chamber studies, only one U.S. study provided sufficient information for the exposure modeling (EPA, 1994a).

# **3.2.2 Overview of the MCCEM**

The MCCEM is an exposure model that estimates airborne concentrations of chemicals released from products in residential settings or other indoor environments (EPA, 2010a). EPA/OPPT relied on a model-based consumer exposure assessment in the absence of sufficient measured data for consumer exposures to DCM-based paint strippers.

The MCCEM incorporates the following features (EPA, 2010a):

- Represents a multiple zone model that uses a deterministic, mass-balance equation to predict time varying indoor air concentrations;
- Uses chemical volatilization rates from chamber test emission data as an input, making it a higher tier model;
- Considers the amount of time individuals spend each day within each zone based on human activity patterns;
- Has been peer reviewed in 1998.

The MCCEM generally uses a two-zone representation of a house to calculate acute air concentrations of DCM for consumers and bystanders for various exposure scenarios. Zone 1 represents the area where the consumer was using the product, whereas Zone 2 represents the rest of the house (ROH). Zone 2 was used for modeling passive exposure to house residents (bystanders), such as children, adults, pregnant women and the elderly (EPA, 2010a). The model assumes complete mixing in each zone.

The MCCEM uses 3 zones to model the bathtub "source cloud" scenario. In this scenario, Zone 1 represents the arbitrary volume close to the tub. Zone 2 represents the bathroom volume, and Zone 3 was the rest of house.

For this assessment, the general steps of the calculation engine within the MCCEM include:

- 1. Introduction of DCM into the room of use by applying the paint stripper on a surface and estimation of the declining emission rate in that room: Consumer products that are applied to surfaces are best represented by the incremental source model. This model assumes a constant application rate over time, coupled with an emission rate for each instantaneously applied segment that declines exponentially over time. Depending on the type of applied product, either one or two exponential expressions may be needed to characterize the declining emission rate (EPA, 2010a). From an analysis of chamber test data, EPA/OPPT determined that a single-exponential expression was appropriate for paint strippers with DCM as a primary ingredient.
- 2. Transfer of DCM to the rest of the house as a function of the rate of chemical loss and gain for that zone: MCCEM requires the conservation of pollutant mass as well as the conservation of air mass when predicting indoor air concentrations in different house zones. The modeled concentration in each zone is a function of the time-varying emission rate in the room of use, the zone volumes, the air exchange rate and the interzonal airflow rates among zones and between each zone and outdoor air (EPA, 2010a).
- **3.** Estimation of the zone-specific airborne concentrations of DCM as the modeled occupant moves around the house: MCCEM estimated detailed time series of zone-specific (*e.g.*, house, workshop, and bathroom) concentrations to account for an individual's location at specific times. The model output was in the form of instantaneous values at the end of consecutive one-minute time intervals for the entire duration of the model run (*i.e.*, 24 hrs in this case) for both the user and residential bystander. The one-minute intervals were used to calculate acute maximum TWA concentrations for certain averaging periods for the user and residential bystanders (*i.e.*, one, 10, and 30 minutes; 1-, 4-, 8- and 24-hrs). The maximum TWA concentration for any averaging period was defined as the highest value of the consecutive running averages for that averaging period. These general steps are explained in greater detail in Appendix H.

EPA/OPPT used the DCM air concentrations for the different averaging periods to evaluate the human health risks of acute, but not chronic, exposures to DCM-based paint strippers in residential settings. The focus on acute exposures is based on the assumption that DCM is not expected to significantly buildup in the body between exposure events. DCM's plasma half-life is estimated to be 40 minutes after inhalation exposure (<u>DiVincenzo et al., 1972</u>). Moreover, EPA/OPPT assumed that consumers would not generally do paint stripping jobs on a regular basis in their residences, allowing sufficient time between exposures to clear DCM and its metabolites from the body.

# **3.2.3 MCCEM Input Parameters and Assumptions**

EPA/OPPT identified and used published data for current product characteristics, use patterns, exposure factors, and air monitoring data to set the model input parameters and develop appropriate consumer exposure scenarios.

Brown (2012) reported a list of DCM-containing products currently available for consumer purchase. EPA/OPPT used the list of consumer products to determine reasonable percentage of DCM in products and product densities. Other resources providing information on product characteristics included the NIH Household Products Database, Material Safety Data Sheets (MSDS), and Product Labels and Technical Data Sheets. Additional data sources were identified and used to support model assumptions and input parameters and they are discussed below. EPA/OPPT assessed the data quality of the identified sources before using the information for the modeling approach. Data quality criteria were similar to those used for evaluating occupational data (Appendix H, section H-1-3 and Table H-1).

The model assumptions and input parameters are summarized in section 3.2.2 and explained more fully in Appendix H.

#### 3.2.3.1 Estimation of Emission Profiles for Paint Removers/Strippers

EPA/OPPT identified air monitoring studies for consumer paint strippers using DCM-containing products, including the Midwest Research Institute (MRI) chamber study (EPA, 1994a), the European Commission (EC) study (EC, 2004), and a study conducted in the Netherlands by (van Veen et al., 2002). Data from the MRI chamber study were used as the basis for developing emission profiles for both brush-on and spray-on applications for this assessment (EPA, 1994a). The MRI chamber data were considered adequate to support the exposure estimation effort and the products studied were considered to be the most representative of paint strippers available in the U.S. consumer product market.

The <u>EC (2004)</u> study is the most current experimental study conducted for paint strippers. However, one of its main limitations was the failure to provide the raw data in the report. Thus, the overall findings of the EC study could not be verified. Additionally, the study may not be representative of use patterns and DCM-containing products in the U.S.

Although the <u>van Veen et al. (2002)</u> study provided useful information, the study was conducted on a small scale and the exposure scenario assessed did not represent well the use patterns in the U.S.

Further discussion and comparison of the air monitoring/chamber test studies above is provided in Appendix H, section H-5.

#### 3.2.3.2 Method of Application

Product labels and technical data sheets indicate that DCM-based paint strippers are sold as brush-on or spray-on products. Thus, EPA/OPPT assessed consumer exposures to products applied by these two application methods. Each application method is characterized by specific chemical release characteristics, DCM weight fractions, application rates, and time required for application. The modeling approach was designed to consider these differences between brushon and spray-on products.

#### 3.2.3.3 Amount Applied to the Surface (Product Mass)

The product application mass (grams of product) was determined for each of the cases examined using application rates (in  $g/ft^2$ ) calculated from the <u>EPA (1994a)</u> chamber tests and the surface areas of objects (in  $ft^2$ ) to be stripped.

EPA (1994a) reported the most complete air monitoring data for the consumer use of paint strippers containing DCM<sup>7</sup>. The study documented chamber experiments for five paint stripping products used in the U.S., including two paint-stripping products containing DCM. The two DCM products were: 1) a spray-on product containing 80 to 85 percent DCM; and, 2) a brush-on product containing >10 percent DCM. EPA/OPPT used descriptions of the study design and the results to determine product application rates (*i.e.*, in g/ft<sup>2</sup> and g/min) and estimated the fraction of applied chemical mass that ultimately was released to the indoor air. Unfortunately, the experimental data could not be used directly to assess indoor residential inhalation exposures in this assessment because the values for the required exposure factors, (*e.g.*, room/house volume, airflow rates, and surface area of object) did not reflect the range of possible residential values. Furthermore, the experiments did not provide concentrations for areas in the rest of the house where the product was not being used.

The calculated application rates were ~90 g/ft<sup>2</sup> and ~68 g/ft<sup>2</sup> for a brush-on and spray-on application, respectively. These application rates are similar to those recommended by Savogran (i.e., 42 to 83 g/ft<sup>2</sup> based on a nominal density of  $1.1 \text{ g/cm}^3$ )<sup>8</sup>.

Surface areas for the consumer exposure modeling were selected so that the resulting mass (g) of the applied product corresponded approximately to the <u>CPSC (1992)</u> survey results for amount of paint stripper used, as reported in the latest Exposure Factors Handbook (EFH) (<u>EPA</u>, <u>2011a</u>). The <u>CPSC (1992)</u> survey reported the following central (near the median) and upperend estimates for the amount of paint stripper product used per event:

<sup>&</sup>lt;sup>7</sup> Appendix H discusses other studies that were reviewed, but were not used to estimate the emission profiles of DCM-based paint strippers.

<sup>&</sup>lt;sup>8</sup> Savogran sells retail and industrial cleaning and paint preparation products, including paint removers <u>http://www.savogran.com/</u>

- 50<sup>th</sup> percentile value of 32 ounces or 1,000 g for the central-tendency surface area of 10 ft<sup>2</sup>. The median value is also supported by Riley et al. (2001)<sup>9</sup>;
- ~80<sup>th</sup> percentile value of 80 ounces or 2,500 g for the upper-end surface area of 25 ft<sup>2</sup>.

To assist the reader to visualize the exposure scenarios, a coffee table of 4 x 2.5 ft could represent the central-tendency surface area of 10 ft<sup>2</sup>, while a chest of drawers 4 ft high x 2.5 ft wide x 1.5 ft deep could represent the upper-end surface area of 25 ft<sup>2</sup>. The model used median product masses of 900 and 680 g for the brush-on and spray-on scenarios, respectively. Upper-end product masses for the brush-on and spray-on scenarios were 2,250 and 1700 g, respectively.

The bathroom scenario occurred in a confined space and was assumed to be performed by a home contractor, as opposed to a consumer. A lower mass of 477 g was used for the brush-on bathroom scenario. The lower mass value was derived from the largest application amount identified in the NIOSH report (<u>CDC, 2012</u>). A surface area of 36 ft<sup>2</sup> was calculated for a bathtub, resulting in an application rate of 13.25 g/ft<sup>2</sup>.

#### 3.2.3.4 Stripping Sequence

The stripping sequence was based in part on product label instructions, which for some DCMcontaining products (*i.e.*, Klean Strip<sup>®</sup> products) indicate that no more than 9 ft<sup>2</sup> should be stripped at a time. Product label information also indicated that the stripping should be repeated to remove multiple coats of paint. As a result, the surface areas of the coffee table, chest and bathtub were divided as follows:

- 10-ft<sup>2</sup> coffee table: Surface area was divided into 2 application segments of 5 ft<sup>2</sup> each with repeat application for a total of 4 segments;
- 25-ft<sup>2</sup> chest: Surface area was divided into 4 application segments of 6.25 ft<sup>2</sup> each with repeat application for a total of 8 segments;
- 36-ft<sup>2</sup> bathtub: Surface area was divided into 4 application segments of 9 ft<sup>2</sup> each with repeat application for a total of 8 segments.

The stripping sequence for brush-on and spray-on applications was divided into 3 steps: (1) product application, (2) wait period, and (3) scraping. EPA/OPPT used product label information to establish the time durations (in minutes) that the user would require to complete each step. Table H-7 in Appendix H describes the detailed stripping sequence for the brush-on application to the chest surface.

It was further assumed that the paint scrapings were removed from the house as soon as scraping was completed for the last segment. In addition, back-to-back stripping sequences

 <sup>&</sup>lt;sup>9</sup> <u>Riley et al. (2001)</u> represents the most current use-pattern survey available for paint strippers. Refer to Appendix H for more information on this study.

with no overlapping activities were modeled because it is likely that the user would take breaks during the wait period.

#### 3.2.3.5 Amount of Chemical Released

The amount of chemical released during and after the stripping event is the product of three parameters: amount applied to the surface (discussed above), weight fraction of chemical in the applied product, and fraction of the chemical that is released to indoor air.

From the product list developed by (<u>Brown, 2012</u>), the median DCM weight fraction was determined to be 0.53 for the brush-on application and 0.8 for the spray-on application. The corresponding 90<sup>th</sup> percentile weight fractions were 0.88 for brush-on and 0.87 for spray-on. A weight fraction of 1.0 (maximum exposure estimate derived from product label) was assumed for the bathtub application.

Release fractions of 0.33 and 0.66 were used for brush-on and spray-on applications, respectively, based on the analysis of the MRI chamber data (<u>EPA, 1994a</u>). Appendix H lists the resultant mass released for the different application targets and methods.

#### 3.2.3.6 Airflow Rates and Volumes

Information about the zone volumes, air exchange rates and interzonal air flows was obtained from published sources including the 2011 EFH (<u>EPA, 2011a</u>), <u>Riley et al. (2001)</u>, <u>EPA (1995a)</u>, <u>Matthews et al. (1989)</u> and <u>CDC (2012)</u>.

The house volume chosen for the model runs (492 m<sup>3</sup>) was the central value listed in the 2011 EFH (EPA, 2011a). The volume assigned to the in-house workshop area was 54 m<sup>3</sup>, which is similar to the value reported in <u>Riley et al. (2001)</u> for the mean volume of the room used for paint stripping (51 m<sup>3</sup>). The volume for the ROH (438 m<sup>3</sup>) is determined by subtraction (492 m<sup>3</sup>-54 m<sup>3</sup>). For the bathtub scenario, the bathroom volume was set at 9 m<sup>3</sup> for consistency with that reported in <u>CDC (2012)</u>.

The air exchange rate (ACH) values for the ROH were the central and low-end values of 0.45/hr and 0.18/hr, respectively. The ACH values corresponded to the mean and 10<sup>th</sup> percentile values reported by the 2011 EFH (<u>EPA, 2011a</u>) and represented the indoor-outdoor airflow rate for the ROH.

For the workshop scenarios, it was assumed that multiple windows were opened. This assumption was supported by both product's labeling instructions and survey data that found the majority of paint stripper users kept a window or door open during use (<u>CPSC, 1992</u>; <u>EPA, 1987</u>; <u>Pollack-Nelson, 1995</u>; <u>Riley et al., 2001</u>). The indoor-outdoor airflow rate assigned to the workshop (68 m<sup>3</sup>/hr) was obtained by multiplying the room volume of 54 m<sup>3</sup> by the 90<sup>th</sup> percentile of the air-exchange-rate distribution from the EFH (1.26 hr; <u>EPA, 2011a</u>), as it was thought to be a reasonable representation of the open-window case.

ACH values and house volumes described above were used to derive the interzonal airflow rates for the workshop scenarios. Appendix H describes how the interzonal airflow rates were estimated using an algorithm developed by (EPA, 1995a).

The modeling of the tub stripping for the bathroom scenario considered a source-cloud effect to better represent the user's exposure to DCM emitted from the paint stripper. The concept of a "source cloud" in the bathroom scenario assumes that the user is typically exposed to elevated concentrations in the immediate vicinity of the application area while stripping the bathtub for an extended period. To account for the source-cloud effect, the model was designed to create a third zone ("source cloud") within the bathroom to represent the DCM concentrations in the vicinity of the tub. The airflow rate between the cloud and the rest of the bathroom was based on work by <u>Matthews et al. (1989)</u>. The indoor-outdoor airflows were based on the air exchange rate of 0.18 ACH assuming windows closed and no exhaust fan. Please refer to Appendix H, section H-3 (*Inhalation Exposure Scenario Inputs: Airflow Rates and Volumes*) for more information.

#### 3.2.3.7 Locations of Exposed Individuals

The model places the user in the work area for stripper application and scraping, which is either in the workshop or a bathroom. During the waiting phase of the stripping process, the user may be placed in the ROH as a central-tendency assumption or in the room of use as an upper-end assumption. However, residential bystanders are located in the ROH.

<u>Riley et al. (2001)</u> supports the reasonableness of placing the user in the ROH during the wait period. The survey reported that 65 percent of users take breaks outside the work area. EPA/OPPT also assumed that users leaving the room of use would be aware of inhalation health hazards from the product's labeling warnings.

However, EPA assumes that some users would stay in the workshop because they do not read the product's labels and may therefore not be aware of health concerns or precautionary techniques. <u>Pollack-Nelson (1995)</u> reported that ~28 percent of consumers did not read the product labels while using paint strippers. Moreover, many labels do not specifically recommend users to leave the room during the wait period. <u>Riley et al. (2001)</u> indicated that 20 percent of participants reported taking breaks inside the work area. EPA/OPPT assumed that the user left the workshop during the wait period for most scenarios, but also included two scenarios with the users staying in the workshop during the wait time.

# 3.2.4 MCCEM Modeling Scenarios

Changing the values for various combinations of input parameters generates a wide range of plausible exposure scenarios and can increase the level of confidence in the model results. Thus, EPA/OPPT conducted a sensitivity analysis as a first step to guide the development of

exposure scenarios for the inhalation exposure assessment of DCM-based paint strippers. The sensitivity analysis helped us to determine which parameters used in the model have the most influence over the results of the assessment.

The types of factors that can be varied in MCCEM include the following:

- The configuration of the structure (residence in this case) being modeled, including the number of zones, volume of each zone, airflow rates between each zone and outdoor air, and airflow rates between zones (*i.e.*, interzonal airflow rates);
- The quantity of DCM emitted from the applied product and the time-varying emission rate, which are related to: (1) the type and area of surface being stripped; (2) the type of application (*e.g.*, brush-on *vs.* spray-on); and (3) the rate at which the product is applied to the surface; and
- Locations during and after stripping of users and residential bystanders.

The methods for and results of this sensitivity analysis are described immediately below followed by discussion of the consumer exposure scenarios supporting the risk assessment.

#### 3.2.4.1 Sensitivity Analysis

The sensitivity analysis was conducted using an approach that has been termed a "nominal range sensitivity analysis" (Frey and Patil, 2002). With this approach, a "base case" is defined first, typically consisting of central values for each model input. The base case for the sensitivity analysis was formed as follows:

- House volume of 492 m<sup>3</sup> (corresponds to a 36 ft × 30 ft, two-story house with 8-ft ceiling);
- Workshop (area of product use) volume of 54 m<sup>3</sup> (corresponds to a 20 ft × 12 ft room with 8-ft ceiling) and an indoor-outdoor airflow rate of 68 m<sup>3</sup>/hr (expected value for a room with multiple open windows);
- Airflow rate of 197 m<sup>3</sup>/hr for the ROH, assuming windows closed, corresponding to an air exchange rate of 0.45 ACH;
- Brush-on application with a target surface area of 10 ft<sup>2</sup>;
- Applied product mass of 900 g (90 g/ft<sup>2</sup>) and emitted (released to indoor air) DCM mass of 148.5 g, assuming a DCM weight fraction of 0.5 in the product and a release fraction of 0.33;
- User located in workshop during application and scraping periods but in ROH during wait periods between applying/scraping and after completion of all applying/scraping.

The time required to apply and scrape the paint stripper, including the wait time between applying and scraping, is about an hour, according to <u>CPSC (1992)</u>. Consequently, the model was run for a 24-hr period to capture all or most of the declining indoor-air concentrations following the episode of product use.

For this assessment, the relevant exposure measures included the maximum TWA concentrations for certain averaging periods (*i.e.*, one, 10, and 30 minutes and 1, 4, and 8 hrs)

in addition to the 24-hr TWA value. All the exposure durations were reported in the model runs; but only the maximum 1-hr and the 24-hr TWA were used for the sensitivity analysis. Figures 3-1 and 3-2 show a generic example of the user and bystander exposure to DCM for selected averaging times.

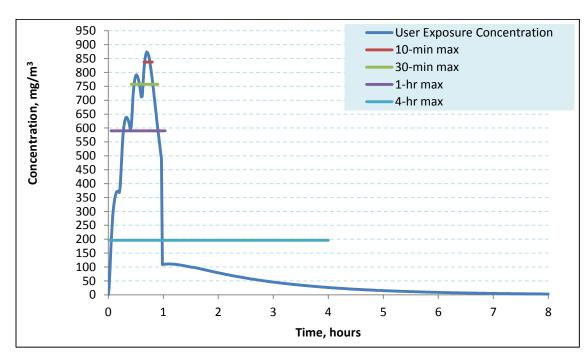
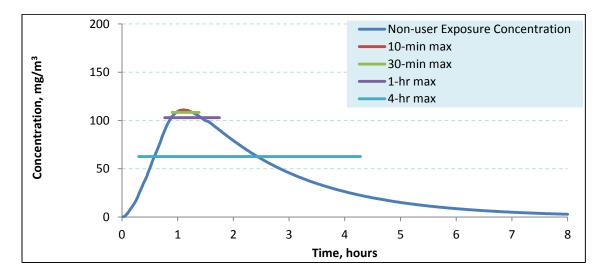




Figure 3-2. Example of Time-Varying Residential Bystander Exposure Concentration and Maximum TWA Values for Selected Averaging Times



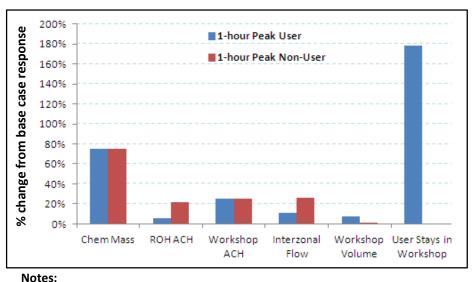
The next step after running the base case consists of varying the input parameters—one at a time—and recording the model response (*i.e.*, average or peak concentrations to which individuals are exposed). The "index of sensitivity" is the magnitude of change in model response, typically expressed as a percent change from that of the base case. Details about the computation approach for the sensitivity analysis are described in Appendix H, Section H-2 (*Sensitivity Analysis for Inhalation Scenarios*).

Figures 3-3 and 3-4 display the sensitivity results for two exposure measures, maximum 1-hr TWA and 24-hr TWA, respectively. The results can be summarized as follows:

- The model is highly sensitive to changes in chemical mass as shown by a 75 percent change from the base case response in both the user and residential bystander exposed to DCM for 1- and 24-hrs. This is indicative of a linear and proportional response.
- The model is even more sensitive to changes in the user location during the wait period between applying and scraping (*i.e.*, user stays in workshop *vs.* moves to ROH) irrespective of whether the user is exposed to DCM for 1- and 24-hrs.
- The model response is somewhat sensitive to the ROH air exchange rate with outdoor air (ROH ACH) for the bystander, but not for the user.

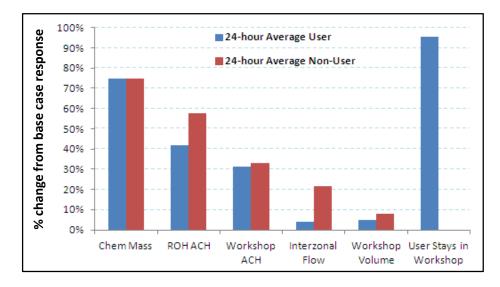
As a result of the sensitivity analysis, EPA/OPPT determined that the chosen modeling scenarios should include some variations in each of these three factors (i.e., DCM chemical mass emitted, user location during the wait period, and the ROH ACH with outdoor air) to address greater model sensitivity.

#### Figure 3-3. Model Sensitivity Results: Percent Change from Base-Case Response for Maximum 1-hr TWA for User and Residential Bystander



Chem Mass= Chemical mass ROH ACH= Rest of the house air exchange rate Non-user= Residential bystander

Figure 3-4. Model Sensitivity Results: Percent Change from Base-case Response for 24-hr TWA for User and Residential Bystander



#### Notes:

Chem Mass= Chemical mass ROH ACH= Rest of the house air exchange rate Non-user= Residential bystander

#### **3.2.4.2 Exposure Scenarios for the DCM Inhalation Exposure Assessment**

Table 3-5 lists the seven indoor exposure scenarios evaluated for this risk assessment. Also, Table 3-6 summarizes the input parameters and assumptions that were used to build the scenarios.

The following factors were considered in developing the scenarios:

- The type of application (*i.e.*, brush-on or spray-on), weight fraction of applied product, application rate, surface area of object to be stripped, and emission rate of the chemical concern, which can affect the amount of DCM that ultimately is released to the indoor environment;
- The location where the product is applied, which relates to exposure factors such as the room volume and its air exchange rate with outdoor air;
- The house volume and air exchange rate, for reasons similar to those for the product use location;
- Precautionary behaviors such as opening windows in the application room and the user leaving the application room during the effect period, and related changes to the air exchange rates and the proximity of the user to the source of DCM emissions.

Table 3 5. Consumer Exposure Scenarios for the DCM Inhalation Exposure Assessment									
	Scenario Description								
Scenario ID	Type of Application	Location of Product Use	Concentration Characterization <sup>a</sup>						
1	Brush-on	Workshop	Central tendency						
2	Brush-on	Workshop	User Upper-end						
3	Brush-on	Workshop	User and Bystander upper-end						
4	Spray-on Workshop Central tendency								
5	Spray-on	Workshop	User Upper-end						
6	Spray-on	Workshop	User and Bystander Upper-end						
7	7 Brush-on Bathroom Bystander Upper-end								
Note: <sup>a</sup> Conditions obtained by varying the most sensitive parameters within application type: DCM mass emitted; user location during the wait period; and the rest of the house (ROH) air exchange rate with outdoor air.									

Table 3	6. Summa	ry of DCN	/I Consum	er Paint Str	ipping Sc	enario Descriptions and	Paramete	ers				
		DCM Released					Ro	Room of Use		House		
Scenario ID	Conc. Characte- rization	Weight Fraction	Surface Area Treated <sup>a</sup> , ft <sup>2</sup>	Application Rate, g/ft <sup>2</sup>	Release Fraction	Stripping Method	Volume, m <sup>3</sup>	Ventilation/ACH, 1/hr	Volume, m³	ROH ACH, hr-1	Location During Wait Period <sup>b</sup>	By- stander Location
					Bru	ish-on Exposure Scenarios in Wo	orkshop					
1	Central	0.53 (central)	10 coffee			<ul> <li>Four segments for coffee table (<i>i.e.</i>, two 5-ft<sup>2</sup></li> </ul>				0.45	ROH	
2	Upper-end for user <sup>c</sup>		table (central)			segments with repeat application) and eight				(central)	Workshop	
3	Upper-end for user and bystander <sup>c</sup>	0.88 (upper- end)	25 chest of drawers (upper- end)	90	0.33	<ul> <li>segments for chest of drawers (<i>i.e.</i>, four 6.25-ft<sup>2</sup> segments with repeat application)</li> <li>2-minute application, 15-minute wait, and 4- minute scrape per segment</li> <li>No overlapping activities</li> <li>Scrapings removed from house after last scraping</li> </ul>	54 (central)	Open windows/ 1.26 (professional judgment, 90 <sup>th</sup> percentile)	492 (central)	0.18 (low- end)	ROH	ROH (entire time)

		DCM Released				Room of Use		House		User		
Scenario ID	Conc. Characte- rization	Weight Fraction	Surface Area Treated <sup>a</sup> , ft <sup>2</sup>	Application Rate, g/ft <sup>2</sup>	Release Fraction	Stripping Method	Volume, m³	Ventilation/ACH, 1/hr	Volume, m³	ROH ACH, hr-1	Location During Wait Period <sup>b</sup>	By- stander Location
			•		Spi	ay-on Exposure Scenarios in Wo	rkshop	•		•		•
4	Central	0.80 (central)	10 coffee			<ul> <li>Four segments for coffee table (<i>i.e.</i>, two 5-ft<sup>2</sup></li> </ul>				0.45	ROH	
5	Upper-end for user <sup>c</sup>		table (central)			segments with repeat application) and eight				(central)	Workshop	
6	Upper-end for user and bystander <sup>c</sup>	0.87 (upper- end)	25 chest of drawers (upper- end)	68	0.66	<ul> <li>segments for chest of drawers (<i>i.e.</i>, four 6.25-ft<sup>2</sup> segments with repeat application)</li> <li>1-minute application, 15-minute wait, and 4- minute scrape per segment</li> <li>No overlapping activities</li> <li>Scrapings removed from house after last scraping</li> </ul>	54 (central)	Open windows/ 1.26 (professional judgment, 90 <sup>th</sup> percentile)	492 (central)	0.18 (low- end)	ROH	ROH (entire time)

Scenario ID	Conc. Characte- rization	DCM Released				Room of Use		House		User		
		Weight Fraction	Surface Area Treated <sup>a</sup> , ft <sup>2</sup>	Application Rate, g/ft <sup>2</sup>	Release Fraction	Stripping Method	Volume, m <sup>3</sup>	Ventilation/ACH, 1/hr	Volume, m³	ROH ACH, hr <sup>-1</sup>	Location During Wait Period <sup>b</sup>	By- stande Locatio
					Br	ush-on Exposure Scenario in Bat	hroom					
7	Simulation for bystander exposure	0.88 (upper- end)	36 bathtub (upper- end)	13.25	0.33	<ul> <li>Eight segments (<i>i.e.</i>, four 9 ft<sup>2</sup> segments with repeat application)</li> <li>3-minute application, 15-minute wait, and 6- minute scrape per segment</li> <li>No overlapping activities</li> <li>Scrapings removed from house after last scraping</li> </ul>	9 (low- end) <sup>d</sup>	Window closed, no exhaust fan/ 0.18 ° (low-end)	492 (central)	0.18 (low- end)	ROH	ROH (entire time)
of pain end sur <sup>b</sup> For all s <sup>c</sup> Changes concent <sup>d</sup> 1 m <sup>3</sup> for	t stripper used face area of 2 scenarios, the s in both chem rations for Sco r the vicinity o	d (50 <sup>th</sup> perc 25 ft <sup>2</sup> ). user is in th nical mass a enarios 3 ar of the tub (s	entile value ne treatment nd ACH para nd 6 are highe ource cloud	of 32 ounces room during meters are me er than those ) and 8 m <sup>3</sup> for	or 1,000 g f the applica ore influent for Scenario the rest of	of product applied (in grams) or the central surface area of tion and scraping times and i al than changes in only user lo s 2 and 5, respectively. the bathroom. ) for an extended period, a th	10 ft <sup>2</sup> and ^ n ROH after ocation from	'80 <sup>th</sup> percentile va the last scraping. workshop to the	lue of 80 or	unces or 2,5 nouse. Cons	i00 g for the equently, the	upper- user

the DCM concentrations in the vicinity of the tub; this is a virtual zone, with no physical boundaries. The airflow rate between the cloud and the rest of the bathroom was based on work by <u>Matthews et al. (1989)</u>(for more information, see discussion in Appendix H, H.3. *Inhalation Exposure Scenario Inputs* (Airflow Rates and Volumes).

**Abbreviations:** ROH= room of use; ACH= air exchange rate

The primary distinctions among the seven scenarios were as follows: type of application (*i.e.*, brush *vs.* spray); location of product application (*i.e.*, workshop for most scenarios, bathroom for one scenario); and values used for other inputs including the DCM mass emitted, the user's location during the effect or wait period, and the air exchange rate of the rest of the house (ROH) with outdoor air. The sensitivity analysis indicated that these latter three inputs were the most sensitive variables in the modeling within application type.

Central-tendency or upper-end input parameters were used when building the exposure scenarios. Central-tendency values<sup>10</sup> are exposure values expected to be near the average or median for the range of exposure values. On the other hand, upper–end values<sup>11</sup> are plausible exposure values from the upper half of the range of expected exposure amounts. Of the scenarios listed in Tables 3-5 and 3-6, two are considered central tendency for both the user and the bystander, four had combinations of inputs to estimate upper-end concentrations for the user, and two of the latter also had input combinations to estimate upper-end concentrations for the bystander.

EPA/OPPT developed the seventh scenario to simulate the actual reported conditions from a Centers for Disease Control and Prevention (CDC)/NIOSH occupational exposure case for a DCM paint stripper used on a bathtub (CDC, 2012; Chester et al., 2012). In this case, the user died after using a DCM-based paint strippers in a confined (*i.e.*, closed, poorly ventilated) bathroom. Thus, the purpose of including this latter scenario was to estimate the DCM air concentrations to residential occupants outside the use zone (i.e., bystanders) under conditions of high product use in the room of use. The selected parameter values for scenario 7 (*e.g.*, large surface area, small room size, minimal ventilation, upper-end weight fraction, and low ROH ventilation) would increase concentrations and exposures so that the combinations of parameter values would be expected to result in upper-end to bounding concentrations for the user and residential bystander.

Further details of the exposure scenario inputs are discussed in Appendix H, section H-3 (*Inhalation Exposure Scenario Inputs*).

<sup>&</sup>lt;sup>10</sup> As noted in Section 2.3.1 (Individual Risk) of the EPA (1992b) exposure assessment guidelines, "Individual risk descriptors will generally require the assessor to make estimates of high-end exposure, and sometimes additional estimates (e.g., estimates of central tendency such as average or median exposure)." For this assessment, scenarios with central parameter values refer to a set of inputs that are expected to result in a central (*i.e.*, near the median) estimate of individual exposure.

<sup>&</sup>lt;sup>11</sup> As also noted in Section 2.3.1 of the <u>EPA (1992b)</u> exposure assessment guidelines, "a high end exposure estimate is a plausible estimate of the individual exposure for those persons at the upper end of an exposure distribution. The intent of this designation is to convey an estimate of exposures in the upper range of the distribution, but to avoid estimates that are beyond the true distribution; these latter estimates are called "bounding." Conceptually, the high end of the distribution means above the 90<sup>th</sup> percentile of the population distribution, but not higher than the individual in the population who has the highest exposure." For this assessment, scenarios labeled "upper-end" were modeled by selecting low- and high-end values for sensitive parameters. An "upper-end" exposure estimate is above central tendency and may include the high end of the exposure distribution.

# **3.2.5 Consumer Model Results**

Table 3-7 provides the scenario-specific DCM air concentrations for the consumer user of DCMcontaining paint strippers and residential bystanders. These concentrations were calculated by computing running averages and selecting the maximum of these averages. For example, for the 1-hr averaging period, the 1-hr average concentration was calculated for each one-minute start time during the 24-hr period (*e.g.*, zero to 60, one to 61, and *etc.*), for which the maximum of these averages is reported in Table 3-7. As the averaging time increases, the user to bystander exposure ratio decreases. For example, the ratio of user to bystander maximum oneminute concentration is ~5:1 for scenario 1, whereas the ratio is ~1.5:1 for the 24-hr user and bystander TWA values.

Appendix H provides additional information on various aspects of the model output, such as the following:

- Mathematical description of the calculations (section H-4, *Inhalation Model Outputs and Exposure Calculations*)
- Comparison of results resulting from the MCCEM modeling and the Lawrence Berkley Laboratory (LBL) study monitoring data (section H-5, *Comparison of Modeling-based and Monitoring-based Exposure Estimates*)
- Scenario summaries for each of the modeled scenarios, including both model inputs and results (section H-6, *MCCEM Inhalation Modeling Scenario Summaries*)

		Maximum Values for Averaging Period, mg/m <sup>3</sup> (ppm)								
Scenario	Individual <sup>a</sup>	1 Minute	10 Minutes	30 Minutes	1 Hour	4 Hours	8 Hours	24 Hours		
1. Brush application in workshop,	User	630 (180)	380 (110)	270 (78)	220 (64)	120 (35)	69 (20)	23 (6.7)		
central parameter estimates	Bystander	130 (38)	130 (38)	130 (37)	120 (36)	82 (24)	49 (14)	17 (4.8)		
2. Brush application in workshop,	User	1,300 (370)	1,300 (360)	1,100 (330)	1,100 (300)	420 (120)	220 (64)	75 (22)		
upper-end user estimates <sup>b</sup>	Bystander	220 (63)	220 (63)	220 (62)	210 (59)	140 (39)	82 (24)	28 (8.0)		
3. Brush application in workshop, upper-end user and bystander	User	1,800 (520)	1,200 (340)	900 (260)	760 (220)	560 (160)	400 (120)	160 (45)		
estimates <sup>b</sup>	Bystander	470 (140)	470 (140)	470 (140)	460 (130)	380 (110)	290 (83)	120 (34)		
4. Spray application in workshop,	User	1,500 (430)	780 (220)	600 (170)	490 (140)	270 (77)	150 (44)	52 (15)		
central parameter estimates	Bystander	300 (87)	300 (87)	300 (86)	280 (82)	190 (54)	110 (32)	38 (11)		
5. Spray application in workshop,	User	2,000 (570)	1,900 (550)	1,800 (510)	1,600 (460)	620 (180)	330 (96)	110 (32)		
upper-end user estimates <sup>b</sup>	Bystander	330 (95)	330 (95)	320 (93)	310 (89)	200 (59)	120 (35)	42 (12)		
6. Spray application in workshop, upper-end user and bystander	User	2,800 (810)	1,600 (470)	1,300 (360)	1,100 (320)	810 (230)	580 (170)	230 (66)		
estimates <sup>b</sup>	Bystander	710 (210)	710 (210)	710 (200)	700 (200)	580 (170)	430 (120)	180 (51)		
7. Brush application in bathroom,	User	2,428 (699)	1,455 (419)	887 (255)	799 (230)	536 (154)	340 (98)	135 (39)		
simulation	Bystander	224 (64)	224 (64)	222 (64)	218 (63)	187 (54)	150 (43)	70 (20)		

Notes:

 <sup>a</sup> The bystander was assumed to be in Rest-of-House (ROH).
 <sup>b</sup> Changes in both chemical mass and air changes per hour (ACH) parameters are more influential than changes in only user location from workshop to the rest of the house. Consequently, the user concentrations for Scenarios 3 and 6 are higher than those for Scenarios 2 and 5, respectively.

# 3.3 HAZARD/DOSE-RESPONSE ASSESSMENT

## **3.3.1 Approach and Methodology**

#### 3.3.1.1 Selection of Peer-Reviewed Hazard/Dose-Response Assessments as the Source Documents for the DCM TSCA Assessment

EPA/OPPT's work plan risk assessment for DCM is primarily based on the peer-reviewed hazard and dose-response information<sup>12</sup> published in the following reports:

- *Toxicological Review of Methylene Chloride* published in 2011 by EPA's Integrated Risk Information System (IRIS) (<u>EPA, 2011c</u>);
- Spacecraft Maximum Allowable Concentrations (SMAC) for Selected Airborne Contaminants: Methylene chloride (Volume 2) published by the U.S. National Academies (NRC, 1996);
- Acute Reference Exposure Level (REL) and Toxicity Summary for Methylene Chloride published by the Office of Environmental Health Hazard Assessment (<u>OEHHA, 2008</u>);
- Interim Acute Exposure Guideline Levels (AEGL) for Methylene Chloride developed by the U.S. National Advisory Committee on AEGLs (NAC, 2008).

To a lesser extent, the *Toxicological Profile for Methylene Chloride* published by the Agency for Toxic Substances and Disease Registry (ATSDR) was consulted for hazard information (<u>ATSDR</u>, <u>2000</u>, <u>2010</u>).

EPA/OPPT used the DCM IRIS assessment as the principal data source for chronic toxicity hazard and dose-response information. The DCM IRIS assessment used a weight-of-evidence approach, the latest scientific information and physiologically-based pharmacokinetic (PBPK) modeling to develop hazard and dose-response assessments for carcinogenic and non-carcinogenic health effects resulting from lifetime exposure to DCM.

The DCM IRIS assessment followed the principles set forth by the various risk assessment guidelines issued by the National Research Council (NRC) and EPA. Primary, peer-reviewed literature identified through September 2011 was systematically reviewed and included where that literature was determined to be critical to the assessment (<u>EPA, 2011c</u>).

In addition, EPA/OPPT used the SMAC, the California acute REL and AEGL technical support documents as the data source for acute toxicity hazard and dose-response information. SMACs and the California acute REL for DCM are derived following the *Guidelines for Developing* 

<sup>&</sup>lt;sup>12</sup> EPA/OPPT uses the hazard values (i.e., points of departure) and, in most cases, the same uncertainty factors that were used to derive the SMAC, acute REL and AEGLs and EPA's IRIS cancer/non-cancer values for chronic exposures to DCM. Since EPA/OPPT is using margin of exposures (MOEs) to estimate risk, our approach does not use the derived human health guidelines (e.g., RfC, SMAC, acute ERL and AEGLs) for risk estimation. See sections 3.3.1.2 and 3.3.1.3 for more details.

Spacecraft Maximum Allowable Concentrations for Space Station Contaminants (<u>NRC, 1992</u>) and California's Air Toxics Hot Spots Program risk assessment guidelines for acute RELs (<u>OEHHA, 1999</u>), respectively. AEGLs are developed based on the criteria discussed in the Standing Operating Procedures (SOP) for Developing Acute Exposure Guideline Levels for Hazardous Chemicals (<u>NRC, 2001</u>).

Appendix I provides additional information about the information considered in the development of the DCM IRIS (section I-1), AEGL (section I-2), SMAC (section I-3) and the California acute REL (section I-4) toxicology assessments.

#### 3.3.1.2 Chronic Hazard and Dose-Response Assessment: EPA IRIS Toxicological Review of Methylene Chloride

EPA/OPPT used the DCM cancer and non-cancer hazard/dose-response assessments published by the EPA's IRIS program to estimate chronic risks for the occupational scenarios. A summary of the approach and methodology is provided in sections 3.3.1.2.1 (*Carcinogenic Effects*) and 3.3.1.2.2 (*Non-Cancer Effects*).

### 3.3.1.2.1 Carcinogenic Effects Following Chronic Exposure to DCM

DCM is likely to be carcinogenic in humans by a mutagenic mode of action (<u>EPA, 2011c</u>). The EPA IRIS cancer dose-response analysis used linear low-dose extrapolation to derive an inhalation unit risk (IUR) of  $4 \times 10^{-5}$  per ppm ( $1 \times 10^{-5}$  per mg/m<sup>3</sup>; assuming a 70-year human lifetime)<sup>13</sup>. The IUR was used in the EPA/OPPT risk assessment to estimate excess cancer risks for the inhalation occupational exposures scenarios.

The IUR for DCM was derived from mouse liver and lung tumor incidence data (<u>Mennear et al.,</u> <u>1988</u>; <u>NTP, 1986</u>). The IUR is defined as the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1  $\mu$ g/m<sup>3</sup> in air (<u>EPA</u>, <u>2011c</u>). There is high confidence in the IUR because it was based on the best available dose-response data for liver and lung cancer in mice (<u>EPA</u>, <u>2011c</u>). Moreover, the weight of evidence from multiple *in vivo* and *in vitro* studies supported the mutagenicity of DCM and the key role of glutathione S-transferase (GST) metabolism and the formation of DNA-reactive GST-pathway metabolites (<u>EPA</u>, <u>2011c</u>). Appendix J contains more information on how the cancer IUR was developed for DCM. Table 3-8 lists the cancer dose-response information that EPA/OPPT used in the work plan risk assessment for DCM.

EPA/OPPT decided not to use the IUR to calculate the theoretical cancer risk associated with a single (acute) exposure to paint strippers containing DCM. <u>NRC (2001)</u> published methodology for extrapolating cancer risks from chronic to short-term exposures to mutagenic carcinogens.

<sup>&</sup>lt;sup>13</sup> The inhalation unit risk for dichloromethane should not be used with exposures exceeding the point of departure (BMDL<sub>10</sub> = 7,700 mg/m<sup>3</sup> or 2,200 ppm), because above this level the fitted dose-response model does not characterize what is known about the carcinogenicity of DCM.

These methods were published with the caveat that extrapolation of lifetime theoretical excess cancer risks to single exposures has great uncertainties.

As <u>NRC (2001)</u> explains, "There are no adopted state or federal regulatory methodologies for deriving short-term exposure standards for workplace or ambient air based on carcinogenic risk, because nearly all carcinogenicity studies in animals and retrospective epidemiologic studies have entailed high-dose, long-term exposures. As a result, there is uncertainty regarding the extrapolation from continuous lifetime studies in animals to the case of once-in-a-lifetime human exposures. This is particularly problematical, because the specific biologic mechanisms at the molecular, cellular, and tissue levels leading to cancer are often exceedingly diverse, complex, or not known. It is also possible that the mechanisms of injury of brief, high-dose exposures will often differ from those following long-term exposures. To date, U.S. federal regulatory agencies have not established regulatory standards based on, or applicable to, less than lifetime exposures to carcinogenic substances (NRC, 2001)." Thus, the final EPA/OPPT work plan risk assessment for DCM does not estimate excess cancer risks for acute exposures because the relationship between a single short-term exposure to DCM and the induction of cancer has not been established in the current scientific literature.

#### 3.3.1.2.2 Non-Cancer Effects Following Chronic Exposure to DCM

The EPA IRIS non-cancer dose-response assessment calculated a hazard value of 17.2 mg/m<sup>3</sup> (4.8 ppm) for chronic DCM inhalation exposures (<u>EPA, 2011c</u>). The hazard value was estimated by PBPK modeling and expressed as the 1<sup>st</sup> percentile of the distribution of human equivalent concentrations (HEC) i.e. the HEC<sub>99</sub> the concentration at which there is 99% likelihood an individual would have an internal dose less than or equal to the internal dose of hazard was used to protect toxicokinetically sensitive individuals. EPA/OPPT used the PBPK-derived HEC as the non-cancer hazard value for the occupational risk calculations.

The derivation of the non-cancer hazard value was based on the hepatic effects reported in a 2-year rat study. Specifically, female rats reported liver lesions (i.e., hepatic vacuolation) following exposure to 500 ppm DCM for 6 hrs/day, 5 days/week for 2 years (<u>Nitschke et al., 1988a</u>). The rat data were suitable for non-cancer dose-response analysis in the DCM IRIS assessment. The animal-to-human extrapolation was conducted by PBPK modeling, coupled with benchmark dose<sup>14</sup> estimation. The DCM IRIS assessment chose the 1<sup>st</sup> percentile HEC i.e. the HEC<sub>99</sub> the concentration at which there is 99% likelihood an individual would have an internal dose less than or equal to the internal dose of hazard of 17.2 mg/m<sup>3</sup> as the point of departure (POD)<sup>15</sup> for the non-cancer dose-response assessment because it would protect toxicokinetically sensitive individuals. Appendix J contains more information on how the non-cancer PBPK-derived HEC was developed.

<sup>&</sup>lt;sup>14</sup> The benchmark dose (BMD) is a dose or concentration that produces a predetermined change in response rate of an adverse effect (called the benchmark response or BMR) compared to background (<u>EPA, 2011c</u>).

<sup>&</sup>lt;sup>15</sup> A point of departure (POD) is a dose or concentration that can be considered to be in the range of observed responses, without significant extrapolation. A POD is used to mark the beginning of extrapolation to determine risk associated with lower environmentally relevant human exposures (EPA, 2011b).

There is high confidence in the key study supporting the non-cancer hazard value. <u>Nitschke et al. (1988a)</u> is a well-conducted, peer-reviewed study that used three dose groups plus a control. In addition, the inhalation database contains several studies consistently identifying the liver as the most sensitive non-cancer target organ in rats (<u>EPA, 2011c</u>).

EPA/OPPT used the same endpoint and study-specific uncertainty factors (UFs) that the EPA IRIS program applied to the PBPK-derived HEC to interpret the non-cancer risk estimates (i.e., margin of exposure, MOE<sup>16</sup>) for workers. EPA/OPPT did not use a database uncertainty factor for the benchmark MOE for specific endpoints. This uncertainty in the database is discussed qualitatively in the risk characterization.

A total UF of 10 was used as the benchmark MOE and was allocated as follows:

- interspecies UF (UF<sub>A</sub>) of 3 to account for toxicodynamic differences between animals and humans,
- intraspecies UF (UF<sub>H</sub>) of 3 to account for toxicodynamic differences within humans

Table 3-8 lists the cancer and non-cancer dose-response information that EPA/OPPT used in this assessment to evaluate risks associated with chronic exposures to DCM.

<sup>&</sup>lt;sup>16</sup> Margin of Exposure (MOE) = (Non-cancer hazard value, POD) ÷ (Human Exposure). The benchmark MOE is used to interpret the MOEs and consists of the UFs for interspecies and intraspecies uncertainty set by the IRIS program. Refer to section 3.4 for more information about the MOE calculations.

Effects Category	Target Organ/ System	Species	Route of Exposure and Exposure Concentrations <sup>1</sup>	Duration	РОД Туре	Effect	Uncertainty Factors (UFs) for Benchmark MOE <sup>2</sup>	Hazard Value Used in Chronic Risk Assessment	Additional Information <sup>3</sup>	Reference
CANCER	Liver and lung	Mouse (male)	Inhalation 0 ppm, 2,000 ppm, 4,000 ppm	6 hrs/day, 5 days/week for 2 years, beginning at 7-8 weeks of age	<u>Male liver tumors:</u> Mouse internal BMD <sub>10</sub> and BMDL <sub>10</sub> = 913.9 and 544.4 mg DCM metabolized via GST pathway/liter tissue/day, respectively <u>Male lung tumors:</u> Mouse internal BMD <sub>10</sub> and BMDL <sub>10</sub> = 61.7 and 48.6 mg DCM metabolized via GST pathway/liter tissue/day, respectively	Liver and lung tumors	Not applicable	Inhalation Unit Risk (IUR): 4 x 10 <sup>-5</sup> per ppm (1 x 10 <sup>-5</sup> per mg/m <sup>3</sup> )	Internal dose BMDL <sub>10</sub> values for each type of tumor were converted into an IUR that combined both types of tumors.	<u>Mennear</u> <u>et al.</u> (1988) <u>NTP</u> (1986)
NON-CANCER	Liver	Rat (female)	Inhalation 0 ppm, 50 ppm, 200 ppm, 500 ppm	6 hrs/day, 5 days/week for 2 years	Rat internal BMDL <sub>10</sub> = 531.82 mg DCM metabolized via cytochrome P450 (CYP) pathway/liter liver tissue/day	Hepatic effects (vacuol ation)	UF <sub>A</sub> = 3; UF <sub>H</sub> =3; Total UF=10	1 <sup>st</sup> percentile human equivalent concentratio n (HEC) i.e. the HEC <sub>99</sub> : 17.2 mg/m <sup>3</sup> (4.8 ppm)	Allometric scaling and probabilistic modeling were used to calculate the hazard value (i.e., HEC99) from the rat internal BMDL10.	<u>Nitschke</u> <u>et al.</u> (1988a)

Margin of Exposure (MOE) = (Non-cancer hazard value)  $\div$  (Human Exposure). The benchmark MOE is used to interpret the MOEs and intraspecies (UF<sub>H</sub>) uncertainty factors. UF values were those used in the DCM IRIS assessment (EPA, 2011c).

<sup>3</sup> for further information see Appendix J

#### 3.3.1.3 Acute Hazard and Dose-Response Assessment

Workers and consumers can be exposed to a single (acute) exposure to DCM when handling DCM-containing paint strippers. In this assessment, non-cancer risks following acute exposures to DCM were assessed using the dose-response information (i.e., PODs) supporting the derivations of the *Spacecraft Maximum Allowable Concentrations* (SMACs)(NRC, 1996) and the *Acute Exposure Guideline Levels* (AEGLs)(NAC, 2008). The assessment also evaluated acute risks with the POD from the California acute reference exposure level (REL)(<u>OEHHA, 2008</u>), but the SMAC POD was preferred over the REL POD for reasons explained in Sections 3.3.1.3.1 (*SMACs*) and 3.3.1.3.2 (*California's Acute REL*). Although AEGLs are intended for emergency response activities, the AEGL PODs were used in this assessment to evaluate acute risks associated with discomfort/non-disabling (AEGL-1) and incapacitating (AEGL-2) effects following DCM exposure from the use of paint strippers.

EPA/OPPT assumed that consumers would not generally perform paint stripping jobs on a regular basis in their residences allowing sufficient time between exposures to clear DCM and its metabolites from the body. This assumption was supported by DCM's short plasma half-life (~40 min) (DiVincenzo et al., 1972). Evaluation of acute risks in occupational scenarios is appropriate based on the assumption that some workers could be rotating tasks and not necessarily using DCM-based paint strippers on a daily basis. This type of exposure would allow the worker to clear DCM and its metabolites before the next encounter with the DCM-containing paint stripper.

The consumer exposure modeling indicated that virtually all of the DCM release occurs within 2 hrs after product application for both spray and brush paint strippers. This is very shortly after the last scraping is finished due to DCM's relatively high volatility (Appendix H, section H-1-1-4). After the peak concentration is reached, the modeling showed that the concentration decline is due almost exclusively to ventilation rather than to declining emissions. EPA/OPPT used these observations as the basis to select acute hazard values (i.e., SMAC and AEGL PODs) applicable to 1-hr exposures for consumer scenarios.

In contrast, for occupational scenarios, the California REL POD was time scaled to 8 hrs to compare the hazard value to the 8-hr air concentration estimated from the monitoring data. This assumed that the worker would be performing paint stripping activities during the entire 8-hr work shift. The 8-hr AEGL-2 was used to evaluate whether the 8-hr occupational exposures estimates exceeded the threshold for disability. However, comparisons of consumer exposure estimates with AEGLs incorporated AEGL PODs for shorter or longer time durations (i.e., 10-min, 30-min, 4-hr and 8-hr) in addition to the 1-hr POD to evaluate a wider concentration-time response.

Sections 3.3.1.3.1 (*SMACs*), 3.3.1.3.2 (*AEGLs*), and 3.3.1.3.3 (*California's Acute REL*) summarize the approach and methodology used in the acute inhalation risk assessment. Appendix K provides additional information about the definitions of the SMAC, AEGL and the California acute REL values and how their respective PODs were derived.

## 3.3.1.3.1 SMACs

SMACs are developed by the U.S. National Academies (NAS) to provide guidance on chemical exposures that may occur during normal operations of spacecraft as well as emergency situations (NRC, 1996). EPA/OPPT used the SMAC's dose-response assessment as the starting point for deriving acute air concentrations for residential users of DCM-based paint strippers, as well as other residential occupants that may be indirectly exposed (e.g., children, adults, the elderly).

The DCM acute risk assessment used the acute POD of 350 mg/m<sup>3</sup> (100 ppm) supporting the derivation of the 1-hr SMAC. The POD was considered the NOAEL<sup>17</sup> for central nervous system (CNS) effects associated with the formation of 3% carboxyhemoglobin (COHb) in human blood based on various human studies (<u>Andersen et al., 1991</u>; <u>Astrand et al., 1975</u>; <u>DiVincenzo and Kaplan, 1981</u>; <u>Peterson, 1978</u>; <u>Putz et al., 1979</u>; <u>Ratney et al., 1974</u>; <u>Stewart et al., 1972</u>).

The 1-hr SMAC POD derivation relied on COHb levels in human blood as an indicator of CNS depression since the metabolism of DCM produces carbon monoxide (CO) and carbon dioxide (CO<sub>2</sub>). Furthermore, there are extensive studies about the relationship between COHb blood levels and human health adverse effects, primarily CNS effects. Thus, EPA/OPPT preferred the 1-hr SMAC POD over the 1-hr California acute REL (*section 3.3.1.3.2*) as the health protective hazard value used to estimate acute risks for the consumer scenarios. The SMAC POD was based on multiple human observations reporting increased COHb levels after DCM exposure, coupled with the knowledge of what would be considered a NOAEL COHB level based on the extensive CO database (NRC, 1996). However, the California acute REL POD was used to estimate risks for occupational scenarios since an 8-hr SMAC POD was not available for the risk calculations.

The SMAC assessment did not adjust the 1-hr POD with UFs as the intended audience for the values is healthy astronauts. However, EPA/OPPT used a total UF of 10 as the benchmark MOE when interpreting the MOE risk estimates. The total UF took into account a 10-fold factor for variability within the human population based on the following reasons:

an evaluation of the COHb data for different human subpopulations supports the approach of retaining the default intraspecies UF of 10 under the premise that a level of 3% COHb is considered protective of neurotoxic effects in most individuals (*e.g.*, healthy individuals, children), but may not be protective enough for patients with coronary artery disease and the fetus (NRC, 2010). At COHb levels of 2 or 4%, patients with coronary artery disease may experience a reduced time until onset of angina (chest pain) during physical exertion (Allred et al., 1989a; Allred et al., 1989b, 1991). Other studies have also confirmed a reduced time to onset of exercise-induced chest pain at a COHb between 2.5 and 4.5 percent (Anderson et al., 1973; Aronow et al., 1972; Kleinman et al., 1989; Kleinman et al., 1998; Sheps et al., 1987). Fetuses are at higher risk for CO toxicity because of higher CO affinity and slower CO

<sup>&</sup>lt;sup>17</sup> NOAEL= No-observed-adverse-effect level

elimination (<u>NRC, 2010</u>). There are no studies reporting effects on the unborn after a single acute exposure resulting in lower COHb levels (<u>EPA, 2000a</u>; <u>NRC, 2010</u>);

- adult workers and consumers of both sexes are expected to be the users of DCM-based paint strippers, whereas residential bystanders (i.e., individuals of any age) are expected to be indirectly exposed to DCM; and
- no need to apply an interspecies UF for animal-to-human extrapolation because human data were used to support the 1-hr SMAC POD.

Appendix K contains more information on the derivation of the 1-hr SMAC POD. Table 3-9 lists the derivation information for the SMAC POD used in this assessment

## 3.3.1.3.2 California's Acute REL

Acute RELs are developed by the Office of Environmental Health Hazard Assessment (OEHHA) from the State of California. The acute REL is defined as the concentration level at or below which no adverse health effects are anticipated (*i.e.*, one or eight hrs) in a human population, including sensitive subgroups, exposed on an intermittent basis (OEHHA, 1999).

As an alternative approach to estimate acute inhalation risks, this assessment also considered the POD of 840 mg/m<sup>3</sup> (240 ppm) supporting the derivation of the 1-hr acute REL. The POD was considered the LOAEL<sup>18</sup> for subtle impairment of the nervous system function in humans based on human volunteers exposed to 195 ppm DCM (696 mg/m<sup>3</sup>) for 1.5 hrs (Putz et al., 1979). The 1.5-hr exposure concentration was then time-scaled to obtain the 1- or 8-hrs PODs of 840 mg/m<sup>3</sup> (240 ppm) and 290 mg/m<sup>3</sup> (80 ppm), respectively. As discussed in Section 3.3.1.3.1, EPA/OPPT preferred the 1-hr SMAC POD to estimate acute risks because the hazard value was based on multiple human observations reporting increased COHb levels after DCM exposure, coupled with the knowledge of what would be considered a NOAEL COHB level based on the extensive CO database (NRC, 1996).

EPA/OPPT used a total UF of 60 as the benchmark MOE when interpreting the MOE risk estimates based on the acute REL POD. The total UF consisted of an intraspecies UF of 10 to account for human variability and a LOAEL-to-NOAEL UF<sup>19</sup> of 6 (<u>OEHHA, 2008</u>).

Appendix K contains more information on the derivation of the 1-hr REL POD. Table 3-9 lists the derivation information for the REL POD used in this assessment.

<sup>&</sup>lt;sup>18</sup> LOAEL= Lowest-observed-adverse-effect level

<sup>&</sup>lt;sup>19</sup> The acute REL documentation does not provide the basis for the selection of a LOAEL-to-NOAEL UF of 6.

### 3.3.1.3.3 AEGLs

AEGLs are emergency response values designed for once-in-a-lifetime exposures to airborne chemicals. AEGL values are threshold levels developed for three different health effect end point tiers (discomfort/non-disabling effects = AEGL-1 threshold; disability = AEGL-2 threshold; and death = AEGL-3 threshold) and different durations of exposure (10 min; 30 min; 1 hr; 4 hrs; and 8 hrs) within the constraints of available data.

An AEGL threshold represents an estimated point of transition between one defined set of symptoms or adverse effects in one tier and another defined set of symptoms or adverse effects in the next tier (NRC, 2001). This concept is reflected in the definition of AEGLs which describe AEGLs as maximum airborne concentrations above which there is an increasing likelihood of the adverse effects associated with the respective AEGL tiers. In other words, AEGL-2 and -3 values are not safe and are in the range where some human response may be anticipated. The AEGL values are intended to protect the general public, including susceptible individuals such as infants, children, the elderly, persons with asthma, and those with other illnesses in the context of emergency-related chemical releases, and not consumer exposures (NRC, 2001).

Recent reports have documented human fatalities among bathtub refinishers using DCM-based paint stripping products (CDC, 2012; Chester et al., 2012). Such real-life situations support our current risk approach of evaluating how far the acute consumer and occupational exposure are from the thresholds for discomfort/non-disabling effects (AEGL-1) and disability (AEGL-2). EPA/OPPT used these comparisons to provide an indicator of whether the exposure estimates would be expected to produce human adverse effects following DCM exposure. Please note that the comparisons to the AEGL-3 PODs were not included in this assessment as none of the DCM air concentrations for the occupational and consumer scenarios exceeded the AEGL-3 POD threshold for lethal effects. However, a summary of the AEGL-3 POD derivations is included in Appendix K for reference.

The scientific literature supports two relevant toxicity endpoints for acute exposures to DCM: (1) CNS depression related to the brain concentration of DCM itself; and (2) COHb formation in the blood (<u>NRC, 2008</u>). Taking this into consideration, PBPK modeling was used to calculate AEGL PODs based on DCM concentrations in brain and peak COHb in blood. CNS effects drove the setting of AEGL values for the shorter exposure durations, whereas formation of COHb determined the AEGL values for longer exposure durations. This is consistent with the observations that CNS effects occur soon after the onset of exposure, while peak levels of COHb in blood can be reached hours later after cessation of exposure. Also the metabolic pathway leading to the formation of carbon monoxide is saturable around 500 ppm (<u>NAC, 2008</u>).

Table 3-9 describes the AEGL-1 and -2 PODs that EPA/OPPT used in the acute risk assessment. It also summarizes derivation information for the AEGL PODs with more detailed information found in Appendix K.

Table 3 9.	Non Cano Paint St		rd Values	Used in th	e Risk Evaluation	of Acute Expos	ures to Work	ers and Cons	umers Using DCM	Based
Reference Value	Target Organ/ System	Species	Route of Exposure and Exposure Concen- trations <sup>1</sup>	Duration	POD Type	Effect	Hazard Value Used in Acute Risk Assessment	Uncertainty Factors (UFs) for Benchmark MOE <sup>2,3</sup>	Additional Information	Reference
SMAC	Central Nervous System	Human	Inhalation COHb data from several sources	1 hr	NOAEL = 100 ppm (350 mg/m <sup>3</sup> ) supported by various human studies	CNS depression related to formation of 3% COHb in blood	100 ppm (350 mg/m <sup>3</sup> )	UFs were not applied to the 1-hr SMAC. However, EPA/OPPT applied a $UF_{H}=10$ in the acute risk assessment to account for human variability.	NOAELs for CNS depression have not been reported for DCM exposures. A linear regression analysis estimated the DCM concentration (350 mg/m <sup>3</sup> ) that produces ~3 percent COHb concentration in blood (NOAEL)	<u>NRC</u> (1996, 2008)
California Acute REL	Central Nervous System	Human	Inhalation 195 ppm (696 mg/m <sup>3</sup> )	90 min (1.5 hrs)	LOAEL = 195 ppm (696 mg/m <sup>3</sup> ) at 90 minutes (Putz <i>et al.,</i> 1979) or 240 ppm (840 mg/m <sup>3</sup> ) when time adjusted to a 60-min exposure	Impaired performance on dual-task and auditory vigilance tests in humans	1-hr REL POD= 240 ppm (840 mg/m <sup>3</sup> ) 8-hr REL POD= 80 ppm (290 mg/m <sup>3</sup> )	UF <sub>H</sub> =10; UF <sub>L</sub> =6; <i>Total UF=60</i>	ten Berge equation (C <sup>n</sup> xt = k, n = 2) was used for time adjustment from 90 to 60 min or 480 min (8 hrs).	<u>OEHHA</u> (2008)
AEGL-1 (threshold for discomfort/ non- disabling effects)	Central Nervous System (Direct effect in brain)	Human	Inhalation 213 to 986 ppm	60 - 120 min (1-2 hrs)	No observed effect for slight CNS effects at 1-hr exposure to 514 ppm (1,840 mg/m <sup>3</sup> ) equivalent to a brain concentration of 0.063 mM.	No effect level for light- headedness, difficulties in enunciation	10-min = 870 ppm (3,000 mg/m <sup>3</sup> ) 4 30-min = 690 ppm (2,400 mg/m <sup>3</sup> ) 4 1-hr = 600 ppm (2,130 mg/m <sup>3</sup> ) 4	UF <sub>H</sub> = 3 Total UF = 3	PBPK model was used. Time scaling was based on maximum DCM concentration in human brain. AEGL PODs for 4-and 8-hr were not calculated since they would be above the corresponding AEGL- 2 values.	<u>NAC</u> (2008)

Reference Value	Target Organ/ System	Species	Route of Exposure and Exposure Concen- trations <sup>1</sup>	Duration	POD Type	Effect	Hazard Value Used in Acute Risk Assessment	Uncertainty Factors (UFs) for Benchmark MOE <sup>2,3</sup>	Additional Information	Reference
AEGL-2 (threshold for disability) <sup>5</sup>	Central Nervous System (Direct effect in brain or COHb formation in blood)	Human	Inhalation <u>CNS</u> <u>effects:</u> 195 to 751 ppm <u>COHb</u> <u>formation:</u> 0, 117 or 253 ppm (0, 420, or 900 mg/m <sup>3</sup> )	<u>CNS</u> effects: Up to 230 min (3.8 hr) <u>COHb</u> formation 50-70 min	<u>CNS effects:</u> DCM concentration in human brain of 0.137 mM equivalent to a 230- min exposure to 751 ppm <u>COHb formation:</u> No observed effect level (NOEL) of 4% COHb	CNS effects: Absence of CNS effects in humans COHb formation: COHb formation in patients with coronary artery disease	10-min= 1,700 ppm (6,000 mg/m <sup>3</sup> ) <sup>6</sup> 30-min= 1,200 ppm (4,200 mg/m <sup>3</sup> ) <sup>6</sup> 1-hr= 560 ppm (2,000 mg/m <sup>3</sup> ) <sup>7</sup> 4-hr= 100 ppm (350 mg/m <sup>3</sup> ) <sup>7</sup> 8-hr= 60 ppm (210 mg/m <sup>3</sup> ) <sup>7</sup>	UF <sub>H</sub> = 1 Total UF = 1	PBPK model was used. Time scaling was based on maximum DCM concentration in human brain (10 and 30 minutes) or on COHb formation (1-, 4-, and 8-hr exposure)	<u>NAC</u> (2008) <u>NRC</u> (2010)

Notes:

<sup>1</sup> Airborne concentration conversion factor for DCM is 3.47 mg/m<sup>3</sup> per ppm <u>NIOSH (2011b)</u>

<sup>2</sup> Margin of Exposure (MOE) = (Non-cancer hazard value, POD) ÷ (Human Exposure). The benchmark MOE is used to interpret the MOEs and consists of UFs.

 $^3$  UF\_H=intraspecies UF; UF\_L=LOAEL-to-NOAEL UF

<sup>4</sup> These are the AEGL PODs without the 3X intraspecies UF adjustment.

<sup>5</sup> PBPK modeling was used to predict both the DCM concentration in brain and COHb levels. The toxic endpoint (CNS effects or COHb formation) changed over the exposure range of 10 min to 8 hrs. CNS effects determined the AEGL values for the shorter exposure durations, whereas formation of COHb determined the AEGL values for longer exposure durations. This is consistent with the observations that CNS effects occur soon after the onset of exposure, while peak levels of COHb in blood can be reached hours later after cessation of exposure. Also the metabolic pathway of carbon monoxide is saturable around 500 ppm (<u>NAC, 2008</u>).

<sup>6</sup> AEGL derivations were driven by CNS effects.

<sup>7</sup> AEGL derivations were driven by COHb formation.

## **3.3.2 Human Health Hazard Summary**

The information presented in this section is not intended to be an exhaustive discussion of DCM's toxicity, but rather a summary of its toxicity *via* the inhalation route of exposure. The section also summarizes the absorption, distribution, metabolism and excretion of DCM. Thus, the reader is referred to the original documents for detailed toxicity data supporting the summary presented in this document.

## 3.3.2.1 Absorption, Distribution, Metabolism and Excretion

DCM is rapidly absorbed through inhalation exposure. The pulmonary uptake of DCM ranges roughly from 40 to 60 percent (<u>Andersen et al., 1991</u>; <u>Gamberale et al., 1975</u>; <u>Stewart et al., 1976</u>), but may be up to 70 percent during the first minutes of exposure (<u>Riley et al., 1966</u>). The uptake decreases with exposure duration and concentration (<u>Peterson, 1978</u>; <u>Stewart et al., 1976</u>), and a steady-state absorption rate is generally achieved within 2 hrs for exposures up to 200 ppm (<u>DiVincenzo and Kaplan, 1981</u>; <u>DiVincenzo et al., 1972</u>).

Animal studies show that following absorption, DCM is rapidly distributed throughout the body, including the liver, brain, and subcutaneous adipose tissue (<u>ATSDR, 2000</u>; <u>Carlsson and</u> <u>Hultengren, 1975</u>; <u>EPA, 2011c</u>). DCM's plasma half-life is estimated to be 40 minutes after inhalation exposure (<u>ATSDR, 2000</u>; <u>DiVincenzo et al., 1972</u>). Metabolism occurs predominantly in the liver, although additional transformation occurs in the lungs and kidneys (<u>ATSDR, 2000</u>).

In the liver, metabolism of DCM involves two primary pathways. The first pathway produces CO and  $CO_2$ , and saturation occurs at very low concentrations of a few hundred ppm. The second pathway yields formaldehyde and formic acid, and saturation occurs at very high concentrations (>10,000 ppm).

Acute toxic effects (*i.e.*, CNS depression) may persist for hours after cessation of exposure because of continued metabolism of DCM released from tissue storage (<u>ATSDR, 1990</u>). COHb levels can continue to increase reaching peak levels as much as 5 to 6 hours after exposure (<u>ATSDR, 2000</u>).

Elimination of DCM is predominantly through the lungs. Unchanged DCM also is found in small amounts in the urine and feces (<u>ATSDR, 2000</u>). At low doses, a large percentage of DCM is transformed into COHb and eliminated as CO, while at higher doses, more of the unchanged parent compound is exhaled (<u>ATSDR, 1990</u>).

DCM has been detected in human breast milk (<u>EPA, 1980</u>; <u>Pellizzari et al., 1982</u>); thus, it is possible that infants could be exposed to DCM through maternal exposures. However, PBPK modeling suggests that lactating females who breast feed their infants will not deliver DCM in quantities significant enough to be harmful (<u>Fisher et al., 1997</u>).

Blood concentrations of DCM were below the level of detection in 1,165 individuals who participated in the recent National Health and Nutrition Examination Survey (NHANES) 2003 to 2004 subsample of the U.S. population (CDC, 2009). DCM was present in the urine of workers employed at a pharmaceutical factory. Urine levels appear to be nearly eliminated during the overnight period after exposure has occurred (HSDB, 2012). Human health effects associated with exposure to low environmental levels of DCM or low levels detected in biomonitoring studies are unknown (CDC, 2009).

## 3.3.2.2 Human and Animal Toxicity Following Acute Exposure to DCM

Acute inhalation exposure of humans to DCM decreases the oxygen availability in the blood by COHb formation. Acute exposure to DCM also results in neurological impairment from the interaction of DCM with membranes in the nervous system (<u>ACGIH, 2001</u>; <u>ATSDR, 2000</u>; <u>Bos et al., 2006</u>; <u>Cherry et al., 1983</u>; <u>Gamberale et al., 1975</u>; <u>Putz et al., 1979</u>; <u>Winneke, 1974</u>).

The organ most often affected in exposures to high levels of DCM is the brain. Effects on lung, liver, or kidney have also been reported in humans as primary signs of DCM toxicity (<u>NAC</u>, <u>2008</u>). In some cases, high COHb levels (*i.e.*, up to 40 percent) are measured without serious complaints. The reported COHb levels could not be linked to effects in a dose-related way in any of the human observations (<u>NAC</u>, <u>2008</u>).

Acute lethality in humans following inhalation exposure is related to CNS depressant effects. These effects include loss of consciousness and respiratory depression, resulting in irreversible coma, hypoxia, and eventual death (<u>NAC, 2008</u>). Especially at exposure to high concentrations in which death occurs within a relatively short time, it is unlikely that the formation of CO will have resulted in life-threatening levels of COHb (<u>NAC, 2008</u>). Only one fatal case was reported to be related to a myocardial infarction (*i.e.,* heart attack) without any signs of reported CNS depression (<u>NAC, 2008</u>).

Acute non-lethal effects in humans are most frequently described as CNS-related only (<u>NAC</u>, <u>2008</u>). Acute exposure to humans results in acute neurobehavioral deficits measured in psychomotor tasks including: tests of hand-eye coordination, visual evoked response changes, and auditory vigilance, which may occur at concentrations >200 ppm with 4–8 hrs of exposure (<u>ACGIH</u>, 2001; <u>ATSDR</u>, 2000; <u>Bos et al.</u>, 2006; <u>Cherry et al.</u>, 1983; <u>Gamberale et al.</u>, 1975; <u>Putz et al.</u>, 1979; <u>Winneke</u>, 1974). In few cases, cardiotoxic effects (*i.e.*, evidenced by electrocardiogram [ECG] changes) were reported in humans (<u>EPA</u>, 2011c).

Neurological evaluations in animals during and after acute inhalation exposure to DCM (*i.e.*, >200 to 1000 ppm for 1 to 8 hrs) have resulted in CNS depressant effects with decreased motor activity, impaired memory, and changes in responses to sensory stimuli (EPA, 2011c). Several neurological mediated parameters, including decreased activity (Heppel and Neal, 1944; Heppel et al., 1944; Kjellstrand et al., 1985; Weinstein et al., 1972), impairment of learning and memory (Alexeeff and Kilgore, 1983), and changes in responses to sensory stimuli (Rebert et al., 1989), were reported from acute and short-term DCM exposure. Evidence of a localized

immunosuppressive effect in the lung resulting from inhalation DCM exposure was seen in CD-1 mice acutely exposed to 100 ppm for 3 hrs (<u>Aranyi et al., 1986</u>).

## 3.3.2.3 Human and Animal Toxicity Following Repeated Exposures to DCM

## 3.3.2.3.1 Non-Cancer Effects

Relatively little is known about the long-term neurological effects of chronic low level DCM exposures in humans, although there are studies that provide some evidence of an increased prevalence of neurological symptoms among workers with average exposures of 75 to 100 ppm (<u>Cherry et al., 1981</u>). Long-term effects on some neurological measures (*i.e.*, possible detriments in attention and reaction time in complex tasks) have been observed in retired workers whose past chronic exposures were in the 100 to 200 ppm range (<u>Lash et al., 1991</u>). These studies are limited by the relatively small sample sizes and their low power for detection of statistically significant results (<u>EPA, 2011c</u>).

Following repeated inhalation exposure to DCM, the liver is the most sensitive target for noncancer toxicity in rats and mice. Lifetime exposure was associated with hepatocyte vacuolation and necrosis in F344 rats exposed to 1,000 ppm for 6 hrs/day (<u>Mennear et al., 1988</u>; <u>NTP</u>, <u>1986</u>), hepatocyte vacuolation in Sprague-Dawley rats exposed to 500 ppm for 6 hrs/day (<u>Burek et al., 1984</u>; <u>Nitschke et al., 1988a</u>), and hepatocyte degeneration in B6C3F<sub>1</sub> mice exposed to 2,000 ppm for 6 hrs/day (*i.e.*, lower concentrations were not tested in mice) (<u>Mennear et al.,</u> <u>1988</u>; <u>NTP</u>, <u>1986</u>). Other effects were renal tubular degeneration in F344 rats and B6C3F<sub>1</sub> mice at 2,000 ppm, testicular atrophy in B6C3F<sub>1</sub> mice at 4,000 ppm, and ovarian atrophy in B6C3F<sub>1</sub> mice at 2,000 ppm (<u>EPA, 2011c</u>).

Lung toxicity has also been reported in rodents exposed to DCM. In a 13-week exposure study conducted by <u>NTP (1986)</u>, rats exposed to 8,400 ppm DCM reported an increased incidence of foreign body pneumonia (<u>EPA, 2011c</u>).

A two-generation inhalation exposure to DCM revealed no significant effects on reproductive performance in rats (up to 1,500 ppm) (<u>Nitschke et al., 1988b</u>). Some evidence of a decrease in fertility index was seen in male mice exposed to 150 and 200 ppm (<u>Raje et al., 1988</u>), and no adverse effects on fetal development of mice or rats exposed to up to 1,250 ppm were seen by (<u>Schwetz et al., 1975</u>). Decreases in fetal body weight and changes in behavioral habituation were observed in offspring of Long-Evans rats exposed to 4,500 ppm during the gestational period (<u>Bornschein et al., 1980</u>; <u>Hardin and Manson, 1980</u>).

Though few developmental effects were observed at high exposures to DCM (<u>Bornschein et al.,</u> <u>1980</u>; <u>Schwetz et al., 1975</u>), there are no studies that have adequately evaluated neurobehavioral and neurochemical changes resulting from gestational DCM exposure. The available data identified changes in behavior habituation (<u>Bornschein et al., 1980</u>) and increases in COHb (<u>Schwetz et al., 1975</u>) following DCM exposure (<u>EPA, 2011c</u>). (<u>Bornschein et al., 1980</u>)

study observed developmental neurotoxicity effects at 4,500 ppm, this was the only dose group used in the study. No other neurological endpoints have been evaluated in the available developmental studies of DCM. The potential for developmental neurotoxicity occurring at low exposures to DCM represents a data gap (<u>EPA, 2011c</u>).

The significance of this data gap also is supported by evidence from adult neurotoxicity testing indicating that acute/short term exposures can affect neurotransmission and neurotransmitters levels. These effects on neurotransmitters levels, while transient, may have qualitatively different outcomes if they occur during development of the nervous system when neurotransmitters serve a critical role in patterning the nervous system (<u>Barone et al., 2000</u>; <u>Rice and Barone, 2000</u>).

## 3.3.2.3.2 Carcinogenic Effects

EPA concluded that DCM is likely carcinogenic in humans by a mutagenic mode of action (<u>EPA</u>, <u>2011c</u>). The conclusion was based on evidence from both animal studies and epidemiological data reporting DCM-induced carcinogenicity.

Studies in humans provide evidence for an association between occupational exposure to DCM and increased risk for some specific cancers, including brain cancer (<u>Hearne and Pifer, 1999</u>; <u>Heineman et al., 1994</u>; <u>Tomenson, 2011</u>), liver cancer (<u>Lanes et al., 1990</u>; <u>Lanes et al., 1993</u>), non-Hodgkin lymphoma (<u>Barry et al., 2011</u>; <u>Miligi et al., 2006</u>; <u>Seidler et al., 2007</u>; <u>Wang et al., 2009</u>), and multiple myeloma (<u>Gold et al., 2011</u>).

In addition, several cancer bioassays in animals have identified the liver and lung as the most sensitive target organs for DCM-induced tumor development (EPA, 2011c). For example, B6C3F1 mice reported statistically significant increases in hepatocellular adenomas and carcinomas when exposed to DCM for 2 years via drinking water (NCA, 1983; Serota et al., 1986). Lung and liver tumors were reported in B6C3F1 mice exposed to 2,000 or 4,000 ppm DCM for 6 hrs/day, 5 days/week for 2 years by inhalation (Mennear et al., 1988; NTP, 1986). Inhalation animal studies have also reported benign mammary tumors in F344 rats exposed to 2,000 or 4,000 ppm DCM for 6 hrs/day, 5 days/week for 2 years (Mennear et al., 1988; NTP, 1986). Brain tumors were observed in a 2-year inhalation study that exposed Sprague-Dawley rats to relatively low concentrations of DCM (0-500 ppm) (Nitschke et al., 1988a). These tumors are exceedingly rare in rats, and there are few examples of statistically significant trends in animal bioassays (Sills et al., 1999). Please refer to *Chapter 4 and 5* of the DCM IRIS assessment for detailed information about the epidemiological and animal studies evaluated in the cancer-assessment, as well as their strengths and limitations (EPA, 2011c).

The hypothesized mode of action for DCM-induced lung and liver tumors is through a mutagenic mode of carcinogenic action. A weight-of-evidence analysis of in vivo and in vitro data provide support to the proposed mutagenicity of DCM and the key role of GST metabolism and the formation of DNA-reactive GST-pathway metabolites (<u>EPA, 2011c</u>).

### 3.3.2.4 Susceptible Subpopulations

Certain human subpopulations may be more susceptible to exposure to DCM than others. One basis for this concern is the potential effect of COHb, a metabolic byproduct of DCM exposure. The COHb generated from DCM is expected to be additive to COHb from other sources. Of particular concern are smokers who maintain significant constant levels of COHb and persons with existing cardiovascular disease (<u>ATSDR, 2000</u>).

Varying susceptibility to DCM may be correlated with polymorphism in its metabolizing enzymes. Genetic polymorphisms have been identified for both GST theta-1 and CYP2E1 (Garte and Crosti, 1999).

Hemoglobin in the fetus has a higher affinity for CO than does adult hemoglobin. Thus, the neurotoxic and cardiovascular effects may be exacerbated in fetuses and in infants with higher residual levels of fetal hemoglobin when exposed to high concentrations of DCM (<u>OEHHA</u>, <u>2001</u>).

## 3.3.3 Summary of Hazard Values Used to Evaluate Acute and Chronic Exposures

Table 3-10 summarizes the hazard values (i.e., PODs), adverse effects and UFs that are relevant for the risk evaluation of acute and chronic exposure scenarios.

Table 3 10	<ol> <li>Summary of Inhalation Hazard Information Used i and Chronic Scenarios</li> </ol>	n the Risk Evaluat	ion of Acute
Exposure Duration for Risk Analysis	Hazard Value Used in Risk Assessment	Effect	Total Uncertainty Factor (UF) for Benchmark MOE
CHRONIC	Inhalation Unit Risk (IUR): 4 x 10 <sup>-5</sup> per ppm (1 x 10 <sup>-5</sup> per mg/m <sup>3</sup> )	Liver and lung tumors	Not applicable
EXPOSURE	$1^{st}$ percentile human equivalent concentration (HEC) i.e. the HEC_{_{99}}: 17.2 mg/m^3 (4.8 ppm)	Liver effects	Total UF=10
	1-hr SMAC POD= 100 ppm (350 mg/m <sup>3</sup> )	Central nervous system (CNS) depression related to the formation of 3% COHb in blood	Total UF=10
	1-hr California REL POD= 240 ppm (840 mg/m <sup>3</sup> ) 8-hr California REL POD=80 ppm (290 mg/m <sup>3</sup> ) (for occupational scenarios)	Impairment of the CNS	Total UF=60
ACUTE EXPOSURE	AEGL-1 POD (threshold for discomfort/non-disabling effects) 10-min= 870 ppm (3,000 mg/m <sup>3</sup> ) 30-min= 690 ppm (2,400 mg/m <sup>3</sup> ) 1-hr= 600 ppm (2,130 mg/m <sup>3</sup> )	CNS effects (light headedness, difficulty in enunciation)	Total UF=3
	AEGL-2 POD (threshold for disability) 10-min= 1,700 ppm (6,000 mg/m <sup>3</sup> ) <sup>6</sup> 30-min= 1,200 ppm (4,200 mg/m <sup>3</sup> ) <sup>6</sup> 1-hr= 560 ppm (2,000 mg/m <sup>3</sup> ) <sup>7</sup> 4-hr= 100 ppm (350 mg/m <sup>3</sup> ) <sup>7</sup> 8-hr= 60 ppm (210 mg/m <sup>3</sup> ) <sup>7</sup>	CNS effects for 10- and 30-min AEGL-2 PODs COHb formation for 1-, 4- and 8-hr AEGL-2 PODs	Total UF=1

# 3.4 HUMAN HEALTH RISK CHARACTERIZATION

Exposure to DCM is associated with adverse effects on the nervous system, liver and lung. These non-cancer adverse effects are deemed important for acute and chronic risk estimation for the scenarios and populations addressed in this risk assessment.

DCM is likely to be carcinogenic to humans. The cancer risk assessment uses the IUR derived in the 2011 DCM IRIS assessment based on liver and lung tumors in rodents. The weight-of-evidence analysis for the cancer endpoint was sufficient to conclude that DCM-induced tumor development operates through a mutagenic mode of action (EPA, 2011c).

## **3.4.1 Risk Estimation Approach for Acute and Repeated Exposures**

Tables 3-11 and 3-12 show the use scenarios, populations of interest and toxicological endpoints that were used for estimating acute or chronic risks, respectively.

Use Scenarios Populations And Toxicological Approach	OCCUPATIONAL USE	RESIDENTIAL USE
Population of Interest and Exposure Scenario: <i>Users</i>	Adults of both sexes (>16 years old) exposed to DCM during an 8-hr workday <sup>1, 2</sup>	Adults of both sexes (>16 years old) typically exposed to DCM for 1 hr. Other shorter (10-min, 30-min) or longer exposure times (4-hr, 8-hr) were also assumed when comparing DCM air concentrations with AEGLs.
Population of Interest and Exposure Scenario: <i>Bystander</i>	Adults of both sexes (>16 years old) indirectly exposed to DCM while being in the same building during product use.	Individuals of any age indirectly exposed to DCM while being in the rest of the house during product use.
Health Effects of Concern, Concentration and Time Duration	<u>Non-Cancer Health Effects:</u> CNS effects and C Hazard Values (PODs) for Occupational Scenarios: <sup>3</sup> 8-hr California REL POD= 290 mg/m <sup>3</sup> 8-hr AEGL-2 POD = 210 mg/m <sup>3</sup>	OHb formation in the blood (see Table 3-10). <i>Hazard Values (PODs) for Residential</i> <i>Scenarios:</i> 1-hr SMAC POD= 350 mg/m <sup>3</sup> 1-hr California REL POD= 840 mg/m <sup>3</sup> 10-min AEGL-1 POD= 3,000 mg/m <sup>3</sup> 30-min AEGL-1 POD = 2,400 mg/m <sup>3</sup> 10-min AEGL-1 POD = 2,130 mg/m <sup>3</sup> 10-min AEGL-2 POD = 6,000 mg/m <sup>3</sup> 30-min AEGL-2 POD = 4,200 mg/m <sup>3</sup> 1-hr AEGL-2 POD = 2,000 mg/m <sup>3</sup> 4-hr AEGL-2 POD = 350 mg/m <sup>3</sup> 8-hr AEGL-2 POD = 210 mg/m <sup>3</sup>
	Cancer Health Effects: Acute cancer risks were between a single short-term exposure to DCN	
Uncertainty Factors (UF) used in Non-Cancer Margin of Exposure (MOE) calculations	UF for Californ UF for AEG	AC PODs= 10 nia REL POD= 60 L-1 PODs= 3 L-2 PODs= 1

<sup>3</sup> AEGL-1 POD for 8-hr is not available since the DCM AEGL technical support document did not derive AEGL-1 values for 8-hrs.

Use		
Scenarios		
Populations And Toxicological Approach	OCCUPATIO	NAL USE
Population of Interest and Exposure Scenario: Users	Adults of both sexes (>16 years of an 8-hr workday for up to 250 days per year occupational so	for 40 working years depending on the
Population of Interest and Exposure Scenario: Bystander	Adults of both sexes (>16 years old) indirectly exp building during product use. <sup>3</sup>	oosed to DCM while being in the same
Health Effects of Concern, Concentration and Time Duration	Hazard Value (PODs) for Non-Cancer Effects (liver effects): 1 <sup>st</sup> percentile human equivalent concentration (HEC) i.e. the HEC <sub>99</sub> : 17.2 mg/m <sup>3</sup> (4.8 ppm)	Hazard Value (PODs) for Cancer Effects (liver and lung tumors): Inhalation Unit Risk (IUR): 4 x 10 <sup>-5</sup> per ppm (1 x 10 <sup>-5</sup> per mg/m <sup>3</sup> )
Uncertainty Factors (UF) used in Non-Cancer Margin of Exposure (MOE) calculations	UF for the HE UF is not applied for the ca	

<sup>3</sup> Data sources did not often indicate whether exposure concentrations were for occupational users or bystanders. Therefore, EPA/OPPT assumed that exposures were for a combination of users and bystanders. Some bystanders may have lower exposures than users, especially when they are further away from the source of exposure. Acute or chronic MOEs (MOE<sub>acute</sub> or MOE<sub>chronic</sub>) were used in this assessment to estimate non-cancer risks (Table 3-13).

	of Exposure (MOE) Equation to Estimate Non Cancer Risks Following r Chronic Exposures to DCM
	MOE acute or chronic = Non-cancer Hazard value (POD)
	Human Exposure
MOE =	Margin of exposure (unitless)
Hazard value (POD) =	derived from various toxicological documents (see Tables 3-10, 3-11, 3-12)
Human Exposure =	Exposure estimate (in ppm) from occupational or consumer exposure assessment. ADCs were used for non-cancer risks associated with chronic exposures to DCM. Acute concentrations as expressed as 8-hr TWA DCM air concentrations were used for acute risks.

Study-specific UFs were identified for each hazard value (i.e., POD). These UFs accounted for (1) the variation in susceptibility among the members of the human population (i.e., interindividual or intraspecies variability); (2) the uncertainty in extrapolating animal data to humans (i.e., interspecies uncertainty); and (3) the uncertainty in extrapolating from a LOAEL rather than from a NOAEL.

The total UF for each non-cancer hazard value was the benchmark MOE used to interpret the MOE risk estimates for each use scenario. The MOE estimate was interpreted as human health risk if the MOE estimate was less than the benchmark MOE (i.e. the total UF). On the other hand, the MOE estimate indicated negligible concerns for adverse human health effects if the MOE estimate exceeded the benchmark MOE. Typically, the larger the MOE, the more unlikely it is that a non-cancer adverse effect would occur.

Cancer risks for repeated exposures to DCM were estimated using the equation in Table 3-14. Estimates of cancer risks should be interpreted as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen (i.e., incremental or excess individual lifetime cancer risk).

Table 3 14. Equation	n to Calculate Cancer Risks
	Risk = Human Exposure × IUR
Risk =	Cancer risk (unitless)
Human exposure =	Exposure estimate (LADC in ppm) from occupational exposure assessment
IUR =	Exposure estimate (LADC in ppm) from occupational exposure assessment Inhalation unit risk 4 x 10 <sup>-5</sup> per ppm (1 x 10 <sup>-5</sup> per mg/m <sup>3</sup> ) ( <u>EPA, 2011c</u> )

# 3.4.2 Acute Non-Cancer Risk Estimates for Inhalation Exposures to DCM

The acute inhalation risk assessment used CNS effects to evaluate the acute risks for consumer and occupational use of DCM-containing paint strippers. Health hazard values were derived from the SMAC and the California acute REL hazard/dose-response assessments. This assessment gives preferences to those acute risk estimates derived from the SMAC hazard/dose-response assessment because the SMAC POD was based on multiple human observations reporting increased COHb levels after DCM exposure, coupled with the knowledge of what would be considered a NOAEL COHb level based on the extensive CO database (<u>NRC, 1996</u>).

Hazard values based on the AEGL hazard/dose-response assessment were also included in the acute risk assessment. As discussed in section 3.3.1.3.3, AEGL PODs for the respective tiers (discomfort/non-disabling effects = AEGL-1 threshold; disability = AEGL-2 threshold; and death = AEGL-3 threshold) are selected to represent an estimated point of transition between one defined set of symptoms or adverse effects in one tier and another defined set of symptoms or adverse effects in the next tier (NRC, 2001). Although the AEGL PODs and total UFs do not have the degree of conservatism that other values have, EPA/OPPT used them in this assessment to gauge how far the acute consumer and occupational exposure are from the thresholds for discomfort/non-disabling effects (AEGL-1) and disability (AEGL-2). These comparisons provide an indicator of whether the exposure estimates would be expected to produce human adverse effects following DCM exposure.

## 3.4.2.1 Acute Risks for Consumer Exposure Scenarios

Acute inhalation risks for CNS effects were reported for all of the consumer exposure scenarios when risks were evaluated with the SMAC and the California acute REL PODs and respective benchmark MOEs. There risks were reported for both the product user and the residential bystanders exposed to DCM, irrespective of the type of product used (i.e., brush-on vs. spray-on paint stripper) (Table 3-15).

Consumers using DCM-based paint strippers reported risk concerns for non-disabling effects (AEGL-1) during the first hour of product use (i.e., 10-min, 30-min or 1-hr exposure). For instance, MOEs based on the AEGL-1 PODs were lower than the benchmark MOE for users using brush-on and spray-on products in those scenarios constructed with upper-end estimates for either the user or the user and bystanders (Scenarios 2, 3, 5 and 6) (Table 3-16).

Likewise, risk concerns for incapacitating effects (AEGL-2) in product users were observed in Scenarios 2, 3, 5 and 6 at longer exposure times (i.e., 4-hr or 8-hrs). Interestingly, these risks were also reported for residential bystanders in Scenarios 3 and 6, where upper end user and bystander parameters were used to construct the scenarios (Table 3-16).

The bathroom scenario (#7) was constructed to simulate a human fatality case during a bathtub refinishing project. It was included in the assessment to estimate the DCM air concentrations to

residential occupants outside the use zone (i.e., bystanders) under conditions of high product use in the room of use. As expected, risk concerns for incapacitating effects (AEGL-2) were seen in users exposed to DCM for 4- and 8-hrs. Similarly, the users showed risks for non-disabling effects (AEGL-1) during the first hour of product use (i.e., 10-min, 30-min or 1-hr). Bystanders did not show risk concerns for non-disabling (AEGL-1) and incapacitating (AEGL-2) effects at any of the exposure durations (i.e., 10-min, 30-min, 1-hr, 4-hr or 8-hr) (Table 3-16).

Table 3 15. Acute Risk Es				
			OEs below benchmar ed in bold text	k MOE indicate
potential ne		Maximum		xposure (MOE)
Exposure Scenario	Individual	Value for 1-hr Averaging Period (mg/m <sup>3</sup> )	1-hr SMAC POD Total UF or Benchmark MOE=10*Preferred Approach	1-hr California REL POD Total UF or Benchmark MOE=60
Scenario #1 Brush application in	User	220	1.6	3.8
workshop, central parameter values	Bystander	120	2.9	7.0
Scenario #2 Brush application in	User	1,100	0.3	0.8
workshop, upper-end values for user	Bystander	210	1.7	4.0
Scenario #3 Brush application in workshop, upper-end	User	760	0.5	1.1
values for user and bystander estimates	Bystander	460	0.8	1.8
Scenario #4 Spray application in	User	490	0.7	1.7
workshop, central parameter values	Bystander	280	1.3	3.0
Scenario #5 Spray application in	User	1,600	0.2	0.5
workshop, upper-end values for user	Bystander	310	1.1	2.7
Scenario #6 Spray application in workshop, upper-end	User	1,100	0.3	0.8
values for user and bystander estimates	Bystander	700	0.5	1.2
Scenario #7	User	799	0.4	1.1
Brush application in bathroom, simulation	Bystander	218	1.6	3.9

Table 3 16. Acu Exp	ite Risk Estir Josure Durat													bus
		Max		/alues fo iod, mg/	•	ging	Margin of Exposure (MOE)							
Consumer Scenario	Individual	al 10- min	30-					EGL-1 POD JF or Benc MOE =3	-	AEGL-2 PODs Total UF or Benchmark MOE =1				
			min	1-hr	4-hr	8-hr	10-min (3,000 mg/m <sup>3</sup> )	30-min (2,400 mg/m <sup>3</sup> )	1-hr (2,130 mg/m³)	10-min (6,000 mg/m <sup>3</sup> )	30-min (4,200 mg/m <sup>3</sup> )	1-hr (2,000 mg/m³)	4-hr (350 mg/m <sup>3</sup> )	8-hr (210 mg/m <sup>3</sup> )
Scenario #1: Brush application in	User	380	270	220	120	69	7.9	8.9	9.7	15.8	15.6	9.1	2.9	3.0
workshop, central parameter estimates	Bystander	130	130	120	82	49	23.1	18.5	17.8	46.2	32.3	16.7	4.3	4.3
Scenario #2: Brush application in	User	1,300	1,100	1,100	420	220	2.3	2.2	1.9	4.6	3.8	1.8	0.8	1.0
workshop, upper-end user estimates	Bystander	220	220	210	140	82	13.6	10.9	10.1	27.3	19.1	9.5	2.5	2.6
Scenario #3: Brush application in	User	1,200	900	760	560	400	2.5	2.7	2.8	5.0	4.7	2.6	0.6	0.5
workshop, upper-end user and bystander estimates	Bystander	470	470	460	380	290	6.4	5.1	4.6	12.8	8.9	4.3	0.9	0.7
Scenario #4: Spray application in	User	780	600	490	270	150	3.8	4.0	4.3	7.7	7.0	4.1	1.3	1.4

Table 3 16. Acu Exp	ite Risk Estir Josure Durat													ous
		Max		/alues fo iod, mg/	•	ging	Margin of Exposure (MOE)							
Consumer Scenario	Individual	dual 10- min	30-		4-hr			EGL-1 POD JF or Benc MOE =3	-	AEGL-2 PODs Total UF or Benchmark MOE =1				
			min	1-hr	4-111	8-hr	10-min (3,000 mg/m <sup>3</sup> )	30-min (2,400 mg/m³)	1-hr (2,130 mg/m <sup>3</sup> )	10-min (6,000 mg/m <sup>3</sup> )	30-min (4,200 mg/m <sup>3</sup> )	1-hr (2,000 mg/m³)	4-hr (350 mg/m³)	8-hr (210 mg/m <sup>3</sup> )
workshop, central parameter estimates	Bystander	300	300	280	190	110	10.0	8.0	7.6	20.0	14.0	7.1	1.8	1.9
Scenario #5: Spray application in	User	1,900	1,800	1,600	620	330	1.6	1.3	1.3	3.2	2.3	1.3	0.6	0.6
workshop, upper-end user estimates	Bystander	330	320	310	200	120	9.1	7.5	6.9	18.2	13.1	6.5	1.8	1.8
Scenario #6: Spray application in workshop,	User	1,600	1,300	1,100	810	580	1.9	1.8	1.9	3.8	3.2	1.8	0.4	0.4
upper-end user and bystander estimates	Bystander	710	710	700	580	430	4.2	3.4	3.0	8.5	5.9	2.9	0.6	0.5
Scenario #7: Brush application in	User	1,455	887	799	536	340	2.1	2.7	2.7	4.1	4.7	2.5	0.7	0.6
bathroom, simulation	Bystander	224	222	218	187	150	13.4	10.8	9.8	26.8	18.9	9.2	1.9	1.4

## 3.4.2.2 Acute Risks for Occupational Exposure Scenarios

Acute inhalation risks for CNS effects were reported for most of the relevant industries when occupational risks were evaluated with the California acute REL POD and respective benchmark MOE. These risks were irrespective of the absence or presence of respirators and were observed with central tendency or high-end DCM air concentrations. No risks were found for workers handling DCM-based strippers in the art restoration and conservation industry (Table 3-17).

Workers handling DCM-containing paint strippers with no respirator showed risks for incapacitating effects (AEGL-2) when employed in all of the relevant industries, except the art restoration and conservation industry (Table 3-17). These risks were present with either central tendency or high-end DCM air concentrations of DCM.

Workers employed in industries with high exposure to DCM [i.e., professional contractors, furniture refinishing, aircraft paint stripping, and immersion stripping of wood (non-specific workplace settings)] typically showed risks for incapacitating (AEGL-2) effects when using APF 10 respirators (Scenario 2) during high exposure conditions. The use of APF 25 respirators (Scenario 3) was not protective for workers employed in the immersion stripping of wood (non-specific workplace settings when DCM air concentrations were as high as 7,000 mg/m<sup>3</sup>.

Table 3 17. Acute Expo									ppers: AEGL Ith risks and a					
Professional	Acute	8-hr con	centration (	mg/m³)		•	REL POD=290 enchmark MC	•	Acute MOE (8hr-AEGL-2 POD=210 mg/m <sup>3</sup> ) Total UF or Benchmark MOE=1					
Contractors	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low		
Scenario 1 (No respirator, APF=0)		2,98 0	1,520	60		0.1	0.2	5		0.07	0.1	4		
Scenario 2 (Respirator, APF 10)		298	152	6		1	2	48		0.7	1.4	35		
Scenario 3 (Respirator, APF 25)		119	61	2		2	5	121		1.8	4	88		
Scenario 4 (Respirator, APF 50)		60	30	1		5	10	242		4	7	175		
Automotive	Acute	8-hr con	centration (	mg/m³)		•	REL POD=290 enchmark MC	0. ,	Acute MOE (8hr-AEGL-2 POD=210 mg/m³) Total UF or Benchmark MOE=1					
Refinishing	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low		
Scenario 1 (No respirator, APF=0)	253	416	253	90	1	0.7	1	3	0.8	0.5	0.8	2		
Scenario 2 (Respirator, APF 10)	25	42	25.3	9	12	7	12	32	8	5	8	23		
Scenario 3 (Respirator, APF 25)	10	17	10	4	29	17	29	81	21	13	21	58		
Scenario 4 (Respirator, APF 50)	5	8	5	2	57	35	57	161	42	25	42	117		
Furniture	Acute	8-hr con	centration (	mg/m³)		•	REL POD=290 enchmark MC	0. /		DE (8hr-AEGL-) al UF or Bench	2 POD=210 mg Imark MOE=1	/m³)		
Refinishing	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low		
Scenario 1 (No respirator, APF=0)	499	2,24 5	1,125	4	0.6	0.1	0.3	73	0.4	0.1	0.2	53		
Scenario 2 (Respirator, APF 10)	49.9	225	113	0.4	6	1.3	2.6	725	4	0.9	2	525		
Scenario 3 (Respirator, APF 25)	20	90	45	0.2	15	3	6	1813	11	2	5	1312		
Scenario 4 (Respirator, APF 50)	10	45	23	0.1	29	6	13	3625	21	5	9	2625		

Table 3 17. Acute Expos									ppers: AEGL Ith risks and a				
Art Restoration and	Acute	8-hr con	centration (	mg/m³)		•	REL POD=290 nchmark MC	0. ,		DE (8hr-AEGL-2 al UF or Bench	2 POD=210 mg, mark MOE=1	/m³)	
Conservation	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	
Scenario 1 (No respirator, APF=0)			2				145			105			
Scenario 2 (Respirator, APF 10)			0.2		1450					1050	)		
Scenario 3 (Respirator, APF 25)			0.1		3625					2625	5		
Scenario 4 (Respirator, APF 50)			0.04		7250					5250	)		
Aircraft Paint	Acute	8-hr con	centration (	mg/m³)	Acute MOE (8hr-REL POD=290 mg/m <sup>3</sup> ) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m³) Total UF or Benchmark MOE=1				
Stripping	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	
Scenario 1 (No respirator, APF=0)		3,80 2	1,944	86		0.1	0.2	3		0.1	0.1	2	
Scenario 2 (Respirator, APF 10)		380	194	9		1	1.5	34		0.6	1	24	
Scenario 3 (Respirator, APF 25)		152	78	3		2	4	84		1	3	61	
Scenario 4 (Respirator, APF 50)		76	39	2		4	7	167		3	5	122	
Graffitti	Acute	8-hr con	centration (	mg/m³)	Acute MOE (8hr-REL POD=290 mg/m³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m <sup>3</sup> ) Total UF or Benchmark MOE=1				
Removal	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	
Scenario 1 (No respirator, APF=0)	260	1,18 8	603	18	1	0.2	0.5	16	0.8	0.2	0.4	12	
Scenario 2 (Respirator, APF 10)	26	118. 8	60.3	1.8	11	2	5	161	8	2	3	117	
Scenario 3 (Respirator, APF 25)	10	48	24	0.7	28	6	12	403	20	4	9	292	
Scenario 4 (Respirator, APF 50)	5	24	12	0.4	56	12	24	806	40	9	17	583	

Non-Specific Workplace Settings	Acute	8-hr con	centration (	mg/m³)		•	REL POD=290 nchmark MC	- · ·		DE (8hr-AEGL-) al UF or Bench	2 POD=210 mg, mark MOE=1	/m³)
- Immersion Stripping of Wood	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)		7,00 0	3,518	35		0.04	0.1	8		0.03	0.1	6
Scenario 2 (Respirator, APF 10)		700	352	4		0.4	0.8	83		0.3	0.6	60
Scenario 3 (Respirator, APF 25)		280	141	1		1	2	207		0.8	1.5	150
Scenario 4 (Respirator, APF 50)		140	70	0.7		2	4	414		2	3	300
Non-Specific Workplace Settings	Acute	8-hr con	centration (	mg/m³)		•	REL POD=290 nchmark MC	0. /		DE (8hr-AEGL-) al UF or Bench	2 POD=210 mg, mark MOE=1	/m³)
- Immersion Stripping of Wood and Metal	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)		1,01 7	825	633		0.3	0.4	0.5		0.2	0.3	0.3
Scenario 2 (Respirator, APF 10)		101. 7	83	63		3	4	5		2	3	3
Scenario 3 (Respirator, APF 25)		41	33	25		7	9	11		5	6	8
Scenario 4 (Respirator, APF 50)		20	17	13		14	18	23		10	13	17
Non-Specific Workplace Settings	Acute	8-hr con	centration (	mg/m³)		•	REL POD=290 nchmark MC	0. /		DE (8hr-AEGL- al UF or Bench	2 POD=210 mg, mark MOE=1	/m³)
- Unknown	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)	357	428	357	285	0.8	0.7	0.8	1	0.6	0.5	0.6	0.7
Scenario 2 (Respirator, APF 10)	36	43	36	29	8	7	8	10	6	5	6	7
Scenario 3 (Respirator, APF 25)	14	17	14	11	20	17	20	25	15	12	15	18
Scenario 4 (Respirator, APF 50)	7	9	7	6	41	34	41	51	29	25	29	37

Table 3.17 Acute Risk Estimates for Occupational Exposures to DCM Based Paint Strippers: AEGL 1 and AEGL 2 PODs for Various

## 3.4.3 Non-Cancer and Cancer Risk Estimates for Chronic Inhalation Exposures to DCM

Non-cancer and cancer risk estimates for inhalation exposures to DCM were only derived for occupational scenarios since the exposures for consumer uses were not considered chronic in nature. Hazard values were obtained from the EPA IRIS *Toxicological Review of Methylene Chloride* (EPA, 2011c).

### 3.4.3.1 Cancer Risks for Occupational Exposure Scenarios

The cancer risk assessment evaluated the incremental individual lifetime cancer risks for continuous exposures to DCM occurring during the use of paint stripping products. Excess cancer risks were calculated by multiplying the EPA inhalation unit risk for DCM (EPA, 2011c) by the exposure estimate (i.e., LADC). Cancer risks were expressed as number of cancer cases per million.

Occupational scenarios assumed that the exposure frequency (i.e., the number of days per year workers or bystanders are exposed to DCM) was either 125 or 250 days per year for an occupational exposure duration of 20 or 40 years over a 70-yr lifespan. It is recognized that the combination of these assumptions may yield conservative cancer risk estimates for some of the occupational scenarios evaluated in this assessment. Nevertheless, EPA/OPPT does not have additional information for further refinement of the exposure assumptions.

EPA typically uses a benchmark cancer risk level between 1x10<sup>-4</sup> and 1x10<sup>-6</sup> for determining the acceptability of the cancer risk in a population. Since the benchmark cancer risk level will be determined during risk management, the occupational cancer risk estimates were compared to three benchmark levels within EPA's acceptability range. The benchmark levels were:

- **1.** 1x10<sup>-6</sup>: the probability of 1 chance in 1 million of an individual developing cancer;
- **2.**  $1 \times 10^{-5}$ : the probability of 1 chance in 100,000 of an individual developing cancer, which is equivalent to 10 cancer cases in 1 million;
- **3.**  $1x10^{-4}$ : the probability of 1 chance in 10,000 of an individual developing cancer, which is equivalent to 100 cancer cases in 1 million.

Tables 3-18 to 3-26 show the excess cancer risks calculated for workers of different industries handling DCM-based paint strippers. Selected scenarios ranging from the highest exposure scenario (i.e., no respiratory protection and high end values for EF and WY–i.e., Scenario 1) to the lowest exposure scenario (e.g., respiratory protection APF 50 and midpoints for EF and WY–Scenario 16) were included in the tables. Calculations of cancer risks for the full set of industries and scenarios are provided in the supplemental Excel spreadsheet, *DCM Exposure and Risk Estimates\_081114.xlsx*.

Workers showed excess cancer risks for all of the industries evaluated when working with DCMbased paint strippers for 250 days/year for 40 years with no respiratory protection (Scenario 1). Generally, Scenario 1 exceeded the three target cancer levels with the exception of art restoration and conservation that only exceeded the 1x10<sup>-6</sup> target level. On the other hand, workers showed a reduction in cancer risks when working for 125 days/year for 20 years with adequate respiratory protection (Scenario 16). That reduction in excess cancer risk was one or two orders of magnitude depending on the industry involved in paint stripping activities when compared with Scenario 1.

For Scenarios 3 and 15, occupational cancer risks for the different industries fell between the risks calculated for Scenario 1 and 16, and generally exceeded one or more benchmark cancer levels when workers were exposed to high or midpoint DCM air concentrations.

	18. Occupational Canc Professional Contractors	** LADCs fo	LADC (mg/m <sup>3</sup> ) or scenarios 2 sted with the	to 16 have	Ex (Inha	cess Cancer R alation Unit R 10 <sup>-5</sup> per mg/n	isk isk =
		High	Midpoint	Low	High	Midpoint	Low
Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	389	198	8	3.9E-03	2.0E-03	7.8E-05
H	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	16	8	0.31	1.6E-04	7.9E-05	3.1E-06
oosure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	4	2	0.08	3.9E-05	2.0E-05	7.8E-07
Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	2	1	0.04	1.9E-05	9.9E-06	3.9E-07

Tab	ole 3 1	19. Occupational Canc	er Risks			efinishi	ng (Scen		-	16)
		Automotive Refinishing		Cs for sce	(mg/m <sup>3</sup> ) marios 2 to 1 with the mul			(Inhalatio	Cancer Risk n Unit Risk = per mg/m³)	:
			Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
	Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	33	54	33	12	3.3E-04	5.4E-04	3.3E-04	1.2E-04
	High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	1	2	1	0.48	1.3E-05	2.2E-05	1.3E-05	4.8E-06
	oosure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.3	1	0.33	0.12	3.3E-06	5.4E-06	3.3E-06	1.2E-06
	Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.2	0.3	0.2	0.1	1.7E-06	2.7E-06	1.7E-06	6.0E-07

Table 3	20. Occupational Cano	er Risks	for Fur	niture Refi	nishing	(Scenari	ios 1, 3,	15 and 16	
	Furniture Refinishing		Cs for sce	(mg/m <sup>3</sup> ) marios 2 to 1 with the mul			(Inhalatio	Cancer Risk n Unit Risk = per mg/m³)	:
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	65	293	147	0.5	6.5E-04	2.9E-03	1.5E-03	5.0E-06
High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	3	12	6	0.02	2.6E-05	1.2E-04	5.9E-05	2.0E-07
Exposure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	3	1	0.01	6.5E-06	2.9E-05	1.5E-05	5.0E-08
Lowest Exp	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.3	1.5	0.7	0.003	3.3E-06	1.5E-05	7.4E-06	2.5E-08

	Aircraft Paint Stripping	** LADCs fo	LADC (mg/m <sup>3</sup> ) or scenarios 2 sted with the	to 16 have	(Inh	cess Cancer R alation Unit R 10 <sup>-5</sup> per mg/n	isk =
		High	Midpoint	Low	High	Midpoint	Low
Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	496	254	11	5.0E-03	2.5E-03	1.1E-04
High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	20	10	0.44	2.0E-04	1.0E-04	4.4E-06
Exposure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	5	3	0.11	5.0E-05	2.5E-05	1.1E-06
Lowest Exp	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	2	1	0.06	2.5E-05	1.3E-05	5.5E-07

Table 3	22. Occupational Cano	er Risks			val (Sce	narios 1,	-	-	
	Graffiti Removal		Cs for sce	(mg/m <sup>3</sup> ) enarios 2 to 1 with the mul			(Inhalatio	Cancer Risk n Unit Risk = er mg/m³)	=
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	34	155	79	2.3	3.4E-04	1.6E-03	7.9E-04	2.3E-05
Higt	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	1	6	3	0.092	1.4E-05	6.2E-05	3.2E-05	9.2E-07
Exposure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.340	2	1	0.023	3.4E-06	1.6E-05	7.9E-06	2.3E-07
Lowest Exp	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.2	0.8	0.4	0.012	1.7E-06	7.8E-06	4.0E-06	1.2E-07

	Non-Specific Workplace Settings - Immersion Stripping of Wood	** LADCs f	LADC (mg/m³) or scenarios 2 sted with the	to 16 have	(Inh	cess Cancer Ri alation Unit Ri 10 <sup>-5</sup> per mg/m	isk =
		High	Midpoint	Low	High	Midpoint	Low
Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	913	459	4.6	9.1E-03	4.6E-03	4.6E-05
High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	37	18	0.184	3.7E-04	1.8E-04	1.8E-06
oosure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	9	5	0.046	9.1E-05	4.6E-05	4.6E-07
Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	5	2	0.023	4.6E-05	2.3E-05	2.3E-07

	Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	** LADCs f	LADC (mg/m <sup>3</sup> ) or scenarios 2 sted with the	to 16 have	(Inh	cess Cancer Ri alation Unit R 10 <sup>-5</sup> per mg/n	isk =
		High	Midpoint	Low	High	Midpoint	Low
Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	133	108	83	1.3E-03	1.1E-03	8.3E-04
Higl	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	5	4	3	5.3E-05	4.3E-05	3.3E-05
oosure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	1	1	1.3E-05	1.1E-05	8.3E-06
Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	1	1	0.415	6.7E-06	5.4E-06	4.2E-06

# Table 3 25. Occupational Cancer Risks for Non Specific Workplace SettingsUnknown(Scenarios 1, 3, 15 and 16)

		Scenarios 1, 3, 15 Non-Specific Workplace Settings - Unknown	** LAD	LADC Cs for sce	(mg/m³) narios 2 to 1 with the mul			(Inhalatio	Cancer Risk n Unit Risk = per mg/m³)	=
			Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
1	Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	47	56	47	37	4.7E-04	5.6E-04	4.7E-04	3.7E-04
	Hig	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	2	2	2	1	1.9E-05	2.2E-05	1.9E-05	1.5E-05
	posure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.5	1	0.5	0.4	4.7E-06	5.6E-06	4.7E-06	3.7E-06
	Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.2	0.3	0.2	0.2	2.4E-06	2.8E-06	2.4E-06	1.9E-06

Та	ble 3	26. Occupational Cano and 16)	er Risks			on and (	Conserva	ation (Se	cenarios 1,	3, 15		
		Art Restoration and Conservation		Cs for sce	(mg/m <sup>3</sup> ) enarios 2 to 1 with the mul		Excess Cancer Risk (Inhalation Unit Risk = 1x10 <sup>-5</sup> per mg/m <sup>3</sup> )					
			Mean	High	Midpoint	Low	Mean	High	Midpoint	Low		
1	Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]			0.3			3.	.0E-06			
	High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)		C	).012			1	.2E-07			
	Exposure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)		(	).003			3	.0E-08			
	Lowest Exp	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)		0	.0015			1	.5E-08			

# 3.4.3.2 Non-Cancer Risks for Occupational Exposure Scenarios Following Chronic Exposure to DCM

EPA/OPPT estimated non-cancer risks for the occupational use of DCM-containing paint strippers. Chronic exposure to DCM has been associated with liver effects. As previously discussed, the DCM IRIS assessment developed a non-cancer hazard value (i.e., POD) based on hepatic effects. EPA/OPPT used the PBPK-derived 1<sup>st</sup> percentile HEC i.e. the HEC<sub>99</sub> the concentration at which there is 99% likelihood an individual would have an internal dose less than or equal to the internal dose of hazard reported in the DCM IRIS assessment (EPA, 2011c) to calculate non-cancer risks associated with the repeated use of DCM-based strippers at different workplace settings.

Tables 3-27 to 3-35 show the non-cancer MOE estimates calculated for workers of different industries handling DCM-based paint strippers on a repeated basis. Selected scenarios ranging from the highest exposure scenario (i.e., no respiratory protection and high end values for EF and WY–i.e., Scenario 1) to the lowest exposure scenario (e.g., respiratory protection APF 50 and midpoints for EF and WY–Scenario 16) were included in the tables. Calculations of non-cancer risks for the full set of industries and scenarios are provided in the supplemental Excel spreadsheet, *DCM Exposure and Risk Estimates\_081114.xlsx*.

Most workers using DCM-based paint strippers showed non-cancer risks for liver effects, with the exception of workers employed in the art renovation and conservation industry (Table 3-30). For instance, risk concerns for liver effects were reported for most workers handling DCM-

based paint strippers. These risk findings were reported with or without respiratory protection and using the product in a repeated nature at facilities usually reporting central tendency or high-end DCM air levels. Among all of the occupational scenarios, the greatest risk concern is for workers engaging in long-term use of the product (i.e., 250 days/year for 40 years) with no respiratory protection.

Non-cancer risks were not observed for workers that reduce their exposure to DCM-based strippers by doing all of the following: (1) wearing adequate respiratory protection (i.e., APF 50 respirator), (2) limiting exposure to central tendency exposure conditions (i.e., 125 days/year for 20 years) and (3) working in facilities with low-end DCM air concentrations. This observation was reported in all of the relevant industries.

Table 3	27. Occupational Non Exposure to DCM (				tractors Fo	llowing Chr	onic
	Professional Contractors	** ADCs fo	ADC (mg/m <sup>3</sup> ) or scenarios 2 sted with the	to 16 have		c MOE (24hr   17.2 mg/m <sup>3</sup> ) or Benchmark	
		High	Midpoint	Low	High	Midpoint	Low
Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	680	347	14	0.025	0.050	1
High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	27	14	1	1	1	31
Exposure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	7	3	0.1	3	5	123
Lowest Exp	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	3	2	0.1	5	10	246

Note: MOEs below benchmark MOE indicating risk are denoted in bold text.

Ta	ble 3	28. Occupational Non Exposure to DCM					nishing	Followir	ng Chronic	
		Automotive Refinishing	** AD(	ADC ( Cs for scel	mg/m <sup>3</sup> ) narios 2 to 10 with the mul Midpoint	6 have		17.2	E (24hr HEC9 mg/m³) nchmark MO Midpoint	
	Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	58	95	58	21	0.3	<b>0.2</b>	0.3	0.8
	High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	2	4	2	1	7	5	7	20
	posure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	1	1	0.2	30	18	30	82
	Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.3	0.5	0.3	0.1	59	36	59	164
		Note: MOEs below	<mark>/ bench</mark> m	hark MO	E indicating	risk are	denoted	in bold t	ext.	
Tal	ble 3	29. Occupational Non	Cancer	Risks fo	r Furniture	e Refinis	hing Fol	lowing	Chronic	
		Exposure to DCM	(Scenari	os 1, 3,	15 and 16)					
		Furniture Refinishing		Cs for sce	(mg/m³) narios 2 to 1 with the mul			17.2	E (24hr HEC9 mg/m <sup>3</sup> ) nchmark MO	
			Mean	High	Midpoint	Low	Mean	High		
Î	inre 1	Scenario 1					mean	i iigii	Midpoint	Low
	lest Exposure	[No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	114	513	257	0.9	0.2	<b>0.03</b>	Midpoint 0.1	
	Highest Expos	ends of ranges for	114	513 21	257	0.9		-		Low
		ends of ranges for exposure frequency (EF) and working years (WY)] Scenario 3 (Respirator APF 25, high ends of ranges for EF					0.2	0.03	0.1	<b>Low</b> 19

	Art Restoration/ Conservation	ADC (mg/m <sup>3</sup> ) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier	Chronic MOE (24hr HEC <sub>99</sub> = 17.2 mg/m³) Total UF or Benchmark MOE=1		
		Mean <sup>a</sup>	Mean <sup>a</sup>		
Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	0.5	34		
High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	0.02	860		
oosure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.005	3440		
Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.0025	6880		

Note:

<sup>a</sup> Based on one 8-hr TWA data point reported in the OSHA IMIS database.

Note: MOEs below benchmark MOE indicating risk are denoted in bold text.

Table 3 31. Occupational Non Cancer Risks for Aircraft Stripping Following Chronic Exposure toDCM (Scenarios 1, 3, 15 and 16)										
		Aircraft Paint Stripping	** ADCs fo	ADC (mg/m <sup>3</sup> ) or scenarios 2 sted with the	to 16 have	Chronic MOE (24hr HEC99 = 17.2 mg/m <sup>3</sup> ) Total UF or Benchmark MOE=10				
			High	Midpoint	Low	High	Midpoint	Low		
	Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	868	444	20	0.02	0.04	0.9		
	Hig	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	35	18	1	0.5	1	22		
	Exposure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	9	4	0.2	2	4	86		
	Lowest Exp	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	4	2	0.1	4	8	172		

	DCM (Scenarios 1,	3, 15 ar							
	Graffiti Removal	ADC (mg/m <sup>3</sup> ) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier				Chronic MOE (24hr HEC99 = 17.2 mg/m³) Total UF or Benchmark MOE=10			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
	Scenario 1								
Highest Exposure	[No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	59	271	138	4	0.3	0.1	0.1	4
Highe	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	2	11	6	0.2	7	2	3	105
sure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	3	1	0.04	29	6	12	420
Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.3	1	0.7	0.02	58	13	25	839
	Note: MOEs below	/ Delicili			rick ara	donotod	in hold	toxt	
able 3	33. Occupational Non	Cancer							ion
able 3	33. Occupational Non Stripping of Wood		Risks fo	r Non Spe	cific Wo	orkplace	Setting	s (Immers	
able 3	Stripping of Wood		Risks fo	r Non Spe	cific Wo	orkplace	Setting	s (Immers	
able 3	Stripping of Wood Non-Specific		Risks fo ving Chr	or Non Spe onic Expos	cific Wo	orkplace DCM (Sce	Setting enarios	s (Immers) 1, 3, 15 ar	d 16)
able 3	Stripping of Wood Non-Specific Workplace	) Follow	Risks fo ing Chr ADC	r Non Spe onic Expos (mg/m³)	cific Wo sure to I	orkplace DCM (Sce	Settings enarios ronic MC	s (Immers) 1, 3, 15 an DE (24hr HEC	d 16)
able 3	Stripping of Wood Non-Specific Workplace Settings -	) Follow ** AD	Risks fo ring Chr ADC Cs for sce	or Non Spe onic Expos	cific Wo sure to I	orkplace DCM (Sce Ch	Settings enarios ronic MC 17.2	s (Immers) 1, 3, 15 ar	<b>id 16)</b>
able 3	Stripping of Wood Non-Specific Workplace Settings - Immersion	) Follow ** AD	Risks fo ring Chr ADC Cs for sce	r Non Spe onic Expos (mg/m <sup>3</sup> ) narios 2 to 1	cific Wo sure to I	orkplace DCM (Sce Ch	Settings enarios ronic MC 17.2	s (Immersi 1, 3, 15 ar DE (24hr HEC mg/m <sup>3</sup> )	<b>id 16)</b>
able 3	Stripping of Wood Non-Specific Workplace Settings -	) Follow ** AD( been a	Risks fo ing Chr ADC Cs for sce adjusted	r Non Spe onic Expos (mg/m <sup>3</sup> ) narios 2 to 1 with the mu	cific Wo sure to I	OCM (Sce CM (Sce Ch Total	Settings enarios ronic MC 17.2 UF or Be	s (Immersi 1, 3, 15 ar DE (24hr HEC mg/m <sup>3</sup> ) nchmark M	<b>id 16)</b>
	Stripping of Wood Non-Specific Workplace Settings - Immersion Stripping of Wood Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF)	) Follow ** AD	Risks fo ring Chr ADC Cs for sce adjusted Mi	r Non Spe onic Expos (mg/m <sup>3</sup> ) narios 2 to 1	cific Wo sure to E 6 have Itiplier	orkplace DCM (Sce Ch	Settings enarios ronic MC 17.2 UF or Be Mi	s (Immersi 1, 3, 15 ar DE (24hr HEC mg/m <sup>3</sup> )	nd 16) 599 = OE=10
	Stripping of Wood Non-Specific Workplace Settings - Immersion Stripping of Wood Scenario 1 [No respirator, high ends of ranges for	) Follow ** AD been a High	Risks fo ring Chr ADC Cs for sce adjusted Mi	r Non Spe onic Expos (mg/m <sup>3</sup> ) narios 2 to 1 with the mu dpoint	cific Wo sure to E L6 have Itiplier Low	OCM (Sce CM (Sce Ch Total High	Settings enarios ronic MC 17.2 UF or Be Mi	s (Immersi 1, 3, 15 ar DE (24hr HEC mg/m <sup>3</sup> ) nchmark M dpoint	nd 16) S <sub>99</sub> = OE=10 Low
	Stripping of Wood Non-Specific Workplace Settings - Immersion Stripping of Wood Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)] Scenario 3 (Respirator APF 25, high ends of ranges for EF	) Follow ** AD been a High 1,598	Risks fo ring Chr ADC Cs for sce adjusted Mi	r Non Spe onic Expos (mg/m <sup>3</sup> ) narios 2 to 1 with the mu dpoint 803	cific Wo sure to I L6 have Itiplier Low	OCM (Sce Ch Total High	Settings enarios ronic MC 17.2 UF or Be Mi	s (Immersi 1, 3, 15 ar DE (24hr HEC mg/m <sup>3</sup> ) nchmark M dpoint 0.02	rd 16) 599 = OE=10 Low 2

Tab	ole 3	34. Occupational Non Stripping of Wood 15 and 16)					-			
		Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	ADC (mg/m <sup>3</sup> ) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier				Chronic MOE (24hr HEC99 = 17.2 mg/m³) Total UF or Benchmark MOE=10			
			High	Mi	dpoint	Low	High	Mi	dpoint	Low
	Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	232		188 145		0.07		0.1	
	-	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	9		8	6	2		2	
	Lowest Exposure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	2		2	1	7		9	12 24
	Lowest	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	1		1	1	15		18	
		Note: MOEs below	<sup>,</sup> benchm	nark MO	E indicating	; risk are	denoted	in bold	text.	
Tab	ole 3	35. Occupational Non Following Chronic							s (Unknow	n)
		Non-Specific Workplace Settings - Unknown		Cs for sce adjusted	(mg/m³) narios 2 to 1 with the mu		Chronic MOE (24hr HEC99 = 17.2 mg/m³) Total UF or Benchmark MOE=10			
			Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
	Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	81	98	81	65	0.21	0.18	0.21	0.27
	High	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	3	4	3	3	5	4	5	7
	posure	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	1	1	0.65	21	18	21	26
	Lowest Exposure	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.41	0.49	0.41	0.33	42	35	42	53

# 3.4.4 Human Health Risk Characterization Summary

This risk assessment focused on the occupational and consumer uses of DCM-containing paint strippers. The population of interest consisted of workers and consumers with direct (users) or indirect (bystander) exposure to DCM. Only the inhalation route of exposure was considered in this risk assessment.

The occupational and consumer exposure assessments generated the DCM exposure levels required to derive non-cancer risk estimates associated with acute and chronic exposures to DCM. In addition, cancer risks were estimated for occupational scenarios and expressed as lifetime risks, meaning the risk of developing cancer as a result of the occupational exposure over a normal lifetime of 70 yrs. Lifetime cancer risks from DCM exposure were compared to benchmark cancer risks ranging from 10<sup>-6</sup> to 10<sup>-4</sup>.

Many of the occupational scenarios exceeded the target cancer risks of 10<sup>-6</sup>, 10<sup>-5</sup> and 10<sup>-4</sup> when workers employed at various industries handled DCM-paint strippers for 250 days/year for 40 years with no respiratory protection. Adequate respiratory protection and reduced exposure conditions (e.g., exposure to 125 day/year for 20 years) resulted in reduced cancer risks for workers when compared to conditions of no respiratory protection while working with paint strippers for a 250 days/year for a working lifetime (i.e., 40 years).

To characterize the risks of adverse health effects other than cancer, MOEs were used to evaluate non-cancer risks for both acute and chronic exposures using hazard values derived from peer-reviewed hazard/dose-response assessments. Health protective hazard values were derived from the SMAC and the California acute REL hazard/dose-response assessments, whereas hazard values for non-disabling (AEGL-1) and incapacitating (AEGL-2) effects were obtained from the AEGL hazard/dose-response assessment for DCM.

Workers employed at most industries showed non-cancer risks for liver effects when using DCM-based strippers on a repeated basis. The exception was the art renovation and conservation industry which did not show non-cancer risks for the different scenarios evaluated in the assessment.

Most workers handling DCM-based paint strippers are at risk of developing non-cancer effects when they handle the product on a repeated basis with or without wearing respiratory protection. These observations were seen under various exposure conditions (i.e., exposure frequency and working years) in facilities reporting central tendency or high-end DCM air levels. Of special interest are workers using DCM-containing paint strippers engaging in long-term use of the product (i.e., 250 days/year for 40 years) with no respiratory protection as they showed the greatest risk concern for non-cancer risks.

On the contrary, non-cancer risks were not observed in workers that reduced their chronic exposure to DCM by doing all of the following: (1) wearing adequate respiratory protection (i.e.,

APF 50 respirator), (2) limiting exposure to central tendency exposure conditions (i.e., 125 days/year for 20 years), and (3) working in facilities with low-end DCM air concentrations.

Most occupational and residential users of DCM-based paint strippers reported acute risks for CNS effects when the SMAC and California's acute REL hazard values were used for risk estimation. These risks were observed in workers with or without respiratory protection and residential bystanders indirectly exposed to DCM.

There were concerns for discomfort/non-disabling (AEGL-1) and incapacitating (AEGL-2) effects for residential users exposed to DCM for shorter (10-min, 30-min, 1-hr) or longer exposure durations (4-hr, 8-hr) while doing the product application or staying in the residence after completion of the stripping task. These concerns were present for upper-end exposure conditions in the residential scenario as well as some of the upper-end exposure scenarios for affected bystanders.

Moreover, there were concerns for incapacitating effects (AEGL-2 effects) in workers handing DCM-containing paint strippers on an acute/short-term basis with no respiratory protection while employed in most industries involved in paint stripping. Concerns for incapacitating effects (AEGL-2 effects) were also observed for workers wearing respirators (i.e., APF 10 or APF 25) while performing paint stripping activities in industries with high DCM air concentrations [i.e., professional contractors, furniture refinishing, aircraft paint stripping, and immersion stripping of wood (non-specific workplace settings)].

The bathroom consumer modeling indicated that application of DCM-based paint strippers in a bathroom generate unsafe exposure conditions for the user of the product. Risk concerns for discomfort/non-disabling (AEGL-1) and incapacitating effects (AEGL-2) were seen in users exposed to DCM for shorter (10-min, 30-min, 1-hr) or longer exposure durations (4-hr, 8-hr) while doing the product application or staying in the residence after completion of the stripping task. However, residential bystanders did not report risk concerns for AEGL-1 and AEGL-2 effects.

## **3.5 DISCUSSION OF KEY SOURCES OF UNCERTAINTY AND DATA LIMITATIONS**

The characterization of variability and uncertainty is fundamental to the risk assessment. Variability refers to "the true heterogeneity or diversity in characteristics among members of a population (i.e., inter-individual variability) or for one individual over time (intra-individual variability)" (EPA, 2001). The risk assessment was designed to reflect critical sources of variability to the extent allowed by available methods and data and given the resources and time available.

On the other hand, uncertainty is "the lack of knowledge about specific variables, parameters, models, or other factors" (EPA, 2001) and can be described qualitatively or quantitatively. Uncertainties in the risk assessment can raise or lower the confidence of the risk estimates. In this assessment, the uncertainty analysis also included a discussion of data gaps/limitations.

Below is a discussion of the uncertainties and data gaps in the exposure, hazard/dose-response and risk characterization.

## **3.5.1 Uncertainties in the Occupational Exposure Estimates**

Uncertainties in the occupational exposure assessment arise from the following sources:

1. Inhalation Exposure Estimates: EPA/OPPT did not find enough data to determine complete statistical distributions of actual exposure concentrations for the exposed workers using DCM-based paint strippers. Ideally, EPA/OPPT would like to know 50<sup>th</sup> and 95<sup>th</sup> percentiles for each population. In the absence of percentile data, the air concentration means and midpoints (means are preferred over midpoints) of the data sets served as substitutes for 50<sup>th</sup> percentiles of the actual distributions, whereas high ends of ranges served as substitutes for 95<sup>th</sup> percentiles of the actual distributions.

However, these substitutes are highly uncertain and are weak substitutes for the ideal percentiles. For instance, in the few cases where enough data were found to determine statistical means and 95<sup>th</sup> percentiles (*Appendix G, Table G-2*), the associated substitutes (i.e., midpoints and high ends of ranges) were shown to overestimate exposures, sometimes significantly. While it is clear that the air concentration data represent real exposure levels (*Appendix G, Table G-2*), EPA/OPPT cannot determine whether these concentrations are representative of the statistical distributions of actual DCM air concentrations generated at the workplace during paint stripping activities.

The hypothetical scenario multipliers for workers have significant limitations. EPA/OPPT cannot determine how accurately the hypothetical scenario multipliers reflect real world reductions to exposure concentrations presented for the highest exposed population due to protective equipment and actual exposure frequencies and working years. Moreover, a

probabilistic exposure approach is inappropriate for this assessment due to the lack of statistical data for most of the parameters used in the ADC and LADC equation and the hypothetical scenario multipliers.

In addition, the worker exposure assessment is limited to include exposures from DCMbased strippers only and does not include exposures to DCM from other sources. Evaluation of other DCM uses did not fall within the scope of this assessment.

- 2. Population Exposed: The estimates of numbers of exposed workers are uncertain. The most uncertain parameter used in the method is the number of workers using strippers designated for each particular model plant. EPA/OPPT cannot determine whether the assumed numbers of workers designated for these model plants may underestimate or overestimate numbers of workers. However, the inclusion of only numbers of workers who actually use the strippers will underestimate the total number of workers exposed because non-users (bystanders) are excluded.
- **3. Dermal Exposure:** The worker exposure assessment only includes inhalation exposures from DCM-based strippers. The exclusion of dermal exposure from the assessment is likely to underestimate risks to workers, more so for workers who use respirators. This is also an uncertainty for the consumer exposure assessment.

## **3.5.2 Uncertainties in the Consumer Exposure Estimates**

The inhalation exposure assessment is composed of modeled exposure scenarios for which the inputs are based on experimental data, survey information, and a number of assumptions with varying degrees of uncertainty. The results are characterized as either plausible estimates of individual exposure, *e.g.*, central tendency, or possibly greater than the distribution of actual exposures, *e.g.*, bounding. These individual estimates are based on exposures to the modeled area concentrations in the room of product use (user), and in the rest of house (bystanders, and some user wait periods).

The extent of all of the uncertainties identified below is not known, so the total impact for the parameters that are discussed could result in either larger or smaller exposure estimates.

There is a high degree of confidence in the weight fraction and product density data for the paint stripper products. These values are based on currently available consumer products, as identified in (<u>Brown, 2012</u>). However, these values were not weighted by percent market share.

Similarly, there is a high degree of confidence in the values chosen to represent the house volume and air exchange rate, as they are based on scientifically defensible data cited in the EPA's 2011 EFH (EPA, 2011a).

The confidence level is similarly high for the amount of product applied and application rates, with data ties to surveys cited in the EFH as well as experiments conducted by <u>EPA (1994a)</u>; note that the upper-end amount of product applied is less than the 80<sup>th</sup> percentile value, which should tend to reduce the total exposure estimate.

For the stripping sequence, the wait time per segment has a high level of confidence because the time is based on what is shown on current product labels. The application and scraping times have a lower confidence level because they are based on the <u>EPA (1994a)</u> study, which only included a limited number of experiments using flat panels, which could be considered to require less application and scraping time than more complex shapes. If so, the impact would be to lower the exposure duration. However, no data were available to reasonably determine a range of application and scraping times. This potential impact was mitigated for the upper-end scenarios by either locating the user in the workshop during the wait periods, or by specifying a larger project, which would require more stripper—the most sensitive model input parameters were user location and product amount.

High-quality <u>EPA (1994a)</u> data were available as a quantitative basis for development of the estimates for the fraction of applied chemical mass that is released to the indoor air (see Appendix H-1 – Estimation of Emission Profiles), but there were only a few cases on which the estimates were based. These cases included products with and without vapor retardant ingredients, therefore provide some representation of both types.

Given the potential variability across paint stripping scenarios for estimating consumer exposure, not only for airflow rates, *e.g.*, interzonal air flows, but also for factors such as amount of product used, and application rates and locations in the house, there is some unknown degree of uncertainty in the percentiles of the exposure distribution that are represented by the modeled scenarios. However, as discussed above, input parameter values for the greater-than-central-tendency scenarios were selected to avoid unlikely combinations of high-end or greater values—a "worst-case" scenario.

Therefore, for these scenarios, the general term "upper-end"—instead of more definitive descriptors, *e.g.*, high-end—was used to characterize plausible exposure values greater than central tendency; the more definitive descriptors would imply an inappropriate level of accuracy.

The bathtub stripping scenario is an occupational exposure for the user that was modeled to estimate potential exposures for residential bystanders occupying the ROH during bathtub refinishing. Given the model's sensitivity of concentrations in the ROH to room-of-use air exchange and interzonal air flow, there is uncertainty about the likelihood that a bystander would be exposed to this scenario's ROH concentrations. Thus, EPA characterized the non-user exposures as upper-end to bounding.

### 3.5.3 Uncertainties in the Hazard and Dose-Response Assessments

#### 3.5.3.1 Uncertainties in the Cancer Hazard/Dose-Response Assessments

The cancer IUR for DCM was based on mouse liver and lung tumors reported in a cancer inhalation bioassay (Mennear et al., 1988; NTP, 1986). There is high confidence in the IUR because it was based on the best available dose-response data for liver and lung cancer in mice (EPA, 2011c). In addition, DCM-induced tumorigenesis is supported by both animal and human studies. For instance, female and male rodents (i.e., rats and mice) have reported hepatic and lung cancer following oral or inhalation exposure to DCM. Further support for DCM's carcinogenicity comes from epidemiological studies providing evidence for an association between occupational exposure to DCM and increased risk form some specific cancers (EPA, 2011c). Moreover, multiple *in vivo* and *in vitro* studies support DCM's mutagenic mode of action (EPA, 2011c).

There are a number of uncertainties in the cancer dose-response models and animal-to-human extrapolation methods used to derive the IUR. The major uncertainties are briefly listed and summarized in Table 3-36 from information discussed in the DCM IRIS assessment (EPA, 2011c). Note that the information in Table 3-36 was extracted from Table 5-26 in the DCM IRIS assessment, which covered uncertainties for both oral and inhalation cancer values. Table 3-36 is only summarizing uncertainties for the cancer IUR. Please refer to the DCM IRIS assessment for detailed discussion of these uncertainties (EPA, 2011c).

Table 3 36. Summary of the Uncertainties in the Derivation of the Cancer Inhalation UnitRisk		
Consideration and impact on cancer risk value	Decision	Justification and Discussion
Selection of data set (Selection of an alternative data set could change the recommended cancer risk values.)	NTP (1986) selected as principal inhalation study to derive the cancer IUR.	NTP (1986) inhalation mouse bioassay provides the strongest cancer responses (liver and lung tumors) and the best dose-response data in the animal database.
Selection of target organ (Selection of a target organ could change the recommended cancer risk values.)	Liver and lung were selected as the target organs. Cancer risk values were considered for mammary gland tumors. Potential brain cancer risk and hematopoietic cancer risk were identified as data gaps.	The evidence for DCM-induced mammary gland tumors is less consistent than evidence for liver and lung tumors. Inhalation cancer risk values based on mammary tumors in rats are about one order of magnitude higher than risk values based on liver or lung tumors in mice. No data are available to allow derivation of unit risks based on brain or hematopoietic cancers.

Table 3 36. Summary of the Uncertainties in the Derivation of the Cancer Inhalation UnitRisk				
Selection of extrapolation approach (Selection of extrapolation approach could change the recommended cancer risk values.)	Inhalation data was used to derive IUR.	Uncertainty is lower when deriving cancer IUR from inhalation exposure data rather than using route-to-route extrapolation from oral data.		
Selection of dose metric (Selection of dose metric could change the recommended cancer risk values.)	Tissue-specific GST- metabolism was used as dose metric. Cancer risk estimates based on alternative (whole-body) metrics also examined.	The contribution of CYP pathway to cancer risk is unknown, but strong evidence of GST role in carcinogenesis supports focus on this pathway. Values based on tissue-specific GST metabolism recommended based on evidence of site locality of effects.		
Dose-response modeling (Human risk values could increase or decrease, depending on fits of alternative models)	The multistage dose- response model was used to derive BMD and BMDL values.	The multistage model has biological support and is the model most consistently used in EPA cancer assessments.		
Low-dose extrapolation (Human risk values would be expected to decrease with the application of nonlinear tumor responses in low-dose regions of dose-response curves.)	Inhalation cancer assessment used linear extrapolation of risk in low- dose region.	PBPK model incorporates the metabolic shift and expected nonlinearity (GST dose attenuation) in the exposure-dose relationship across exposure levels. DCM's mutagenic mode of action is supported by <i>in vivo and in</i> <i>vitro</i> studies, resulting in support for the linear low-dose extrapolation approach used in the inhalation cancer assessment.		
Interspecies extrapolation of dosimetry and risk (Alternative values for PBPK model parameters and cross- species scaling factor could increase or decrease human cancer risk values.)	PBPK model and allometric scaling factor were used for the primary dose metric.	Use of rodent and human PBPK models reduced uncertainty due to interspecies differences in toxicokinetics. Examination of impact of different values for key parameters in human model, and sensitivity analysis of rodent PBPK model parameters identified influential metabolic parameters for which limited experimental data exist.		
Sensitive subpopulations (Differences in CYP and GST metabolic rates could change cancer risk values.)	Risk estimates generated for presumed most sensitive (GST-T1+/+) genotype. The CYP variability incorporated into PBPK model.	No data are available to determine the range of human toxicodynamic variability or sensitivity, including whether children are more sensitive than adults.		
Source: Adapted from EPA (2011	Source: Adapted from EPA (2011c) (Table 5-26).			

#### 3.5.3.2 Uncertainties in the Non-Cancer Hazard/Dose-Response Assessments

#### 3.5.3.2.1 Uncertainties in the Acute Hazard/Dose-Response Assessments

Neurotoxicity in adults was the endpoint used to derive the different acute PODs used in the acute inhalation risk assessment. It is possible that younger individuals may respond differently

to DCM exposure in terms of dose, magnitude of response, or different response. Thus, the MOEs presented for the acute occupational and consumer exposure scenarios may under- or overestimate risk to younger age groups for this endpoint.

Furthermore, there are uncertainties about the selected acute PODs since the values (e.g., NOAEL, LOAEL) depend on the current available data and could change as additional studies are published. These uncertainties are minimized in the SMAC POD by considering multiple human observations reporting increased COHb levels after DCM exposure and the extensive CO database supporting a NOAEL COHb level. Likewise, the derivations of the AEGL-1 and -2 PODs considered the DCM and CO human literature in combination with PBPK modeling when setting the AEGL-1 and -2 PODs.

The California acute REL and AEGL PODs were time scaled with the ten Berge equation  $(C^{n*}t=k)^{20}$  and PBPK modeling, respectively, to adjust the experimental exposure duration to the desired acute exposure duration relevant for risk assessment purposes. It is possible that the time extrapolation approach may not accurately represent the concentration-time-response relationship of DCM.

#### 3.5.3.2.2 Uncertainties in the Chronic Hazard/Dose-Response Assessments

There is general high confidence on the hazard database supporting the non-cancer hazard value based on liver toxicity (<u>EPA, 2011c</u>). The inhalation database for DCM includes several well-conducted chronic inhalation studies reporting the liver as the most sensitive target organ (<u>Burek et al., 1984</u>; <u>Nitschke et al., 1988a</u>; <u>NTP, 1986</u>). Both studies identified 500 ppm as the lowest inhalation LOAEL for non-cancer liver lesions.

There is uncertainty about chronic exposure impacts on the nervous system function. The nervous system has been well studied and identified as very sensitive for acute effects. However, there is a paucity of data on chronic neurological impacts, especially developmental neurotoxicity. Likewise, there is limited information about immunotoxicity following chronic exposure to DCM. Existing hazard studies are not sufficient for dose response analysis to provide a lower point of departure than existing adverse findings in the liver from chronic exposures.

A DCM PBPK model was used to extrapolate internal dosimetry from rat liver responses to human risk. Uncertainties in the rat and human dosimetry can arise from the various steps of model development. The DCM PBPK model had a number of uncertainties related to the data set, parameters and assumptions used to simulate the toxicokinetics of DCM for animals and humans (EPA, 2011c). These uncertainties are fully described in the DCM IRIS assessment (EPA, 2011c).

<sup>&</sup>lt;sup>20</sup> In the ten Berge equation ( $C^n * T = k$ , n = 2), C = concentration of the chemical of interest, n=chemical-specific exponent, t=time, and k=constant (NRC, 2001).

The dose metric used in the models is the rate of metabolism to a putative toxic metabolite rather than its concentration expressed as the average or area under the concentration curve of the metabolite. The selected dose-metric fails to account for rodent-to-human differences in clearance or removal of the toxic metabolite. A scaling factor based on body weight (BW) ratios was used to account for this difference assessment (EPA, 2011c). There is uncertainty about the most relevant dose-metric for the noncancer liver effects. This basic research question represents a data gap, and the DCM IRIS assessment did not address this uncertainty quantitatively or qualitatively (EPA, 2011c).

One of the advantages of the DCM PBPK model is that it used a human probabilistic approach to quantitatively address human variability due to pharmacokinetic differences. The model and resulting distributions considered the known differences in human physiology and metabolic capability with regard to DCM dosimetry. The first percentile value of the distributions of HECs served as the non-cancer POD to protect toxicokinetically sensitive individuals. The model did not address toxicodynamic differences in the human population (EPA, 2011c).

## 3.5.4 Uncertainties in the Risk Assessment

MOEs were used to express non-cancer risks associated with acute or chronic exposures to DCM. MOEs are obtained by comparing the hazard values (i.e., PODs) for DCM-related health effects with the exposure concentrations for the specific use scenarios. Given that the MOE is the ratio of the hazard value divided by the exposure, the confidence in the MOEs is directly dependent on the uncertainties in the hazard/dose-response and exposure assessments that supported the hazard and exposure estimates used in the MOE calculations.

The total UF for each acute or chronic POD was the benchmark MOE used to interpret the MOE risk estimates for each use scenario. The UFs accounted for various endpoint and study-specific uncertainties in the hazard values, such as:

1. Animal-to-human extrapolation (UF<sub>A</sub>): The UF<sub>A</sub> accounts for the uncertainties in extrapolating from rodents to humans. In the absence of data, the default UF<sub>A</sub> of 10 is adopted which breaks down to a factor of 3 for toxicokinetic variability and a factor of 3 for pharmacodynamic variability.

For the non-cancer POD reported in the DCM IRIS assessment (i.e., chronic exposure to DCM), the PBPK model accounted for the interspecies extrapolation using rodent pharmacokinetic data to estimate internal doses for a particular dose metric, thus reducing the interspecies toxicokinetic uncertainty to 1. Since the PBPK model did not address interspecies toxicodynamic differences, the total UF<sub>A</sub> of 3 was retained (<u>EPA, 2011c</u>).

2. Inter-individual variation (UF<sub>H</sub>): The UF<sub>H</sub> accounts for the variation in sensitivity within the human population. In the absence of data, the default UF<sub>H</sub> of 10 is adopted which breaks

down to a factor of 3 for toxicokinetic variability and a factor of 3 for toxicodynamic variability.

For the non-cancer POD reported in the DCM IRIS assessment (i.e., chronic exposure to DCM), the PBPK model reduced the human toxicokinetic variability to 1, but not the human toxicodynamic variability. Thus, the total UF<sub>H</sub> was 3. This is because the PBPK model does not address the uncertainties regarding the susceptibility of the human subpopulations to DCM exposure and the extent of toxicodynamics variability (<u>EPA, 2011c</u>).

In the absence of PBPK modeling, a  $UF_H$  of 10 was retained for the SMAC and the California acute REL POD to account for variability within the human population.

As for the AEGL PODs, PBPK modeling was used to derive the AEGL PODs and a UF<sub>H</sub> of 3 and 1 were used for the AEGL-1 and -2 PODs, respectively. Since susceptibility for gross CNS-depressing effects do not vary by more than a factor of 2- to 3-fold in humans, a UF<sub>H</sub> of 3 was applied for the AEGL-1 POD (<u>NAC, 2008</u>). On the other hand, a UF<sub>H</sub> of 1 was considered sufficient for the AEGL-2 POD since the toxic effects studied were less severe than those defined for AEGL-2 and the application of a greater value would result in values that were inconsistent with the available human data. Similarly, an intraspecies UF of 1 was applied for the effects associated with COHb formation because the POD was based on experimental data on the most susceptible individuals (*i.e.*, coronary artery disease patients), which is also protective for other human subpopulations (<u>NRC, 2008, 2010</u>).

LOAEL-to-NOAEL extrapolation (UF<sub>L</sub>): The UF<sub>L</sub> accounts for the uncertainty in extrapolating from a LOAEL to a NOAEL. A value of 10 is the standard default UF<sub>L</sub> value, although lower values (e.g., 3) can be used if the effect is considered minimally adverse at the LOAEL or is an early marker for an adverse effect.

OEHHA applied a UF<sub>L</sub> of 6 to the California acute REL POD to generate a NOAEL (<u>OEHHA</u>, <u>2008</u>), but the basis for the value selection was not explained. EPA/OPPT retained the UF<sub>L</sub> of 6 as part of the composite factors comprising the total UF (i.e., benchmark MOE).

Unlike cancer risks, an MOE exceeding the benchmark MOEs is an indicator that there is a potential risk and cannot be translated to a probability that certain adverse health effects would occur. Also, those MOEs that exceed but remain close to the benchmark MOE do not necessarily mean that adverse effects would occur.

The non-cancer risks for the occupational chronic exposures assumed that the human health risks are constant for specific hypothetical scenarios based on variations of exposure conditions (i.e., type of respiratory, exposure frequency, working years). However, risks could be under- or over-estimated depending on the real exposure profile of the workers using DCM-paint strippers.

Regarding exposure to DCM through the skin, the impact of dermal exposures on human health risks was not assessed in this assessment for the consumer and occupational scenarios. Exclusion of dermal exposures is expected to underestimate the risks of the selected DCM use. This would likely be an issue of concern in those exposure scenarios that resulted in a "no-risk" finding, especially those that reported MOEs close to the benchmark MOE, but still above the benchmark.

The assessment did not consider the cumulative exposure from other uses of DCM around the house or at the workplace setting. Thus, the current risk assessment on the use of DCM-based paint strippers is likely to underestimate the human risks.

As discussed previously, the cancer risk estimates were based on the assumption of linearity in the relationship between DCM exposure and probability of cancer. Uncertainties are introduced in the cancer risks when there is limited information justifying the liner cancer dose-response model when compared to other available models. In the case of DCM, the cancer IUR was based on multiple *in vivo* and *in vitro* studies supporting a mutagenic mode of action (EPA, 2011c).

## 3.6 CONCLUSIONS OF THE HUMAN HEALTH RISK ASSESSMENT

EPA/OPPT's risk assessment focuses on the occupational and consumer use of DCM-based paint strippers. In this assessment, EPA/OPPT estimates that over 230,000 workers nationwide are directly exposed to DCM from DCM-based strippers. This estimate only accounts for workers performing the paint stripping using DCM and does not include other workers ("occupational bystanders") within the facility who are indirectly exposed. No data were available to estimate the number of consumers and residential bystanders exposed to DCM during the use of paint strippers.

In summary, the risk assessment showed the following risk findings:

#### Cancer Risks Associated With Chronic Exposures to DCM:

- There are cancer risk concerns for workers exposed to DCM that are employed at various industries handling DCM-containing paint strippers.
- Many of the occupational scenarios exceed at least one of the target cancer risks of 10<sup>-4</sup>, 10<sup>-5</sup> and 10<sup>-6</sup>.
- The greatest cancer risks occur for workers handling DCM-based paint strippers with no respiratory protection for an extended period of time.

#### Non-Cancer Risks Associated With Chronic Exposures to DCM:

- There are non-cancer risks for liver effects for most workers using DCM-based paint strippers in relevant industries, with the exception of the art renovation and conservation industry.
- Non-cancer risks occur for most workers handling DCM-based paint strippers with or without respiratory protection for various exposure scenarios. Among all of the occupational scenarios, the greatest risk concern is for workers engaging in long-term use of the product (i.e., 250 days/year for 40 years) with no respiratory protection.
- Non-cancer risks are not found when workers reduce their exposure to DCM-based strippers by taking all three of the following actions; wearing respiratory protection (i.e., respirator with at least an assigned protection factor of 50), limiting exposure to central tendency exposure conditions (i.e., 125 days/year for 20 years) and working in facilities with low-end DCM air concentrations.

#### Non-Cancer Risks Associated With Acute Exposures to DCM:

- There are acute risks for neurological effects for most workers using DCM-based paint strippers. These risks are apparent in the presence or absence of respiratory protection.
- There are concerns for incapacitating effects in workers handing DCM-containing paint strippers on an acute/short-term basis with no respiratory protection. These concerns are

also present for workers wearing different types of respirators (e.g., APF 10, APF 25) while performing paint stripping in industries with high exposure to DCM.

- There are acute risks for neurological effects for consumers of DCM-based paint strippers at residential settings. Also, bystanders are at risk while staying in the residence when paint strippers are being applied.
- There are concerns for discomfort/non-disabling and incapacitating effects for consumers exposed to DCM while applying the product or staying in the residence after completion of the stripping task. These concerns are also present for residential bystanders in some scenarios when exposure conditions are at the highest in the rest of the house after completing the paint stripping task.
- Application of DCM-based paint strippers in a bathroom generates unsafe exposure conditions for the user of the product, but not residential bystanders. DCM concentrations may reach levels associated with non-disabling and incapacitating effects for the user applying the product. User relocation to the rest of the house after completing the paint stripping task may also produce non-disabling and incapacitating effects as DCM's internal dose builds up in the body over time.

## **4** References

- ACGIH (American Conference of Governmental Industrial Hygienists). 2001. *Documentation of the Threshold Limit Values and Biological Exposure Indices for Dichloromethane*. Cincinnati, OH. (as cited in EPA, 2011c).
- Alexander, H. C., W. M. McCarty, and E. A. Bartlett. 1978. *Toxicity of Perchloroethylene, Trichloroethylene, 1,1,1-Trichloroethane, and Methylene Chloride to Fathead Minnows*. Bulletin of Environmental Contamination and Toxicology, 20(3), 344-352.
- Alexeeff, G. V., and W. W. Kilgore. 1983. *Learning Impairment in Mice Following Acute Exposure to Dichloromethane and Carbon Tetrachloride*. Journal of Toxicology and Environmental Health, 11(4-6), 569-581. (as cited in EPA, 2011c).
- Allred, E. N., E. R. Bleecker, B. R. Chaitman, T. E. Dahms, S. O. Gottlieb, J. D. Hackney, D. Hayes, M. Pagano, R. H. Selvester, S. M. Walden, and J. Warren. 1989a. Acute Effects of Carbon Monoxide Exposure on Individuals with Coronary Artery Disease. Research Report No. 25. Health Effects Institute, Cambridge, MA. (as cited in NRC, 2010).
- Allred, E. N., E. R. Bleecker, B. R. Chaitman, T. E. Dahms, S. O. Gottlieb, J. D. Hackney, M. Pagano, R. H. Selvester, S. M. Walden, and J. Warren. 1989b. Short-Term Effects of Carbon Monoxide Exposure on the Exercise Performance of Subjects with Coronary Artery Disease. The New England Journal of Medicine, 321(23), 1426-1432. (as cited in NRC, 2010).
- Allred, E. N., E. R. Bleecker, B. R. Chaitman, T. E. Dahms, S. O. Gottlieb, J. D. Hackney, M. Pagano, R. H. Selvester, S. M. Walden, and J. Warren. 1991. *Effects of Carbon Monoxide* on Myocardial Ischemia. Environmental Health Perspectives, 91, 89-132. (as cited in NRC, 2010).
- Andersen, M. E., H. J. Clewell, 3rd, M. L. Gargas, M. G. MacNaughton, R. H. Reitz, R. J. Nolan, and M. J. McKenna. 1991. *Physiologically Based Pharmacokinetic Modeling with Dichloromethane, Its Metabolite, Carbon Monoxide, and Blood Carboxyhemoglobin in Rats and Humans*. Toxicology and Applied Pharmacology, 108(1), 14-27. (as cited in NAC, 2008 and NRC, 1996).
- Anderson, E. W., R. J. Andelman, J. M. Strauch, N. J. Fortuin, and J. H. Knelson. 1973. Effect of Low-Level Carbon Monoxide Exposure on Onset and Duration of Angina Pectoris. A Study in Ten Patients with Ischemic Heart Disease. Annals of Internal Medicine, 79(1), 46-50. (as cited in NRC, 2010).

- Anundi, H., S. Langworth, G. Johanson, M. L. Lind, B. Akesson, L. Friis, N. Itkes, E. Soderman, B.
   A. Jonsson, and C. Edling. 2000. *Air and Biological Monitoring of Solvent Exposure During Graffiti Removal*. Int Arch Occup Environ Health, 73(8), 561-569.
- Anundi, H., M. L. Lind, L. Friis, N. Itkes, S. Langworth, and C. Edling. 1993. *High Exposures to Organic Solvents among Graffiti Removers*. Int Arch Occup Environ Health, 65(4), 247-251.
- Aranyi, C., W. J. O'Shea, J. A. Graham, and F. J. Miller. 1986. *The Effects of Inhalation of Organic Chemical Air Contaminants on Murine Lung Host Defenses*. Fundamental and Applied Toxicology, 6(4), 713-720. (as cited in EPA, 2011c).
- Aronow, W. S., C. N. Harris, M. W. Isbell, S. N. Rokaw, and B. Imparato. 1972. *Effect of Freeway Travel on Angina Pectoris*. Annals of Internal Medicine, 77(5), 669-676. (as cited in NRC, 2010).
- Ash, M., and I. Ash. 2009. Methylene Chloride. In *Specialty Chemicals Monographs* (pp. 1361). Synapse Information Resources, Inc., Endicott, NY.
- ASTM (ASTM International). 1997. Standard Practice for Estimation of Short-Term Inhalation Exposure to Volatile Organic Chemicals Emitted from Bedding Sets. Designation: D 6178-97 (Reapproved 2008). West Conshohocken, PA.
- Astrand, I., P. Ovrum, and A. Carlsson. 1975. *Exposure to Methylene Chloride. I. Its Concentration in Alveolar Air and Blood During Rest and Exercise and Its Metabolism*. Scandinavian Journal of Work, Environment & Health, 1(2), 78-94. (as cited in NRC, 1996).
- ATSDR (Agency for Toxic Substances and Disease Registry). 1990. *Methylene Chloride Toxicity. Case Studies in Environmental Medicine*. PB 85-241529. U.S. Department of Health and Human Services, Atlanta, GA. (as cited in ATSDR, 2000).
- ATSDR (Agency for Toxic Substances and Disease Registry). 2000. *Toxicological Profile for Methylene Chloride*. Division of Toxicology/Toxicology Information Branch, Atlanta, GA. <u>http://www.atsdr.cdc.gov/toxprofiles/tp14.pdf</u>.
- ATSDR (Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services). 2010. Addendum to the Toxicological Profile for Methylene Chloride. Division of Toxicology and Environmental Medicine, Atlanta, GA. <u>http://www.atsdr.cdc.gov/toxprofiles/methylene\_chloride\_addendum.pdf</u>.
- Barone, S., Jr., K. P. Das, T. L. Lassiter, and L. D. White. 2000. Vulnerable Processes of Nervous System Development: A Review of Markers and Methods. Neurotoxicology, 21(1-2), 15-36.

- Barry, K. H., Y. Zhang, Q. Lan, S. H. Zahm, T. R. Holford, B. Leaderer, P. Boyle, H. D. Hosgood, 3rd, S. Chanock, M. Yeager, N. Rothman, and T. Zheng. 2011. *Genetic Variation in Metabolic Genes, Occupational Solvent Exposure, and Risk of Non-Hodgkin Lymphoma*. American Journal of Epidemiology, 173(4), 404-413. (as cited in EPA, 2011c).
- Bornschein, R. L., L. Hastings, and J. M. Manson. 1980. *Behavioral Toxicity in the Offspring of Rats Following Maternal Exposure to Dichloromethane*. Toxicology and Applied Pharmacology, 52(1), 29-37. (as cited in EPA, 2011c).
- Bos, P. M., M. J. Zeilmaker, and J. C. van Eijkeren. 2006. *Application of Physiologically Based Pharmacokinetic Modeling in Setting Acute Exposure Guideline Levels for Methylene Chloride*. Toxicological Sciences, 91(2), 576-585. (as cited in EPA, 2011c).
- Brown, J. (U.S. Environmental Protection Agency). *Formulations Spreadsheet File*. Personal communication with: Conrad, F. (U.S. Environmental Protection Agency, Washington, DC), June 2012.
- Buccafusco, R. J., S. J. Ells, and G. A. LeBlanc. 1981. *Acute Toxicology of Priority Pollutants to Bluegill (Lepomis Macrochirus)*. Bulletin of Environmental Contamination and Toxicology, 26(4), 446-452.
- Burek, J. D., K. D. Nitschke, T. J. Bell, D. L. Wackerle, R. C. Childs, J. E. Beyer, D. A. Dittenber, L. W. Rampy, and M. J. McKenna. 1984. *Methylene Chloride: A Two-Year Inhalation Toxicity and Oncogenicity Study in Rats and Hamsters*. Fundamental and Applied Toxicology, 4(1), 30-47.
- Cal EPA (California Environmental Protection Agency). 2000. *Public Health Goals for Chemicals in Drinking Water. Dichloromethane (Methylene Chloride, DCM)*. Office of Environmental Health Hazard Assessment, Sacramento, CA. <u>http://oehha.ca.gov/water/phg/pdf/dcm.pdf</u>.
- Carlsson, A., and M. Hultengren. 1975. *Exposure to Methylene Chloride. Metabolism of 14c-Labelled Methylene Chloride in Rat.* Scandinavian Journal of Work, Environment & Health, 1(2), 104-108. (as cited in ATSDR, 2000).
- CDC (Centers for Disease Control and Prevention). 2009. *Fourth National Report on Human Exposure to Environmental Chemicals*. Department of Health and Human Services, Atlanta, GA. <u>http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf</u>.
- CDC (Centers for Disease Control and Prevention). 2012. *Tub Refinisher Died Due to Methylene Chloride Overexposure While Stripping a Bathtub*. Michigan case report: 10MI013. Atlanta, GA. <u>http://www.cdc.gov/niosh/face/stateface/mi/10MI013.html</u>.

- CDHS/EPA (California Department of Health Services/U.S. Environmental Protection Agency). 2006. Assessment, Development and Demonstration of Alternatives for Five Emerging Solvents. Prepared by the Institute for Research and Technical Assistance for the California Department of Health Services and U.S. Environmental Protection Agency. www.cdph.ca.gov/programs/hesis/Documents/emergingsolvents.pdf.
- Chen, Q., and W. Xu. 1998. *A Zero-Equation Turbulence Model for Indoor Airflow Simulation*. Energy and Buildings, 28(2), 137-144.
- Cheng, K. C., V. Acevedo-Bolton, R. T. Jiang, N. E. Klepeis, W. R. Ott, O. B. Fringer, and L. M. Hildemann. 2011. *Modeling Exposure Close to Air Pollution Sources in Naturally Ventilated Residences: Association of Turbulent Diffusion Coefficient with Air Change Rate*. Environ Sci Technol, 45(9), 4016-4022.
- Cherrie, J. W. 1999. The Effect of Room Size and General Ventilation on the Relationship between near and Far-Field Concentrations. Appl Occup Environ Hyg, 14(8), 539-546.
- Cherry, N., H. Venables, and H. A. Waldron. 1983. *The Acute Behavioural Effects of Solvent Exposure*. The Journal of the Society of Occupational Medicine, 33(1), 13-18. (as cited in EPA, 2011c).
- Cherry, N., H. Venables, H. A. Waldron, and G. G. Wells. 1981. *Some Observations on Workers Exposed to Methylene Chloride*. British Journal of Industrial Medicine, 38(4), 351-355. (as cited in EPA, 2011c).
- Chester, D., K. D. Rosenman, G. R. Grimes, K. Fagan, and D. N. Castillo. 2012. *Fatal Exposure to Methylene Chloride among Bathtub Refinishers -United States, 2000–2011*. Morbidity and Mortality Weekly Report, 61(7), 119-122.
- CPSC (U.S. Consumer Product Safety Commission). 1987. *Statement of Policy for Methylene Chloride*. Washington, DC. <u>http://www.cpsc.gov/en/newsroom/news-</u> <u>releases/1987/statement-of-policy-for-methylene-chloride/</u> (Accessed June 19, 2014).
- CPSC (U.S. Consumer Product Safety Commission). 1992. *Methylene Chloride Consumer Products Use Survey Findings*. Prepared by L. Boast from Abt Associates, Inc., for the U.S. Consumer Product Safety Commission, Bethesda, MD.
- DHHS (Department of Health and Human Services). 2012. *Household Products Database*. Bethesda, MD. <u>http://householdproducts.nlm.nih.gov/</u> (accessed in June 2014).
- Dill, D. C., P. G. Murphy, and M. A. Mayes. 1987. *Toxicity of Methylene Chloride to Life Stages of the Fathead Minnow, Pimephales Promelas Rafinesque*. Bulletin of Environmental Contamination and Toxicology, 39(5), 869-876.

- Dilling, W. L., N. B. Tefertiller, and G. J. Kallos. 1975. Evaporation Rates and Reactivities of Methylene Chloride, Chloroform, 1,1,1-Trichloroethane, Trichloroethylene, Tetrachloroethylene, and Other Chlorinated Compounds in Dilute Aqueous Solutions. Environ Sci Technol, 9(9), 833-838.
- DiVincenzo, G. D., and C. J. Kaplan. 1981. *Uptake, Metabolism, and Elimination of Methylene Chloride Vapor by Humans*. Toxicology and Applied Pharmacology, 59(1), 130-140. (as cited in NAC, 2008 and NRC, 1996).
- DiVincenzo, G. D., F. J. Yanno, and B. D. Astill. 1972. *Human and Canine Exposures to Methylene Chloride Vapor*. American Industrial Hygiene Association Journal, 33(3), 125-135. (as cited in ATSDR, 2000 and NAC, 2008).
- Dow. 1999. Alternatives to Chlorinated Solvents. Dow Newsletter, 3(4), 1-4.
- DTSC (California Department of Toxic Control Substances). 2010. *Chemical Lists*. Sacramento, CA. https://dtsc.ca.gov/SCP/ChemList.cfm (accessed on July 9, 2014).
- DuPont (DuPont Chemical Company). 1982. *Inhalation Approximate Lethal Concentration of Methylene Chloride with Cover Letter Dated 081092*. Study conducted by Haskell Laboratories,(July 23, 1982), Newark, DE. Doc #88-920009163. (as cited in NAC, 2008).
- EC (European Commision). 1999. *Methylene Chloride: Advantages and Drawbacks of Possible Market Restrictions in the EU*. STB-99-53 Final. Brussels, Belgium.
- EC (European Commission). 2004. Effectiveness of Vapour Retardants in Reducing Risks to Human Health from Paint Strippers Containing Dichloromethane. Brussels, Belgium.
- EC (European Commission). 2010. Commission Regulation (EU) No 276/2010 of 31 March 2010 Amending Regulation (EC) No 1907/2006 of the European Parliament and of the Council on the Registration, Evaluation, Authorisation and Restriction of Chemicals (Reach) as Regards Annex Xvii (Dichloromethane, Lamp Oils and Grill Lighter Fluids and Organostannic Compounds). Official Journal of the European Union. <u>http://eurlex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2010:086:0007:0012:en:PDF</u>.
- ECB (European Chemicals Bureau). 2000. *IUCLID Dataset for Dichloromethane*. European Chemical Substances Information System, Joint Research Centre, Institute for Health and Consumer Protection, European Chemicals Bureau, Helsinki, Finland.
- ECHA (European Chemicals Agency). 2011. Proposal for Identification of a Substance as a Category 1a or 1b CMR, PBT, vPvB or a Substance of an Equivalent Level of Concern: Annex Xv Dossier: Identification of 1-Methyl-2-Pyrrolidone as SVHC. Helsinki, Finland.

- Enander, R. T., H. J. Cohen, D. M. Gute, L. C. Brown, A. M. Desmaris, and R. Missaghian. 2004. *Lead and Methylene Chloride Exposures among Automotive Repair Technicians*. Journal of occupational and environmental hygiene, 1(2), 119-125.
- Environment Canada. 2003a. Code of Practice for the Reduction of Dichloromethane Emissions from the Use of Paint Strippers in Commercial Furniture Refinishing and Other Stripping Applications. EPS 1/CC/4. June 2003, Ottawa, Canada. <u>http://www.ec.gc.ca/lcpe-</u> <u>cepa/default.asp?lang=En&n=B7812356-1</u>.
- Environment Canada. 2003b. Notice Requiring the Preparation and Implementation of Pollution Prevention Plans in Respect of Dichloromethane, November 29, 2003. Ottawa, Canada. <u>http://www.ec.gc.ca/planp2-p2plan/default.asp?lang=En&n=540C2673-1</u>.
- EPA (US Environmental Protection Agency). 1978. *In-Depth Studies on Health and Environmental Impacts of Selected Water Pollutants*. Duluth, MN.
- EPA (US Environmental Protection Agency). 1980. Acquisition and Chemical Analysis of Mother's Milk for Selected Toxic Substances. EPA 560/13-80-029 Office of Pesticides and Toxic Substances, Washington, DC. (as cited in ATSDR, 2000).
- EPA (US Environmental Protection Agency). 1986a. *Guidelines for Mutagenicity Risk Assessment*. EPA/630/R-98/003. Risk Assessment Forum, Washington, DC. <u>http://www.epa.gov/iris/backgrd.html</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Agency). 1986b. *Guidelines for the Health Risk Assessment of Chemical Mixtures*. EPA/630/R-98/002. Risk Assessment Forum, Washington, DC. <a href="http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=22567">http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=22567</a>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 1987. *Household Solvent Products: A National Usage Survey*. Office of Toxic Substances, Washington, DC.
- EPA (US Environmental Protection Agency). 1988. Recommendations for and Documentation of Biological Values for Use in Risk Assessment. EPA/600/6-87/008. Office of Research and Development, Cincinnati, OH. <u>http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=34855</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 1991. *Guidelines for Developmental Toxicity Risk Assessment*. EPA/600/FR-91/001. Risk Assessment Forum, Washington, DC. <u>http://ofmpub.epa.gov/eims/eimscomm.getfile?p\_download\_id=4560</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 1992a. *Classification Criteria for Environmental Toxicity and Fate of Industrial Chemical*. Office of Pollution Prevention and Toxics, Washington, DC.

- EPA (US Environmental Protection Agency). 1992b. *Guidelines for Exposure Assessment*. EPA/600/Z-92-001. Risk Assessment Forum, Washington, DC. <u>http://ofmpub.epa.gov/eims/eimscomm.getfile?p\_download\_id=429103</u>.
- EPA (US Enviornmental Protection Agency). 1993a. *Draft Guidance on LADDs and APDRs*. Office of Pollution Prevention and Toxics, Washington, DC.
- EPA (US Environmental Protection Agency). 1993b. *Locating and Estimating Air Emissions from Sources of Methylene Chloride*. EPA-454/R-93-006. Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- EPA (US Environmental Protection Agency). 1994a. *Consumer Exposure to Paint Stripper Solvents*. Office of Pollution Prevention and Toxics, Washington, DC.
- EPA (US Environmental Protection Agency). 1994b. *Interim Policy for Particle Size and Limit Concentration Issues in Inhalation Toxicity Studies*. Office of Pesticide Products, Washington, DC. <u>http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=186068</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 1994c. Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry. EPA/600/8-90/066F.
   Office of Research and Development, Research Triangle Park, NC.
   <a href="http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=71993">http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=71993</a>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 1994d. OPPT Chemical Fact Sheet. Chemicals in the Environment: Methylene Chloride (Dichloromethane) (CAS No. 75-09-2). EPA 749-F-94-018. Office of Pollution Prevention and Toxics, Washington, DC.
   www.epa.gov/chemfact/f\_dcm.txt.

EPA (US Environmental Protection Agency). 1995a. *Estimation of Distributions for Residential Air Exchange Rates*. Office of Pollution Prevention and Toxics, Washington, DC. <u>http://nepis.epa.gov/Exe/ZyNET.exe/910063GS.TXT?ZyActionD=ZyDocument&Client=EP</u> <u>A&Index=1995+Thru+1999&Docs=&Query=&Time=&EndTime=&SearchMethod=1&Toc</u> <u>Restrict=n&Toc=&TocEntry=&QField=&QFieldYear=&QFieldMonth=&QFieldDay=&IntQFi</u> <u>eldOp=0&ExtQFieldOp=0&XmlQuery=&File=D%3A%5Czyfiles%5CIndex%20Data%5C95th</u> <u>ru99%5CTxt%5C0000025%5C910063GS.txt&User=ANONYMOUS&Password=anonymo</u> <u>us&SortMethod=h%7C-</u> <u>&MaximumDocuments=1&FuzzyDegree=0&ImageQuality=r75g8/r75g8/x150y150g16/i4</u> <u>25&Display=p%7Cf&DefSeekPage=x&SearchBack=ZyActionL&Back</u>.

EPA (US Environmental Protection Agency). 1995b. *The Use of the Benchmark Dose Approach in Health Risk Assessment*. EPA/630/R-94/007. Risk Assessment Forum, Washington, DC. http://www.epa.gov/raf/publications/useof-bda-healthrisk.htm. (as cited in EPA, 2011c).

- EPA (US Environmental Protection Agency). 1996a. *Consumer/Small Shop Paint Stripping Use Cluster. Draft Risk Assessment Report: Engineering Assessment*. Chemical Engineering Branch, Economics, Exposure and Technology Division, Washington, DC.
- EPA (US Environmental Protection Agency). 1996b. *Guidelines for Reproductive Toxicity Risk Assessment*. EPA/630/R-96/009. Risk Assessment Forum, Washington, DC. <u>http://www.epa.gov/raf/publications/pdfs/REPRO51.PDF</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 1997. Use of Small Chamber Data to Estimate and Model Chemical Emissions from Latex and Alkyd Paints. Prepared by GEOMET Technologies, Inc., under EPA contract# 68-D3-0013 for the Office of Pollution Prevention and Toxics, Washington, DC.
- EPA (US Environmental Protection Agency). 1998a. *Guidelines for Neurotoxicity Risk Assessment*. EPA/630/R-95/001F. Risk Assessment Forum, Washington, DC. <u>http://www.epa.gov/raf/publications/pdfs/NEUROTOX.PDF</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 1998b. *Methods for Exposure-Response Analysis for Acute Inhalation Exposure to Chemicals (External Review Draft)*. EPA/600/R-98/051. Office of Research and Development, Washington, DC.
- EPA (US Environmental Protection Agency). 1999. *Category for Persistent, Bioacculative, and Toxic New Chemical Substances*. 64 Federal Register 213 (November 4, 1999), pp. 60194-60204.
- EPA (US Environmental Protection Agency). 2000a. *Air Quality Criteria for Carbon Monoxide*. EPA 600/P-99/001F. Office of Research and Development, Washington, DC. <u>http://www.epa.gov/NCEA/pdfs/coaqcd.pdf</u>. (as cited in NRC, 2010).
- EPA (US Environmental Protection Agency). 2000b. Benchmark Dose Technical Guidance Document [External Review Draft]. EPA/630/R-00/001. Risk Assessment Forum, Washington, DC. <u>http://www.epa.gov/raf/publications/benchmark-dose-doc-draft.htm</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 2000c. *Science Policy Council Handbook: Risk Characterization*. EPA/100/B-00/002. Office of Science Policy, Washington, DC. <a href="http://www.epa.gov/osa/spc/pdfs/rchandbk.pdf">http://www.epa.gov/osa/spc/pdfs/rchandbk.pdf</a>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 2000d. *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures*. EPA/630/R-00/002. Risk Assessment

Forum, Washington, DC. <u>http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20533</u>. (as cited in EPA, 2011c).

- EPA (US Environmental Protection Agency). 2001. Risk Assessment Guidance for Superfund: Volume 3 - Part a, Process for Conducting Probabilistic Risk Assessment. EPA/540-R-02-002. Office of Emergency and Remedial Response, Washington, DC. <u>http://www.epa.gov/oswer/riskassessment/rags3adt/</u>.
- EPA (US Environmental Protection Agency). 2002. A Review of the Reference Dose and Reference Concentration Processes. EPA/630/P-02/002F. Risk Assessment Forum, Washington, DC. <u>http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=51717</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 2003. *Source Ranking Database, Volume 1: Guide and Documentation*. Office of Pollution Prevention and Toxics, Washington, DC. <u>http://www.epa.gov/oppt/exposure/pubs/srd.htm</u>.
- EPA (US Environmental Protection Agency). 2005a. *Guidelines for Carcinogen Risk Assessment*. EPA/630/P-03/001F. Risk Assessment Forum, Washington, DC. <u>http://www.epa.gov/cancerguidelines/</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 2005b. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. EPA/630/R-03/003F. Risk Assessment Forum, Washington, DC. <u>http://www.epa.gov/cancerguidelines/guidelinescarcinogen-supplement.htm</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 2006a. A Framework for Assessing Health Risk of Environmental Exposures to Children. EPA/600/R-05/093F. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. <u>http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=158363</u>. (as cited in EPA, 2011c).
- EPA (US Environmental Protection Agency). 2006b. *Peer Review Handbook (3rd Edition)*. EPA/100/B-06/002. Science Policy Council, Washington, DC. <u>http://www.epa.gov/peerreview/pdfs/peer review handbook 2006.pdf</u>. (as cited in EPA, 2011c).
- EPA (US Enviornmental Protection Agency). 2006c. *Proposed Test Rule for Certain Chemicals on the ATSDR/EPA CERCLA Priority List of Hazardous Substances*. 71 Federal Register 203 (October 20, 2006), pp. 61926-61944.
- EPA (US Environmental Protection Agency). 2007. *Technical Support Document for Proposed Rule: National Emission Standards for Hazardous Air Pollutants: Paint Stripping Operations at Area Sources*. OAQPS/Sector Policies and Programs Division, Research

Triangle Park, NC. <u>http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OAR-2005-0526-0019</u>.

- EPA (US Environmental Protection Agency). 2008. National Emission Standards for Hazardous Air Pollutants: Paint Stripping and Miscellaneous Surface Coating Operations at Area Sources. 73 Federal Register 6 (January 9, 2008), pp. 1738-1768.
- EPA (US Environmental Protection Agency). 2009. Risk Assessment Guidance for Superfund.
   Volume I: Human Health Evaluation Manual (Part F, Supplemental Guidance for Inhalation Risk Assessment). EPA-540-R-070-002. Office of Superfund Remediation and Technology Innovation, Washington, DC.
   www.epa.gov/oswer/riskassessment/ragsf/pdf/partf\_200901\_final.pdf.
- EPA (US Environmental Protection Agency). 2010a. *Multi-Chamber Concentration and Exposure Model (MCCEM) Version 1.2*. Washington, DC. <u>http://www.epa.gov/oppt/exposure/pubs/mccem.htm</u> (accessed on October 31, 2012).
- EPA (US Environmental Protection Agency). 2010b. *National Primary Drinking Water Regulations*. 40 CFR 141.61, Washington, DC. <u>http://www.gpo.gov/fdsys/pkg/CFR-2010-title40-vol1/content-detail.html</u>.
- EPA (US Environmental Protection Agency). 2011a. *Exposure Factors Handbook*. EPA/600R-090052F. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. <u>http://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252</u>.
- EPA (US Environmental Protection Agency). 2011b. Integrated Risk Information System (IRIS) Glossary. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. <u>http://ofmpub.epa.gov/sor\_internet/registry/termreg/searchandretrieve/glossariesand keywordlists/search.do?details=&glossaryName=IRIS%20Glossary</u> (accessed on March 1, 2014).
- EPA (US Environmental Protection Agency). 2011c. *Toxicological Review of Dichloromethane* (*Methylene Chloride; CAS No. 75-09-2*). EPA/635/R-10/003F. Integrated Risk Information System, Office of Research and Development, Washington, DC. <u>http://www.epa.gov/iris/toxreviews/0070tr.pdf</u>.
- EPA (US Environmental Protection Agency). 2011d. *TRI.Net*. Washington, DC. <u>http://www.epa.gov/tri/tridotnet/index.html</u> (accessed on August 15, 2012).
- EPA (US Environmental Protection Agency). 2011e. *TSCA Inventory Update Modifications; Chemical Data Reporting*. 76 Federal Register 158, pp. 50816-50879.

- EPA (US Environmental Protection Agency). 2012a. *Benchmark Dose Technical Guidance*. EPA/100/R-12/001. Risk Assessment Forum, Washington, DC. <u>http://www.epa.gov/raf/publications/pdfs/benchmark\_dose\_guidance.pdf</u>.
- EPA (US Environmental Protection Agency). 2012b. Estimation Programs Interface Suite™ for Microsoft® Windows, V4.10. Office of Pollution Prevention and Toxics, Washington, DC. <u>http://www.epa.gov/opptintr/exposure/pubs/episuitedl.htm</u> (accessed on March 19, 2012).
- EPA (US Environmental Protection Agency). 2012c. *Identification and Listing of Hazardous Waste*. 40 CFR 261, Washington, DC. <u>http://www.gpo.gov/fdsys/pkg/CFR-2012-title40-vol27/xml/CFR-2012-title40-vol27-part261.xml#seqnum261.30</u>.
- EPA (US Environmental Protection Agency). 2012d. *TSCA Work Plan Chemicals: Methods Document*. Office of Pollution Prevention and Toxics, Washington, DC. <u>www.epa.gov/oppt/existingchemicals/pubs/wpmethods.pdf</u>.
- EPA (US Environmental Protection Agency). 2013. 2012 Chemical Data Report (CDR). Washington, DC. <u>http://www.epa.gov/tri/index.htm</u> (accessed in June 2014).
- EPA (US Environmental Protection Agency). 2014. *Toxic Chemical Release Reporting: Community Right-to-Know*. 40 CFR 372.65, Washington, DC. <u>http://www.ecfr.gov/cgi-bin/text-idx?c=ecfr&tpl=/ecfrbrowse/Title40/40cfr372\_main\_02.tpl</u>.
- Estill, C. F., and A. B. Spencer. 1996. *Case Study: Control of Methylene Chloride Exposures During Furniture Stripping*. Am Ind Hyg Assoc J, 57(1), 43-49.
- EU (European Commission). 2007. Impact Assessment of Potential Restrictions on the Marketing and Use of Dichloromethane in Paint Strippers. Revised Final Report-Annexes. Directorate-General Enterprise and Industry. <u>http://ec.europa.eu/enterprise/sectors/chemicals/files/markrestr/j549\_dcm\_annex\_en.</u> <u>pdf</u>.
- FDA (U.S. Food and Drug Administration). 1989. Use of Methylene Chloride as an Ingredient of Cosmetic Products. Washington, DC. <u>http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?fr=700.19</u>.
- Fisher, J., D. Mahle, L. Bankston, R. Greene, and J. Gearhart. 1997. Lactational Transfer of Volatile Chemicals in Breast Milk. American Industrial Hygiene Association Journal, 58(6), 425-431. (as cited in ATSDR, 2000).
- Forster, P., V. Ramaswamy, P. Artaxo, T. Berntsen, R. Betts, D.W. Fahey, J. Haywood, J. Lean,D.C. Lowe, G. Myhre, J. Nganga, R. Prinn, G. Raga, M. Schulz and R. Van Dorland. 2007.Changes in Atmospheric Constituents and in Radiative Forcing. In Solomon, S., D. Qin, M.

Manning, Z. Chen, M. Marquis, K.B. Averyt, M.Tignor and H.L. Miller, *Climate Change* 2007: *The Physical Science Basis*. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change, Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA. http://www.ipcc.ch/pdf/assessment-report/ar4/wg1/ar4\_wg1\_full\_report.pdf.

- Frey, H. C., and S. R. Patil. 2002. *Identification and Review of Sensitivity Analysis Methods*. Risk analysis : an official publication of the Society for Risk Analysis, 22(3), 553-578.
- Furtaw, E. J., Jr., M. D. Pandian, D. R. Nelson, and J. V. Behar. 1996. Modeling Indoor Air Concentrations near Emission Sources in Imperfectly Mixed Rooms. J Air Waste Manag Assoc, 46(9), 861-868.
- Gamberale, F., G. Annwall, and M. Hultengren. 1975. *Exposure to Methylene Chloride: Psychological Functions*. Scandinavian Journal of Work, Environment & Health, 1(2), 95-103. (as cited in EPA, 2011c and NAC, 2008).
- Garte, S., and F. Crosti. 1999. *A Nomenclature System for Metabolic Gene Polymorphisms*. IARC Scientific Publications(148), 5-12. (as cited in ATSDR, 2000).
- Geiger, D. L., S. H. Poirier, L. T. Brooke, and D. J. Call. 1986. *Acute Toxicities of Organic Chemicals to Fathead Minnows (Pimephales Promelas)*. Superior, WI: Center for Lake Superior Environmental Studies, University of Wisconsin.
- Gold, L. S., P. A. Stewart, K. Milliken, M. Purdue, R. Severson, N. Seixas, A. Blair, P. Hartge, S. Davis, and A. J. De Roos. 2011. *The Relationship between Multiple Myeloma and Occupational Exposure to Six Chlorinated Solvents*. Occupational and Environmental Medicine, 68(6), 391-399. (as cited in EPA, 2011c).
- Grevenkamp, A. 2007. *Overexposure and Control of Methylene Chloride in a Furniture Stripping Operation*. Journal of occupational and environmental hygiene, 4(5), D39-D41.
- Hall, R. M., K. F. Martinez, and P. A. Jensen. 1995. *Control of Methylene Chloride—Furniture Stripping Dip Tank*. Applied Occupational and Environmental Hygiene, 10(3), 188-195.
- Hardin, B. D., and J. M. Manson. 1980. *Absence of Dichloromethane Teratogenicity with Inhalation Exposure in Rats*. Toxicology and Applied Pharmacology, 52(1), 22-28. (as cited in EPA, 2011c).
- Hearne, F. T., and J. W. Pifer. 1999. Mortality Study of Two Overlapping Cohorts of Photographic Film Base Manufacturing Employees Exposed to Methylene Chloride. Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine, 41(12), 1154-1169. (as cited in EPA, 2011c).

- Heineman, E. F., P. Cocco, M. R. Gomez, M. Dosemeci, P. A. Stewart, R. B. Hayes, S. H. Zahm, T. L. Thomas, and A. Blair. 1994. Occupational Exposure to Chlorinated Aliphatic Hydrocarbons and Risk of Astrocytic Brain Cancer. American Journal of Industrial Medicine, 26(2), 155-169. (as cited in EPA, 2011c).
- Heitmuller, P. T., T. A. Hollister, and P. R. Parrish. 1981. Acute Toxicity of 54 Industrial Chemicals to Sheepshead Minnows (Cyprinodon Variegatus). Bulletin of Environmental Contamination and Toxicology, 27(5), 596-604.
- Heppel, L. A., and P. A. Neal. 1944. Toxicology of Dichloromethane (Methylene Chloride): Its Effect Upon Running Activity in the Male Rat. Journal of Industrial Hygiene and Toxicology, 26(1), 17-21. (as cited in EPA, 2011c).
- Heppel, L. A., P. A. Neal, T. L. Perrin, M. L. Orr, and V. T. Porterfield. 1944. Toxicology of Dichloromethane (Methylene Chloride): Studies on Effects of Daily Inhalation. Journal of Industrial Hygiene and Toxicology, 26, 8-16. (as cited in EPA, 2011c).
- HSDB (Hazardous Substances Data Bank). 2012. Dichloromethane. National Library of Medicine, Bethesda, MD. http://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm (accessed on March 19, 2012).
- HSE (Health and Safety Executive). 2001. Health Risks During Furniture Stripping Using Dichloromethane (DCM). Woodworking Sheet No 19 (Revised). United Kingdom. www.hse.gov.uk/pubns/wis19.pdf.
- HSIA (Halogenated Solvents Industry Alliance, Inc.). 2008. White Paper on Methylene Chloride. Arlington, VA.
- HSIA (Halogenated Solvents Industry Alliance, Inc.). 2010. Dichloromethane (Methylene Chloride). Arlington, VA.
- IAQUK (Indoor Air Quality UK). 2014. IAQUK Resources: Methylene Chloride. Somerset, UK. http://www.iaguk.org.uk/ResourcesMethylene.html (accessed on July 29, 2014).
- IARC (International Agency for Research on Cancer). 1999. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Dichloromethane, Volume 71. World Health Organization, Lyon, France. http://monographs.iarc.fr/ENG/Monographs/vol71/mono71-10.pdf.
- IARC (International Agency for Research on Cancer). 2010. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Occupational Exposure as a Painter, Volume 98. World Health Organization, Lyon, France.

http://monographs.iarc.fr/ENG/Monographs/vol98/mono98-6.pdf.

- ICIS. 2007. *Chemical Profile for Methylene Chloride*. ICIS Chemical Business Americas, April 2-8, 2007.
- Kjellstrand, P., B. Holmquist, I. Jonsson, S. Romare, and L. Mansson. 1985. *Effects of Organic Solvents on Motor Activity in Mice*. Toxicology, 35(1), 35-46. (as cited in EPA, 2011c).
- Kleinman, M. T., D. M. Davidson, R. B. Vandagriff, V. J. Caiozzo, and J. L. Whittenberger. 1989. Effects of Short-Term Exposure to Carbon Monoxide in Subjects with Coronary Artery Disease. Archives of Environmental Health, 44(6), 361-369. (as cited in NRC, 2010).
- Kleinman, M. T., D. A. Leaf, E. Kelly, V. Caiozzo, K. Osann, and T. O'Niell. 1998. Urban Angina in the Mountains: Effects of Carbon Monoxide and Mild Hypoxemia on Subjects with Chronic Stable Angina. Archives of Environmental Health, 53(6), 388-397. (as cited in NRC, 2010).
- Kühn, R., M. Pattard, K.-D. Pernak, and A. Winter. 1989. Results of the Harmful Effects of Selected Water Pollutants (Anilines, Phenols, Aliphatic Compounds) to Daphnia Magna. Water Research, 23(4), 495-499.
- Lanes, S. F., A. Cohen, K. J. Rothman, N. A. Dreyer, and K. J. Soden. 1990. Mortality of Cellulose Fiber Production Workers. Scandinavian Journal of Work, Environment & Health, 16(4), 247-251. (as cited in EPA, 2011c).
- Lanes, S. F., K. J. Rothman, N. A. Dreyer, and K. J. Soden. 1993. Mortality Update of Cellulose Fiber Production Workers. Scandinavian Journal of Work, Environment & Health, 19(6), 426-428. (as cited in EPA, 2011c).
- Lash, A. A., C. E. Becker, Y. So, and M. Shore. 1991. *Neurotoxic Effects of Methylene Chloride: Are They Long Lasting in Humans?* British Journal of Industrial Medicine, 48(6), 418-426. (as cited in EPA, 2011c).
- LBL (Lawrence Berkeley Laboratory). 1986. Source Characterization and Personal Exposure to Methylene Chloride from Consumer Products. Report Number LBL-20227. Indoor Environment Program, Applied Science Division, Berkeley, CA.
- LBL (Lawrence Berkeley Laboratory). 1987. *Exposure to Methylene Chloride from Controlled Use* of a Paint Remover in Residences. Report Number LBL-23078. Indoor Environment Program, Applied Science Division, Berkeley, CA.
- LeBlanc, G. A. 1980. *Acute Toxicity of Priority Pollutants to Water Flea (Daphnia Magna)*. Bulletin of Environmental Contamination and Toxicology, 24(5), 684-691.
- Lide, D. R. 2001. Physical Constants of Organic Compounds. In *CRC Handbook of Chemistry and Physics, 82nd Edition* (pp. 3-206). Taylor and Francis, Boca Raton, FL.

- Lofgren, D. J., C. K. Reeb-Whitaker, and D. Adams. 2010. *Surveillance of Washington OSHA Exposure Data to Identify Uncharacterized or Emerging Occupational Health Hazards*. Journal of occupational and environmental hygiene, 7(7), 375-388.
- Mannsville (Mannsville Chemical Products Corp). 1999. *Chemical Products Synopsis. Methylene Chloride*. Adams, NY. <u>http://mannsvillechemical.com/synopsis/</u>.
- Matthews, T. G., C. V. Thompson, D. L. Wilson, A. R. Hawthorne, and D. T. Mage. 1989. Air Velocities inside Domestic Environments: An Important Parameter in the Study of Indoor Air Quality and Climate. Environment International, 15(1–6), 545-550.
- McCammon, C. S., R. A. Glaser, V. E. Wells, F. C. Phipps, and W. E. Halperin. 1991. Exposure of Workers Engaged in Furniture Stripping to Methylene Chloride as Determined by Environmental and Biological Monitoring. Applied Occupational and Environmental Hygiene, 6(5), 371-379.
- MDH (Minnesota Department of Health). 2013. *Chemicals of High Concern List (July 1, 2013)*. St. Paul, MN. <u>http://www.health.state.mn.us/divs/eh/hazardous/topics/toxfreekids/chclist/mdhchc2</u> <u>013.pdf</u> (accessed on July 9, 2014).
- Mennear, J. H., E. E. McConnell, J. E. Huff, R. A. Renne, and E. Giddens. 1988. Inhalation Toxicity and Carcinogenesis Studies of Methylene Chloride (Dichloromethane) in F344/N Rats and B6C3F1 Mice. Annals of the New York Academy of Sciences, 534, 343-351. (as cited in EPA, 2011c).
- Merlin, G., H. Thiebaud, G. Blake, S. Sembiring, and J. Alary. 1992. *Mesocosms' and Microcosms' Utilization for Ecotoxicity Evaluation of Dichloromethane, a Chlorinated Solvent*. Chemosphere, 24(1), 37-50.
- Miligi, L., A. S. Costantini, A. Benvenuti, D. Kriebel, V. Bolejack, R. Tumino, V. Ramazzotti, S. Rodella, E. Stagnaro, P. Crosignani, D. Amadori, D. Mirabelli, L. Sommani, I. Belletti, L. Troschel, L. Romeo, G. Miceli, G. A. Tozzi, I. Mendico, and P. Vineis. 2006. *Occupational Exposure to Solvents and the Risk of Lymphomas*. Epidemiology (Cambridge, Mass.), 17(5), 552-561. (as cited in EPA, 2011c).
- MSU/MIFACE (Michigan State University/Michigan Fatality Assessment and Control Evaluation). 2011. *Tub Refinisher Died Due to Methylene Chloride Overexposure While Stripping a Bathtub*. MIFACE investigation report #10MI013. East Lansing, MI. <u>http://www.oem.msu.edu/MiFace/10MI013Report.pdf</u>

- Musy, M., E. Wurtz, and J. M. Nataf. 1999. *An Intermediate Model to Predict Thermal Comfort and Air Quality in a Building*. Proceedings of the 8th International Conference on Indoor Air Quality and Climate, Vol. 1, pp. 685, Edinburgh, Scotland.
- NAC (National Advisory Committee). 2008. Interim Acute Exposure Guideline Levels (AEGL) for Methylene Chloride. Washington, DC. http://www.epa.gov/oppt/aegl/pubs/methylene chloride interim dec 2008 v1.pdf.
- NCA (National Coffee Association). 1983. 24-Month Oncogenicity Study of Methylene Chloride in Mice: Final Report. Study conducted by Hazleton Laboratories, Vienna, VA. Doc #45-8303005. (as cited in EPA, 2011c).
- NIH (National Institutes of Health). 2005. *Substance Profiles for Dichloromethane*. Bethesda, MD.
- NIOSH (National Institute for Occupational Safety and Health). 1977. *Health Hazard Evaluation* Determination Report 75-195-396. United Airlines Maintenance Base San Francisco International Airport Burlingame, California. U.S. Department of Health, Education and Welfare, Cincinnati, OH. <u>www.ntis.gov/search/product.aspx?ABBR=PB273779</u>.
- NIOSH (National Institute for Occupational Safety and Health). 1990. *Walk-Though Survey Report: Control of Methylene Chloride in Furniture Stripping at Colonial Furniture Stripping*. ECTB number: 170-14a. U.S. Department of Health and Human Services, Cincinnati, OH. <u>www.cdc.gov/niosh/surveyreports/pdfs/170-14a.pdf</u>.
- NIOSH (National Institute for Occupational Safety and Health). 1991. In-Depth Survey Report. The Control of Methylene Chloride in Furniture Stripping at Association for Retarded Citizens. ECTB number: 170-18a. Division of Physical Sciences and Engineering, Cincinnati, OH. <u>http://www.cdc.gov/niosh/surveyreports/pdfs/170-18a.pdf</u>.
- NIOSH (National Institute for Occupational Safety and Health). 1993. *Health Hazard Evaluation. Ackerman and Sons, Littleton, Colorado*. Report No. 92-0360. Cincinnati, Ohio. <u>www.cdc.gov/niosh/hhe/reports/pdfs/1992-0360-2372.pdf</u>.
- NIOSH (National Institute for Occupational Safety and Health). 2011a. NIOSH Pocket Guide to Chemical Hazards: Introduction. Atlanta, GA. <u>http://www.cdc.gov/niosh/npg/pgintrod.html</u> (accesed on July 20, 2012).
- NIOSH (National Institute for Occupational Safety and Health). 2011b. *NIOSH Pocket Guide to Chemical Hazards: Methylene Chloride*. <u>http://www.cdc.gov/niosh/npg/npgd0414.html</u> (accessed on July 20, 2012).

- NITE (National Institute of Technology and Evaluation). 2002. *Biodegradation and Bioaccumulation of the Existing Chemical Substances under the Chemical Substances Control Law.* Japan.
- Nitschke, K. D., J. D. Burek, T. J. Bell, R. J. Kociba, L. W. Rampy, and M. J. McKenna. 1988a. *Methylene Chloride: A 2-Year Inhalation Toxicity and Oncogenicity Study in Rats*. Fundamental and applied toxicology : official journal of the Society of Toxicology, 11(1), 48-59. (as cited in EPA 2011b).
- Nitschke, K. D., D. L. Eisenbrandt, L. G. Lomax, and K. S. Rao. 1988b. *Methylene Chloride: Two-Generation Inhalation Reproductive Study in Rats*. Fundamental and applied toxicology : official journal of the Society of Toxicology, 11(1), 60-67. (as cited in EPA 2011b).
- NRC (National Research Council). 1983. *Risk Assessment in the Federal Government: Managing the Process*. National Academy Press, Washington, DC. <a href="http://www.nap.edu/openbook.php?record\_id=366&page=R1">http://www.nap.edu/openbook.php?record\_id=366&page=R1</a>. (as cited in EPA, 2011c).
- NRC (National Research Council). 1992. *Guidelines for Developing Spacecraft Maximum Allowable Concentrations for Space Station Contaminants* National Academy Press, Washington, DC.
- NRC (National Research Council). 1996. Spacecraft Maximum Allowable Concentration for Selected Airborne Contaminants: Methylene Chloride (Volume 2). National Academy Press, Washington, DC. <u>http://www.nap.edu/catalog.php?record\_id=5170</u>.
- NRC (National Research Council). 2001. *Standing Operating Procedures for Developing Acute Exposure Guideline Levels for Hazardous Chemicals*. National Academy Press, Washington DC. <u>http://www.epa.gov/oppt/aegl/pubs/sop.pdf</u>.
- NRC (National Research Council). 2008. Spacecraft Maximum Allowable Concentration for Selected Airborne Contaminants: Methylene Chloride (Volume 5). National Academy Press, Washington, DC. <u>http://www.nap.edu/catalog.php?record\_id=12529</u>.
- NRC (National Research Council). 2010. *Final Acute Exposure Guideline Levels for Carbon Monoxide*. Volume 8. National Academy Press, Washington, DC. <u>http://www.nap.edu/catalog.php?record\_id=12770</u>.
- NTP (National Toxicolgy Program). 1986. Toxicology and Carcinogenesis Studies of Dichloromethane (Methylene Chloride) (CAS No. 75-09-2) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). Report No. 306. U.S. Department of Health and Human Services, Research Triangle Park, NC. (as cited in EPA, 2011c).
- O'Neil, M. J. 2001. Methylene Chloride. In *The Merck Index: An Encyclopedia of Chemicals, Drugs and Biologicals* (13th ed., pp. 1082). Merck & Co., Inc, Whitehouse Station, NJ.

- OECD (Organization for Economic Co-operation and Development). 2010. *Emission Scenario Document on Coating Application Via Spray Painting in the Automotive Refinishing Industry (Draft Final)*. OECD Environmental health and safety publications. Series on emission scenario documents, Paris, France.
- OECD (Organization for Economic Co-operation and Development). 2011. *SIDS Initial Assessment Profile for Methylene Chloride*. CoCAM 1, October 10-12, 2011, Paris, France.
- OEHHA (Office of Environmental Health Hazard Assessment). 1999. Air Toxics Hot Spots Program Risk Assessment Guidelines. Part I: The Determination of Acute Reference Exposure Levels for Airborne Toxicants. State of California Environmental Protection Agency, Sacramento, CA. <u>http://oehha.ca.gov/air/pdf/acuterel.pdf</u>.
- OEHHA (Office of Environmental Health Hazard Assessment). 2001. *Methylene Chloride: Prioritization of Toxic Air Contamination-Children's Environmental Health Protection Act.* State of California Environmental Protection Agency, Sacramento, CA. <u>http://oehha.ca.gov/air/toxic\_contaminants/pdf\_zip/methylene%20chloride\_final.pdf</u>.
- OEHHA (Office of Environmental Health Hazard Assessment). 2008. Acute Reference Exposure Level (REL) and Toxicity Summary for Methylene Chloride. State of California Environmental Protection Agency, Sacramento, CA. <u>http://oehha.ca.gov/air/hot\_spots/2008/AppendixD2\_final.pdf#page=187</u>.
- OEHHA (Office of Environmental Health Hazard Assessment). 2014a. *Chemicals Biomonitored in California*. State of California Environmental Protection Agency, Sacramento, CA. <a href="http://www.biomonitoring.ca.gov/chemicals/chemicals-biomonitored-california">http://www.biomonitoring.ca.gov/chemicals/chemicals-biomonitored-california</a> (accessed on July 9, 2014).
- OEHHA (Office of Environmental Health Hazard Assessment). 2014b. *Chemicals Known to the State to Cause Cancer or Reproductive Toxicity (May 2, 2014)*. State of California Environmental Protection Agency, Sacramento, CA. <u>http://oehha.ca.gov/prop65/prop65\_list/files/P65single050214.pdf</u>.
- OSHA (Occupational Safety and Health Administration). 1997a. Intro to 29 CFR Parts 1010, 1915 and 1926; Occupational Exposure to Methylene Chloride. 29 CFR 1910, 1915, 1926, Washington, DC. https://www.osha.gov/pls/oshaweb/owadisp.show\_document?p\_table=PREAMBLES&p\_\_\_\_\_id=998.
- OSHA (Occupational Safety and Health Administration). 1997b. Substance Safety Data Sheet and Technical Guidance for Methylene Chloride. 29 CFR 1910.1052 App. A, Washington, DC.

https://www.osha.gov/pls/oshaweb/owadisp.show\_document?p\_table=STANDARDS&p\_\_id=10095.

- OSHA (Occupational Safety & Health Administration). 2010. *Regulatory Review of 29 CFR* 1910.1052: Methylene Chloride. Directorate of Evaluation and Analysis, Washington, DC.
- OSHA (Occupational Safety and Health Administration). 2012a. Integrated Management Information System (IMIS): Methylene Chloride (CASRN 75-09-2). Washington, DC.
- OSHA (Occupational Safety and Health Administration). 2012b. *Methylene Chloride Safety and Health Topics*. Washington, DC. <u>http://www.osha.gov/SLTC/methylenechloride/</u> (accessed on August 15, 2012).
- Pauli, R. 1996. *Alternative Processes to Methylene Chloride*. National Metal Finishing Resource Center, Fairfield, CA. <u>http://www.nmfrc.org/pdf/pf0697b.htm</u>.
- Pellizzari, E. D., T. D. Hartwell, B. S. Harris, 3rd, R. D. Waddell, D. A. Whitaker, and M. D.
   Erickson. 1982. *Purgeable Organic Compounds in Mother's Milk*. Bulletin of
   Environmental Contamination and Toxicology, 28(3), 322-328. (as cited in ATSDR, 2000).
- Peterson, J. E. 1978. *Modeling the Uptake, Metabolism and Excretion of Dichloromethane by Man.* American Industrial Hygiene Association Journal, 39(1), 41-47. (as cited in NAC, 2008 and NRC, 1996).
- Pollack-Nelson, C. 1995. Analysis of Methylene Chloride Product Labelling. Ergonomics, 38(11), 2176-2187.
- Putz, V. R., B. L. Johnson, and J. V. Setzer. 1979. A Comparative Study of the Effects of Carbon Monoxide and Methylene Chloride on Human Performance. Journal of Environmental Pathology and Toxicology, 2(5), 97-112. (as cited in NRC, 1996; OEHHA, 2008; and EPA, 2011c).
- Raje, R., M. Basso, T. Tolen, and M. Greening. 1988. *Evaluation of in Vivo Mutagenicity of Low-Dose Methylene Chloride in Mice*. International Journal of Toxicology, 7, 699-703. (as cited in EPA, 2011c).
- Ratney, R. S., D. H. Wegman, and H. B. Elkins. 1974. *In Vivo Conversion of Methylene Chloride to Carbon Monoxide*. Archives of Environmental Health, 28(4), 223-226. (as cited in NRC, 1996).
- Rebert, C. S., M. J. Matteucci, and G. T. Pryor. 1989. Acute Effects of Inhaled Dichloromethane on the Eeg and Sensory-Evoked Potentials of Fischer-344 Rats. Pharmacology, Biochemistry and Behavior, 34(3), 619-629. (as cited in EPA, 2011c).

- Rice, D., and S. Barone, Jr. 2000. *Critical Periods of Vulnerability for the Developing Nervous System: Evidence from Humans and Animal Models*. Environ Health Perspect, 108 Suppl 3, 511-533.
- RIDEM (Rhode Island Department of Environmental Management). 2011. *Environmental/Occupational Health Complicance Certification Program. Certification Workbook for Auto Body Repair Facilities*. Office of Customer and Technical Assistance, Providence, RI. <u>http://www.dem.ri.gov</u>.
- Riley, D. M., B. Fischhoff, M. J. Small, and P. Fischbeck. 2001. *Evaluating the Effectivenes of Risk-Reduction Strategies for Consumer Chemical Products*. Risk Analysis, 21(2), 357-369.
- Riley, E. C., D. W. Fassett, and W. L. Sutton. 1966. *Methylene Chloride Vapor in Expired Air of Human Subjects*. American Industrial Hygiene Association Journal, 27(4), 341-348. (as cited in NAC, 2008).
- Roederer, G. 1990. *Testung Wassergefaehrdender Stoffe Als Grundlage Fuer Wasserqualitaetsstandards*. Fraunhofer-Institutfuer Umweltchemie und Oekotoxikologie, 5948 Schmallenberg, UFOPLAN-Nr. 116 08 071/01.
- Schwetz, B. A., K. J. Leong, and P. J. Gehring. 1975. The Effect of Maternally Inhaled Trichloroethylene, Perchloroethylene, Methyl Chloroform, and Methylene Chloride on Embryonal and Fetal Development in Mice and Rats. Toxicology and Applied Pharmacology, 32(1), 84-96. (as cited in EPA, 2011c).
- Seidler, A., M. Mohner, J. Berger, B. Mester, E. Deeg, G. Elsner, A. Nieters, and N. Becker. 2007. Solvent Exposure and Malignant Lymphoma: A Population-Based Case-Control Study in Germany. Journal of occupational medicine and toxicology (London, England), 2, pp. 2. (as cited in EPA, 2011c).
- Serota, D. G., A. K. Thakur, B. M. Ulland, J. C. Kirschman, N. M. Brown, R. H. Coots, and K. Morgareidge. 1986. A Two-Year Drinking-Water Study of Dichloromethane in Rodents. Ii. Mice. Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association, 24(9), 959-963. (as cited in EPA, 2011c).
- Sheps, D. S., K. F. Adams, Jr., P. A. Bromberg, G. M. Goldstein, J. J. O'Neil, D. Horstman, and G. Koch. 1987. Lack of Effect of Low Levels of Carboxyhemoglobin on Cardiovascular Function in Patients with Ischemic Heart Disease. Archives of Environmental Health, 42(2), 108-116. (as cited in NRC, 2010).
- Sills, R. C., J. R. Hailey, J. Neal, G. A. Boorman, J. K. Haseman, and R. L. Melnick. 1999. Examination of Low-Incidence Brain Tumor Responses in F344 Rats Following Chemical Exposures in National Toxicology Program Carcinogenicity Studies. Toxicologic Pathology, 27(5), 589-599. (as cited in EPA, 2011c).

Smithsonian (Smithsonian Museum Conservation Institute). 2012a. *Does My Painting Need to Be Cleaned*. Suitland, MD.

http://si.edu/mci/english/learn\_more/taking\_care/painting\_clean.html (accessed on July 20, 2012).

- Smithsonian (Smithsonian Museum Conservation Institute). 2012b. What Does It Mean to Have a Painting Restored and How Do I Pick a Conservator. Suitland, MD. <u>http://si.edu/mci/english/learn\_more/taking\_care/conservation\_meaning.html</u> (accessed on July 20, 2012).
- SRC (Syracuse Research Corporation). 1978. *Results of Continuous Exposure of Fathead Minnow Embryo to 21 Priority Pollutants*. OTS#0511060. Doc#40-7848049.
- SRRP (Source Reduction Research Partnership). 1992. Source Reduction and Recycling of Halogenated Solvents in the Adhesives Industry. Prepared by Jacobs Engineering Group, Pasadena, CA.
- Stewart, R. D., T. N. Fisher, M. J. Hosko, J. E. Peterson, E. D. Baretta, and H. C. Dodd. 1972. *Experimental Human Exposure to Methylene Chloride*. Archives of Environmental Health, 25(5), 342-348. (as cited in NAC, 2008 and NRC, 1996).
- Stewart, R. D., C. L. Hake, and A. Wu. 1976. *Use of Breath Analysis to Monitor Methylene Chloride Exposure*. Scandinavian Journal of Work, Environment & Health, 2(2), 57-70. (as cited in NAC, 2008).
- Tabak, H. H., S. A. Quave, C. I. Mashni, and E. F. Barth. 1981. *Biodegradability Studies with Organic Priority Pollutant Compounds*. Journal of the Water Pollution Control Federation, 53(10), 1503-1518.
- Tomenson, J. A. 2011. Update of a Cohort Mortality Study of Workers Exposed to Methylene Chloride Employed at a Plant Producing Cellulose Triacetate Film Base. Int Arch Occup Environ Health, 84(8), 889-897. (as cited in EPA, 2011c).
- USDOC (U.S. Department of Commerce). 2007a. American Factfinder: 2007 Economic Census. U.S. Census Bureau, Washington, DC. <u>http://factfinder2.census.gov/faces/nav/isf/pages/programs.xhtml?program=econ</u> (accessed in August 2012).
- USDOC (U.S. Department of Commerce). 2007b. North American Industry Classification System (NAICS): 2007 NAICS Index File. U.S. Census Bureau, Washington, DC. <u>http://www.census.gov/cgi-bin/sssd/naics/naicsrch?chart=2007</u> (accessed on August 9, 2012).

- van Veen, M. P., F. Fortezza, E. Spaans, and T. T. Mensinga. 2002. Non-Professional Paint Stripping, Model Prediction and Experimental Validation of Indoor Dichloromethane Levels. Indoor Air, 12(2), 92-97.
- Vincent, R., P. Poirot, I. Subra, B. Rieger, and A. Cicolella. 1994. *Occupational Exposure to Organic Solvents During Paint Stripping and Painting Operations in the Aeronautical Industry*. Int Arch Occup Environ Health, 65(6), 377-380.
- von Bringmann, G., and F. Meinck. 1964. *Wassertoxikologische Beurteilung Von Industrieabwassern*. Gesundheits-Ingenieur, 85, 229-260.
- Wang, R., Y. Zhang, Q. Lan, T. R. Holford, B. Leaderer, S. H. Zahm, P. Boyle, M. Dosemeci, N. Rothman, Y. Zhu, Q. Qin, and T. Zheng. 2009. *Occupational Exposure to Solvents and Risk of Non-Hodgkin Lymphoma in Connecticut Women*. American Journal of Epidemiology, 169(2), 176-185. (as cited in EPA, 2011c).
- Weinstein, R. S., D. D. Boyd, and K. C. Back. 1972. Effects of Continuous Inhalation of Dichloromethane in the Mouse: Morphologic and Functional Observations. Toxicology and Applied Pharmacology, 23(4), 660-679. (as cited in EPA, 2011c).
- Winneke, G. 1974. Behavioral Effects of Methylene Chloride and Carbon Monoxide as Assessed by Sensory and Psychomotor Performance. In Xintaras, C., B. L. Johnson, and I. de Groot, *Behavioral Toxicology: Early Detection of Occupational Hazards* (pp. 130-144).
  U.S. Department of Health, Education and Welfare Washington, DC. (as cited in EPA, 2011c and NAC, 2008).
- WM Barr (W.M. Barr and Company). 2008. *Material Safety Data Sheet: Klean-Strip Color Change Stripper*. Memphis, TN. <u>http://www.wmbarr.com/ProductFiles/GKCC00326.pdf</u>.
- WM Barr (W.M. Barr and Company). 2009a. *Material Safety Data Sheet. Klean Strip Aircraft Remover*. Memphis, TN. <u>http://www.wmbarr.com/ProductFiles/3404.11.pdf</u>.
- WM Barr (W.M. Barr and Company). 2009b. *Material Safety Data Sheet. Klean Strip Klean Kutter*. Memphis, TN. <u>http://www.wmbarr.com/ProductFiles/130%20(Klean%20Kutter).pdf</u>.
- WM Barr (W.M. Barr and Company). 2010a. *Material Safety Data Sheet. Klean-Strip Naked Gun* Spray Gun Paint Remover. Memphis, TN.
- WM Barr (W.M. Barr and Company). 2010b. *Material Safety Data Sheet. Klean-Strip Peeler*. Memphis, TN. <u>http://www.wmbarr.com/ProductFiles/KS%20Peeler%20(A223.2)%207-23-10.pdf</u>.

- WM Barr (W.M. Barr and Company). 2011a. *Material Safety Data Sheet. Klean-Strip Premium Sprayable Stripper*. Memphis, TN. <u>http://www.wmbarr.com/ProductFiles/MSWRPTM.pdf</u>.
- WM Barr (W.M. Barr and Company). 2011b. *Material Safety Data Sheet. Klean-Strip Strip X Stripper*. Memphis, TN.
- WM Barr (W.M. Barr and Company). 2011c. Material Safety Data Sheet. Klean Strip Adhesive Remover/Klean Strip Premium Stripper. Memphis, TN. <u>http://www.wmbarr.com/ProductFiles/KS%20Adhesive%20Remover%20(4015-</u>26)%205-17-11.pdf.
- WM Barr (W.M. Barr and Company). 2012. Material Safety Data Sheet. Premium Stripper. Memphis, TN. <u>http://www.wmbarr.com/ProductFiles/KS%20Premium%20Stripper%203%2028%20201</u> <u>2.pdf</u>.
- Wollbrinck, T. 1993. *The Composition of Proprietary Paint Strippers*. Journal of the Amercian Institute for Conservation, 32(1), Article 5 (pp. 43-57).
- Wong, K. L. 1990. Carbon Monoxide. In Spacecraft Maximum Allowable Concentrations for Selected Airborne Contaminants (Vol. 1, pp. 61-90). National Academy Press, Washington, D.C. (as cited in NRC 1996).
- WSDE (Washington State Department of Ecology). 2013. *Chemicals of High Concern to Children*. Lacey, WA. <u>http://www.ecy.wa.gov/programs/swfa/cspa/chcc.html</u> (accessed on July 9, 2014).
- Xu, J. Q., K. D. Kochanek, S. L. Murphy, and B. Tejada-Vera. 2010. *Deaths: Final Data for 2007*. National Center for Health Statistics, Hyattsville, MD.
   <u>http://www.cdc.gov/nchs/data/nvsr/nvsr58/nvsr58\_19.pdf</u>. (as cited in EPA, 2011a).

## APPENDICES

# Appendix A REGULATORY HISTORY OF DCM IN THE U.S. AND ABROAD

### A-1 DCM Regulatory History in the U.S.

DCM has been the subject of US federal regulations by the Environmental Protection Agency (EPA), the Consumer Product Safety Commission (CPSC), the Food and Drug Administration (FDA), and the Occupational Safety and Health Administration (OSHA).

EPA lists DCM as a toxic (*i.e.*, non-acute) hazardous waste under the Resource Conservation and Recovery Act (RCRA) (Code U080) (<u>EPA, 2012c</u>), and DCM is listed on the Toxics Release Inventory (TRI) pursuant to section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) (<u>EPA, 2014</u>). DCM is also listed on the TSCA Inventory of Chemical Substances and is subject to reporting under the TSCA Chemical Data Reporting (CDR) rule (<u>EPA, 2011e</u>).

EPA's Office of Air Quality Planning and Standards issued a final rule in January 2008, under the National Emission Standards for Hazardous Air Pollutants (NESHAP) that established national emission standards for using DCM to remove dried paint (*i.e.*, including, but not limited to: paint, enamel, varnish, shellac, and lacquer) from wood, metal, plastic, and other substrates (EPA, 2008). The NESHAP also implemented management practices that minimize DCM emissions.

Additionally, the Safe Drinking Water Act (SDWA) requires EPA to determine the level of contaminants in drinking water at which no adverse health effects are likely to occur. EPA has set an enforceable maximum contaminant level (MCL) for DCM at 0.005 mg/L or 5 ppb (EPA, 2010b).

In 1987, CPSC issued a statement of policy regarding its decision to require labeling of consumer products that contain DCM (<u>CPSC, 1987</u>). Labels indicated that inhalation of DCM vapor has caused cancer in certain laboratory animals, and the labels specified precautions to be taken during use by consumers.

DCM was previously used in aerosol cosmetic products, such as hairspray. In 1989, FDA banned DCM as an ingredient in all cosmetic products because of its animal carcinogenicity and likely hazard to human health (FDA, 1989).

OSHA also took steps to reduce the DCM exposure in occupational settings. OSHA lowered the permissible exposure limit (PEL) for DCM from 500 parts per million (ppm) to 25 ppm (<u>OSHA</u>, <u>1997a</u>, <u>1997b</u>).

DCM is listed as an informational initial candidate chemical under California's Safer Consumer Products regulations (<u>DTSC, 2010</u>). The chemical is also listed on the state's Proposition 65 list because it is known to cause cancer or birth defects or other reproductive harm (<u>OEHHA</u>, <u>2014b</u>). In addition, California lists DCM as a designated chemical for biomonitoring (<u>OEHHA</u>, <u>2014a</u>). The States of Washington and Minnesota classify DCM as a chemical of high concern (<u>MDH</u>, 2013; <u>WSDE</u>, 2013).

# A-2 DCM Regulatory History in Canada and Europe

In 2003, the Canadian Minister of the Environment published a Notice under Part 4 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) requiring the preparation and implementation of pollution prevention plans for DCM (<u>Environment Canada, 2003b</u>). This Notice targets persons involved in the use of DCM for the following activities: aircraft paint stripping; flexible polyurethane foam blowing; pharmaceuticals and chemical intermediates manufacturing and tablet coating; industrial cleaning; and adhesive formulations.

Also in 2003, Environment Canada published a Code of Practice for the reduction of dichloromethane emissions from the use of paint strippers in commercial furniture refinishing and other stripping applications (Environment Canada, 2003a). The Code of Practice was developed by a multi-stakeholder technical working committee, which consisted of industry representatives (i.e., furniture strippers, auto body shops, paint stripper formulators, solvent recovery firms), government personnel, and environmental non-governmental organizations.

The European Commission (EC) amended its Registration, Evaluation, Authorization, and Restriction of Chemical substances in 2010 to incorporate restrictions for the use of DCM in paint strippers (EC, 2010). DCM is banned from: (1) placement on the market in a new product for consumers/professionals after December 2010, (2) placement on the market in any product for consumers/professionals after December 2011, and (3) use by professionals after June 2012, unless the professionals are appropriately licensed and trained in the following: awareness, evaluation and management of risks, use of adequate ventilation, and use of appropriate personal protective equipment. In addition, industrial installations using DCM must have effective ventilation, minimize evaporation from tanks, and have measures for safe handling of DCM in tanks, adequate personal protective equipment, and adequate information and training for operators. Pain strippers containing DCM in a concentration equal to or greater than 0.1% by weight must include a label: "*Restricted to industrial use and to professionals approved in certain EU Member States – verify where use is allowed*. (EC, 2010)"

# Appendix B SUMMARY OF ENVIRONMENTAL EFFECTS: AQUATIC TOXICITY

The aquatic toxicity of DCM for fish, aquatic invertebrates, and aquatic plants is low based on EPA/OPPT criteria described in the *TSCA Work Plan Chemicals Methods Document* (EPA, 2012d) and the *Classification Criteria for Environmental Toxicity and Fate of Industrial Chemicals* (EPA, 1992a). The sections below summarize the aquatic toxicity studies considered in the evaluation of environmental hazards of DCM.

## B-1 Acute Toxicity to Fish

Fathead minnows (*Pimephales promelas*) were exposed to unspecified measured concentrations of DCM under flow-through conditions for 96 hrs. A 96-hr LC<sub>50</sub> of 99 mg/L was reported (<u>Alexander et al., 1978</u>).

Fathead minnows (*P. promelas*) were exposed to unspecified measured concentrations of DCM under flow-through conditions for 96 hrs. A 96-hr LC<sub>50</sub> of 193 mg/L was reported (<u>Alexander et al., 1978</u>).

Fathead minnows (*P. promelas*) were exposed to unspecified nominal concentrations of DCM under static conditions for 96 hrs. A 96-hr  $LC_{50}$  of 310 mg/L was reported (<u>Alexander et al., 1978</u>).

Fathead minnows (*P. promelas*) were exposed to unspecified measured concentrations of DCM under flow-through conditions for 96 hours. A 96-hr LC<sub>50</sub> of 193 mg/L was reported (<u>Geiger et al., 1986</u>).

Fathead minnows (*P. promelas;* 10/replicate) were exposed to measured concentrations of 79, 135, 207, 357, 527, and 855 DCM under flow-through conditions for 96 hrs. A 96-hr  $LC_{50}$  of 502 mg/L was reported (<u>Dill et al., 1987</u>).

Sheepshead minnows (*Cyprinodon variegates*) were exposed to unspecified nominal concentrations of DCM under static conditions for 96 hrs. A 96-hr LC<sub>50</sub> of 330 mg/L was reported (<u>Heitmuller et al., 1981</u>).

Zebrafish (*Danio rerio*) were exposed to unspecified concentrations of DCM under unspecified conditions for 96 hrs. The 96-hr LC<sub>50</sub> of 254 mg/L was reported (<u>Roederer, 1990</u>).

Bluegill sunfish (*Lepomis macrochirus*) were exposed to unspecified nominal concentrations of DCM under static conditions for 96 hrs. A 96-hr  $LC_{50}$  of 220 mg/L was reported (<u>Buccafusco et al., 1981</u>).

# B-2 Chronic Toxicity to Fish

Fathead minnows (*P. promelas*) were exposed to measured concentrations 29, 55, 82, 142, 209, and 321 mg/L of DCM under flow-through conditions for 28 days. The 28-day lowest-observed-effect concentration (LOEC) ranged between 82.5 and 142 mg/L and a maximum acceptable toxicant concentration (MATC) of 108 mg/L was reported (<u>Dill et al., 1987</u>).

### **B-3** Acute Toxicity to Aquatic Invertebrates

Water fleas (*Daphnia magna*) were exposed to unspecified nominal concentrations of DCM under static conditions for 48 hrs. A 48-hr  $EC_{50}$  of 1,682 mg/L was reported (<u>Kühn et al., 1989</u>).

Water fleas (*D. magna*) were exposed to unspecified nominal concentrations of DCM under static conditions for 48 hrs. A 48-hr EC<sub>50</sub> of 1,250 mg/L was reported (<u>von Bringmann and Meinck, 1964</u>).

Water fleas (*D. magna*) were exposed to unspecified nominal concentrations of DCM under static conditions for 48 hrs. A 48-hr EC<sub>50</sub> of 220 mg/L was reported (<u>LeBlanc, 1980</u>).

Opossum shrimp (*Americamysis bahia*) were exposed to unspecified nominal concentrations of DCM under static conditions for 96 hrs. A 96 hr  $EC_{50}$  of 256 mg/L was reported (<u>SRC, 1978</u>).

### **B-4** Toxicity to Aquatic Plants

Green algae (*Pseudokirchneriella subcapitata*) were exposed to unspecified nominal concentrations of DCM under static conditions for 96 hrs. A 96-hr EC<sub>50</sub> of 500 mg/L was reported (EPA, 1978).

Diatoms (*Skeletomema costatum*) were exposed to unspecified nominal concentrations of DCM under static conditions for 96 hrs. A 96-hr EC<sub>50</sub> of 662 mg/L was reported (<u>SRC, 1978</u>).

Green algae (*Scenedesmus subspicatus*) were exposed to unspecified nominal concentrations of DCM under static conditions for 96 hrs. A 96-hr  $EC_{50}$  of 1,000 mg/L was reported (<u>Merlin et al.</u>, <u>1992</u>).

# Appendix C INVENTORY UPDATE REPORTING RULE DATA FOR DCM

EPA's 2012 Chemical Data Report (CDR) reported a DCM production volume of 261.5 million pounds. Two companies reported domestic manufacturing of DCM: Dow Chemical Company and Occidental Chemical Corporation (EPA, 2013). There were also some companies that reported to 2012 CDR, but much of this information was claimed confidential business information and cannot be made available to the public. Data in tables C-1 to C-3 were extracted from the 2012 CDR records (EPA, 2013).

Table C 1. National Chemical Information for DCM from 2012 CDR	
Production Volume (aggregate)	261.5 million pounds
Maximum Concentration (at manufacture or import site)	>90%
Physical form(s)	Liquid
Number of reasonably likely to be exposed industrial manufacturing, processing, and use workers (aggregated)	>1,000
Was industrial processing or use information reported?	Yes
Was commercial or consumer use information reported?	Yes

Industrial Sector (Based on NAICS)	Industrial Function	Type of Processing
Adhesive Manufacturing	Solvents (for cleaning or degreasing)	Use-non-incorporative activities
Adhesive Manufacturing	Not Known or Reasonably Ascertainable	Processing-incorporation into formulation, mixture, or reaction product
All Other Basic Organic Chemical Manufacturing	Solvents (for cleaning or degreasing)	Processing-incorporation into formulation, mixture, or reaction product
All Other Basic Organic Chemical Manufacturing	Processing aids, not otherwise listed	Use-non-incorporative activities
All Other Basic Organic Chemical Manufacturing	Solvents (for cleaning or degreasing)	Processing-incorporation into formulation, mixture, or reaction product
All Other Basic Organic Chemical Manufacturing	Processing aids, specific to petroleum production	Use-non-incorporative activities
All Other Chemical Product and Preparation Manufacturing	Propellants and blowing agents	Processing-incorporation into formulation, mixture, or reaction product

Industrial Sector (Based on NAICS)	Industrial Function	Type of Processing
All Other Chemical Product and Preparation Manufacturing	Solvents (for cleaning or degreasing)	Use-non-incorporative activities
All Other Chemical Product and Preparation Manufacturing	Adhesives and sealant chemicals	Processing-incorporation into formulation, mixture, or reaction product
All Other Chemical Product and Preparation Manufacturing	Solvents (which become part of product formulation or mixture)	Processing-incorporation into formulation, mixture, or reaction product
Paint and Coating Manufacturing	Solvents (which become part of product formulation or mixture)	Processing-incorporation into formulation, mixture, or reaction product
Pesticide, Fertilizer, and Other Agricultural Chemical Manufacturing	Processing aids, not otherwise listed	Use-non-incorporative activities
Pesticide, Fertilizer, and Other Agricultural Chemical Manufacturing	Processing aids, specific to petroleum production	Use-non-incorporative activities
Petrochemical Manufacturing	Processing aids, not otherwise listed	Processing-incorporation into formulation, mixture, or reaction product
Plastics Material and Resin Manufacturing	Processing aids, not otherwise listed	Use-non-incorporative activities
Plastics Material and Resin Manufacturing	Processing aids, specific to petroleum production	Use-non-incorporative activities

Table C 3. DCM Commercial/Consumer Use Category Summary					
Commercial/Consumer Product Category	Intended for Commercial and/or Consumer Uses or Both	Intended for Use in Children's Products in Related Product Category			
Adhesives and Sealants	Both	Not Known or Reasonably Ascertainable			
Automotive Care Products	Both	No			
Metal Products not covered elsewhere	Commercial	No			
Paints and Coatings	Both	No			

# Appendix D HOUSEHOLD PRODUCTS DATA FOR DCM

EPA/OPPT searched the National Institute of Health (NIH) Household Database, which links over 13,000 consumer brands to health effects reported in Material Safety Data Sheets (MSDS) (<u>DHHS, 2012</u>). The database also allows scientists and consumers to research products based on chemical ingredients. Table D-1 lists the household products containing DCM in their formulations.

Table D 1. Household Products Containing I         Product Brand	Category	Form	% DCM Content by Weight (as of June 2014) <sup>a</sup>	
Jasco Brushable Semi-Paste Premium Paint & Epoxy Remover	Arts and crafts	Liquid	60-100	
Savogran Liquid Kutzit-08/31/2007	Arts and crafts	Liquid	20-25	
Carb Medic Carburetor Choke and Valve Cleaner- 08/01/2002-old product	Auto products	Liquid	60-70	
Sprayway Industrial Gasket Remover No. 719	Auto products	Aerosol	70-80	
Gunk Carb Medic Carburetor and Choke Cleaner- 04/07/2010	Auto products	Aerosol	15-40	
Carb Medic Carb/Choke/Valve Cleaner-old product	Auto products	Aerosol	40-50	
Gumout Professional Non Flammable Brake Parts Cleaner	Auto products	Aerosol	5-30	
Espree Tire Shine	Auto products	Aerosol	50	
Lectra Motive Auto Care-old product	Auto products	Aerosol	1-20	
Anti-Seize Lubricant-old product	Auto products	Aerosol	60-65	
Carb Medic Carburetor Choke and Valve Cleaner- old product	Auto products	Liquid	40-50	
Champion Sprayon Degreasing Solvent	Auto products	Aerosol	70-75	
ProsALL Prosolv	Auto products	Aerosol	70-75	
Zinsser Brush & Roller Wash	Home maintenance	Liquid		
Crown Handi-Strip All Purpose Liquid Stripper	Home maintenance	Liquid	40-60	
Crown Tuff-Strip Heavy Duty Semi-Paste Stripper	Home maintenance	Liquid	80-90	
UGL ZAR Paint and Varnish Remover	Home maintenance	Liquid	90	
Savogran Prepaint Deglosser	Home maintenance	Liquid	35-40	
Savogran Water Rinsing Kwikeeze	Home maintenance	Liquid	5-10	
Savogran Sprayable Strypeeze-08/22/2001	Home maintenance	Aerosol	85-90	
Klean-Strip Klean Kutter	Home maintenance	Liquid	25-30	
Klean-Strip Metal & Masonry Paint Remover	Home maintenance	Liquid	75-85	
Klean-Strip Premium Stripper, Aerosol	Home maintenance	Aerosol	70-95	
Klean-Strip Deep Down Stain Stripper-old product	Home maintenance	Aerosol	<60	
Zinc It Electric Grade Lubricant	Home maintenance	Aerosol	32	

Product Brand	Category	Form	% DCM Content by Weight (as of June 2014) <sup>a</sup>
Savogran Kutzit Paint & Varnish Remover-old product	Home maintenance	Liquid	>24
Champion Sprayon Paint Off	Home maintenance	Aerosol	80 – 85
Sprayway Vandalism Mark and Stain Remover No. 870	Home maintenance	Aerosol	43
Zinsser Adhesive Remover	Home maintenance	Liquid	71
Crown Solu-Strip Semi-Paste Adhesive Remover	Home maintenance	Liquid	80-90
Crown Handi-Strip All Purpose Sprayable Stripper, Aerosol	Home maintenance	Aerosol	45-60
Savogran Adhesive Remover	Home maintenance	Liquid	85-90
Savogran Paint Stripper, Aerosol	Home maintenance	Aerosol	25-30
Savogran Heavy Duty SuperStrip	Home maintenance	Liquid	85-90
Klean-Strip Brush Cleaner	Home maintenance	Liquid	1-3
Klean-Strip KS-3 Premium Stripper	Home maintenance	Liquid	60-100
Klean-Strip Premium Sprayable Stripper	Home maintenance	Liquid	70-85
Klean-Strip Strip-X Stripper	Home maintenance	Liquid	30-50
Jasco Semi-Paste Varnish & Stain Remover	Home maintenance	Liquid	25-40
Klean-Strip Graffiti Remover-old product	Home maintenance	Aerosol	75-80
Savogran Strypeeze Paint/Varnish Remover-old product	Home maintenance	Liquid	>10
Parks Pro Liquid Paint Stripper-discontinued	Home maintenance	Liquid	40-90
Paint & Varnish Remover No. 2600, Aerosol	Home maintenance	Aerosol	
Klean-Strip Adhesive Remover	Inside the home	Liquid	60-100
Aqua Mix Sealer and Adhesive Remover-old product	Inside the home	Liquid	
Parks Adhesive Remover-discontinued	Inside the home	Liquid	40-90
Radio Shack Rosin Flux Stripper	Inside the home	Liquid	39.83
Parks Adhesive Remover-09/04/1998- discontinued	Inside the home	Liquid	65-70
Monsanto Amplify Herbicide (agricultural)	Pesticides	Granules	<16

Notes:

<sup>a</sup> EPA/OPPT searched the NIH Household Products Database in August 2012 and June 2014 (DHHS, 2012). Both searches reported the same list of consumer products and %DCM content with the exception of one product that showed up in the 2014 search, but not in the 2012 search. This product is *Canberra Husky 1229 Vandalism Mark and Stain Remover*. It is a home maintenance product available in aerosol form with 40-50% DCM content. In addition, five products had different category classifications in the 2012 and 2014 searches. Below are the product names and their categories (in parenthesis) as of June 2014.

- 1. Savogran Liquid Kutzit-08/31/2007 (Inside Home)
- 2. Savogran Sprayable Strypeeze-08/22/2001 (Arts/Crafts)
- 3. Savogran Heavy Duty SuperStrip (Arts/Crafts)
- 4. Aqua Mix Sealer and Adhesive Remover-old product (Home maintenance)
- 5. *Parks Adhesive Remover-09/04/1998-discontinued* (Arts/crafts)

# Appendix E ENVIRONMENTAL FATE OF DCM

Knowledge of the environmental fate (transport and transformation) of a compound is important to understanding its potential impact on specific environmental media (*e.g.*, water, sediment, soil) and exposures to target organisms of concern.

Releases of DCM to soil can volatilize from soil surfaces or migrate through soil and contaminate groundwater. DCM has high mobility in soil. It is not readily biodegradable, but biodegrades at varying rates under both aerobic and anaerobic conditions. The rate of hydrolysis is negligible.

The high vapor pressure and Henry's Law constant,  $2.19 \times 10^{-3}$  atm-m<sup>3</sup>/mole, indicate DCM has a tendency to partition to the atmosphere. DCM is expected to undergo slow photooxidation in the atmosphere and is considered moderately persistent and has low bioaccumulation potential (EPA, 1999, 2012b; NITE, 2002; OECD, 2011).

Due to its volatility, DCM enters the atmosphere where it reacts slowly enough to undergo atmospheric transport and act as a greenhouse gas. DCM has been reported to the Intergovernmental Panel on Climate Change (IPCC) as a global warming potential (GWP) chemical with a value of 8.7 [*i.e.,* or approximately 8.7 times more heat absorptive than carbon dioxide ( $CO_2$ )] GWP (Forster, 2007).

Table E-1 provides a summary of the environmental fate information for DCM. The sections below summarize current knowledge of the transport, persistence, bioaccumulation, and bioconcentration of DCM in the environment including biological and abiotic reactions and environmental distribution.

Table E         1. Environmental Fate Characteristics of DCM <sup>1</sup>			
Property	Value		
CASRN	79-09-2		
Photodegradation Half-life	107 days (estimated)		
Hydrolysis Half-life	18 months (measured)		
Biodegradation	13 % in 28 days (not readily biodegradable) <sup>2</sup>		
Bioconcentration Factor (BCF)	BCF = $2.0$ to $5.4$ (measured in carp) <sup>2</sup> ;		
	BCF = $<6.4$ to 40.0 (measured in carp) <sup>2</sup>		
	BAF = 2.6 (estimated) <sup>3</sup>		
Log K <sub>oc</sub>	1.4 (estimated) <sup>3</sup>		
Fugacity (Level III Model) <sup>3</sup>			
Air (%)	43.8		
Water (%)	45.0		
Soil (%)	11.0		
Sediment (%)	0.1		
Persistence <sup>4</sup>	Moderate		
Bioaccumulation <sup>4</sup>	Low		
Sources: <sup>1</sup> <u>OECD (2011)</u> <sup>2</sup> <u>NITE (2002)</u> <sup>3</sup>	EPA (2012b) <sup>4</sup> EPA (1999)		

# E-1 Fate in Air

If released to the atmosphere, DCM is expected to exist solely in the vapor-phase based on its vapor pressure. Vapor-phase DCM is degraded slowly in air by reaction with photochemically produced hydroxyl radicals. The half-life of this reaction is approximately 107 days. Thus, it is considered persistent in the atmosphere and subject to transport (<u>OECD, 2011</u>).

# E-2 Fate in Water

The low soil organic carbon partition coefficient ( $K_{oc}$ ) value (i.e., 25) suggests that DCM is not expected to adsorb to suspended solids and sediment when released to water. In the water column, DCM's rate of volatilization is expected to be high based on a Henry's Law constant of 2.19 × 10<sup>-3</sup> atm-m<sup>3</sup>/mole (<u>OECD, 2011</u>). A volatilization half-life of 21 minutes was measured for DCM when stirred in distilled water in a laboratory beaker at 25 °C (<u>Dilling et al., 1975</u>). The volatilization half-life increased to over 90 minutes when the solution was not stirred.

The rate of hydrolysis of DCM under environmental conditions is expected to be negligible. The hydrolysis half-life reported at neutral pH, is approximately 18 months at 25 °C (<u>Dilling et al., 1975</u>). Biodegradation is expected to occur slowly under aerobic conditions, but DCM may degrade more rapidly in anaerobic waters. A half-life of 11 days was reported for DCM in a 2 month laboratory study using bacteria isolated from an anaerobic aquifer as inoculum (Hazardous Substance Data Bank (<u>HSDB, 2012</u>). A half-life of 108 days was observed for DCM in contaminated groundwater under methanogenic conditions (<u>HSDB, 2012</u>).

# E-3 Fate in Soil/Sediment

Based on its low soil organic carbon partitioning coefficient ( $K_{oc}$ =25), DCM is likely to possess high mobility in soils and may be expected to leach from soils into groundwater (<u>ATSDR, 2000</u>). This is supported by the results of screening studies measuring DCM's biodegradability.

DCM present at 100 mg/L achieved 13 percent of its theoretical biochemical oxygen demand (BOD) using an activated sludge inoculum at 30 mg/L and the modified Ministry of International Trade and Industry (MITI) test (OECD 301C) over the course of a 4-week incubation period. The study findings indicated that DCM is not readily biodegradable (<u>NITE, 2002</u>).

DCM was shown to degrade under aerobic conditions in static culture screening studies that used domestic wastewater amended with yeast as inoculum (<u>Tabak et al., 1981</u>). Complete loss of DCM was observed within 7 days in the static culture tests. However, up to 25% of the loss could have arisen from volatilization. Taken together, these studies suggest that DCM is mobile in soils and persists long enough to migrate to groundwater given its low affinity for soil and potential to degrade somewhat slowly.

## E-4 Bioconcentration and Persistence

Bioconcentration and persistence are qualitatively characterized according to the criteria set forth in EPA's TSCA New Chemical Premanufacture Notification Program (PMN) (EPA, 1999). Though biodegradation tests of this substance found DCM not readily biodegradable, there is evidence that metabolism occurs under both aerobic and anaerobic conditions (HSDB, 2012; Tabak et al., 1981).

Bioconcentration factor (BCF) values ranging from 2.0 to 5.4 were measured for DCM in carp over a 6 week incubation period at an initial concentration of 0.25 mg/L (<u>NITE, 2002</u>). BCF values of <6.4 to 40.0 were observed when the concentration was 0.025 mg/L. Based on these studies, DCM is not expected to bioconcentrate significantly in aquatic organisms.

# Appendix F PAINT STRIPPING PROCESSES AND ASSOCIATED WORKERS ACTIVITIES, AND FACILITY AND POPULATION INFORMATION

Appendix F presents information about industries engaging in paint stripping activities, stripping processes, and facility and worker population data. This information serves as background information for the worker exposure estimates described in Appendix G.

# F-1 Identification of Industrial Sectors

Because a variety of industries include paint stripping among their business activities, an effort was made to determine and characterize these industries, especially for the "small commercial shops" of interest to EPA/OPPT. Note that the terms for commercial, industrial, and small shops often are difficult to distinguish, particularly as related to exposure data.

EPA/OPPT reviewed the published literature and evaluated the 2007 North American Industry Classification System (NAICS) codes to determine industries that likely included paint stripping activities. These industries are presented in Table F-1.

Table F 1. 2	2007 NAICS Codes Identi	fied that Include Paint Stripping Activities
2007	2007 NAICS Title	Rationale for Inclusion of NAICS
NAICS <sup>a</sup>		with Paint Stripping Activities
238320	Painting and wall	US Census reports an index entry of "Paint and wallpaper
	covering contractors	stripping" ( <u>USDOC, 2007b</u> ).
238330	Flooring contractors	US Census reports index entries of "Floor laying, scraping, finishing, and refinishing" and "Resurfacing hardwood flooring" ( <u>USDOC, 2007b</u> ). <u>NIOSH (1993)</u> cites the paint stripping of flooring by a wood flooring and restoration company.
811121	Automotive body, paint, and interior repair and maintenance	NAICS code 811121 is identified as the NAICS code for automobile refinishing per the Organisation for Economic Co-operation and Development (OECD) Coating Application <i>via</i> Spray-Painting in the Automotive Refinishing Industry ESD ( <u>OECD, 2010</u> ).
811420	Reupholstery and furniture repair	US Census reports index entries of "Furniture refinishing shops" and "Restoration and repair of antique furniture" (USDOC, 2007b).
711510	Independent artists, writers, and performers	US Census reports index entries of "Painting restorers, independent" and "Conservators ( <i>i.e.</i> , art, artifact restorers), independent" ( <u>USDOC, 2007b</u> ). Research has shown art conservation to use paint strippers based on DCM or NMP ( <u>Wollbrinck, 1993</u> ).
712110	Museums	Research has shown art conservation to use paint strippers based on DCM or NMP ( <u>Wollbrinck, 1993</u> ).

2007 NAICS <sup>a</sup>	2007 NAICS Title	Rationale for Inclusion of NAICS with Paint Stripping Activities
336411	Aircraft manufacturing	US Census reports an index entry of "Aircraft rebuilding ( <i>i.e.</i> , restoration to original design specifications)" ( <u>USDOC</u> , <u>2007b</u> ). Paint removal during the restoration process may use DCM- or NMP-based paint strippers.
336611	Ship building and repairing	US Census NAICS definition includes shipyards involved in the construction of ships as well as "their repair and conversion and alteration" ( <u>USDOC, 2007b</u> ). Any paint removal activities during repair, conversion, and alteration may use DCM- or NMP-based paint strippers.

NAICS codes were identified by performing general internet searches to identify workplace-related activities that involve paint stripping, and searching the U.S. Census 2007 NAICS website for keywords related to paint stripping, including those determined from the general internet search, such as "refinish," "stripping," "paint," "restorer," and "conservator" (<u>USDOC, 2007b</u>).

# F-2 Descriptions of Paint Stripping Processes and Activities in Relevant Industries

Techniques for paint stripping typically include manual coating, tank dipping, and spray application (<u>EC, 1999</u>). Pouring, wiping, and rolling are also possible application techniques, and application can be manual or automated (<u>ECHA, 2011</u>). An individual's exposure to paint stripping chemicals greatly depends on control measures taken and work practices adopted (<u>EC, 1999</u>). The following sections summarize processes and activities for the industries found to employ paint stripping.

### *F-2-1 Paint Stripping By Professional Contractors*

Paint strippers can be used by professional contractors to strip paint and varnish from walls, wood flooring, and kitchen and wood cabinets. Professional contractors are expected to purchase strippers in commercially available container sizes that commonly range from one liter up to 5 gallons, although they may also purchase consumer paint stripper products from hardware stores.

Stripper is typically applied to wall or floor surfaces using a hand-held brush. Strippers used in these applications often have a high viscosity since they can be applied to vertical surfaces. After application, the stripper is allowed to set and soften the old coating. Once the stripper has finished setting, the old coating is removed from the surface by scraping and brushing. During wood floor stripping, old coating and stripper may also be removed using an electric floor buffer. After the old coating is removed, the surface is wiped clean before moving to the next stages of the job. The stripping process is often completed on an incremental basis with treatment for one section of wall or flooring being completed before moving to the next section

(<u>CDHS/EPA, 2006</u>; <u>EC, 1999</u>; <u>EU, 2007</u>; <u>NIOSH, 1993</u>). Professional contractors can use portable local exhaust ventilation machines to increase ventilation in the vicinity of the paint stripping (<u>EU, 2007</u>).

Professional contractors may also be employed to refinish or reglaze bathtubs. Various health case studies have noted the use of DCM-based strippers during bathtub refinishing or reglazing (<u>CDC, 2012</u>; <u>Chester et al., 2012</u>; <u>MSU/MIFACE, 2011</u>). Case studies have identified professional bathtub refinishers that repaired and resurfaced countertops, tubs, and sinks in both apartment buildings and private homes (<u>Chester et al., 2012</u>; <u>MSU/MIFACE, 2011</u>).

In addition, the OSHA IMIS data identified two OSHA or state health inspections in 2004 and 2007 of two bathtub reglazers/refinishers. The bathtub reglazers' company in the 2007 inspection was identified under NAICS code 811420 – Reupholstery and Furniture Repair (CDC, 2012). However, this assessment discusses bathtub reglazing/refinishing in the context of professional contractors, as professional contractors and professional bathtub refinishers or reglazers are both expected to perform their work at customer sites (for example, in the cited case studies of bathtub refinishers/reglazers, apartment buildings, and private homes). This professional contractor-type work differs from furniture refinishing, which typically entails the refinishing of customer furniture at fixed furniture refinishing facilities.

Bathtub refinishing or reglazing can involve a worker pouring and brushing stripper onto a bathtub using a paintbrush. The worker then scrapes the finish from the bathtub after leaving the stripper in contact with the bathtub for 20 to 30 minutes (<u>Chester et al., 2012</u>; <u>MSU/MIFACE, 2011</u>). This information was obtained from a case study that noted a stripper DCM concentration of 60 to 100 percent (<u>Chester et al., 2012</u>; <u>MSU/MIFACE, 2011</u>). However, multiple health case studies have reported the use of aircraft and marine coating remover in bathtub refinishing/reglazing (<u>CDC, 2012</u>).

## F-2-2 Graffiti Removal

Unlike fixed facility operations, graffiti removal is expected to employ similar job-site characteristics as professional contractors. Swedish studies of graffiti removal companies (using both DCM- and NMP-based solvents) identified that solvents are either spray or brush applied. Sprayed solvents can be swabbed or wiped with a cloth or tissue. After spraying and wiping or brushing the solvent on the surface, the surface is then washed with heated (70°C) wash water using a high-pressure spray.

The observed work was performed in train depots and underground stations and included confined spaces, such as elevators and train cars. The study authors noted poor ventilation in the confined spaces. The authors also noted the potential for members of the general public to be indirectly exposed as work was conducted during the day while travelers were occupying the train depots and stations (<u>Anundi et al., 2000</u>; <u>Anundi et al., 1993</u>). The prevalence of graffiti removal companies in the U.S. is uncertain. Graffiti removal in the U.S. may be performed by public works municipal workers or contractors.

#### *F-2-3 Paint Stripping at Automotive Body Repair and Maintenance Shops*

Automotive refinishing shops apply coatings to motor vehicles subsequent to the original manufacturing process. The overall refinishing process typically involves the following steps:

- Structural repair;
- Surface preparation (cleaning and sanding);
- Primer coat mixing;
- Spray application of primer coat;
- Curing;
- Sanding;
- Solvent wipe-down;
- Topcoat (basecoat color and clearcoat) mixing;
- Spray application of topcoat; and
- Curing.

As stated in <u>OECD (2010)</u>, the surface preparation step of the refinishing process involves "removing residual wax, grease, or other contaminants from the surface to be painted, to ensure adhesion of the new coating. The new coating may be applied over an existing coating if it is free of chips or cracks after it has been roughened through sanding. Alternatively, the previous coating may be removed using a mechanical method (e.g., sanding) or a paint-removing solvent. After the coating is roughened or removed, the surface is typically wiped down with a solvent- or water-based surface preparation product".

#### F-2-4 Wood Furniture Stripping

During furniture stripping, paint stripper may be applied to the furniture by either dipping the furniture in an open tank containing the stripper, brushing or spraying the stripper onto the furniture surface, or manually applying the stripper. Larger facilities may pump the stripper through a brush. The application method depends on the size and structure of the furniture as well as the capabilities of the facility.

The application area typically has a sloped surface to allow for collection and recycling of unused stripper. Larger facilities use a flow tray to apply the stripper to parts. The flow tray is a sloped, shallow tank with a drain at the lower end.

After application, the stripper is left to soak on the furniture surface to soften the surface coating. Once soaking is complete, the unwanted coating is scraped and brushed from the furniture surface. The furniture is then transferred to a washing area where residuals are washed from the furniture.

Washing can be performed using low-pressure washing operations or high-pressure water jets or high-pressure wands. Wash water may contain oxalic acid to brighten the wood surface. Wash water is collected and either recycled or disposed of as waste. After washing, the

furniture is transferred to a drying area where it is allowed to dry before being transferred to other refinishing processes (*e.g.*, sanding, painting, reupholstery)(<u>CDHS/EPA, 2006</u>; <u>HSE, 2001</u>; <u>NIOSH, 1990</u>, <u>1993</u>).

Larger facilities likely purchase stripper in drum quantities from suppliers. Smaller facilities that use hand stripping instead of stripping equipment likely purchase their stripper from hardware and home improvement stores. Stripper applied using application equipment has low viscosity, so it can be pumped through the pumps in the flow tray. Strippers applied using hand stripping are typically more viscous, so they will remain on the part long enough to strip the coating (CDHS/EPA, 2006).

Figure F-1 shows a typical flow tray used by larger furniture strippers to apply stripper to furniture parts, obtained from <u>CDHS/EPA (2006)</u>. Figure F-2 shows a typical water wash booth used to wash stripper and coating residue from stripped furniture, obtained from (<u>CDHS/EPA</u>, <u>2006</u>). Figure F-3 shows an example diagram of a dipping tank for furniture stripping complete with local exhaust ventilation, obtained from (<u>HSE</u>, 2001).



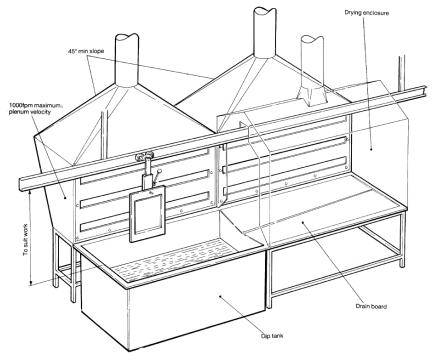
#### Figure F-1. Typical Flow Tray for Applying Stripper to Furniture

Source: CDHS/EPA (2006)

Figure F-2. Typical Water Wash Booth Used to Wash Stripper and Coating Residue from Furniture



Source: CDHS/EPA (2006)



Source: <u>HSE (2001)</u>

#### *F-2-5* Art Restoration and Conservation

Art restoration and conservation can include the care and maintenance of paintings to reverse negative effects of aging and dirt accumulation. It can also include repairing paintings that have suffered paint loss, weakened canvas, tears, water damage, fire damage, and insect damage (<u>Smithsonian, 2012b</u>). Art restoration and conservation can include paint cleaning, which can entail removing dirt and other obscuring material, removing varnish, or removing overpaint while maintaining the original layer of paint (<u>Smithsonian, 2012a</u>). These activities can involve the use of paint strippers.

Although paint strippers used in this field can contain DCM, the use of DCM is not always favored, as DCM can penetrate through the overpaint layer that is being removed and into the original paint layer that is being conserved. Products marketed for use in this field that do not contain DCM may contain N-Methylpyrrolidone (NMP) (<u>Wollbrinck, 1993</u>). More detailed information on the use of paint strippers in art restoration and conservation was not identified. It is anticipated that paint strippers are applied manually in this field.

#### F-2-6 Aircraft Paint Stripping

During aircraft paint stripping, paint stripper is pumped from bulk storage containers or tanks and applied to the body of the aircraft using hoses. Once the paint stripper has been applied, it is allowed to set for a certain period of time (usually about 30 minutes) to allow the paint to soften. Once setting is complete, the stripper and loose paint are scraped down into a collection area. Any remaining stripper and paint residue are then brushed or washed away with water and brushes. Once the surface of the aircraft has dried, a new layer of primer, paint, and top coat are applied (NIOSH, 1977).

#### F-2-7 Ship Paint Stripping

Process description information for paint stripping of ships has not been identified. It is anticipated that paint stripping of ships may involve similar processes as the paint stripping of aircraft.

#### F-2-8 Respiratory Protection

OSHA requires NIOSH-approved supplied-air respirators when respiratory protection is required to protect against DCM. Air-purifying respirators do not provide adequate respiratory protection against DCM (<u>OSHA, 1997b</u>).

EPA/OPPT examined 13 MSDS for paint strippers and checked the recommendations for respiratory protection. Ten of the MSDS were for DCM-containing paint strippers. Eight of the 10 MSDS recommended a NIOSH-approved, self-contained breathing apparatus or air-supplied respirator if respiratory protection is required. One MSDS recommended NIOSH-approved

respiratory protection for organic solvent vapors, which may include the use of supplied air. The remaining MSDS only recommended a NIOSH-approved respirator for organic solvent vapors without further specification of the respirator type (<u>WM Barr, 2008</u>, <u>2009a</u>, <u>2009b</u>, <u>2010a</u>, <u>2010b</u>, <u>2011a</u>, <u>2011b</u>, <u>2011c</u>, <u>2012</u>).

## F-3 Facility and Worker Population Data

This section summarizes data on the number of facilities and workers nationwide that perform DCM-based paint stripping activities. It also includes data on the number of workers per facility, which can be a factor in determining shop sizes.

#### *F-3-1 Potentially Exposed Population in the U.S.*

EPA/OPPT estimated that over 230,000 workers, who directly use DCM-based strippers, are potentially exposed to DCM from these products (Table F-2). EPA/OPPT cannot estimate the numbers of workers exposed in each of the individual industries that may use DCM-based strippers. EPA/OPPT cannot estimate the numbers of workers exposed in small shops. Also, there was no information that EPA/OPPT could use to estimate the number of additional workers within the facility who are indirectly exposed to DCM.

with DCM					
	AREA SOURCE FACILITIES		MAJOR SOURCE FACILITIES	TOTAL FACILITIES NATIONWIDE	
	Model Plant Type		Assumed Model Plant Type		
Model Plant Type	1	2	3	3	
Workers per site (assumed)	2	7	20	20	
Total number of sites <sup>a</sup>	1,470	780	750	10,500	13,500
Total number of workers <sup>b</sup>	2,940	5,460	15,000	210,000	233,400
Notes:					

<sup>a</sup> A total of 3,000 area source facilities is obtained by summing the number of sites for model plants #1, 2 and 3 (i.e., 1,470 + 780 + 750, respectively).

<sup>b</sup> A total of 23,400 workers is obtained by summing the number of workers for model plants #1, 2 and 3 (i.e., 2,940 + 5,460 + 15,000, respectively).

Workers who are bystanders and not directly involved in using DCM-based strippers were not included in this estimate. The remainder of this section and Table F-2 describe EPA/OPPT's approach for estimating this population.

EPA/OPPT estimates given above were based on the following data and assumptions. EPA/OPPT compiled information from the exposure literature sources and the technical support document for the National Emission Standards for Hazardous Air Pollutants (NESHAP) Paint Stripping Operations at Area Sources proposed rule (EPA, 2007).

The NESHAP technical support document estimated the number of workers performing paint stripping using DCM at area source facilities, specifically a total of 23,400 workers among 3,000 area source facilities. In the NESHAP analysis, area sources were defined as facilities that emit less than 10 tons/yr of DCM, and major sources were defined as facilities that emit at least 10 tons/yr of DCM. When estimating the number of workers, and area source facilities, the NESHAP document assumed three types of model plants (each with a different average number of workers per site) and estimated the number of each model plant type (Table F-2).

However, the estimate of 3,000 area source facilities represented only 22 percent of the total facilities nationwide that use DCM in paint stripping operations. The remaining facilities (78%) include major source facilities (or area sources covered by other area source rules) and consumer uses. The estimate of 3,000 area source facilities did not include paint stripping operations in private-sector aircraft maintenance or military maintenance activities. It also did not include paint stripping during original equipment manufacturing (such as the manufacture of automobiles, furniture, and other equipment).

The total number of all paint stripping facilities (excluding consumer uses) that use DCM has been previously estimated at approximately 13,500 (Johnson, 2007)(Table F-2). Assuming the additional 10,500 facilities not accounted for in the area source estimate are larger, major source facilities, and assuming major source facilities are equal in size to Model Plant Type 3, the total number of workers nationwide that perform paint stripping using DCM could be well over 230,000 (Table F-2).

The estimates of numbers of exposed workers are highly uncertain. The most uncertain parameter used in the method is the number of workers using strippers designated for each particular model plant. EPA/OPPT cannot determine whether the assumed numbers of workers designated for these model plants may underestimate or overestimate the numbers of workers. However, the inclusion of only numbers of workers who actually use the strippers would underestimate the total number of workers exposed because non-users (bystanders) are excluded.

#### *F-3-2 Numbers of Workers per Facility by Industry*

This section summarizes data on the number of establishments, number of paid employees and workers, and production hours and work day estimates (for manufacturing industries). Some of these data are useful for determining the average number of workers per establishment, which can indicate relative sizes of the businesses.

# *F-3-2-1 Paint Stripping By Professional Contractors, Bathtub Refinishing, and Graffiti Removal*

Table F-3 summarizes the number of establishments and average number of workers for painting and wall covering contractors and flooring contractors according to the 2007 U.S. Economic Census (<u>USDOC, 2007a</u>).

Table F 3. 2007 U.S. Economic Census Data for Painting and Wall Covering and         Flooring Contractors					
2007 NAICS	2007 NAICS Title	2007 Number of Establishments	2007 Average Number of Construction Workers		
238320	Painting and wall covering contractors	35,619	174,276		
<b>238330</b> Flooring contractors 14,575 49,085					
Source: USDOC (2007a)					

The Census data did not include hours worked for construction industry sectors nor data about bathtub refinishers/reglazers or graffiti removal. Also, there were no data about the number of painting and wall covering contractors and flooring contractors who use DCM-based paint strippers, the number of jobs per year a contractor uses DCM-based paint strippers, and the number of workers within a job site exposed to DCM-based paint strippers.

The number of establishments and workers from the U.S. Census provided some context for potential numbers of establishments and workers potentially exposed to DCM during paint stripping. While some fraction of these workers may be exposed to DCM, the Census data did not include self-employed, single person businesses, and some of these workers may also be exposed to DCM. The Census data indicated an average of approximately 4 to 5 workers per establishment.

Many bathtub refinishers are self-employed or a small business (<u>Chester et al., 2012</u>). Past investigations of fatalities that occurred during bathtub refinishing indicate it is likely that only one contractor refinishes a bathtub at a time (<u>CDC, 2012</u>; <u>Chester et al., 2012</u>; <u>MSU/MIFACE, 2011</u>).

Swedish studies of graffiti removal companies identified one company with 12 workers (<u>Anundi</u> <u>et al., 1993</u>), and a separate study monitored a total of 38 workers over five companies (an average of seven to eight workers monitored per company)(<u>Anundi et al., 2000</u>). As previously discussed, the prevalence of graffiti removal companies in the U.S. is uncertain as graffiti removal may be performed by public works municipal workers or contractors.

#### *F-3-2-2 Paint Stripping at Automotive Body Repair and Maintenance Shops*

Table F-4 summarizes the number of establishments and average number of paid employees for automotive body, paint, and interior repair and maintenance according to the 2007 U.S.

Economic Census (<u>USDOC, 2007a</u>). The Census data did not include hours worked for this industry sector.

Table F 4. 2007 U.S. Economic Census Data for Automotive Body, Paint, and InteriorRepair and Maintenance				
2007 NAICS	2007 NAICS Title	2007 Number of Establishments	2007 Number of Paid Employees	
811121	Automotive body, paint, and interior repair and maintenance	35,581	223,942	
Source: USDOC (2007a)				

The Census data indicated an average of approximately 6 employees per facility (<u>USDOC</u>, <u>2007a</u>). A 2003 Rhode Island study observed two comparably-sized vehicle repainting shops. One of the two shops had a total of 14 employees (<u>Enander et al., 2004</u>).

In 1998, the Rhode Island Department of Environmental Management (DEM) surveyed over 350 body shops and found that 20 percent of the shops still used DCM as a paint stripper at that time. It is unknown if this fraction of body shops in Rhode Island in 1998 (that used DCM) is representative of body shops within the entire U.S. Rhode Island DEM recommends eliminating the use of DCM-based paint strippers as a pollution prevention measure (<u>RIDEM, 2011</u>). Therefore, it is uncertain if the 20 percent of shops that used DCM in 1998 is representative of the fraction of shops that use DCM in the present day.

EPA/OPPT did not find information about the current number of automotive body repair and maintenance shops within the U.S. that use DCM-based paint strippers, nor the number of employees within an establishment exposed to DCM-based paint strippers. Therefore, the number of establishments and employees from the U.S. Census are possibly overestimates of the number of establishments and employees potentially exposed to DCM during paint stripping.

A 2003 Rhode Island study that monitored exposures to DCM in a vehicle repainting shop noted a use rate of DCM of 1 to 2 gallons per week for that particular facility (<u>Enander et al., 2004</u>).

### F-3-2-3 Wood Furniture Stripping

Table F-5 summarizes the number of establishments and average number of paid employees for reupholstery and furniture repair according to the 2007 U.S. Economic Census (<u>USDOC</u>, <u>2007a</u>). The Census data also indicated an average of approximately 3 employees per facility (<u>USDOC</u>, <u>2007a</u>). However, the Census data did not include hours worked for this industry sector.

Table F 5. 2	007 U.S. Economic Census D	ata for Reupholstery a	and Furniture Repair
2007 NAICS	2007 NAICS Title	2007 Number of Establishments	2007 Number of Paid Employees
811420	Reupholstery and furniture repair	4,693	16,142
Source: USDO	<u>C (2007a)</u>		

A total of 10 furniture refinishing shops were identified among the exposure studies. Of these 10 shops, only one was confirmed to have greater than 10 total employees (this shop had 18 workers) (Grevenkamp, 2007). Three of the shop studies only monitored a single refinisher (one shop was owned and operated by the single refinisher) (Estill and Spencer, 1996; McCammon et al., 1991; NIOSH, 1990, 1991). The remaining shop studies monitored two to four refinishers (and one shop was confirmed to have a total of only six employees)(Hall et al., 1995; McCammon et al., 1991).

OSHA conducted a Regulatory Impact Analysis (RIA) in 1996 for the 1997 OSHA Methylene Chloride Standard. In the RIA, OSHA estimated 6,152 establishments engaged in furniture paint stripping using DCM and estimated 7,872 workers exposed during this activity. The 2010 OSHA Regulatory Review of the Methylene Chloride Standard estimates that the number of reupholstery and furniture repair facilities decreased to fewer than 6,000 by 2003. The 2007 U.S. Economic Census reported a further decline in the total number of establishments. However, it is unknown if these data are representative of the population of establishments and workers that use DCM in the present day (<u>OSHA, 2010</u>).

EPA/OPPT did not have information about the current population of reupholstery and furniture repair establishments that use DCM-based paint strippers and the number of employees within an establishment exposed to DCM-based paint strippers. Therefore, the number of establishments and employees from the U.S. Census are possibly overestimates of the population of establishments and employees potentially exposed to DCM during paint stripping.

The Institute for Research and Technical Assistance (IRTA) surveyed the furniture stripping industry in the South Coast Basin in Southern California to determine the usage of DCM-based strippers (Table F-6). IRTA then used these data to estimate the number of firms in the state of California that use DCM-based strippers (Table F-6)(<u>CDHS/EPA, 2006</u>). The source did not identify the year in which these data were obtained. It is unknown the representativeness of the distribution of facility annual use rate of stripper across the entire U.S.

<u>CDHS/EPA (2006)</u> identifies the facilities that use >200 gallons/yr of stripper as larger facilities that purchase stripper in drum quantities from suppliers. The firms that use <200 gallons/yr of stripper likely use hand stripping and purchase their stripper from hardware and home improvement stores (<u>CDHS/EPA, 2006</u>).

Table F 6. Estimated Annual DCM Based Stripper Usage in California <sup>a</sup>							
Annual Stripper Usage (Gallons per Year)	Number of Firms in California						
1,200-2,000	6						
700-1,200	30						
200-700	40						
5-200	172						
<5	248						
Total	596 <sup>b</sup>						
Notes:							
<sup>a</sup> Source: <u>CDHS/EPA (2006)</u>							
<sup>b</sup> <u>CDHS/EPA (2006)</u> notes a total of 596 firms in Calife	ornia. However, an actual summation						
of the firms gives a total of 496.							

#### F-3-2-4 Art Restoration and Conservation

Table F-7 summarizes the number of establishments and average number of paid employees for independent artists, writers, and performers and museums according to the 2007 U.S. Economic Census (<u>USDOC, 2007a</u>). The Census data did not include hours worked for these industry sectors.

	007 U.S. Economic Census D Art Restoration and Conser		rs that May Engage in
2007 NAICS	2007 NAICS Title	2007 Number of Establishments	2007 Number of Paid Employees
711510	Independent Artists, Writers, and Performers	20,612	48,321
712110	Museums	4,664	83,899
Source: USDO	<u>C (2007a)</u>		

NAICS code 711510 includes a wide variety of professions, including independent art restorers and independent conservators. According to the U.S. Census Bureau, the majority of the professions listed within this NAICS code are not expected to engage in paint stripping. Furthermore, the extent that art restorers and conservators engage in paint stripping is unknown particularly for the use of DCM-based paint strippers.

Similarly, it is unknown the number of museums within NAICS code 712110 that use DCMbased paint strippers. Therefore, the number of establishments and employees from the U.S. Census are likely overestimates of the number of establishments and employees potentially exposed to DCM during paint stripping.

#### F-3-2-5 Aircraft Paint Stripping

Table F-8 summarizes the number of establishments, average number of production workers, and production workers hours for aircraft manufacturing according to the 2007 U.S. Economic Census (<u>USDOC</u>, 2007a). The table also estimates the average worker days per yr and average worker hrs per day. These parameters were estimated from the production workers hours and the average number of production workers. The calculations for the average worker days per yr when estimating the average worker hrs per day. The calculations also assumed 250 worker days per yr when estimating the average worker hours per day. The estimates of worker days per yr and worker hrs per day were within 10 percent of the EPA/OPPTs' New Chemicals Program default values of 250 days/yr and 8 hr/day, respectively (<u>EPA</u>, 1993a).

The Census data indicated an average of approximately 320 production workers per facility. This observation is consistent with the exposure studies identified in the literature. A 1977 NIOSH study of an aircraft refinishing facility observed approximately 1,400 employees working in the dock area, which constituted seven refinishing docks but appeared to exclude workers and employees associated with security checkpoints, the front lobby, cafeterias, the credit union, the turbine shop, the medical bay, and maintenance activities (NIOSH, 1977). Similarly, a 1994 French study of an aeronautical workshop monitored 30 painters, although the total number of employees was not identified (Vincent et al., 1994).

Table F	8. 2007 U.S.	Economic Censu	ıs Data for Ai	rcraft Manuf	acturing	
	200	the Corresp	alculated from oonding 2007 Census Data			
2007 NAICS Code	2007 NAICS Title	Number of Establishments	Average Number of Production Workers	Production Workers Hours (1,000 hr)	Average Worker Days per Year (Assuming 8 hr/day)	Average Worker Hrs per Day (Assuming 250 days/year)
336411	Aircraft manufact- uring	254	81,456	157,589	242	7.74
Source:	JSDOC (2007a)					

In the 1996 RIA, OSHA estimated 300 establishments engaged in paint stripping of aircrafts using DCM and estimated 2,470 workers potentially exposed during this activity. Further, the 1996 RIA estimate of number of establishments using DCM-using establishments was similar to the estimate provided by the 2007 U.S. Economic Census (i.e., 254). However, the 2007 U.S. Economic Census presented a much greater number of workers (i.e., 81,456) than the RIA estimate of number of workers exposed to DCM (i.e., 2,470). It is unknown if these data are representative of the number of establishments and workers that use DCM in the present day (OSHA, 2010).

EPA/OPPT did not have information about the current number of aircraft manufacturing establishments that use DCM-based paint strippers and the number of employees within an establishment exposed to DCM-based paint strippers. Therefore, the number of establishments and employees from the U.S. Census are possibly overestimates of the number of establishments and employees potentially exposed to DCM during paint stripping.

### F-3-2-6 Ship Paint Stripping

Table F-9 summarizes the number of establishments, average number of production workers, and production workers hours for ship building and repairing according to the 2007 U.S. Economic Census (<u>USDOC</u>, 2007a). The table also estimates the average worker days per year and average worker hours per day. These parameters were estimated from the production workers hours and the average number of production workers. The calculations for the average worker days per year assumed 8 worker hrs per day. The calculations also assumed 250 worker days per yr when estimating the average worker hrs per day. The estimates of worker days per yr and worker hours per day were within 10 percent of the EPA/OPPTs' New Chemicals Program default values of 250 days/yr and 8 hr/day, respectively (<u>EPA</u>, 1993a).

Table F	9. 2007 U.S	. Economic Cens	us Data for Sl	nip Building a	nd Repairing	
	20	007 Economic Cens	sus Data		the Corres	Calculated from conding 2007 Census Data
2007 NAICS Code	2007 NAICS Title	Number of Establishments	Average Number of Production Workers	Production Workers Hours (1,000 hr)	Average Worker Days per Year (Assuming 8 hr/day)	Average Worker Hrs per Day (Assuming 250 days/year)
336611	Ship building and repairing	656	65,737	136,929	260	8.33
Source: U	<u>ISDOC (2007a)</u>					

The Census data also indicated an average of approximately 100 production workers per facility.

EPA/OPPT did not have information about the number of ship building and repair establishments that use DCM-based paint strippers and the number of employees within an establishment exposed to DCM-based paint strippers. Therefore, the number of establishments and employees from the U.S. Census are possibly overestimates of the number of establishments and employees potentially exposed to DCM during paint stripping.

# Appendix G OCCUPATIONAL EXPOSURE LITERATURE DATA AND EXPOSURE CALCULATIONS

# G-1 Data Needs, Data Collection Strategy and Data Quality Criteria for the Occupational Exposure Analysis

#### G-1-1 Data Needs

EPA/OPPT defined the data needs for the completion of the occupational exposure assessment for the use of DCM-based strippers before starting data collection. These data needs included both quantitative data (e.g., exposure measurements) and qualitative information (e.g., descriptions of worker activities).

The following data needs were required for the occupational exposure assessment:

- Inhalation exposure monitoring data of DCM during paint stripping, specifically full-shift 8-hour (hr) time-weighted average (TWA) personal breathing zone samples
  - Monitoring of over 5-hr duration was assumed adequate to represent full-shift exposure levels. Area and short-term samples were found and presented in the discussions of literature data for perspective and completeness but were not used in the occupational exposure concentration calculations and risk analyses. Personal samples provide a better representation of the amount of DCM inhaled by the worker when compared to area samples.
- Description of processes and worker activities used to perform paint stripping
- Description of engineering controls and personal protective equipment used during paint stripping
- Estimates of number of workers exposed to DCM during paint stripping in the U.S.
- Estimates of the number of facilities that perform DCM-based paint stripping in the U.S.

In general, the inhalation exposure monitoring data were from occupational settings representing the relevant industry. However, there were instances that surrogate personal inhalation data were used when no data were available for the relevant industry. These cases are discussed below in the summary of the occupational literature and corresponding uncertainties.

The following data were not considered in the occupational analysis:

- Modeling results: Monitoring data were preferred to modeling unless there were known data quality issues, including data representativeness, or if modeling results were expected to be useful for filling data gaps or addressing other data weaknesses;
- Biological measurements (e.g., blood or urine samples): The types of approaches used in this risk assessment did not require these types of data;
- Recreated exposure conditions from case studies: Monitoring data were preferred to
  recreated exposure conditions unless the monitoring data had known data quality issues or
  the data representativeness was believed to be weak;
- Exposure data from non-paint stripping industries: Paint stripping exposure data would better reflect the exposure conditions when compared to exposures from non-paint stripping industries.

The detailed summaries of the literature studies presented below in this appendix include mention of some data that did not meet the data needs described above (e.g., there is mention of modeled, and not measured, exposure data that were discussed in an investigation of a bathtub refinishing fatality). These data are presented for perspective only. The data in the detailed summaries and in Table G-2 meet the first bulleted data need above (breathing zone monitoring data of DCM during paint stripping).

#### G-1-2 Data Collection Strategy

EPA/OPPT's literature search comprised a general Internet search and a targeted search of specific Internet resources. To begin the literature search, EPA/OPPT defined primary keywords to use in the search queries. The defined primary keywords were:

- dichloromethane
- methylene chloride
- paint stripp\*

EPA/OPPT included the preferred chemical name "dichloromethane" as well as the chemical synonym "methylene chloride." The wildcard (\*) allows for variations of the word "strip", including "stripper" and "stripping." To sort through extensive search results, EPA/OPPT used secondary keywords including, but not limited to, the following:

- expos\*
- inhal\*
- breathing zone

Here, the wildcard (\*) allows for the variations: "exposure", "exposures", "exposed", "inhale", and "inhalation."

EPA/OPPT used these keywords in queries performed in an Internet search engine (e.g., Google) for the general Internet search and in the following targeted NIOSH online resources.

- NIOSH Workplace Survey Reports: <u>http://www.cdc.gov/niosh/surveyreports/</u>
- NIOSH Health Hazard Evaluations (HHEs): <a href="http://www2a.cdc.gov/hhe/">http://www2a.cdc.gov/hhe/</a>

EPA/OPPT obtained inhalation exposure data from OSHA and state health inspections from the OSHA's Integrated Management Information System (IMIS) database. Also, some additional studies were identified during the public and peer reviews of the 2012 draft DCM risk assessment.

#### G-1-3 Data Quality Criteria

EPA/OPPT defined criteria to evaluate the quality of collected data. Also, EPA/OPPT developed and used acceptance specifications for each data quality criterion to determine if the collected data were of acceptable quality for use in this risk assessment. Table G-1 summarizes the data quality criteria, the definition or description of each criterion, and the corresponding acceptance specifications used to determine if the data were acceptable for use.

EPA/OPPT accepted surrogate data for two industries (professional contractors and art restoration and conservation) for use in the occupational exposure assessment. For professional contractors, EPA/OPPT accepted for use some consumer paint stripping exposure data from U.S. and European studies. The uncertainties associated with these surrogate data are described in the Paint Stripping by Professional Contractors section of this appendix.

EPA/OPPT also accepted surrogate data for art restoration and conservation because no other data were identified in the literature search. The surrogate data for air restoration and conservation were obtained from the OSHA IMIS database. Although the relevance of the surrogate data is uncertain, the data point met the data need of personal, inhalation monitoring of DCM (see *section G-3-6* for more information).

Table G 1. Data C	Quality Criteria and Acceptance Speci	fications for Occupational Data
Quality Criterion	Description/Definition	Acceptance Specification
Currency	The information reflects present	Data from all years are acceptable.
(up to date)	conditions.	
Geographic Scope	The information reported reflects an area relevant to the assessment.	Exposure and process description data from the United States and the rest of world are acceptable. Only US estimates of number of workers and number of facilities that perform paint stripping are acceptable.
	<ul> <li>The information reported is reliable. For example, this criterion may include the following acceptance specifications:</li> <li>The information or data are from a peer-reviewed, government, or industry-</li> </ul>	Data are reliable if they are from one of the following sources: US or other government publication.
	<ul><li>specific source.</li><li>The source is published.</li></ul>	<ul> <li>Sources by an academic researcher where:</li> <li>Publication is in peer-reviewed journal; or</li> </ul>
Reliability	• The author is engaged in a relevant field such that competent knowledge is expected (i.e., the author writes for an industry trade association publication	<ul> <li>Presented at a technical conference; or</li> <li>Source has documented qualifications or credentials to discuss particular topic.</li> </ul>
	<ul> <li>versus a general newspaper).</li> <li>The information was presented in a technical conference where it is subject to review by other industry experts.</li> </ul>	<ul> <li>Sources by an industry expert or trade group where:</li> <li>Presented at a technical conference where the information is subject to review by other industry experts; or</li> <li>Source has documented qualifications or credentials to discuss particular topic; or</li> <li>Source represents a large portion of the industry of interest.</li> </ul>
Unbiased	The information is not biased towards a particular product or outcome.	<ul> <li>Objective of the information is clear.</li> <li>Methodology is designed to answer a specific question.</li> </ul>
Comparability	The data are comparable to other sources that have been identified.	Data sources will not be accepted or rejected based on their comparison to data from other sources.
Representativeness	The data reflect the typical industry practices. The data are based on a large industry survey or study, as opposed to a case study or sample from a limited number of sites.	Literature sources are not rejected based on the sample size of sites. Large industry surveys as well as case studies and limited sample sizes are acceptable.
Applicability	For surrogate data, the data are expected to be similar for the industry or property of interest.	Surrogate data deemed applicable if they are inhalation exposure or airborne concentration data of DCM measured during paint stripping.

# G-2 Approach and Methodology for Estimating Occupational Exposure

#### G-2-1 Identification of Relevant Industries

Because a variety of industries include paint stripping among their business activities, EPA/OPPT made the effort to determine and characterize these industries, with a special interest in small commercial shops.<sup>21</sup>

EPA/OPPT reviewed the published literature and evaluated the 2007 North American Industry Classification System (NAICS) codes to determine industries that likely include paint stripping activities (see *Appendix F, Table F-1*).

The identified industries were the following:

- Professional contractors;
- Bathtub refinishing;
- Automotive refinishing;
- Furniture refinishing;
- Art restoration and conservation;
- Aircraft paint stripping;
- Ship paint stripping; and
- Graffiti removal

By identifying these industries, EPA/OPPT determined worker subpopulations that may be exposed to DCM due to the use of these strippers. Appendix F details the industries identified and processes and worker activities that may contribute to worker exposures.

# *G-2-2 Estimation of Potential Workplace Exposures for DCM-Based Paint Strippers*

#### G-2-2-1 Workplace Exposures Based on Monitoring Data

EPA/OPPT used air concentration data and estimates found in literature sources to serve as exposure concentrations for occupational inhalation exposures to DCM. These air concentrations were used to estimate the exposure for workers exposed to DCM as a result of the use of DCM-based paint strippers.

<sup>&</sup>lt;sup>21</sup> Please note that differences among commercial, industrial, and small shops are often difficult to distinguish, particularly as related to exposure data. For more information about shop size determination, see section 3.1.1.2 and Appendix F.

EPA/OPPT did not find enough monitoring data to determine complete statistical distributions of actual exposure concentrations for the exposed populations. Ideally, EPA/OPPT would like to know 50<sup>th</sup> and 95<sup>th</sup> percentiles for each population. The air concentration means and midpoints (means are preferred over midpoints) served as substitutes for 50<sup>th</sup> percentiles, and high ends of ranges served as substitutes for 95<sup>th</sup> percentiles.

In compiling the results from the individual literature sources into the results summary for this risk assessment, EPA/OPPT classified exposure durations of 5 hrs or greater as "8-hr TWA" exposures. Exposure durations less than 5 hrs and unknown durations were classified as "STEL, peak, short-task based, and unknown" exposures.

Data sources did not often indicate whether exposure concentrations were for occupational users or bystanders. Therefore, EPA/OPPT assumed that occupational exposures were for a combination of users and bystanders. Some bystanders may have lower exposures than users, especially when they are further away from the source of exposure.

Additionally, inhalation exposure data from OSHA and state health inspections were obtained from the OSHA IMIS database. However, OSHA IMIS data were generally excluded to estimate workplace exposure estimates, except where noted, because (1) inhalation exposures for DCM found in IMIS may or may not be caused by DCM-based strippers; and (2) data from literature were deemed adequate to estimate exposures from DCM-based strippers. In this assessment, the IMIS data were useful for examining the impact of the OSHA PEL update in 1997 on exposures in the industries that are most likely to employ DCM-based strippers (*see section G-3-10*).

Table G-2 presents a summary of the exposure data collected for each industry. The risk characterization of occupational exposures was based on the 8-hr TWA data in Table G-2. The data met the data needs and data quality criteria described in *section G-1*.

Table G 2. DCM Cano				Used for Estimat 8 hr Air Concent					nic Expo	osure Cor	ncentrat	tions fo	r Non
Industry / Activity						8-hr TW	A (mg/m³)ª		-	Peak, Short Id Unknow		-	Data Source for 8-hr TWA and short- term values
	Number of Studies	Time Range of Studies	Number of Sites	Total Number of Measurements <sup>c</sup>	Mean	High	Midpoint <sup>d</sup>	Low	Mean	High	Mid- point	Low	
Professional Contractors	4	1981-2004	Unk	>4 (8-hr TWA); >38 (STEL/Other)		2,980	1,520	60		14,100	7,050	0 <sup>g</sup>	<u>EPA (1994a);</u> <u>EU (2007) EC</u> <u>(1999)</u>
Bathtub Refinishing	1	Unk	1	2						7,565	7,252	6,940	<u>MSU/MIFACE</u> (2011)
Automotive Refinishing	1	2003	1	2 (8-hr TWA); 3 (STEL/Other)	253	416	253	90	330	416	333	250	<u>Enander et al.</u> (2004)
Furniture Refinishing	7	1989-2007	>10	43 (8-hr TWA); >63 (STEL/Other)	499	2,245 (1,266) e	1,125	4.0		6,992	3,506	19	Estill and Spencer (1996); Grevenkamp (2007); Hall et al. (1995); McCammon et al. (1991); NIOSH (1990, 1991, 1993)
Art Restoration and Conservation <sup>f</sup>	1	2005	1	1			2.0						<u>OSHA (2012a)</u>
Aircraft Paint Stripping	5	1977-2006	>5	>35 (8-hr TWA); 130 (STEL/Other)		3,802	1,944	86		5,400	2,719	38	<u>EU (2007);</u> <u>IARC (2010);</u> <u>Vincent et al.</u> <u>(1994) EC</u> <u>(1999)</u>
Ship Paint Stripping	1	1980	1	>=1					215				<u>IARC (2010)</u>

Table G 2. DCM Cano				Used for Estimat 8 hr Air Concent		_			nic Expo	osure Cor	ncentrat	ions fo	r Non
Industry / Activity						8-hr TW	A (mg/m³)ª		-	Peak, Short Id Unknow		-	Data Source for 8-hr TWA and short- term values
	Number of Studies	Time Range of Studies	Number of Sites	Total Number of Measurements <sup>c</sup>	Mean	High	Midpoint <sup>d</sup>	Low	Mean	High	Mid- point	Low	
Graffiti Removal	1	1993	Unk	12 (8-hr TWA); >=10 (STEL/Other)	260	1,188	603	18	1,117	5,315	2,661	6.0	<u>Anundi et al.</u> (1993)
Non-Specific Workplace Settings - Immersion Stripping of Wood	>1	1980-1994	>2	>4		7,000	3,518	35					<u>EC (1999)</u>
Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	1	1980	>=1	7		1,017	825	633					IARC (2010)
Non-Specific Workplace Settings - Immersion Stripping of Metal	>=1	Unk	>=1	>=1						350			<u>EC (1999)</u>
Non-Specific Workplace Settings – Unknown	>=6	1997-2004	>=6	2 (8-hr TWA); >=227 (STEL/Other)	357	428	357	285		3,035	1,518	0.25	<u>EU (2007)</u>

						8-hr TW	/A (mg/m³)ª	STEL, Peak, Short-Tasked Based, and Unknown (mg/m <sup>3</sup> ) <sup>b</sup>				Data Source for 8-hr TWA and short- term values	
	Number of Studies	Time Range of Studies	Number of Sites	Total Number of Measurements <sup>c</sup>	Mean	High	Midpoint <sup>d</sup>	Low	Mean	High	Mid- point	Low	
Data sources are reported <sup>a</sup> These concentrations in study authors; area san <sup>b</sup> These concentrations in durations.	nclude 8-hr mples and r nclude 15-n	TWA concentr modeling result ninute STEL an	rations from ts are not ind d other shor	personal sampling tha luded. Airborne conce t, task-based concentr	entration or rations from	conversion m personal	factor for DCM is sampling that a	s 3.47 m	g/m³ per p	pm ( <u>NIOSH</u>	<u>, 2011b</u> ).		-
durations. These values <sup>c</sup> The total number of me measurements.			•		•		• •	ions in A	oppendix F	may not ide	entify ever	y instance	of number of
<ul> <li><sup>d</sup> EPA/OPPT calculated th</li> <li><sup>e</sup> The value in parenthese</li> <li><sup>f</sup> The data point provided</li> <li><sup>g</sup> The study that reported</li> </ul>	es is the 95 I for this in	<sup>th</sup> percentile of dustry was obt	f the collecte ained from (	d 8-hr TWA exposure OSHA IMIS. No other li	concentra	tions for th	is industry.	n the dat	a were ade	equate.			

#### *G-2-2-2* Workplace Exposure Scenarios Evaluated in this Assessment

**Occupational scenarios for acute and chronic exposures:** Workers performing DCM-based stripping might or might not use a respirator or be exposed to DCM at different exposure frequencies (days per year) or working years. Thus, EPA/OPPT assessed acute risks for 4 occupational scenarios and chronic risks for 16 occupational scenarios based on 8-hr TWA exposure concentrations and different variations in exposure conditions.

For the acute scenarios, EPA/OPPT defined 4 scenarios to reflect a combination of the following (Table G-3):

- No use of a respirator (APF = zero);
- Use of a respirator with an APF of 10, 25, or 50.

Acute Scenario	Respirator APF <sup>a</sup>	8-hr TWA Acute Exposure Concentration Multiplier <sup>a</sup>	Scenario Description
1	0	1	No respirator, APF = 0
2	10	0.1	Respirator APF 10
3	25	0.04	Respirator APF 25
4	50	0.02	Respirator APF 50

<sup>b</sup> As indicated in equation G-2, these multipliers are applied to the 8-hr time-weighted average (TWA) acute exposure concentrations in Table G-5.

For the chronic scenarios, EPA/OPPT defined 16 scenarios to reflect a combination of the following (Table G-4):

- No use of a respirator (APF = zero)<sup>22</sup>;
- Use of a respirator with an APF of 10, 25, or 50;
- An exposure frequency (EF) of the assumed Scenario 1 value of 250 days per year or half of the assumed Scenario 1 value (the midpoint between the assumed Scenario 1 value and zero: 125 days per year); and
- Exposed working years (WY) of the assumed Scenario 1 value of 40 years or half of the assumed Scenario 1 value (the midpoint between the assumed Scenario 1 value and zero: 20 years).

<sup>&</sup>lt;sup>22</sup> APF assumptions are the same for both acute and chronic scenarios.

Chronic Scenario	Strippers Respirator APF	Exposure Frequency (EF) (days/yr)	Working Years (WY) (years)	ADC/LADC Multiplier <sup>a</sup>	Scenario Description
1	0	250	40	1	No respirator, high ends of ranges for EF and WY
2	10	250	40	0.1	Respirator APF 10, high ends of ranges for EF and WY
3	25	250	40	0.04	Respirator APF 25, high ends of ranges for EF and WY
4	50	250	40	0.02	Respirator APF 50, high ends of ranges for EF and WY
5/9	0	250/ 125	20/ 40	0.5	No respirator, one midpoint and one high end of range fc EF and WY
6 / 10	10	250/ 125	20/ 40	0.05	Respirator APF 10, one midpoint and one high end c range for EF and WY
7 / 11	25	250/ 125	20/ 40	0.02	Respirator APF 25, one midpoint and one high end o range for EF and WY
8 / 12	50	250/ 125	20/ 40	0.01	Respirator APF 50, one midpoint and one high end o range for EF and WY
13	0	125	20	0.25	No respirator, midpoints of ranges for EF and WY
14	10	125	20	0.025	Respirator APF 10, midpoint of ranges for EF and WY
15	25	125	20	0.01	Respirator APF 25, midpoint of ranges for EF and WY
16	50	125	20	0.005	Respirator APF 50, midpoint of ranges for EF and WY

and lifetime average daily concentrations (LADCs) shown in Table G-5.

The multipliers in Tables G-3 and G-4 were used to adjust the exposure estimates of acute and chronic Scenario 1 to obtain the exposure estimates for the other exposure scenarios. Additional information is presented below in the sections discussing the approach to calculate the acute and chronic exposure estimates used in the risk characterization.

EPA/OPPT made assumptions about types of respirators used because no data were found about the overall prevalence of the use of respirators to reduce DCM exposures. While it was

not possible to estimate the numbers of workers who have reduced exposures due to the use of respirators, EPA/OPPT believes that the prevalence of respirator use would be high for most industries conducting paint stripping.

Likewise, EPA/OPPT made assumptions about the exposure frequencies and working years because data were not found to allow statistical distributions to be characterized for these parameters. Thus, EPA/OPPT evaluated occupational risks by developing hypothetical scenarios under varying exposure conditions (i.e., different respiratory protection factors, exposure frequencies and working years).

**Approach for calculating acute and chronic workplace exposures:** To facilitate the exposure calculations for the occupational scenarios, EPA/OPPT first estimated the acute and chronic exposure estimates for Scenario 1 (highest exposure group). Equations are described below.

The exposure estimates for Acute Scenarios 2 to 4 and Chronic Scenarios 2 to 16 were obtained by adjusting scenario 1 (highest exposure group) with various multipliers (Tables G-3 and G-4 for acute and chronic, respectively). The acute multipliers reflected the numerical reduction in exposure when respirators were used. The chronic multipliers reflected the numerical reduction in exposure when respirators were used and/or other EF and WY values were used. Although 16 chronic scenarios were possible, scenarios 5 through 8 and 9 through 12 resulted in the same multiplier regardless of whether the scenario used an EF of 250 days/year and a WY of 20 years or an EF of 125 days/year and a WY of 40 years.

#### Acute occupational exposure estimates

For single (acute) workplace exposure estimates, the DCM single (acute) exposure concentration was set to the 8-hour time-weighted average (TWA) air concentration in mg/m<sup>3</sup> reported for the various relevant industries. EPA/OPPT assumed that some workers could be rotating tasks and not necessarily using DCM-based paint strippers on a daily basis. This type of exposure was characterized as acute in this assessment as the worker would clear DCM and its metabolites before the next encounter with the DCM-containing paint stripper. Equation G-1 was used to estimate the single (acute) exposure estimates for acute scenario 1 (EPA, 2009).

EC scenario 
$$1 = C$$
 (Equation G-1)

where:

EC scenario 1

=

exposure concentration for a single 8-hr exposure to DCM (mg/m<sup>3</sup>) for scenario 1;

С

contaminant concentration in air for relevant industry (central tendency, low- or high-end 8-hr TWA in mg/m<sup>3</sup> from Table G-2 or G-5).

Equation G-2 was used to calculate the acute exposure estimates for scenarios 2 through 4.

# EC scenario $2 \rightarrow 4 = EC$ scenario $1 \times M$ acute (Equation G-2)

where:		
EC scenario $2 \rightarrow 4$	=	exposure concentration for a single 8-hr exposure to DCM
		(mg/m <sup>3</sup> ) for acute scenarios 2 through 4;
EC scenario 1	=	single (acute) exposure concentration for relevant industry (8-hr
		TWA in mg/m <sup>3</sup> from Table G-2 or G-5);
M acute	=	Scenario-specific acute exposure multiplier (unitless) for relevant
		industry (see Table G-3).

Acute exposure estimates for scenario 1 are presented in Table G-5. Acute exposure estimates for scenarios 2 through 4 were integrated into the risk calculations by applying the scenario-specific multipliers. Thus, separate tables listing the acute exposure estimates for scenarios 2 through 4 are not provided in this section, but are available in a supplemental Excel spreadsheet documenting the risk calculations for this assessment (*DCM Exposure and Risk Estimates\_081114.xlsx*).

#### Chronic occupational exposure estimates

The worker exposure estimates for the non-cancer and cancer risk calculations were estimated as average daily concentrations (ADCs) and lifetime average daily concentrations (LADCs), respectively. Both ADC and LADC calculations for Scenario 1 were based on the 8-hr TWA air concentration in mg/m<sup>3</sup> reported for the various relevant industries (Table G-5). EPA/OPPT assumed that the worker would be doing paint stripping activities during the entire 8-hr work shift on a daily basis. Equation G-3 was used to estimate the chronic ADCs and LADCs for Scenario 1 (EPA, 2009).

EC scenario 1 = 
$$\frac{C \times ED \times EF \times WY}{AT}$$
 (Equation G-3)

where:

EC scenario 1	=	exposure concentration (mg/m <sup>3</sup> ) for Scenario 1 = ADC for chronic non-
		cancer risks or LADC for chronic cancer risks for Scenario 1;
С	=	contaminant concentration in air for relevant industry (low- or high-end
		8-hr TWA in mg/m <sup>3</sup> from Table G-2);
ED	=	exposure duration (hrs/day) = 8 hrs/day;
EF	=	exposure frequency (days/year) = 250 days/year for high-end of range
		for both ADC and LADC calculations;
WY	=	working years per lifetime (years) = 40 years for high end of range
		for both ADC and LADC calculations; and

AT = averaging time (years × 365 days/years × 24 hrs/day) = 40 years for high end of range for ADC calculations; 70 years for LADC calculations, which is used to match the years used to calculate EPA's cancer inhalation unit risk (IUR).

Equation G-4 was used to estimate the chronic ADCs and LADCs for scenarios 2 through 16.

# $EC \text{ scenario } 2 \rightarrow 16 = EC \text{ scenario } 1 \times M \text{ chronic} \quad \text{(Equation G-4)}$

where:		
EC scenario $2 \rightarrow 16$	=	exposure concentration for chronic exposure concentration (ADC or LADC) to DCM (mg/m <sup>3</sup> ) for chronic scenarios 2 through 16
EC scenario 1	=	chronic exposure concentration (ADC or LADC) for relevant industry, chronic scenario 1 (in mg/m <sup>3</sup> from Table G-5);
M chronic	=	scenario-specific ADC/LADC chronic multiplier for relevant industry (see Table G-4)

Non-cancer and cancer exposure estimates (i.e., ADC and LADC, respectively) for scenario 1 are in presented in Table G-5. The estimates for scenarios 2 through 16 were integrated into the risk calculations by applying the scenario-specific ADC/LADC multipliers. Thus, separate tables listing the chronic exposure estimates for scenarios 2 through 16 are not provided in this section, but are available in a supplemental Excel spreadsheet documenting the risk calculations for this assessment (*DCM Exposure and Risk Estimates\_081114.xlsx*).

Table G 5. DCM A Scena	Acute and Chr ario Group	onic Ex	posure Co	ncentratio	ons (AD	Cs and L	ADCs) 1	for Workeı	rs Sce	nario 1	Highe	st Exposed	
Industry / Activity	Time Range of Studies	-		JRE ESTIMAT ntration (mg Midpoint	-	USED	IN THE N	DSURE ESTIN ION-CANCER [ADC (mg/m Midpoint	R RISK	USE	D IN TH	DSURE ESTIM E CANCER RI LADC (mg/m Midpoint	SK
Professional Contractors	1981-2004		2,980	1,520	60		680	347	14		389	198	7.8
Bathtub Refinishing													
Automotive Refinishing	2003	253	416	253	90	58	95	58	21	33	54	33	12
Furniture Refinishing	1989-2007	499	2,245 (1,266) <sup>c</sup>	1,125	4.0	114	513 (289) c	257	0.9	65	293 (165) c	147	0.5
Art Restoration and Conservation	2005		2	.0				0.5				0.3	
Aircraft Paint Stripping	1977-2006		3,802	1,944	86		868	444	20	-	496	254	11
Ship Paint Stripping	1980									-			
Graffiti Removal	1993	260	1,188	603	18	59	271	138	4.1	34	155	79	2.3
Non-Specific Workplace Settings - Immersion Stripping of Wood	1980-1994	1	7,000	3,518	35		1,598	803	8.0		913	459	4.6
Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	1980	1	1,017	825	633		232	188	145	-	133	108	83
Non-Specific Workplace Settings - Immersion Stripping of Metal													

Non-Specific													
Workplace Settings	1997-2004	357	428	357	285	81	98	81	65	47	56	47	37
– Unknown													
Notes:													
Sources are reported in	Table G-2 and disc	cussed in s	ection G-3.										
<sup>a</sup> Calculated acute singl	e 8-hr concentratio	ons are on	ly estimated f	from 8-hr TW،	A exposure	es; see Equ	uation 3-1	or F-1. Airbor	ne concer	ntration co	onversion	factor for DCN	/is
3.47 mg/m <sup>3</sup> per ppm (NIOSH, 2011b).													
3.47 mg/m <sup>3</sup> per ppm	( <u>NIOSH, 2011b</u> ).				<sup>b</sup> Calculated ADCs and LADCs are only calculated from 8-hr TWA exposures; see Equation 3-3 or F-3.								
0, 1 11	(/	culated fro	om 8-hr TWA e	exposures; see	e Equation	3-3 or F-3	3.						

#### G-2-3 Worker Exposure Limits for DCM

Both regulatory and non-regulatory worker exposure limits have been established for DCM by OSHA, the National Institute for Occupational Safety and Health (NIOSH), and the American Conference of Governmental Industrial Hygienists (ACGIH). Table G-6 provides a summary of the occupational exposure values established. Appendix F presents additional background on processes, respiratory protection, facilities and worker populations.

OSHA's amended regulatory occupational exposure limits for DCM were effective April 10, 1997. The amendments included reducing the permissible exposure limit (PEL), reducing and changing the averaging time of the short-term exposure limit (STEL), adding an Action Level, and removing the ceiling limit (OSHA, 1997a). Our analysis showed that the OSHA PEL and Action Level values were exceeded for some industries using DCM-based strippers when the OSHA values were compared to the air concentrations. Workplaces may consider these levels when instituting respiratory protections. Table G-6 also includes the pre-1997 OSHA limits to provide context when analyzing exposure data measured on or before 1997.

Table G 6. Regulatory and	Recommended Exposure Limits for DCM <sup>a</sup>	
Source	Limit Type	Exposure Limit
OSHA PEL	PEL (8-hr TWA) <sup>b</sup>	25 ppm <sup>c</sup>
(1997 and forward)	STEL (15-minute TWA)	125 ppm
	Action Level (8-hr TWA)	12.5 ppm
OSHA PEL	PEL (8-hr TWA)	500 ppm
(pre-1997)	Ceiling	1,000 ppm
	STEL (5-minute average in any 2-hr period)	2,000 ppm
NIOSH exposure limits	IDLH <sup>d</sup>	2,300 ppm
	REL <sup>e</sup>	Са
ACGIH TLV <sup>f</sup>	8-hr TWA	50 ppm

Notes:

<sup>a</sup> Source: OSHA (1997a)

<sup>b</sup> PEL= Permissible exposure limit ; TWA= Time-weighted average

<sup>c</sup> Airborne concentration conversion factor for DCM is 3.47 mg/m<sup>3</sup> per ppm (NIOSH, 2011b).

<sup>d</sup> IDLH = Immediately dangerous to life and health. IDLH values are based on effects that might occur from a 30-minute exposure.

<sup>e</sup> REL = Recommended Exposure Limit. The REL notation "Ca" is for a potential occupational carcinogen. The NIOSH Pocket Guide website has detailed policy recommendations for chemicals with "Ca" notations (NIOSH, 2011a).

<sup>f</sup> TLV = Threshold limit value

# G-3 Summary of Inhalation Monitoring Data

Data summaries from the literature search are presented below by sector. Inhalation exposure monitoring data of DCM during paint stripping, specifically full-shift 8-hr TWA breathing zone or personal samples, were used for risk analyses. Data monitoring of over 5 hour duration are assumed adequate to represent full shift exposure levels. Area and short-term samples were found and presented in the discussions of literature data for perspective and completeness, but were not used in the occupational exposure concentration calculations and risk analyses. Personal breathing zone samples provide a better representation of the amount of DCM inhaled by the workers when compared to area samples.

#### G-3-1 Bathtub Refinishing Exposures and Fatalities

In 2012, the U.S. Centers for Disease Control and Prevention (CDC) reported on bathtub refinisher fatalities associated with DCM-based stripping agents. Key excerpts from the CDC Morbidity and Mortality Weekly Report (MMWR) are discussed below (<u>CDC, 2012</u>).

In addition to 3 deaths identified by the Michigan Fatality Assessment and Control Evaluation (FACE) program, OSHA identified 10 other bathtub refinisher fatalities associated with DCMbased stripping agents that had been investigated in 9 states during 2000 to 2011. Each death occurred in a residential bathroom with inadequate ventilation. Protective equipment, including a respirator, either was not used or was inadequate to protect against DCM vapor. Inhalation of DCM vapors has been recognized as potentially fatal to furniture strippers and factory workers, but has not been reported previously as a cause of death among bathtub refinishers (<u>CDC, 2012</u>).

A review of the IMIS, a database for federal and state OSHA investigations, identified 12 DCM-related deaths associated with professional bathtub refinishing operations during 2000 to 2011. One of the 3 deaths identified by the Michigan program was not in IMIS because the decedent was self-employed and was therefore outside OSHA's enforcement jurisdiction. The ages of the 13 decedents ranged from 23 to 57 years (median: 39 years) and 12 were male. Ten different products were associated with the 13 deaths. Six of the products were marketed for use in the aircraft industry, the rest for use on wood, metal, glass, and masonry. None of the product labels mentioned bathtub refinishing. The percentage of DCM in the products ranged from 60 to 100 percent (CDC, 2012).

Moreover, analysis of IMIS data regarding deaths from inhalation of DCM vapor showed an increase in cases involving bathtub refinishing since 2000. During 1976 to 1999, only two of all DCM deaths (i.e., 8 percent) investigated by OSHA were linked to bathtub refinishing. Since 2000, 13 of the DCM deaths (i.e., 75 percent) investigated by OSHA occurred during bathtub refinishing (<u>CDC, 2012</u>).

The Michigan FACE program estimated DCM exposure concentrations as part of an investigation of a bathtub refinishing fatality reported in May 2010 (MSU/MIFACE, 2011). The concentration of DCM vapor was estimated at 92,949 ppm to 154,916 ppm (322,533 to 537,559 mg/m<sup>3</sup>) in the bathtub and 5,099 ppm to 8,499 ppm (17,694 to 29,492 mg/m<sup>3</sup>) in the bathroom. The concentration ranges were estimated using a simple modeling technique that considered the size of the bathroom, size of the tub, and an estimate that six fluid ounces (177 mL) of DCM-based stripper were used during a typical job. Also, the product used was an aircraft paint stripper product containing 60 to 100 percent DCM. The estimated concentrations exceeded the NIOSH Immediately Dangerous to Life and Health (IDLH) level of 2,300 ppm (7,981 mg/m<sup>3</sup>) (CDC, 2012). Further details about the fatality case are available in the MMWR (CDC, 2012), the NIOSH FACE Program Michigan Case Report 10MI013 (Chester et al., 2012), and the Michigan State University (MSU)/FACE Report (MSU/MIFACE, 2011).

The MSU/Michigan FACE reported a case of high DCM exposure while stripping a bathtub (<u>MSU/MIFACE, 2011</u>). The case was noted after an inspection conducted by the Washington State's Department of Labor and Industries Division of Occupational Safety and Health (DOSH). Although the exact date of the Washington DOSH inspection was not cited, the inspection occurred between April 2003 and August 2008 (<u>Lofgren et al., 2010</u>).

During the inspection, a bathtub refinishing employee was monitored while stripping a residential bathtub with Kleen Strip Aircraft Remover. The product contained less than 85 percent DCM based on the MSDS. The product information sheets recommended the use of supplied air while using the stripper. The employee had purchased ventilation equipment, which was in use at the time of monitoring, and wore a half-face air purifying respirator. Two personal samples were taken in the breathing zone of the employee for 15 minutes during the stripping task. The DCM concentrations for the personal breathing samples were 2,180 ppm (7,565 mg/m<sup>3</sup>) and 2,000 ppm (6,940 mg/m<sup>3</sup>)<sup>23</sup>. Two area samples were also taken and the DCM concentrations were 545 ppm (1,891 mg/m<sup>3</sup>) and 314 ppm (1,090 mg/m<sup>3</sup>). All of the samples significantly exceeded the OSHA 15-minute TWA STEL of 125 ppm (434 mg/m<sup>3</sup>), and the two breathing zone samples were close to the NIOSH IDLH value of 2,300 ppm (7,981 mg/m<sup>3</sup>) (MSU/MIFACE, 2011).

#### G-3-2 Paint Stripping by Professional Contractors

DCM exposure data for paint stripping conducted by professional contractors were not identified in the literature search. However, <u>EC (1999)</u> reported some DCM exposure data for consumer use of DCM-based paint strippers. The EU report states that there is "*probably...no fundamental difference between the application of paint removers by professional painters and consumers*" and goes on to further state that, in regard to the cited consumer exposure studies,

<sup>&</sup>lt;sup>23</sup> The 15-min DCM air concentrations of 6,940 mg/m<sup>3</sup> (2,000 ppm) and 7,565 mg/m<sup>3</sup> (2,180 ppm) were selected to represent the low and high ends of the range of short-term and other non-8-hr TWA values, respectively, for the breathing zone of bathtub refinishers in Table G-2 (<u>MSU/MIFACE, 2011</u>). EPA/OPPT calculated midpoint values from the high and low values reported by the study authors.

"the test situations and data described...are assumed valid for occupational exposure during professional use as well" (EC, 1999).

There are differences between the consumer and occupational use of DCM-based paint strippers by professional contractors. For instance, professional contractors are expected to have higher frequencies and durations of exposure, and a likely higher prevalence of respirator use, as compared to consumers. It is also not clear whether overall activity patterns and practices of contractors match those of consumers or whether the overall distributions of exposures of contractors and consumers have any semblance to one another. Despite these uncertainties, EPA/OPPT considered some of the literature data for consumers in the occupational exposure assessment of paint strippers.

The EU report conducted a literature review and identified the following consumer exposures to DCM during paint stripping (<u>EC, 1999</u>):

- A 1990 EPA investigation estimated consumer exposure levels ranging from 35 mg/m<sup>3</sup> (10 ppm) to a few short-term exposures of over 14,100 mg/m<sup>3</sup> (4,063 ppm)<sup>24</sup>. The majority of the exposures were below 1,770 mg/m<sup>3</sup> (510 ppm) (<u>EC, 1999</u>).
- A separate study conducted by a solvent manufacturer measured DCM exposures during testing in a small room. One test conducted with ventilation measured a 2-hr TWA exposure of 289 mg/m<sup>3</sup> (83.3 ppm), but the ventilation rate or air change rate was not specified. The peak exposure during application was 460 mg/m<sup>3</sup> (133 ppm). The peak exposure during scrape-off ranged from 710 to 1,410 mg/m<sup>3</sup> (205 to 406 ppm), and the observed maximum during the study was 3,530 mg/m<sup>3</sup> (1,017 ppm). When no ventilation was used, the worst-case exposure exceeded 14,000 mg/m<sup>3</sup> (4,035 ppm). Based on the solvent manufacturer, 8-hr TWA exposures under supplier-recommended ventilation would be 187 to 226 mg/m<sup>3</sup> (54 to 65 ppm) (<u>EC, 1999</u>).
- A literature review conducted by the United Kingdom (UK) in 1998 identified 1-hr TWA exposures of 840 to 2,765 mg/m<sup>3</sup> (240 to 790 ppm) in an unventilated room, and 129.5 to 948 mg/m<sup>3</sup> (37 to 270 ppm) with the door open (<u>EC, 1999</u>).

<sup>&</sup>lt;sup>24</sup> The short-term exposure of over 14,100 mg/m<sup>3</sup> (4,063 ppm) was selected to represent the high end of the range of short-term and other non-8-hr TWA values for professional contractors in Table G-2 (<u>EC, 1999</u>). EPA/OPPT calculated the midpoint values from the high-end values reported by the study authors.

An older study from 1981 found 8-hr TWA exposures of 460 to 2,980 mg/m<sup>3</sup> (133 to 859 ppm)<sup>25</sup> in unventilated rooms and 60 to 400 mg/m<sup>3</sup> (17 to 115 ppm)<sup>21</sup> in ventilated rooms (<u>EC, 1999</u>).

Another EU report described a 2004 study that cited several case studies of DCM monitoring during paint stripping of buildings in the UK (<u>EU, 2007</u>).

- An average personal DCM exposure of 182 mg/m<sup>3</sup> (52 ppm), ranging from 21 to 318 mg/m<sup>3</sup> (6 to 92 ppm), was reported for "paint stripping at a block of flats" (<u>EU, 2007</u>).
- A case study of paint stripping in a building stairway reported an average personal DCM exposure of 86 mg/m<sup>3</sup> (25 ppm) (<u>EU, 2007</u>).
- Another case study observed an average personal DCM exposure of 710 mg/m<sup>3</sup> (205 ppm) while paint stripping a ceiling. The DCM air concentration was measured during brush application and stripping over approximately 40 minutes (<u>EU, 2007</u>).
- A 2003 case study of the paint stripping of an external façade observed personal monitoring DCM concentrations with a maximum of 400 mg/m<sup>3</sup> (115 ppm) and a minimum of zero mg/m<sup>3 26</sup>. The average of all of the reported means was approximately 62 mg/m<sup>3</sup> (18 ppm) (<u>EU, 2007</u>).

Midwest Research Institute (MRI) prepared a report for EPA in 1994 that documented an experimental investigation of consumer exposures to solvents used in paint stripping products with eliminated or reduced DCM content. MRI investigated five paint strippers, two of which contained DCM (along with other solvents, but the concentrations were not specified). The paint stripping was conducted in a laboratory-based, environment-controlled, room-sized test chamber. The paint strippers were used on a plywood panel coated with a primer coat and two finish coats. The air exchange rate for the experiments ranged from 0.54 to 0.76 air changes per hr (ACH), with an average of 0.58 ACH. The air exchange rate of approximately 0.5 ACH was intended to replicate the ventilation rate of an enclosed room in a typical residence as a worst-case scenario (EPA, 1994a).

During each experiment, the following samples were taken for the spray and brush applications: a personal breathing zone sample of the test subject using the paint stripper; two stationary air samples for the duration of the paint stripping task; and one stationary air sample

<sup>&</sup>lt;sup>25</sup> The DCM air concentrations of 60 mg/m<sup>3</sup> (17 ppm) and 2,980 mg/m<sup>3</sup> (859 ppm) were selected to represent the low and high ends of the range of 8-hr TWA values, respectively, for professional contractors in Table G-2 (<u>EC</u>, <u>1999</u>). EPA/OPPT calculated midpoint values from the high and low values reported by the study authors.

<sup>&</sup>lt;sup>26</sup> The short-term exposure of 0 mg/m<sup>3</sup> was selected to represent the low end of the range of short-term and other non-8-hr TWA values for professional contractors in Table G-2 (<u>EC, 1999</u>).

beginning at the start of the paint stripping and lasting for 8 hrs (EPA, 1994a). The results are summarized below.

- For the spray application of the DCM-based paint stripper, MRI reported breathing zone DCM concentrations of 3,000 and 3,400 mg/m<sup>3</sup> (865 and 980 ppm) over 1.7- and 1.5-hour sampling times, respectively. The stationary length-of-task concentrations ranged from 2,900 to 3,600 mg/m<sup>3</sup> (836 to 1,037 ppm). The stationary, 8-hr TWA concentration ranged from 1,700 to 2,000 mg/m<sup>3</sup> (490 to 576 ppm) (EPA, 1994a).
- MRI reported breathing zone concentrations of 380 and 430 mg/m<sup>3</sup> (110 and 124 ppm) over sampling times of approximately 2 hours for the brush application. The stationary length-oftask concentrations ranged from 300 to 490 mg/m<sup>3</sup> (86 to 141 ppm). The stationary, 8-hr TWA concentration ranged from 230 to 270 mg/m<sup>3</sup> (66 to 78 ppm) (EPA, 1994a).

## G-3-3 Graffiti Removal

<u>Anundi et al. (1993)</u> described a study of personal monitoring conducted on 12 workers of a Swedish graffiti removal company. The study authors observed the workers remove graffiti from underground stations and noted that some of the graffiti removal was conducted in confined spaces. None of the workers were observed to wear respirators.

The study authors measured half-day DCM concentrations for the 12 workers and then calculated an 8-hr TWA concentration for each worker. Additionally, the study authors measured 15-min samples for 10 of the 12 workers (<u>Anundi et al., 1993</u>). Table G-7 summarizes the DCM personal sample concentration results for the 12 graffiti removal workers.

The study authors noted that the highest 15-min sample concentration (5,315 mg/m<sup>3</sup>) was measured while the worker was working in an elevator (<u>Anundi et al., 1993</u>). This observation illustrates how working in a confined space, with limited ventilation, can lead to high DCM exposures

Table G 7. Summa	ry of DCM Personal Concentrations	during Graffiti Removal
	Calculated 8-hr TWA Concentrations (mg/m <sup>3</sup> ) (values in parentheses are in ppm) <sup>b</sup>	15-min Sample Concentrations (mg/m <sup>3</sup> ) (values in parentheses are in ppm) <sup>b</sup>
Arithmetic Mean	260 (75) <sup>d</sup>	1,117 (322) <sup>e</sup>
Geometric Mean	127 (37)	400 (115)
High Value	1,188 (342) <sup>d</sup>	5,315 (1,532) <sup>e</sup>
Midpoint <sup>c</sup>	603 (174) <sup>d</sup>	2,661 (767) <sup>e</sup>
Low Value	18 (5.2) <sup>d</sup>	6 (1.7) <sup>e</sup>
Geometric Standard Deviation	3.6 (1.0)	5.6 (1.6)
Number of Workers	12	10

#### Notes:

<sup>a</sup> Source: <u>Anundi et al. (1993)</u>

<sup>b</sup> EPA/OPPT converted concentrations reported by the study authors in units of mg/m<sup>3</sup> to units of ppm.

<sup>c</sup> EPA/OPPT calculated midpoint values from the high and low values reported by the study authors.

<sup>d</sup> The DCM air concentrations of 18 mg/m<sup>3</sup>, 260 mg/m<sup>3</sup>, 603 mg/m<sup>3</sup>, and 1,188 mg/m<sup>3</sup> were selected to represent the low end of range, mean, midpoint, and high end of range for 8-hr TWA values, respectively, for graffiti removal in Table G-2.

<sup>e</sup> The DCM air concentrations of 6 mg/m<sup>3</sup>, 1,117 mg/m<sup>3</sup>, 2,661 mg/m<sup>3</sup>, and 5,315 mg/m<sup>3</sup> were selected to represent the low end of range, mean, midpoint, and high end of range for short-term values, respectively, for graffiti removal in Table G-2.

#### *G*-3-4 *Paint Stripping at Automotive Body Repair and Maintenance Shops*

<u>Enander et al. (2004)</u> described a study in Rhode Island that conducted personal air sampling of workers in two complete vehicle repainting facilities <sup>27</sup> and one vocational technical school. The DCM monitoring was conducted on a single worker in a vehicle repainting shop for one day in the spring and one day in the fall. This worker engaged in paint stripping one to two times per week and 3 to 4 hrs per day. The spring 8-hr TWA exposure was 26 ppm (90 mg/m<sup>3</sup>) <sup>28</sup> and the fall 8-hr TWA exposure was 120 ppm (416 mg/m<sup>3</sup>). These exposures exceeded OSHA's 8-hr TWA action level (12.5 ppm or 43 mg/m<sup>3</sup>) and PEL (25 ppm or 87 mg/m<sup>3</sup>), respectively.

Additionally, three task-based samples were taken in the spring (with sampling times ranging from 9 to 18 minutes). These exposures were 72 ppm (250 mg/m<sup>3</sup>)<sup>29</sup>, 93 ppm (323 mg/m<sup>3</sup>), and

<sup>&</sup>lt;sup>27</sup> Repainting facilities are shops that specialize in repainting the entire surface of cars and small trucks.

<sup>&</sup>lt;sup>28</sup> The DCM air concentrations of 90 mg/m<sup>3</sup> and 416 mg/m<sup>3</sup> were selected to represent the low and high ends of range for 8-hr TWA values, respectively, for automotive refinishing in Table G-2 (Enander et al., 2004). EPA/OPPT calculated the mean and the midpoint values from the high and low values reported by the study authors. The mean and the midpoint values are the same because there are only two samples for this data set.

<sup>&</sup>lt;sup>29</sup> The DCM air concentrations of 250 mg/m<sup>3</sup> and 416 mg/m<sup>3</sup> were selected to represent the low and high ends of the range for short-term and other non-8 hr TWA values, respectively, for automotive refinishing in Table G-2. EPA/OPPT calculated the mean value for the three task-based samples, as well as the midpoint value from the high and low values reported by the study authors.

120 ppm (416 mg/m<sup>3</sup>), which had a mean value of 95 ppm (330 mg/m<sup>3</sup>) and were all below the OSHA STEL (125 ppm or 434 mg/m<sup>3</sup>) (Enander et al., 2004).

## G-3-5 Wood Furniture Stripping

NIOSH surveyed a furniture refinishing shop located in Littleton, Colorado after receiving an invitation from the facility owner (NIOSH, 1993). The facility employed 5 refinishers, although the total number of workers was not specified. The facility stripped furniture in a separate room using a pump and brush technique to apply paint stripper and remove the paint. The stripper was pumped from a 55-gallon drum to the brush, collected from the table, and recycled. The stripping time of each worker averaged at a few hours per week, but reached as high as 3 to 4 hrs in a day. While stripping, the workers wore rubber aprons, full-length rubber gauntlets, a face shield, and plastic upper arm covers, and they also may have worn the safety goggles they wore in the wood shop area (NIOSH, 1993).

NIOSH surveyed the facility three times: an initial survey on October 10, 1992; a second survey on November 20, 1992 after an exhaust ventilation system was installed on the furniture stripping booth; and a third survey on February 10, 1993 after an exhaust ventilation system was installed on the wash booth (<u>NIOSH, 1993</u>). Tables G-8, G-9, and G-10 present the measurements that NIOSH made during these three surveys.

The results indicate that the addition of engineering controls reduced exposure concentrations in both the personal and area samples. For instance, the initial survey indicated that personal exposure concentrations associated with stripping activities ranged from 83 ppm (288 mg/m<sup>3</sup>) to 523 ppm (1815 mg/m<sup>3</sup>) over a range of sampling times with an average of 347 ppm (1,204 mg/m<sup>3</sup>) (Table G-8). After the addition of the stripping booth exhaust ventilation system, the second survey indicated that personal exposure concentrations associated with stripping activities dropped to a range of 10 ppm (35 mg/m<sup>3</sup>) to 110 ppm (382 mg/m<sup>3</sup>) over a range of sampling times with an average of 72 ppm (249 mg/m<sup>3</sup>) (Table G-9). This range and average concentration are generally consistent with the personal exposure concentrations associated with stripping activities observed on the third survey (after controls were added to the wash booth) (Table G-10) (NIOSH, 1993).

Personal samples were not taken during washing activities, and area samples were not taken at consistent, wash-booth area locations during all surveys. However, the area samples altogether do indicate a reduction in concentrations after installation of the ventilation systems. The 8-hr TWA exposures, which were 8-hr averages of the individual samples and not the samples themselves, were below the pre-1997 OSHA PEL of 500 ppm (1,735 mg/m<sup>3</sup>) but above the ACGIH Threshold Limit Value (TLV) of 50 ppm (174 mg/m<sup>3</sup>). NIOSH noted that after the ventilation systems were installed, the 8-hr TWA exposures were all below 50 ppm (174 mg/m<sup>3</sup>). (NIOSH, 1993).

<u>Grevenkamp (2007)</u> described follow-up activities by OSHA in the inspection of a small business that repaired and restored custom-made furniture. The facility employed 18 workers, but only a

single worker worked 3 to 4 days per week using DCM to remove paint and varnish from furniture in one section of the facility. The worker used a compressed-air system to spray DCM onto the furniture on a shallow tray. Excess stripper drained through the tray and was recirculated into a 208-L drum for reuse.

OSHA conducted personal monitoring of the worker while he was wearing a full-face elastomeric respirator with organic vapor cartridges and impervious gloves and an apron. OSHA measured an 8-hr TWA exposure of 108 ppm (375 mg/m<sup>3</sup>), which was above the OSHA PEL of 25 ppm (87 mg/m<sup>3</sup>). OSHA also measured 7 STEL sampling results, which ranged from 153 ppm (531 mg/m<sup>3</sup>) to 662 ppm (2,297 mg/m<sup>3</sup>), with an average of 404 ppm (1,402 mg/m<sup>3</sup>). These measurements were all above the OSHA STEL of 125 ppm (434 mg/m<sup>3</sup>). The facility was cited for the overexposure and required to implement controls, including work practice and engineering controls and the use of a NIOSH-approved supplied-air respirator (<u>Grevenkamp, 2007</u>).

		rvey (Befor	e Controls Added	-	
Activity	Personal Samples Concentration (mg/m <sup>3</sup> ) (values in parentheses are in ppm) <sup>b</sup>	Sampling Time (Minutes)	Location	Area Samples Concentration (mg/m <sup>3</sup> ) (values in parentheses are in ppm) <sup>b</sup>	Sampling Time (Minutes
Stripping large chest	1,437 (414)	78	Door at room entrance	576 (166)	102
Stripping headboard	1,815 (523)	101	Door at room entrance	579 (167)	78
Stripping wicker chairs	1,381 (398)	23	Edge of stripping booth above recycle can	854 (246)	59
Stripping large dresser and drawers	1,544 (445)	58	Above water reservoir between wash booth and outside wall	180 (52)	62
Stripping large dresser and drawers	1,364 (393)	30	Workbench south of wash booth	128 (37)	182
Hand strip table with Palco gel stripper	288 (83)	14	Above drum of bulk stripper	416 (120)	60
Hand strip table with gel	604 (174)	16	Above drum of bulk stripper	298 (86)	102

Table G 8. Summary of Personal and Area Concentrations in a Furniture Refinishing Shop

<sup>b</sup> EPA/OPPT converted concentrations reported by the study authors in units of ppm to units of mg/m<sup>3</sup>.

	Second	Survey (Afte	r Controls Added to Stripping	Booth) <sup>a</sup>	
Р	ersonal Samples	5	Area S	amples	
Activity	Concentration (mg/m <sup>3</sup> ) (values in parentheses are in ppm) <sup>b</sup>	Sampling Time (Minutes)	Location	Concentration (mg/m <sup>3</sup> ) (values in parentheses are in ppm) <sup>b</sup>	Sampling Time (Minutes)
Stripping six chairs	35 (10)	35	Center of room (before ventilation system turned on)	111 (32)	18
Stripping crib	330 (95)	33	Above newly stripped chairs	295 (85)	82
Strip large desk	382 (110)	103	South central part of room near newly stripped crib	87 (25)	42
			Near newly stripped desk drawers	111 (32)	59
			Edge of wash booth (near worker breathing zone)	416 (120)	33
			Edge of wash booth (near worker breathing zone)	201 (58)	34
			Edge of wash booth (near worker breathing zone)	205 (59)	101
			On work bench behind wash booth	16 (4.6)	102
			Above DCM waste buckets	52 (15)	215
			Above wash sludge tank	22 (6.3)	57

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<sup>a</sup> Source: <u>NIOSH (1993)</u>
 <sup>b</sup> EPA/OPPT converted concentrations reported by the study authors in units of ppm to units of mg/m<sup>3</sup>.

		trols Added t	o Stripping Booth and		
Per	sonal Samples Concentration (mg/m <sup>3</sup> ) (values in parentheses are in ppm) <sup>b</sup>	Sampling Time (Minutes)	Location	ea Samples Concentration (mg/m <sup>3</sup> ) (values in parentheses are in ppm) <sup>b</sup>	Sampling Time (Minutes)
Stripping rocking chair	66 (19)	24	Center of room	7 (2.0)	30
Stripping seven chairs	312 (90)	52	Near entrance to room	19 (5.5)	102
Stripping chairs	125 (36)	97	Above door to entrance of room	14 (4.0)	84
Stripping large dresser	243 (70)	113	Above door to entrance of room	29 (8.4)	178
Stripping dresser drawers	382 (110)	61	Above stripping drum	<14 (<4.0)	25
	-		Above stripping drum	9 (2.6)	75
			Above stripping drum	17 (4.9)	87
			Above stripping drum	ND <sup>c</sup>	182
			Above wash sludge tank	17 (4.9)	92
			Near drying chairs	38 (11)	113
			Above dresser while drying	62 (18)	107

ND – not detected at a limit of 0.01 mg per sample

OSHA followed-up with the facility 10 months later and found the worker using a supplied-air respirator, but the facility had only added a wall-mounted fan to blow vapors away from the work area towards the ventilation hood at the rear of the tray. OSHA measured an 8-hr TWA personal exposure of 61 ppm (212 mg/m<sup>3</sup>), which was still above the OSHA PEL. OSHA also measured two STEL exposures of 330 ppm (1,145 mg/m<sup>3</sup>) and 380 ppm (1,319 mg/m<sup>3</sup>), which were still above the OSHA STEL (Grevenkamp, 2007).

The facility hired a consultant who would recommend the installation of engineering controls and the implementation of exposure reduction work practices. The consultant's preliminary investigation of the facility observed an 8-hr TWA exposure of 208 ppm (722 mg/m<sup>3</sup>) and a STEL exposure of 1,072 ppm (3,720 mg/m<sup>3</sup>). Subsequently, the facility installed additional fans, and the consultant made a series of 6 visits to the facility to conduct personal monitoring during the paint stripping and further advice on the exposures. Over these six visits, the consultant measured five 8-hr TWA exposures ranging from 44 ppm (153 mg/m<sup>3</sup>) to 647 ppm (2,245 mg/m<sup>3</sup>)<sup>30</sup> with an average of 278 ppm (965 mg/m<sup>3</sup>). The consultant also measured 12 STEL or task-based samples (approximately 3 hrs), ranging from 298 ppm (1,034 mg/m<sup>3</sup>) to 2,015 ppm (6,992 mg/m<sup>3</sup>)<sup>31</sup> with an average of 926 ppm (3,213 mg/m<sup>3</sup>) (Grevenkamp, 2007).

OSHA visited the facility again and measured an 8-hr TWA exposure of 192 ppm (666 mg/m<sup>3</sup>) and three STEL exposures ranging from 300 ppm (1,041 mg/m<sup>3</sup>) to 811 ppm (2,814 mg/m<sup>3</sup>) with an average of 481 ppm (1,669 mg/m<sup>3</sup>). OSHA then recommended improved engineering controls consisting of a combination of a slotted back draft hood coupled with a downdraft ventilation system. After the facility implemented these controls, OSHA measured an 8-hr TWA exposure of 1.16 ppm (4 mg/m<sup>3</sup>)<sup>32</sup> and a STEL exposure of 5.5 ppm (19 mg/m<sup>3</sup>)<sup>33</sup>, both of which were well below their respective OSHA limits (Grevenkamp, 2007). This case study illustrates that the implementation of engineering controls to reduce DCM exposures during furniture paint stripping may not be a trivial exercise and careful engineering may be required to achieve reduced exposures.

<u>Hall et al. (1995)</u> described a NIOSH visit to a furniture stripping and refinishing facility. The purpose of the visit was to evaluate the facility's current ventilation system and recommend a new system, if needed. The facility employed a total of 6 full-time employees, including two co-owners. Two employees regularly stripped furniture on a daily basis while the other employees performed other refinishing operations. The facility used a dip tank for stripping, followed by a rinse, drying, and then applying the new finish. The stripping solutions were prepared by the facility and contained 60 to 80 percent DCM.

<sup>&</sup>lt;sup>30</sup> The DCM air concentrations of 2,245 mg/m<sup>3</sup> was selected to represent the high end of the range of 8-hr TWA values for furniture refinishing in Table G-2 (Grevenkamp, 2007).

<sup>&</sup>lt;sup>31</sup> The DCM air concentrations of 6,992 mg/m<sup>3</sup> was selected to represent the high end of the range of short-term and other non-8-hr TWA values for furniture refinishing in Table G-2 (Grevenkamp, 2007).

<sup>&</sup>lt;sup>32</sup> The DCM air concentrations of 4 mg/m<sup>3</sup> was selected to represent the low end of the range of 8-hr TWA values for furniture refinishing in Table G-2 (Grevenkamp, 2007).

<sup>&</sup>lt;sup>33</sup> The DCM air concentrations of 19 mg/m<sup>3</sup> was selected to represent the low end of the range of short-term and other non-8-hr TWA values for furniture refinishing in Table G-2 (Grevenkamp, 2007).

NIOSH reported that DCM levels were 2,160 ppm (7,495 mg/m<sup>3</sup>) without the ventilation system, which prompted the facility to activate the existing ventilation system. The existing ventilation system reduced DCM levels to 230 ppm (798 mg/m<sup>3</sup>). After completing the initial air sampling, NIOSH evaluated the existing ventilation system and recommended a new design. The facility installed the NIOSH-recommended design and NIOSH evaluated this new slotted hood ventilation system. During this evaluation, NIOSH measured the exposures of the 2 workers performing the stripping operations over a period of 3 days (<u>Hall et al., 1995</u>).

Table G-11 summarizes NIOSH's personal monitoring results of the two workers. NIOSH noted that, after the implementation of the recommended ventilation system, the exposures during rinsing were greater than exposures during stripping since the rinsing area was still not being locally ventilated. The NIOSH researchers felt that exposures could be reduced to meet the (at the time proposed) OSHA PEL of 25 ppm (87 mg/m<sup>3</sup>) if the rinse area controls were improved (Hall et al., 1995).

<u>McCammon et al. (1991)</u> described a NIOSH industrial hygiene survey of 14 furniture stripping workers exposed to DCM across 5 furniture stripping shops. The number of workers monitored per shop ranged from 1 to 4. These monitored workers performed tasks including stripping, washing, and refinishing.

Personal air sampling of these facilities reported TWA exposures to DCM ranging from 15 ppm (52 mg/m<sup>3</sup>) to 366 ppm (1270 mg/m<sup>3</sup>) over 5 to 8 hrs with an overall average of 133 ppm (462 mg/m<sup>3</sup>). A shop where a single worker was monitored (who performed both stripping and washing) had a 4-hr TWA exposure of 57 ppm (198 mg/m<sup>3</sup>). The highest average exposures to DCM by job category were: 191 ppm (663 mg/m<sup>3</sup>) for strippers; 145 ppm (503 mg/m<sup>3</sup>) for washers; and 31 ppm (108 mg/m<sup>3</sup>) for refinishers. These TWA exposures were below the pre-1997 OSHA PEL of 500 ppm (1,735 mg/m<sup>3</sup>). However, NIOSH noted that the monitoring was conducted in the summer and the shop doors were open to allow increased ventilation. The NIOSH researchers postulated that the exposures may be among the lowest for the work year since the doors were open and if all other relevant parameters were constant throughout the year (McCammon et al., 1991).

Table G	Dip Ta	nary of Worker Exposur ank after Implementatic lation System <sup>a</sup>			niture Paint Stripping using a ended Slotted Hood
	Number of	Sample Concentration Ranges (mg/m <sup>3</sup> )	Total Time	Total Time	Personal TWA Concentration (mg/m <sup>3</sup> ) <sup>c</sup>
	Samples	(values in parentheses are in ppm) <sup>b</sup>	Sampled (min)	Sampled (hr)	(values in parentheses are in ppm) <sup>b</sup>
WORKE	٩A		-		
Day 1	5	35-274 (10-79)	407	6.78	125 (36)
		21-115			49
Day 2	4	(6-33)	224	3.73	(14)
		90-239			160
Day 3	3	(26-69)	227	3.78	(46)
WORKE	R B				
		45-278			243
Day 1	5	(13-80)	461	7.68	(70)
		45-323			160
Day 2	6	(13-93)	549	9.15	(46)
		28-239			167
Day 3	4	(8-69)	287	4.78	(48)
Notes: <sup>a</sup> Source	: Hall et al. (:	<u>1995)</u>			

<sup>b</sup> EPA/OPPT converted concentrations reported by the study authors in units of ppm to units of mg/m<sup>3</sup>.

<sup>c</sup> The personal samples over 5 hours (300 minutes) were assumed to be representative of full-shift 8-hr TWA exposure concentrations.

In 1990, NIOSH conducted surveys in 2 furniture stripping facilities: one in Pennsylvania and the other one in Ohio. These surveys are described below.

 Furniture stripping workshop in Meadow Lands, Pennsylvania: The workshop used a flowover tank with a solution recycling system to strip furniture. The furniture was placed in tank, covered with stripping solution, and then scrubbed by a worker. During scrubbing, the worker alternated between brushing the furniture and covering it with more stripping solution. After stripping, the furniture was rinsed and brushed, dried, sanded, and refinished. The facility used a stripping solution that contained 60 volume percent DCM (Estill and Spencer, 1996; NIOSH, 1991).

The workshop installed a ventilation system in response to the results of an OSHA inspection. The NIOSH survey was conducted after installation of the ventilation system to determine its adequacy. Measurements conducted by NIOSH found personal TWA exposure levels ranging from 613 ppm (2,127 mg/m<sup>3</sup>) to 1,152 ppm (3997 mg/m<sup>3</sup>) during stripping (averaged over stripping times of 177 to 260 minutes). NIOSH found the ventilation system to be inadequate as exposure levels exceeded the pre-1997 OSHA PEL of 500 ppm

(1,735 mg/m<sup>3</sup>). After completing the air sampling, NIOSH made recommendations for improving the ventilation system (<u>Estill and Spencer, 1996</u>; <u>NIOSH, 1991</u>).

After modifications to the ventilation system were made, NIOSH measured personal exposures during stripping on 3 different days while varying the ventilation system configuration between slot hood, downdraft, and combination modes on each day. The three daily average DCM exposures were 25 ppm (87 mg/m<sup>3</sup>), 41 ppm (142 mg/m<sup>3</sup>), and 22 ppm (76 mg/m<sup>3</sup>), which were sampled during stripping operations over 4.6, 5.5, and 4.7 hrs, respectively. The ranges of breathing zone concentrations for the 3 days were: 13 to 64 ppm (45 to 222 mg/m<sup>3</sup>); 17 to 106 ppm (59 to 368 mg/m<sup>3</sup>); and 6 to 32 ppm (21 to 111 mg/m<sup>3</sup>), respectively. The corresponding calculated 8-hr TWA exposures were 15, 29, and 13 ppm (52, 101, and 45 mg/m<sup>3</sup>), respectively. The 9 individual measurements taken over the 3 days and over the 3 different ventilation system configurations ranged from 7 to 59 ppm (24 to 205 mg/m<sup>3</sup>) averaged over time periods ranging from 49 to 147 minutes (Estill and Spencer, 1996; NIOSH, 1991).

- Furniture stripping facility in Cincinnati, Ohio: This facility was operated solely by the owner. The facility conducted dip-tank paint stripping using a DCM-based paint stripper (72 weight percent DCM) and hand stripping using a paint stripper that did not contain DCM. NIOSH measured 1-hr TWA concentrations of breathing zone and area samples. NIOSH observed breathing zone concentrations of 100 ppm (347 mg/m<sup>3</sup>) and 77 ppm (267 mg/m<sup>3</sup>) of the facility owner and the NIOSH employee, respectively. NIOSH observed three area concentrations of 20 ppm (69 mg/m<sup>3</sup>), 63 ppm (219 mg/m<sup>3</sup>), and 90 ppm (312 mg/m<sup>3</sup>). The highest area concentration was observed near the dip tank, the middle concentration was observed near the term the dip tank, the middle concentration was observed near the stripping area. These measurements were below the pre-1997 OSHA PEL of 500 ppm (1,735 mg/m<sup>3</sup>) (NIOSH, 1990).
- NIOSH noted the local exhaust near the dip tank had very low intake velocity and the air movement in the stripping area was generally inadequate. NIOSH also noted that the facility owner wore neoprene gloves and boots while stripping, rinsing, and handling the solutionsoaked furniture, but no other personal protective equipment was worn (<u>NIOSH, 1990</u>).

The <u>EC (1999)</u> report described a 1990 EPA source that cited exposure levels in furniture paint stripping ranging from 258 to 3,812 mg/m<sup>3</sup> (74 to 1,099 ppm) in the absence of adequate control measures.

EPA/OPPT was able to calculate several key statistical values from the forty-three 8-hr TWA samples reported in the literature for this industry. This data set was found to have a mean value of 499 mg/m<sup>3</sup> (144 ppm) and a 95<sup>th</sup>-percentile value of 1,266 mg/m<sup>3</sup> (365 ppm) (Table G-2).

EPA/OPPT did not identify exposure data associated with art restoration and conservation. The exception was a single exposure data point reported in the OSHA IMIS data set (Table G-12). The data point was an 8-hr TWA exposure of 2 mg/m<sup>3</sup> (0.58 ppm) and corresponded to the exposure of a manager (<u>OSHA, 2012a</u>). This is the only value reported for art restoration and conservation in Table G-4. The relevance of this exposure to DCM-based paint stripping is uncertain but is assumed to have been caused by a DCM-based stripper.

#### G-3-7 Aircraft Paint Stripping

<u>NIOSH (1977)</u> identified DCM exposure data corresponding to the breathing zone of workers engaged in aircraft paint stripping in the U.S. in 1977. The breathing zone samples are summarized below.

- *Paint stripping of a wide body aircraft*: A set of 23 breathing zone samples were collected over a range of sampling times of 11 to 52 minutes. Personal air samples ranged from 79 to 950 mg/m<sup>3</sup> (23 to 274 ppm) with a mean of 379 mg/m<sup>3</sup> (109 ppm).
- Paint stripping of a narrow body aircraft: A set of 20 breathing zone samples were collected over sampling times generally less than 33 minutes with one sampling time of 267 minutes. Personal air samples ranged from 38<sup>34</sup> to 2,820 mg/m<sup>3</sup> (11 to 813 ppm) with a mean of 795 mg/m<sup>3</sup> (229 ppm).

A UK study observed aircraft paint stripping using a spray process and found DCM 8-hr TWA exposures of 29 to 95 ppm (101 to 330 mg/m<sup>3</sup>), with a mean of 62 ppm (215 mg/m<sup>3</sup>). Peak levels were as high as 1,600 ppm (5,552 mg/m<sup>3</sup>)(<u>EC, 1999</u>; <u>EU, 2007</u>).

<u>Vincent et al. (1994)</u> observed the paint stripping of a Boeing 747 in an aeronautical workshop. Personal monitoring of 30 painters, working in teams of 6 to 10 in three, 8-hr shifts, was conducted over 2 work days. During paint stripping operations, DCM concentrations ranged from 299.2 mg/m<sup>3</sup> (86 ppm) to 1,888.9 mg/m<sup>3</sup> (544 ppm) over 38 data points with a mean of 783.4 mg/m<sup>3</sup> (226 ppm) for directly exposed workers performing the stripping operations. These measurements were taken over sampling times ranging from 120 to 330 minutes. The calculated 8-hr TWA exposures to DCM ranged from 86 mg/m<sup>3</sup> (25 ppm)<sup>35</sup> to 1,239.5 mg/m<sup>3</sup> (357 ppm) with a mean of 382 mg/m<sup>3</sup> (110 ppm).

Additional data points (n=7) were collected for indirectly exposed workers applying masking film to non-stripped surfaces. These measurements ranged from 317.2 to 762.5 mg/m<sup>3</sup> (91 to 220 ppm) with a mean concentration of 464 mg/m<sup>3</sup> (134 ppm). The calculated 8-hr TWA

<sup>&</sup>lt;sup>34</sup> The DCM air concentration of 38 mg/m<sup>3</sup> was selected to represent the low end of the range of short-term and other non-8-hr TWA values for aircraft paint stripping in Table G-2 (NIOSH, 1977).

<sup>&</sup>lt;sup>35</sup> The DCM air concentration of 86 mg/m<sup>3</sup> was selected to represent the low end of the range of 8-hr TWA values for aircraft paint stripping in Table G-2 (Vincent et al., 1994).

exposures to DCM ranged from 97.2 to 174.6 mg/m<sup>3</sup> (28 to 50 ppm) with a mean of 128.2 mg/m<sup>3</sup> (37 ppm)(<u>EU, 2007</u>; <u>IARC, 1999</u>; <u>Vincent et al., 1994</u>).

Norwegian studies from 2001 and 2002 found 8-hr TWA exposures to DCM associated with paint removal of aircraft of 1,444, 2,319, and 3,802 mg/m<sup>3</sup> (416, 668, and 1,096 ppm, respectively)<sup>36</sup> from personal samples (<u>EU, 2007</u>).

The <u>EC (1999)</u> report cited a 1998 UK study that reported 8-hour TWA exposures to DCM during aircraft paint stripping. The 8-hr TWA air concentrations ranged from 101 to 330 mg/m<sup>3</sup> (29 to 95 ppm) with a mean of 210 mg/m<sup>3</sup> (61 ppm). The same study also stated that peak exposures as high as 5,400 mg/m<sup>3</sup> (1,556 ppm)<sup>37</sup> were possible (<u>EC, 1999</u>).

A 2006 study cited DCM exposures during aircraft paint stripping in Taiwan. Personal samples were collected during activities at 4 different locations of the aircraft: the ground, the nose, the right wing, and the left wing. The number of personal samples taken at each location ranged from 8 to 13. The resulting 2-hr average concentrations were 146, 75, 81, and 71 mg/m<sup>3</sup> (42, 22, 23, and 20 ppm) for the ground, nose, right wing, and left wing, respectively. The standard deviations of each average ranged from 40 to 111 mg/m<sup>3</sup> (12 to 32 ppm), indicating a significant degree of scatter for each data set (<u>IARC, 2010</u>).

## G-3-8 Ship Paint Stripping

EPA/OPPT identified limited data for paint stripping of ships. <u>IARC (2010)</u> described a 1980 UK monitoring study of 8 painters over two days in a dockyard. The study results included a mean concentration of DCM of 214.7 mg/m<sup>3</sup> (62 ppm). The exposure was associated with one painter conducting paint stripping.

## G-3-9 Paint Stripping in Non-specific Workplace Settings

EPA/OPPT identified EU exposure data that were characterized for "general industrial use." However, more specific information on the industries (e.g., applicable NAICS or Standard Industrial Classification [SIC] codes, primary industrial functions or products, or number of sites or workers) was not provided in the identified references.

<sup>&</sup>lt;sup>36</sup> The DCM air concentration of 3,802 mg/m<sup>3</sup> was selected to represent the high end of the range of 8-hr TWA values for aircraft paint stripping in Table G-2 (EU, 2007).

<sup>&</sup>lt;sup>37</sup> The DCM air concentration of 5,400 mg/m<sup>3</sup> was selected to represent the high end of the range of short-term and other non-8-hr TWA values for aircraft paint stripping in Table G-2 (EC, 1999).

The <u>EC (1999)</u> report described a 1998 UK report that identified exposures during immersion stripping of wood. The 8-hr TWA exposures ranged from 38.5 to 7,000 mg/m<sup>3</sup> (11 to 2,017 ppm)<sup>38</sup> with a mean of 700 mg/m<sup>3</sup> (202 ppm) for the period of 1980 to 1994. The 8-hr TWA exposures were lower during the period of 1990 and 1994, with a range of 35 to 2,100 mg/m<sup>3</sup> (10 to 605 ppm)<sup>39</sup> and a mean of 350 to 420 mg/m<sup>3</sup> (101 to 121 ppm).

Moreover, the same 1998 UK report described in the <u>EC (1999)</u> document reported DCM exposure data during the immersion stripping of metal. The exposures were characterized as less than 350 mg/m<sup>3</sup> (101 ppm)<sup>40</sup> "if appropriate protection measures [were] implemented".

A 1980 U.S. study observed the breathing zone of 3 workers engaged in paint stripping from wood and metal. Seven samples were collected and the 8-hr TWA DCM concentrations ranged from 633 to 1,017 mg/m<sup>3</sup> (182 to 293 ppm)<sup>41</sup> (<u>IARC, 2010</u>).

A 2004 report reported concentration measurements of DCM during an activity described only as "paint stripping from an article." The means of six different measurements were reported, although sampling times were not reported. The range of these six means was 35 to 707 mg/m<sup>3</sup> (10 to 204 ppm) with an overall average of 324 mg/m<sup>3</sup> (93 ppm). Two maximums of 459 and 1,413 mg/m<sup>3</sup> (132 and 407 ppm) were also presented (<u>EU, 2007</u>).

The <u>EU (2007)</u> report discussed a 2004 report about DCM exposure monitoring data in Germany associated with both indoor and outdoor paint stripping in 1997. The 62 indoor measurements ranged from 294 to 3,035 mg/m<sup>3</sup> (85 to 875 ppm)<sup>42</sup> over unknown sampling times. The mean was 1,373 mg/m<sup>3</sup> (396 ppm) and the 95<sup>th</sup> percentile was 2,457 mg/m<sup>3</sup> (708 ppm). The 37 outdoor measurements were only slightly lower with a range of 158 to 2,275 mg/m<sup>3</sup> (46 to 656 ppm) (the sampling times ranged from three to 295 minutes). The mean was 524 mg/m<sup>3</sup> (151 ppm) and the 95<sup>th</sup> percentile was 1,339 mg/m<sup>3</sup> (386 ppm).

The same report also cited 122 air measurements of DCM during non-specified paint stripping in France from 1998 to 2002. The concentrations, sampled over a range of 1 to 8 hrs, ranged from 0.25 to 2,723 mg/m<sup>3</sup> (0.07 to 785 ppm)<sup>43</sup>. The arithmetic mean was 163 mg/m<sup>3</sup> (47 ppm),

<sup>&</sup>lt;sup>38</sup> The DCM air concentration of 7,000 mg/m<sup>3</sup> was selected to represent the high end of the range for 8-hr TWA values for non-specific workplace settings (immersion stripping of wood) in Table G-2 (EC, 1999).

<sup>&</sup>lt;sup>39</sup> The DCM air concentration of 35 mg/m<sup>3</sup> was selected to represent the low end of the range for 8-hr TWA values for non-specific workplace settings (immersion stripping of wood) in Table G-2 (EC, 1999).

<sup>&</sup>lt;sup>40</sup> The DCM air concentration of 350 mg/m<sup>3</sup> was selected as the high end of the range of short-term and other non-8-hr TWA values for non-specific workplace settings (immersion stripping of metal) in Table G-2 (EC, 1999). No other values of the range were given.

<sup>&</sup>lt;sup>41</sup> The DCM air concentrations of 633 and 1,017 mg/m<sup>3</sup> were selected to represent the low and high ends of the range for 8-hr TWA values, respectively, for non-specific workplace settings (immersion stripping of wood and metal) in Table G-2 (IARC, 2010).

<sup>&</sup>lt;sup>42</sup> The DCM air concentration of 3,035 mg/m<sup>3</sup> was selected as the high end of the range of short-term and other non-8-hr TWA values for non-specific workplace settings (unknown) in Table G-2 (EC, 1999).

<sup>&</sup>lt;sup>43</sup> The DCM air concentration of 0.25 mg/m<sup>3</sup> was selected as the low end of the range of short-term and other non-8-hr TWA values for non-specific workplace settings (unknown) in Table G-2 (EU, 2007).

the geometric mean was 17.2 mg/m<sup>3</sup> (5 ppm), the median was 17.5 mg/m<sup>3</sup> (5 ppm), and the  $95^{th}$  percentile was 956 mg/m<sup>3</sup> (276 ppm) (<u>EU, 2007</u>).

The <u>EU (2007)</u> report also discussed a 2004 report that documented two studies in Finland related to the effectiveness of respirators during non-specified paint stripping. The first study was conducted in 1997 and did not specify the respirator type. The 8-hr TWA concentrations were 285 mg/m<sup>3</sup> (82 ppm)<sup>44</sup> and 5 mg/m<sup>3</sup> (1.4 ppm) for outside and inside of the respirator, respectively. This equates to a reduction of approximately 98.2 percent and a respirator protection factor of approximately 57. The second study was conducted in 1998 and measured 8-hr TWA concentrations of 428 mg/m<sup>3</sup> (123 ppm)<sup>39</sup> and 2.2 mg/m<sup>3</sup> (0.63 ppm) for outside and inside the respirator, respectively. This equates to a reduction of approximately 99.5 percent and a respirator protection factor of approximately 195 (<u>EU</u>, 2007).

## G-3-10 Summary of OSHA IMIS Data

OSHA IMIS data were among the data collected during the literature search for occupational exposure data. The sources of DCM exposure in IMIS are generally not provided and may or may not include DCM-containing paint stripping products. In some circumstances, EPA/OPPT examined IMIS data to provide insights in some occupational categories where no other data were found to be directly attributable to the use of DCM-containing paint stripping products. Table G-12 summarizes the personal DCM measurements obtained from OSHA IMIS for the industries of interest. Area measurements were excluded from this summary. Additionally, non-detect results were excluded from this summary. A non-detect result is not meaningful for risk analyses as it could be the result of the site not using any DCM as opposed to a lack of worker exposure to DCM during its use.

Although not used in the risk analyses (except for art restoration and conservation), these OSHA IMIS data were useful for providing perspective on the temporal variation of DCM exposures. EPA/OPPT aggregated the exposure data into two categories: before and after the 1997 promulgation of the current OSHA PEL for DCM (25 ppm or 87 mg/m<sup>3</sup>).

For the industries that have data both before and after 1997 (aircraft refinishing, ship and boat refinishing, automotive refinishing, and furniture refinishing), little variation was observed in the statistics of the exposure data. There was little variation in the TWA and STEL maximum values between the post-1997 and pre-1997 data sets. The other statistical values did not show clear trends as a result of the change in PEL. In many cases, the post-1997 exposures were greater than the pre-1997 exposures. Of note, the 90<sup>th</sup> percentile of TWA exposures in ship and boat refinishing increased by more than 100 percent from pre-1997 to post-1997.

<sup>&</sup>lt;sup>44</sup> The DCM air concentrations of 285 and 428 mg/m<sup>3</sup> were selected to represent the low and high ends of the range for 8-hr TWA values, respectively, for non-specific workplace settings (unknown) in Table G-2 (EU, 2007). EPA/OPPT calculated the mean and midpoint values from the high and low values reported by the study authors. The mean and the midpoint values are the same because there are only two samples for this data set.

There are many reasons for OSHA or a State to conduct a health inspection. Reasons can include (but are not limited to) random, programmatic selections within an industry; past health problems at the facility; employee complaints; or a safety inspection in which the inspector felt a health inspection was also warranted. The lack of randomness in the selection of facilities for health inspections reduces the utility of the IMIS data of informing representative temporal trends.

Table G 12.	Summary by Industry	of OSHA IN	AIS Perso	nal Monitor	ing Data	a for DCM fr	om 1992	to 2012	
				Pe	rsonal Me	easurements			
		Р	ost-1997 P	EL Update			Pre-1997 P	EL Update	
		TWA	۱.	STEL		TWA	4	ST	EL
		mg/m <sup>3</sup>	ppm	mg/m <sup>3</sup>	ppm	mg/m <sup>3</sup>	ppm	mg/m <sup>3</sup>	ppm
	Max			0.0	0.0				
	90th Percentile			N/A	N/A				
Bathtub	Median			N/A	N/A				
Refinishing	Min			0.0	0.0				
	Mean			N/A	N/A				
	Number of Data Points	Non	e	3		Non	e	No	ne
	Max	28.2	8.1	27.8	8.0				
Professional	90th Percentile	23.2	6.7	13.8	4.0				
	Median	13.9	4	0.0	0.0				
Contractors	Min	0.0	0	0.0	0.0				
	Mean	12.8	3.7	5.1	1.5				-
	Number of Data Points	5		7		Non	e	No	ne
	Max	34.5	9.9	31.2	9.0	33.3	9.6		
	90th Percentile	32.4	9.3	29.5	8.5	28.1	8.1		
Aircraft	Median	15.6	4.5	12.7	3.7	10.1	2.9		
Refinishing	Min	0.0	0	0.0	0.0	0.0	0.0		
	Mean	17.8	5.1	15.2	4.4	13.3	3.8		
	Number of Data Points	15		6		21		No	ne
	Max	33.7	9.7	10.4	3.0	27.8	8.0		
	90th Percentile	32.4	9.3	6.2	1.8	15.6	4.5		
Ship & Boat	Median	6.1	1.8	0.0	0.0	5.7	1.6		
Refinishing	Min	0.0	0.0	0.0	0.0	0.0	0.0		
	Mean	11.3	3.3	2.1	0.6	7.6	2.2		
	Number of Data Points	11		5		8		No	ne

		Personal Measurements							
		Post-1997 PEL Update Pre-1997 PEL Update							
		TWA		STEL		TWA		STEL	
		mg/m <sup>3</sup>	ppm	mg/m <sup>3</sup>	ppm	mg/m³	ppm	mg/m <sup>3</sup>	ppm
Automotive Refinishing	Max	31.2	9.0	34.2	9.9	24.3	7.0	27.8	8.01
	90th Percentile	28.9	8.3	27.8	8.0	20.1	5.8	N/A	N/A
	Median	4.7	1.4	10.3	3.0	12.3	3.6	N/A	N/A
	Min	0.0	0.0	0.0	0.0	1.1	0.3	6.9	2.0
	Mean	10.0	2.9	13.3	3.8	11.1	3.2	17.4	5.0
	Number of Data Points	10		11		5		2	
Furniture Refinishing	Max	33.9	9.8	34.01	9.8	31.2	9	31.2	9.0
	90th Percentile	28.9	8.3	24.64	7.1	30.2	8.7	N/A	N/A
	Median	10.6	3.1	13.01	3.8	17.2	5.0	N/A	N/A
	Min	0.0	0.0	0.0	0.0	3.5	1.0	0	0
	Mean	13.3	3.8	12.91	3.7	18.8	5.4	15.6	4.5
	Number of Data Points	78		70		12		2	
Art Restoration and Conservation	Single Data Point	2.01	0.58	None		None		None	

## Appendix H RESIDENTIAL/CONSUMER EXPOSURE ASSESSMENT

Appendix H contains detailed information about the modeling approach used to estimate consumer exposures for the use of DCM in paint strippers.

## H-1 Estimation of Emission Profiles for Paint Removers/Strippers

Various studies were considered in developing DCM emission profiles as model inputs for subsequent exposure-estimation efforts.

Four chamber studies were analyzed for DCM emission characteristics. Each of these studies was reviewed to estimate the fraction of applied DCM mass that was emitted. In selected cases, single-exponential representations of time-varying emission profiles were developed. The studies are the following:

- MRI Chamber Study Midwest Research Institute. Consumer Exposure to Paint Stripper Solvents, Final Report. Report to the USEPA, EPA Contract No. 68-DO-0137, Work Assignment No. 4-06 (EPA, 1994a);
- EC Chamber Study European Commission, ETVAREAD. Effectiveness of vapour retardants in reducing risks to human health from paint strippers containing dichloromethane (<u>EC,</u> <u>2004</u>);
- van Veen Chamber Study van Veen, M.P., Fortezza, E.S. and Mensinga, T.T. Nonprofessional paint stripping and experimental validation of indoor dichloromethane levels. Indoor Air, 12:92-97 (van Veen et al., 2002);
- Lawrence Berkley Laboratory (LBL Chamber Study) Girman, J.R. and Hodgson, A. T. Source Characterization and Personal Exposure to Methylene Chloride from Consumer Products, Lawrence Berkeley Laboratory, Report No. LBL-20227 (<u>LBL, 1986</u>).

Data from the MRI chamber study were used as the basis for developing emission profiles for the brush-on and spray-on applications evaluated in this assessment (EPA, 1994a). The advantages of the MRI study include the following: (1) the chamber data were adequate to support the estimation effort; and (2) the products studied were considered to be the most representative of paint strippers available in the U.S. consumer product market.

The <u>EC (2004)</u> chamber study was not used for the current assessment due to several limitations, including: (1) too little information to confirm model parameters or study results; (2) lack of information on study design and product formulation or percent DCM in the tested products; (3) use of European products that may not be representative of U.S. products; (4) use

of a ventilation rate considered to be high for indoor rooms, unless mechanical ventilation were used (*e.g.*, a fan venting directly to outdoors); and, (5) differences from typical U.S. room size (*i.e.*, U.S. room sizes may be larger) (EC, 2004).

The <u>van Veen et al. (2002)</u> chamber study was also reviewed but not used because it used non-U.S. products. Moreover, the study had limitations similar to the EC study, although room volumes and ventilation rates were more in line with values that might be expected for typical uses in U.S. residences.

Subsequent to the preparation of the draft risk assessment, EPA/OPPT gained access to a report on a chamber study conducted by Lawrence Berkeley Laboratory (LBL) (<u>LBL, 1986</u>). The study used a protocol similar to that used in the MRI study<sup>45</sup>. The LBL data were analyzed and are presented in section H-1-1-2, and were used for comparative purposes in section H-5.

The sections below present the analysis and fitted exponential representations of the MRI data for both brush-on and spray-on applications. There are also descriptions of other chamber studies, which were considered but not used in EPA/OPPT's modeling study. The discussion also includes a comparison of the estimates from the MRI chamber study with those from the other chamber studies.

## H-1-1 Conceptual Approach

5. For each chamber test, the applied DCM mass was calculated by multiplying the applied product mass by the assumed DCM content (% by weight). The DCM content (83 - 87%) was determined analytically by LBL (LBL, 1986). In the van Veen study, the DCM content was reported, but not its basis (van Veen et al., 2002). For the EC experiments, the DCM content was assumed to be 82.5% (midpoint of range for their "typical formulation") (EC, 2004). For the MRI experiments, DCM contents of 16.8% for a brush-on product and 85.0% for a spray-on product were assumed based on formulation data (*e.g.*, per MSDS) near the time when these experiments were conducted (EPA, 1994a).

The DCM mass emitted per experiment was estimated by two alternative methods:

- <u>Mass-balance Calculation</u> this method consisted of (a) using the starting and ending concentrations for successive brief (≤ 10-minute) time intervals, together with knowledge of the airflow rate into and out of the chamber, to determine the mass emitted during each interval; and (b) summing these estimates to determine the total DCM mass emitted during the entire experiment.
- <u>Model Fit to Chamber Data</u> this method consisted of (a) using nonlinear least squares (NLS) analysis to fit an incremental source model governed by a single exponential; and (b) calculating the mass emitted by integrating the fitted model over the duration of the experiment.

<sup>&</sup>lt;sup>45</sup> The MRI study cited the LBL protocol.

#### Mass-Balance Calculation

The mass released during chamber studies was estimated using a numerical mass-balance integration method, where the mass released over each interval was determined as follows:

$$MR_{i, i+1} = (C_{i+1} - C_i)^* V + Q^* (\Delta t)^* (C_{i+1} + C_i)/2)$$
 (Equation H-1)

Where:

 $MR_{i, i+1}$  = Mass Released over interval *i* to *i+1*, mg = the change in mass in the chamber plus the amount of mass removed through ventilation

- C = concentration in the compartment, mg/m<sup>3</sup>
- V = compartment volume, m<sup>3</sup>
- Q = compartment ventilation rate, m<sup>3</sup>/hr
- $\Delta t = time interval from$ *i to i+1*, hr

The intervals were chosen such that the concentration was relatively well behaved (*i.e.*, without significant changes in slope) during each interval. The estimated masses for the intervals covering the duration of interest were then summed to estimate the total mass released.

#### Model Fit to Chamber Data

An exponential representation of the time-varying emission rate was chosen in evaluating the experimental data because of the general shape of the concentration profile and the similarity to other emission behaviors (*e.g.*, chemicals emitted from paint). The emission equation has the following form:

$$E = E_0 e^{-kt}$$
 (Equation H-2)

Where:

E = emission rate, mg/hr E<sub>0</sub> = initial emission rate (the emission rate at t = 0), mg/hr k = first-order rate constant, hr<sup>-1</sup> t = time since application, hr

Integrating Equation H-2 to infinity gives the mass released according to the exponential, as follows:

Mass Released (mg) = 
$$\frac{E_0}{k}$$
 (Equation H-3)

Or:

$$E_0 = (Mass Released) * k$$
 (Equation G-4)

Integrations of single-compartment, mass-balance equations for single- and double-exponential representations of the emissions are given in Equations H-5 and H-6 (EPA, 1997), respectively:

Single-exponential representation

$$C(t) = \frac{E_0}{V*\left(\frac{Q}{V}-k\right)} \left( e^{-kt} - e^{-\frac{Q}{V}t} \right)$$

(Equation H-5)

Where:

C(t)= concentration, mg/m<sup>3</sup> V = chamber volume, m<sup>3</sup> Q = air flow rate in and out of the chamber, m<sup>3</sup>/hr

Double-exponential representation

$$C(t) = \frac{E_{01}}{V*(\frac{Q}{V}-k_1)} \left( e^{-k_1 t} - e^{-\frac{Q}{V}t} \right) + \frac{E_{02}}{V*(\frac{Q}{V}-k_2)} \left( e^{-k_2 t} - e^{-\frac{Q}{V}t} \right)$$
(Equation H-6)

Where:

 $E_{01}$  = initial emission rate for the first exponential, mg/hr  $E_{02}$  = initial emission rate for the second exponential, mg/hr  $k_1$  = first-order rate constant for the first exponential, hr<sup>-1</sup>  $k_2$  = first-order rate constant for the second exponential, hr<sup>-1</sup>

#### H-1-1-1 MRI Chamber Study (EPA, 1994a)

In 1993, MRI conducted a series of chamber experiments for EPA on paint stripping products (EPA, 1994a). For each experiment, continuous air concentrations were measured using a Fourier transform infrared (FTIR) spectrometer. In addition, three stationary samplers and a personal sampler were used to collect time-integrated samples on activated charcoal. The resultant data were analyzed and a process was undertaken to fit the data to equations with an exponential to represent the time-varying emission profile that led to the air concentrations.

The MRI study tested five products, two of which contained DCM, as listed in Table H-1.

Table H 1. DCM containing Products Used in the MRI Chamber Studies					
Product	Application Type	Chemical			
BIX Spray-On Stripper	Spray	DCM			
Strypeeze	Brush	DCM			
Source: EPA (1994a)					

Each product used in the study was applied in eight, approximately 1-minute segments, with each 1-minute application followed by an approximately 10-minute wait time prior to the start of the next, resulting in about 11 minutes between successive applications. In each case, the

emissions from each application were represented by a single exponential, with each exponential identical to the other seven but with a different start time set at the midpoint of the corresponding application period. Based on this approach, the start times for the eight DCM exponentials were 0.5, 11.5, 22.5, 33.5, 44.5, 55.5, 66.5, and 77.5 minutes from the start of the paint-stripping activity.

A single exponential was found to provide a good fit to the data corresponding to the two DCM products reported in the MRI study. The fitting process involved:

- 1. Extracting measured concentration values from the MRI chamber study data and co-plotting the points with fits to Equation H-5. The concentration values were extracted, for runs 4, 5, and 6 for BIX Spray-on and for runs 7, 8, and 9 for Strypeeze Brush-on, at each 0.5-hr time point as well as at times of peaks and significant changes in slope, resulting in eight or nine data points per run.
- 2. Calculating the mass of DCM applied during the test and assigning 1/8<sup>th</sup> of the applied mass to each of the eight exponentials.
- 3. Iterating to find the best fit to the concentration data by varying the "fraction released" and the first-order rate constant (*k*), using Equation H-5 and the following relationship:

$$E_0 = (Mass Applied) * (Fraction Released) * k$$
 (Equation H-7)

The analysis was conducted using Excel to solve the equations and plot the results. The best fit to each data set was determined via visual comparison of the results of applying Equation H-5 to the extracted MRI data. Combined data for Runs 4, 5 and 6 (with very similar concentration profiles; see Figure H-1) were used for the BIX spray-on product, attempting to fit the Equation H-5 curve midway between maximum and minimum values of the data at each point in time. Concentration profiles were more disparate for the Strypeeze brush-on product (Figure H-2). In this case, the visual fit was applied only to the Run 7 data, which appeared to be better behaved and also were approximately midway between the data from Runs 8 and 9.

In general, the height of the concentration curve is related primarily to the DCM mass released, and the length and shape of the decay portion of the curve is closely related to the first-order rate constant (k) and the chamber ventilation rate (Q). The resulting fit for the BIX Spray-on product is shown in Figure H-1, and the fit for the Strypeeze Brush-on product is shown in Figure H-2. In each figure, the underlying eight exponentials are shown in the lower part of the figure, with the sum shown as the fitted, dashed line. The fitted parameters for these two DCM cases are shown in Table H-2.

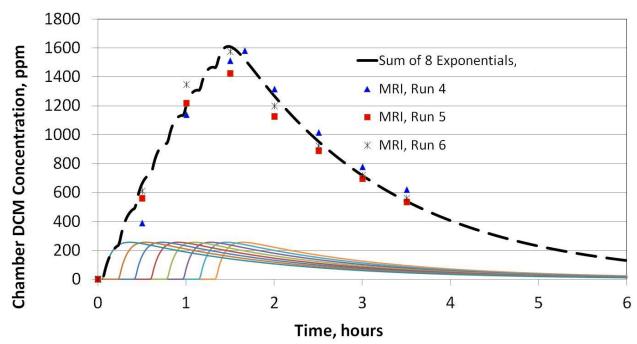
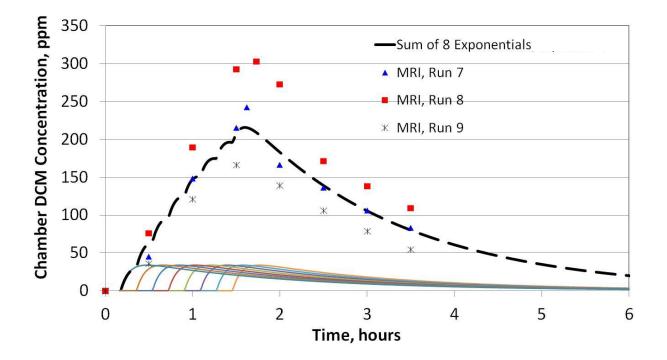


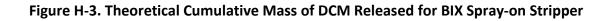
Figure H-1. Model Fit to Data Extracted from MRI Chamber Study for BIX Spray-on Product

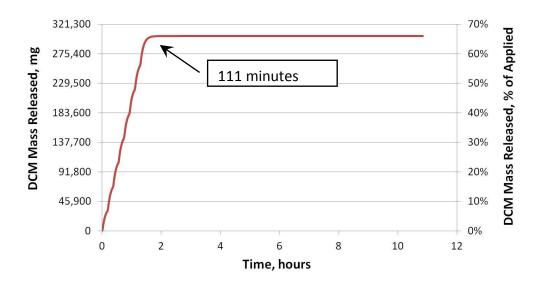
Figure H-2. Model Fit to Data Extracted from MRI Chamber Study Report for Strypeeze Brushon Product



Product	Experiment	Product Mass Applied, g	DCM Weight Fraction	DCM Mass Applied, g	DCM Theoretical Fraction Released <sup>a</sup>	First-Order Rate Constant, hr <sup>-1</sup>
BIX Spray-On Stripper	Runs 4, 5, 6	540	0.85 <sup>b</sup>	459.0	0.66	10.0
Strypeeze (Brush-On)	Run 7	724	0.168 <sup>c</sup>	121.62	0.33	3.9
<b>Notes:</b> <sup>a</sup> The theoretical I <sup>b</sup> <u>EPA (1996a)</u> <sup>c</sup> <u>EPA (2003)</u> Source: <u>EPA (1994a</u> )		eased was estima	ited by integ	rating the fitted	exponential.	

A numerical integration of the fitted "sum of 8 exponentials" shown in Figures H-1 and H-2 was performed by using the average concentration for each one-minute interval. Then a massbalance calculation was conducted for the test chamber, which accounted for the mass in the chamber and the mass that had been removed through ventilation. The estimated cumulative mass released from the product as a function of time is shown in Figures H-3 and H-4 for the BIX Spray-on and Strypeeze Brush-on strippers, respectively.





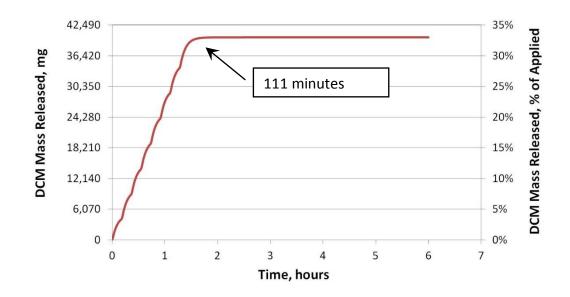


Figure H-4. Theoretical Cumulative Mass of DCM Released for Strypeeze Brush-on Stripper

#### H-1-1-2 LBL Chamber Study (LBL, 1986)

Brush-on product formulations used in the MRI and LBL studies are summarized in Table H-3. The MRI and LBL studies used very similar experimental protocols, with the only notable difference being their product formulations. The actual product formulation (from product label or MSDS) was not reported by either study, but the LBL study did include a bulk analysis of the paint-remover products. The 1988 and 2013 MSDS listed in Table H-3 for Strypeeze bracket the 1994 MRI study, with the 1988 MSDS likely more indicative of the actual composition of the paint remover used by MRI. This inference is supported by a Strypeeze formulation (*circa* 1994) in the Source Ranking Database (SRD), indicating 16.8% DCM by weight (EPA, 2003).

Ingredient	Strypeeze 1988 MSDS <sup>a</sup>	Strypeeze 2013 MSDS <sup>a</sup>	LBL (1986), Paint Remover (PR)-A	LBL (1986), PR-B	
DCM	>10%	25 - 30%	83.0%	86.6%	
Toluene	>35%	15 - 20%			
Methanol	25%	25 - 30%	Present <sup>▶</sup>	Present <sup>ь</sup>	
Acetone	<25%	15 - 20%			
Paraffin Wax	<5%	0 - 5%			
Aliphatic Hydrocarbon		0 - 5%			
Isopropanol				9.4%	
Xylenes			Present <sup>b</sup>		
Non-volatile			3.6%	2.8%	

<sup>a</sup> Proxies for the composition of the MRI product (<u>EPA, 1994a</u>).

Indicates that the ingredient was determined to be present, but the exact mass was not quantified.

The LBL and MRI studies used similar ventilation rates — ~3.0 hr<sup>-1</sup> and 0.6 hr<sup>-1</sup> for LBL Experiments 2 and 5, respectively; and ~0.55 hr<sup>-1</sup> for each of MRI Runs 7, 8 and 9. However, the formulations used in the two studies are quite different, with the most noteworthy differences being the amount of DCM applied as well as the presence of Paraffin Wax vapor retardant in the MRI paint-remover product. Table H-4 summarizes the chamber characteristics, the DCM mass applied, and the estimated percent of applied DCM mass that was released during each LBL/MRI test.

Table H 4. C	Table H 4. Comparison of DCM Mass Released for LBL and MRI Studies of Brush on Products											
Study and	Cha	mber	Duration,	Applied Product	Applied DCM	% DCM						
Expt/Run <sup>a</sup>	Vol, m <sup>3</sup>	ACH, hr <sup>-1</sup>	min	Mass, mg	Mass, mg	Released						
LBL, Expt 2	20.0	3.23	89	363,000	314,358	83%						
LBL, Expt 5	20.0	0.62	86	325,000	269,750	93%						
MRI, Run 7	35.7	0.56	105	724,000	121,632	35%						
MRI, Run 8	35.7	0.54	105	676,000	113,568	51%						
MRI, Run 9	35.7	0.55	105	765,000	128,520	30%						
Note:			•									
<sup>a</sup> Expt= Experir	nent											

The results in Tables H-3 and H-4 provide some insights regarding the factors that can influence DCM emissions:

- The two LBL studies contain high DCM weight fractions (83% and 86.6%) compared to the MRI brush-on product (estimated to be 16.8%).
- The applied product mass is lower for the LBL products as compared to the MRI product, but the applied DCM mass is higher due to the higher DCM weight fraction.
- The MRI product contains a vapor retardant (Paraffin) whereas the LBL products do not.

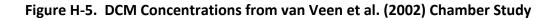
Further insights from these observations and the chamber test results are as follows:

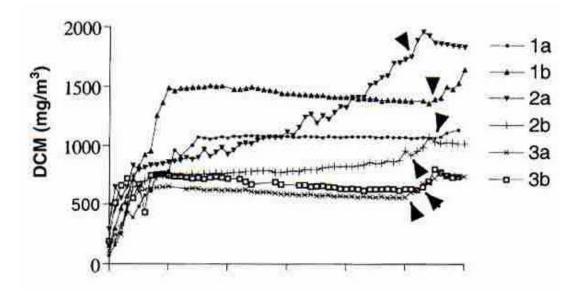
- The ventilation rate appears to have a minimal impact on the DCM emission rate and total mass emitted. LBL Experiments 2 and 5 are very similar with the exception of the ventilation rate. A greater fraction of the mass might be expected to be released in an environment with a higher ventilation rate, but that does not occur in experiment 2.
- The vapor retardant appears to cause more than a 50% reduction in DCM emissions for the MRI stripper, although the lower weight fraction could be contributing to the reduction.

## H-1-1-3 van Veen Chamber Study (van Veen et al., 2002) and EC Chamber Study (EC, 2004)

<u>EC (2004)</u> and <u>van Veen et al. (2002)</u> conducted chamber experiments with a brush-on paint stripper. In the van Veen study, volunteers applied a commercially available paint stripper to a 1.28-m<sup>2</sup> horizontal surface under a range of ventilation rates (0.26-0.73 hr<sup>-1</sup>). The substrate material was not specified. The study reported that the product mass and DCM weight fraction (65.9%) were "measured from the product by GC and ECD detector<sup>46</sup>" with no additional details provided in the report. Also, the product name and other ingredients in the paint stripper were not specified. A single application of the paint stripper was made to the entire substrate, with scraping of the entire substrate occurring after an effect time of approximately 60 minutes.

Figure H-5 shows the concentration profiles for each experiment. The DCM mass released during the study was estimated for four of the experiments using the mass balance method, described above, as shown in Table H-5. The estimated mass of DCM released ranged from 18% to 39% (Table H-5). The results indicated no obvious correlation between the ventilation rate and the mass released. The relatively low fraction released suggests that these paint stripper products contain a vapor retardant.



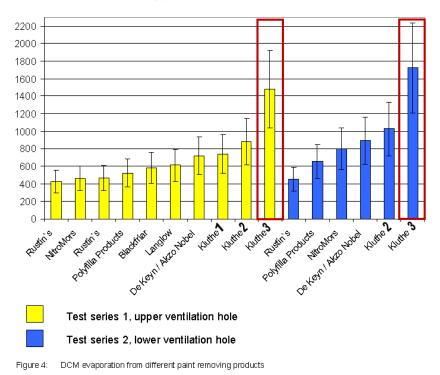


<sup>&</sup>lt;sup>46</sup> Gas chromatography and electron capture detector (GC/ECD)

Table H 5. E	Table H 5. Estimated DCM Released for Four Experiments by van Veen et al. (2002)											
van Veen Experiment	Cha	mber	Duration,	Applied	Applied	% DCM						
	Vol, m <sup>3</sup>	ACH, hr <sup>-1</sup>	min	Product Mass, mg	DCM Mass, mg	Released						
1a	47.65	0.31	60	364,000	239,876	28%						
1b	47.65	0.26	60	335,000	220,765	39%						
3a	47.65	0.53	60	392,000	258,328	18%						
3b	47.65	0.75	60	386,000	254,374	22%						

The <u>EC (2004)</u> study was designed to assess health risks related to the use of defined vaporretarded, DCM-containing paint strippers. TWA concentrations were reported for ten such products available on the European market, as shown in Figure H-6. Of these ten products, six were described as "fluid" and the remaining four as "paste." The chamber was a 15 m<sup>3</sup> room with a ventilation rate of 60 m<sup>3</sup>/hr (4 hr<sup>-1</sup>) for the standard-condition studies. The substrate was a 1.0-m<sup>2</sup> chipboard in a vertical orientation. The composition of specific products was not provided, but the study reported a DCM content ranging from 75 to 90% for the typical formulation of DCM-containing paint strippers on the European market. A single application of the paint stripper lasting about 5 minutes was made to the entire substrate, with scraping occurring after a typical "effecting" time of about 10 minutes.

### Figure H-6. TWA Concentrations for Ten Paint Removing Products (Reproduced from EC, 2004)



TWA 25 min Active Carbon (ppm)

In summary, the data reported in <u>EC (2004)</u> were of marginal utility for this assessment, with the exception of two primary contributions as discussed below.

**Impact of vapor retardants**: The Kluthe 3 product did not contain a vapor retardant while Kluthe 2 contained an unspecified quantity of vapor retardant. Kluthe 1 contained twice the vapor retardant as Kluthe 2.

Assuming that the formulations were similar with the exception of the quantity of vapor retardant, a comparison of the three Kluthe products was used to establish the general impact of the vapor retardant. The following observations are based on Figure H-6 together with the Kluthe vapor-retardant ratios described above:

- The presence of vapor retardant appears to be able to reduce the emissions by approximately 50%. It is possible that formulation with a larger percentage of vapor retardant could lead to an even greater reduction in emissions.
- The first unit of vapor retardant (as represented by Kluthe 2) reduces DCM emissions by approximately 40%. The second unit (as represented by Kluthe 1) reduces emissions by another 10% of the total DCM applied.

The above observations collectively suggest that there may be an optimal vapor retardant quantity for lowering DCM emissions.

**Significance of vertical stratification**: The chamber studies described in <u>EC (2004)</u> measured DCM air concentrations at a lower ventilation hole (10 cm above the floor) and an upper ventilation hole (1.5 m above the floor). DCM is significantly heavier than air since its molecular weight is 84.9 g/mole. Therefore, some vertical stratification would be expected with higher concentrations at lower heights.

Although this phenomenon is beyond the scope of the emissions analysis in this appendix, the extent of stratification can be discerned from the results of the EC study. The ratio of upper to lower concentrations ranged from 0.59 to 0.94 (mean=0.81) for the six products reporting DCM TWA concentrations for both the lower and upper ventilation holes. The observed stratification in the <u>EC (2004)</u> data may have been minimized by the relatively high ventilation rate (4 hr<sup>-1</sup>). The actual extent of stratification may be larger under lower ventilation rates.

# H-1-1-4 Discussion and Conclusions

The four studies reviewed in section H-1 (<u>EC, 2004</u>; <u>EPA, 1994a</u>; <u>LBL, 1986</u>; <u>van Veen et al.,</u> <u>2002</u>) represent the available scientific literature on DCM emissions from consumer use of paint-removal products. Although paint-stripper ingredients other than DCM are not well quantified, the results across the studies appear to be consistent, with DCM-containing products generally categorized as having or not having a vapor retardant. Other paint-stripper ingredients appear to have less impact on DCM emissions.

Table H-6 summarizes the results of the analysis of these studies. Table H-6 also lists some distinguishing features of each experiment along with DCM release fractions as estimated by the two methods described in section H-1-1 (*Conceptual Approach*).

The following additional insights are apparent from Table H-6:

- 1. The estimated release fraction for the product used in the van Veen experiments ranges from 15 to 39%, strongly suggesting the presence of a vapor retardant.
- 2. The two LBL tests have similar estimates, ranging from 84 to 97%, despite distinctly different air exchange rates. These results indicate the absence of vapor retardants.
- 3. The results of the three MRI tests, with release-fraction estimates ranging from approximately 25 to 50%, suggest the presence of a vapor retardant. As discussed in section H-1-1-2, the assumed weight fraction of 16.8% was presented in the 1994 SRD (EPA, 2003), which is a data source contemporary to the MRI study. The assumed weight fraction is also approximately in the middle of the range indicated by the Strypeeze 1988 and 2013 MSDS (Table H-3).

From the exponential fits to the MRI data (EPA, 1994a), it is estimated that 66 percent of the applied DCM in the spray product (Figure H-1, Table H-2) was released to air, as compared to 33 percent of the applied DCM in the brush product (Figure H-2, Table H-2). Further, virtually all of the DCM release occurs within two hours after application for both spray and brush products, very shortly after the last scraping is finished due to DCM's relatively high volatility. Thus, the concentration-decline part of the time series in Figures H-1 and H-2, after the peak, is due almost exclusively to ventilation rather than to declining emissions. Consequently, exposures during this time period could be virtually eliminated through ventilation.

	Experiment		Applied Product	DCM	Applied	Chamb	per	DCM Mass	NLS Fit <sup>b</sup>
Study	Title <sup>a</sup>	Duration, min	Mass, mg	Weight Fraction	DCM Mass, mg	Volume, m <sup>3</sup>	ACH	Released at Duration	Theoretical Mass Released
van Veen et al. 2002	1a	60	364,000	0.659	239,876	47.65	0.31	27.6%	25.5%
	1b	60	335,000	0.659	220,765	47.65	0.26	39.0%	35.1%
	3a	60	392,000	0.659	258,328	47.65	0.53	17.6%	14.9%
	3b	60	386,000	0.659	254,374	47.65	0.75	21.8%	18.0%
	Exp 2	89	363,000	0.866	314,358	20.0	3.23	83.3%	83.9%
LBL (1986)	Exp 5	86	325,000	0.830	269,750	20.0	0.62	92.9%	97.0%
	Run 7	105	724000	0.168	121,632	35.7	0.56	35.0%	34.4%
MRI (FDA 1004a)	Run 8	105	676000	0.168	113,568	35.7	0.54	50.7%	50.4%
(EPA, 1994a)	Run 9	105	765000	0.168	128,520	35.7	0.55	29.5%	24.6%
EC (2004)	VP03	25	465500	0.825 °	384,038	15	4	13.9%	NA

Notes:

 <sup>a</sup> These are the experiment names used in the reports.
 <sup>b</sup> The nonlinear least squares (NLS) fit minimizes the squared difference between observations and model predictions using the model described in section H-1-1. The theoretical mass released is determined by integrating the fitted exponential to infinity.

<sup>c</sup> The EC report did list product- specific formulations, instead providing a "typical formulation of vapour retarded paint strippers" with DCM content ranging from 75 – 90%; the midpoint of this range was used.

# H-2 Sensitivity Analysis for Inhalation Scenarios

For this analysis, each input that could be measured on a continuum (*e.g.*, emission rate, airflow rate) was first halved and then doubled while holding all others at their base-case values. For an input to which the model output is directly and linearly proportional, and for which the exposure measure for the base case is denoted as X, the result for the halved case would be  $\frac{1}{2}X$  and the result for the doubled case would be 2X. Computing and averaging the two differences from the base case gives the following result:

 $([X-1/2X] + [2X-X])/2)/X = \frac{3}{4} \text{ or } 75 \text{ percent}$  (Equation H-8)

For an input that cannot be varied over a continuum, or that can be dealt with only discretely or perhaps dichotomously (*e.g.*, in the use zone or not at certain key times), the above procedure can still be used, but the sensitivity measure reduces to:

|Y-X| / X (expressed as a percent) (Equation H-9)

where Y is the output associated with the change in location pattern from the base case.

## H-3 Inhalation Exposure Scenario Inputs

*Method of Application.* A review of product labels and technical data sheets indicates that paint stripping products can be applied using either brush-on or spray-on (*i.e.*, aerosol or trigger-pump) application methods. In this assessment, exposures were assessed for both brush-on and spray-on products due to differences in chemical release characteristics, DCM weight fraction of products, application rates, and time required for application.

Application Amount (Product Mass). The product application mass (grams of product) was determined for each of the cases examined using application rates (g/ft<sup>2</sup>) and the surface areas of objects to be stripped (ft<sup>2</sup>). The application rates were calculated from the MRI chamber tests (EPA, 1994a). Surface areas were selected so that the resulting product mass corresponded approximately to central (near the median) and upper-end estimates for the amount of paint stripper product used per event from the large nationwide survey by <u>CPSC</u> (1992), as reported in Table 17-20 of the Exposure Factors Handbook (EFH)(EPA, 2011a).

The EFH reported a median value of 32 fluid ounces or ¼ gallon of paint stripper product. Conversion to metric units (3.75 liters per gallon) and consideration of the nominal product density (~1.1 g/cm<sup>3</sup>) (calculated from Brown, 2012) yielded a product mass on the order of 1,000 grams as a central estimate. An upper-end application amount (~80<sup>th</sup> percentile) from the same survey was 80 ounces or 2,500 g. Similarly, the <u>Riley et al. (2001)</u> survey reported 32 ounces as the median amount of paint stripper product used. Median product masses of 900 and 680 g were input into the model for the brush-on and sprayon scenarios, respectively. Upper-end product masses for the brush-on and spray-on scenarios were 2,250 and 1700 g, respectively.

The bathroom scenario occurred in a confined space and was assumed to be performed by a professional, as opposed to a consumer. A lower mass of 477 g was used for the brush-on bathroom scenario. The lower mass value was derived from the largest application amount identified in the NIOSH report (<u>CDC, 2012</u>).

The application amounts for Scenarios 1 through 6 were obtained by multiplying the application rates calculated from the MRI experiments (EPA, 1994a) and the surface area of objects to be treated. The calculated application rates were ~90 g/ft<sup>2</sup> for a brush-on application of a DCM-containing product (722 g of product applied to 8 ft<sup>2</sup>) and ~68 g/ft<sup>2</sup> for a spray-on application of a DCM-containing product (540 g of product applied to 8 ft<sup>2</sup>). These application rates were similar to those recommended on the Savogran Company website for paint strippers in general—1 gallon per 50 to 100 ft<sup>2</sup> (~42 to 83 g/ft<sup>2</sup> based on a nominal density of 1.1 g/cm<sup>3</sup>)<sup>47</sup>.

The applied surface areas selected for central and upper-end values were 10 and 25 ft<sup>2</sup>. The upper-end surface area was 2.5 times higher than the central surface area and provided sufficient distinction from the central case. Application targets with surface areas close to the two specified surface areas (10 and 25 ft<sup>2</sup>) were used in the exposure scenarios to reflect real-world situations. A coffee table with nominal dimensions of 4 ft × 2.5 ft for the top surface was selected for the central case (10 ft<sup>2</sup>)<sup>48</sup>. A chest of drawers with nominal dimensions of 4 ft high by 2.5 ft wide by 1.5 ft deep<sup>49</sup> was selected for the upper-end case (4 × 2.5 ft<sup>2</sup> for front + 2.5 × 1.5 ft<sup>2</sup> for top + 2 × 4 × 1.5 ft<sup>2</sup> for sides ≈ 25 ft<sup>2</sup>). For the bathroom scenario, a bathtub surface area of 36 ft<sup>2</sup> was calculated assuming nominal dimensions of 5 ft wide by 2.5 ft deep by 1.5 ft high.

Stripping Sequence. The stripping sequence chosen to characterize product application was based in part on product label instructions. Labeling information for some DCM-containing products (*i.e.*, Klean Strip<sup>®</sup> products) indicate that no more than 9 ft<sup>2</sup> should be stripped at a time. Accordingly, the 10-ft<sup>2</sup> surface for the coffee table was divided into two application segments of 5 ft<sup>2</sup> each. The 25-ft<sup>2</sup> surface for the chest was divided into four application segments of 6.25 ft<sup>2</sup> each. The 36-ft<sup>2</sup> surface for the bathtub was divided into four application segments of 9 ft<sup>2</sup> each.

The segments were assumed to be treated in sequence with a repeat application of the sequence. Thus, in effect, there were four segments for the coffee table and eight segments each for the chest and bathtub. Repeating the stripping sequence was consistent with

<sup>&</sup>lt;sup>47</sup> See the following URL: <u>http://www.savogran.com/materials.html</u>

<sup>&</sup>lt;sup>48</sup> See the following URL: <u>http://furniture.about.com/od/furnishingdesignresources/a/measurements.htm</u>

<sup>&</sup>lt;sup>49</sup> See the following URL for an illustrative chest of drawers with nearly the same dimensions: http://www.furnitureunfinished.com/product\_info.php?cPath=116\_135&products\_id=1093

instructions on the majority of product labels indicating that the stripping procedures should be repeated to remove multiple coats of paint or stubborn paint. All residuals from the scraping were assumed to be removed from the house on completion of the last segment/application.

The entire stripping sequence for each segment consisted of applying the product to the target surface followed by a wait period and then scraping the treated surface. The application and scrape times were deduced from the protocol description in the MRI chamber study (EPA, 1994a). For both the coffee table and the chest scenarios, a two-minute applying time per segment was used for brush applications and a one-minute applying time per segment was used for spray applications. Application was followed by a 15-minute wait period and then a four-minute scraping period per segment.

As an example, the timing of the entire sequence for a brush-on application to the chest surface is shown in Table H-7. For this scenario, the total duration is 21 minutes per segment or 168 minutes for the entire episode of product use. For the coffee table, the duration was 84 minutes for the entire episode with half the number of segments. For the bathtub scenario, the total duration increased to 24 minutes per segment or 192 minutes for the entire episode. For the spray-on application (applicable to the coffee table and chest but not the tub), the application time was cut in half (*i.e.*, from two minutes to one) while retaining the waiting time of 15 minutes and the scraping time of four minutes, resulting in slightly lower total durations than the brush-on scenarios.

For the bathtub scenario (brush-on only) with a larger surface area, the applying time was increased to three minutes per segment and the scraping time was increased to six minutes per segment. The wait time remained the same at 15 minutes per segment. The wait time of 15 minutes was selected based on wait times on the product labels, which varied from five minutes to two hours, with the majority of labels indicating a wait time of 15 minutes.

Back-to-back stripping sequences with no overlapping activities were modeled because it is likely that the user takes breaks during the wait time. In the Riley survey, 65 percent of the participants reported taking breaks outside the work area and 20 percent of the participants reported taking breaks inside the work area, with break times ranging from five to 30 minutes (<u>Riley et al., 2001</u>). The number of breaks was not reported. Additionally, conducting overlapping stripping activities for multiple segments (*i.e.*, applying or scraping one segment during the wait period of another segment) would be unrealistic for most consumer users.

Segment/Application	Elapsed Time from Time Zero, Minutes							
	Apply	Wait	Scrape					
First 1/4, 1 <sup>st</sup> time	0-2	2-17	17-21					
Second 1/4, 1 <sup>st</sup> time	21-23	23-38	38-42					
Third 1/4, 1 <sup>st</sup> time	42-44	44-59	59-63					
Fourth 1/4, 1 <sup>st</sup> time	63-65	65-80	80-84					
First 1/4, 2 <sup>nd</sup> time	84-86	86-101	101-105					
Second 1/4, 2 <sup>nd</sup> time	105-107	107-122	122-126					
Third 1/4, 2 <sup>nd</sup> time	126-128	128-143	143-147					
Fourth 1/4, 2 <sup>nd</sup> time	147-149	149-164	164-168					

Table H 7 Schedule for Brush on Application to Chest Surface: Four Segments with

Amount of Chemical Released. The amount of chemical released during and after the stripping event is the product of three parameters: amount applied (discussed above), weight fraction of chemical in the applied product, and fraction of the chemical that is released to indoor air.

From the product list developed by Brown (2012), the median DCM weight fraction was determined to be 0.53 for the brush-on application (range of 0.20 to 0.93) and 0.8 for the spray-on application (range of 0.45 to 0.88). The corresponding 90<sup>th</sup> percentile weight fractions were 0.88 for brush-on and 0.87 for spray-on. A weight fraction of 1.0 (maximum exposure estimate derived from product label) was assumed for the bathtub application. The weight fractions were determined from the Brown (2012) spreadsheet by using only products intended for consumer use (*i.e.*, adhesive removers, paint brush cleaners, deglossers, and industrial/commercial use products were removed).

The application method (brush- or spray-on) for a product was determined by examining the product labels/technical data sheets and product names, and through Internet research. If an application method could not be determined through the above methods, then the product was assigned to the brush category. Most paint stripping products are applied by the brush method and formulations, such as semi-paste, would be difficult to apply using a sprayer. If a weight-fraction range was provided in the product list, then the average of the minimum and maximum weight fractions was used in the calculations. The weight fractions were not weighted to reflect the market share of products.

This assessment used the DCM release fractions of 0.33 for brush-on and 0.66 for spray-on based on the analysis of the MRI chamber data (EPA, 1994a) (see Section H-1). The resultant mass released for the different application targets and methods is summarized in Table H-8.

Target (Surface Area) and Method	Application Rate, g/ft <sup>2 a</sup>	Weight Fraction <sup>b</sup>	Release Fraction	DCM Mass Released, g				
Coffee table (10 ft <sup>2</sup> )								
Brush-on	90	0.53   0.88	0.33	157.4   261.4				
Spray-on	68	0.80   0.87	0.66	359.0   390.5				
Chest of drawers (25 ft <sup>2</sup> )								
Brush-on	90	0.88	0.33	653.4				
Spray-on	68	0.87	0.66	976.1				
Bathroom tub (36 ft²)								
Brush-on	13.25	1.0	0.33	157.4				
Notes:								
<ul> <li><sup>a</sup> Reflects repeat application for each segment.</li> <li><sup>b</sup> For the coffee-table case, two weight fractions are given; one for central and one for near-high-end.</li> </ul>								

Airflow Rates and Volumes. The model run requires conceptualization of a residence in terms of the number of zones and their respective volumes. The airflow rates needed to model the central and upper-end scenarios are: (1) rates between indoors and outdoors for each zone; and (2) rates between the zones. The bathroom scenario simulation is somewhat more complex to conceptualize and is described below after the central and upper-end scenarios.

For the central and upper-end scenarios, the house in which the modeled stripper application occurs is conceptualized as having two zones: (1) the workshop where application occurs; and (2) the rest of the house (ROH). The house volume chosen for the model runs (492 m<sup>3</sup>) is the central value listed in the 2011 EFH (EPA, 2011a). The volume assigned to the in-house workshop area was 54 m<sup>3</sup>, corresponding to a 12 ft × 20 ft room with an 8-ft ceiling ( $20 \times 12 \times 8 = 1,920$  ft<sup>3</sup> or ~54 m<sup>3</sup>). This room volume is similar to the value reported in <u>Riley et al. (2001)</u> for the mean volume of the room used for paint stripping ( $51 \text{ m}^3$ ). The volume for the ROH ( $438 \text{ m}^3$ ) is determined by subtraction ( $492 \text{ m}^3 - 54 \text{ m}^3$ ). For the bathtub scenario, the bathroom volume was set at 9 m<sup>3</sup> for consistency with that reported in (<u>CDC, 2012</u>).

The indoor-outdoor airflow for any zone of the house is governed by the choice of air exchange rate (ACH).

The central and low-end ACH values were 0.45/hr and 0.18/hr and corresponded to the mean and 10<sup>th</sup> percentile values, respectively, reported in the 2011 EFH (<u>EPA, 2011a</u>). These values were used for assigning the indoor-outdoor airflow rate for the ROH. Note that a low-end ACH would be expected to contribute to upper-end concentration estimates.

For the workshop, it was assumed that multiple windows were opened. The indoor-outdoor airflow rate assigned to this zone (68 m<sup>3</sup>/hr) was obtained by multiplying the room volume of 54 m<sup>3</sup> by the 90<sup>th</sup> percentile (1.26/hr) of the air-exchange-rate distribution from the 2011 EFH. This indoor-outdoor airflow rate was thought to reasonably represent the open-window assumption.

The use of open windows in the room of use is supported by both label instructions and survey data. The majority of the labels indicate that adequate ventilation must be used and that, to prevent build-up of vapors, windows and doors should be opened to achieve cross ventilation. Additionally, <u>Pollack-Nelson (1995)</u> reported that an average of 70.7 percent of paint stripper users (all products) kept a window or door open during use based on data from the <u>EPA (1987)</u> survey. The <u>CPSC (1992)</u> survey reported that 88.8 percent of paint stripper users (all products) kept a window or door open during use. The increase was significant between the survey years (before and after CPSC labeling requirements took effect). The <u>Riley et al. (2001)</u> survey also indicates that the majority of paint-stripper users (55 percent) opened a window.

Both <u>Pollack-Nelson (1995)</u> and <u>Riley et al. (2001)</u> also reported that some users used an exhaust fan during the stripping process, which would affect the air exchange rate. The percentage of fan users was not reported in <u>Pollack-Nelson (1995)</u>. The <u>Riley et al. (2001)</u> data suggest that only ~27 percent of the users who worked indoors used an open window and fan. Due to the small percentage of respondents who reported using a fan, coupled with the fact that some of labels indicate that the product should be kept away from heat, sparks, flame, and all other sources of ignition, none of the scenarios were assumed to involve use of a fan in the room of product application.

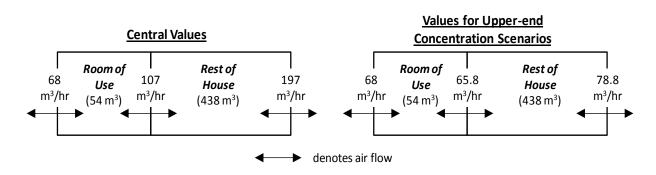
The interzonal airflow rate was estimated using the following algorithm, developed by <u>EPA</u> (1995a):

Q = (0.078 + 0.31\*ACH) \* house volume (Equation H-10)

where Q is the interzonal airflow rate, in m<sup>3</sup>/hr, and ACH is the air exchange rate, in 1/hr. Substitution of the central air exchange rate of 0.45/hr and the house volume of 492 m<sup>3</sup> yields an estimated interzonal airflow rate of 107 m<sup>3</sup>/hr.

The algorithm was derived from empirical ventilation data collected in over 4,000 U.S. residences by the perfluorocarbon tracer (PFT) technique (EPA, 1995a). In the EPA (1995a) analysis, the doors between residential zones were generally considered to be open, and thus EPA/OPPT set up the residential zones to be consistent with EPA (1995a).

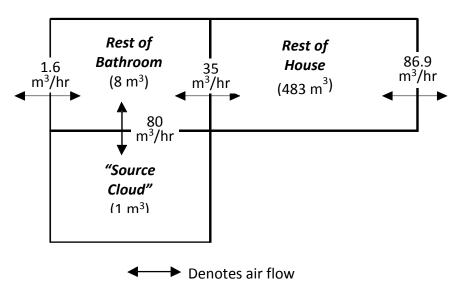
The corresponding interzonal airflow rate for the upper-end scenario, with an air exchange rate of 0.18/hr, is 65.8 m<sup>3</sup>/hr. Figure H-7 depicts the volumes and airflows that were used for the workshop scenarios.



## Figure H-7. Zone Volumes and Airflow Rates for Workshop Scenarios

As previously mentioned, the bathroom scenario is more complex (Figure H-8). While working in close proximity to the target (bathtub) for an extended period, the product user is typically exposed to elevated concentrations in the immediate vicinity of the application area, a concept that has been termed the "source cloud" in the scientific literature. There is considerable evidence of a source-cloud effect around sources (<u>Cheng et al., 2011</u>; <u>Furtaw et al., 1996</u>; <u>Matthews et al., 1989</u>), which generally relates the size of the source cloud and the ratio of the near- vs. far-field concentrations to the room turbulence (*e.g.*, due to natural and mechanical ventilation) and other mixing forces such as thermal gradients.





Several studies have investigated methods for modeling a source cloud, including use of a virtual compartment around the source (<u>Cherrie, 1999</u>), rough partitioning (<u>Musy et al., 1999</u>), and a zero-equation turbulence model (<u>Chen and Xu, 1998</u>). The virtual-compartment method also has been discussed in ASTM Standard Practice D 6178-97 (<u>ASTM, 1997</u>). Although the ideal size of the virtual compartment has not been discussed in the literature, <u>Furtaw et al. (1996</u>) successfully represented concentrations using a sphere around the source (with an unspecified

volume). Thus, both the presence of higher concentrations near a source and the concept of using a source cloud to better represent these near-field elevated concentrations appear to be well founded in the scientific literature.

For the purpose of this exposure assessment, a source cloud is used for the bathroom scenario to better represent the user's exposure to DCM emitted from the paint stripper. The bathroom scenario involves application of a relatively large amount of the product within a semi-enclosed, concave workspace, resulting in accumulation of the heavier-than-air DCM vapors toward the lower tub surfaces in particular (see the *Vertical Stratification Analysis* in section H-1-1-3). Moreover, accessibility constraints and the concave shape of the workspace would require the user to work in close proximity to the surface being stripped, particularly when working on the lower portions of the tub. For these reasons, a source-cloud representation is appropriate for the bathroom scenario.

The source cloud representation was not deemed necessary for the workshop scenarios because work areas within such a space typically are not so confined and are less likely to promote localized accumulation of DCM vapors. The MRI test chamber inlet and outlet concentration values were consistently higher than those in the worker's breathing zone and at the center of the chamber. On the other hand, the LBL test chamber results showed relatively uniform mixing at lower ventilation rates and higher concentrations near the source at higher ventilation rates. The ventilation rate in a chamber can be varied only by mechanical means (e.g., via exhaust fans). In contrast, the ventilation rate in residential settings can be increased by natural means such as opening windows. Air transfer with the ROH can be increased by opening interior doors. The extent of concentration gradients within a workshop would be highly dependent on physical characteristics such as the size, shape and orientation of the stripping target (certainly less well defined than a bathtub) as well as conditions such as air flows through windows and interior doors. Given this variability and dependency, modeling the workshop scenario as a well-mixed zone is considered appropriate for a Tier 2 exposure assessment.

Recognizing that the source cloud is not a well-defined area, but rather a gradual transition between near- and far-field concentrations, and further recognizing that the purpose of this volume is to represent average air concentrations in the breathing zone of the product user, the approach to defining the virtual volume was to establish some geometry around the source that represents the approximate work space. Figure H-9 shows a schematic representation of the bathtub and virtual compartment representing the source cloud. Consistent with this representation, a source-cloud volume of 1.0 m<sup>3</sup> was assumed for the bathroom scenario.

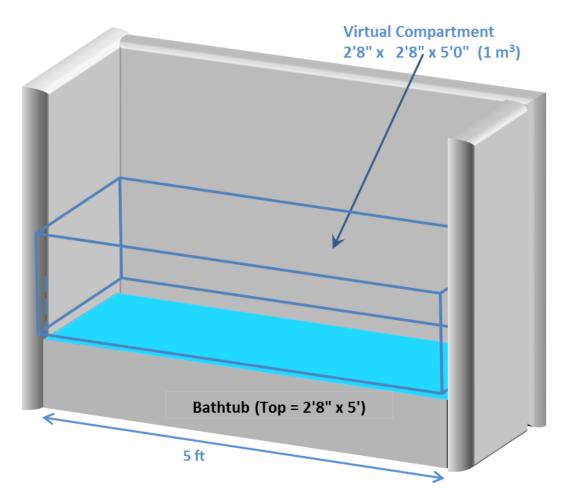
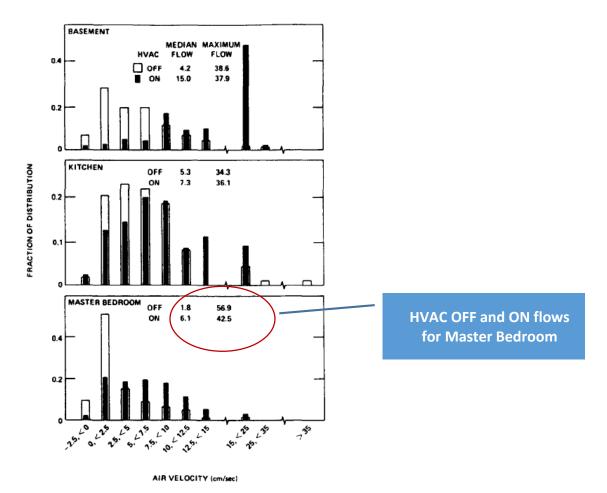
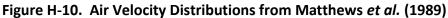


Figure H-9. Modeling Representation of the Bathtub and Virtual Compartment

Matthews et al. (1989) analyzed the impact of a central, forced-air heating, ventilating and air conditioning (HVAC) system on the distribution of air velocities in three of their six study homes. The remaining three homes were not included in the analysis because in two cases the fan was operated continuously and a probe malfunctioned in the third home. In Figure H-10, the results for the three analyzed homes are presented at three different indoor locations (basement, kitchen, and master bedroom). For the bedroom (most similar of the three locations to the bathroom), the Matthews results include a median air velocity of 1.8 cm/sec with the fan off and 6.1 cm/sec with the fan on.





With the fan cycling on and off the air velocity would be between 1.8 and 6.1 cm/sec, with the average velocity dependent on the on-time for the fan. As of 2008, at least 25% of U.S. homes did not have a central, forced-air heating system (EFH Table 19-13; EPA, 2011a). Homes with alternative systems (e.g., steam or hot-water system; baseboard/portable electric heat) would be expected to have a velocity similar to that for the fan-off case. Similarly, ~40% of U.S. homes had either no cooling equipment or room/window cooling units (EFH Table 19-15; EPA, 2011a). Consequently, a velocity of 1.8 cm/sec (65 m/hr) was used for the bathroom scenario, to represent such homes as well as those with a central forced-air system that is off during paint stripping either by intent or due to mild weather.

The assumed airflow rate between the source cloud and the rest of the bathroom was based on a relationship developed by <u>Matthews et al. (1989)</u>, who determined experimentally that such an airflow could be estimated as the product of the room air velocity (m/hr) and the entry/exit surface area (m<sup>2</sup>). An assumed air velocity of 65 m/hr, representing the fan-off case, together with an assumed entry/exit surface area of 5 ft by 2 ft, 8 in (13.35 ft<sup>2</sup> or 1.24 m<sup>2</sup>) resulted in an estimated airflow rate of 80 m<sup>3</sup>/hr between the source cloud and the rest of the bathroom.

*Locations of Exposed Individuals.* Two location patterns were specified, one for a product user and one for a non-user (bystander). The user was assumed to be in the work area for stripper application and scraping for all scenarios. For the waiting phase of the stripping process, the user was assumed to be in the ROH as a central-tendency assumption for the user (Scenarios 1 and 4), in the workshop as an upper-end assumption for the user (Scenarios 2 and 5), and in the ROH for Scenarios 3, 6, and 7, which were developed to model upper-end concentrations primarily for the non-user.

The user was placed in the remainder of the house during the waiting phase for Scenarios 1, 3, 4, 6, and 7 because the user was assumed to be aware of inhalation health concerns from the label warnings ("Vapor Harmful"). Also, some labels such as the Klean Strip<sup>®</sup> products specifically stated that the user should leave the room during the wait period.

As previously mentioned, the <u>Riley et al. (2001)</u> survey also reported that 65 percent of users reported taking breaks outside the work area. Breaks typically involved a specific break activity and location, such as going to the kitchen and making a sandwich or going outside to do yard work.

For the upper-end Scenarios 2 and 5, it was assumed that the user would stay in the workshop, based on the fact that some people do not read/skim labels (~28 percent in 1990) (Pollack-Nelson, 1995) and may therefore not be aware of health concerns or precautionary techniques. Many labels do not specifically state to leave room during the wait period, and the <u>Riley et al.</u> (2001) survey indicated that 20 percent of participants reported taking breaks inside the work area. For all scenarios, the user was assumed to leave the workroom immediately after the stripping job was completed. This assumption was based on the <u>EPA (1987)</u> and <u>CPSC (1992)</u> survey findings of a median value of zero minutes spent in the room after using the product.

The non-user (bystander) was assumed to be in the ROH throughout the model run, as was the user for the portion of the run after all applying/scraping was completed. For the bathroom scenario, the user was assumed to be in the ROH during the wait times. It was further assumed that the scrapings were removed from the house as soon as scraping was completed for the last segment. The implication for modeling purposes is that any remaining DCM emissions would be truncated at that time. However, the modeled DCM emissions were not truncated as an expedience. Given the high volatility of DCM (vapor pressure = 352.5 Torr), its emission rate would essentially drop to zero in a relatively short time, on the order of 15-20 minutes, which coincides with the end of the scraping period. Our calculations indicate that, at this time, less than 1 % of the DCM mass released to air remains.

## H-4 Inhalation Model Outputs and Exposure Calculations

## H-4-1 Exposure Calculations

Maximum TWA concentrations for different averaging periods, described below, were calculated from the one-minute averages for both the user and non-user (bystander) based on their respective exposure concentration time series. The calculations took into account the possibility that the user can change zones within a one-minute interval (*e.g.*, at an elapsed time of 6.25 minutes). The exposure concentration was calculated for each one-minute interval in the modeling period (24 hours or 1,440 one-minute intervals) as follows:

For each time interval, *i* to *i* +1, for *i* = 0 to 1,440:

$$EC_{i,i+1} = \left[ \binom{(C_{1,i} + C_{1,i+1})}{2} * F_{i,i+1} + \left[ \binom{(C_{ROH,i} + C_{ROH,i+1})}{2} * (1 - F_{i,i+1}) \right]$$
(Equation H-11)

where:

 $EC_{i,i+1}$  = the exposure concentration over the time interval *i* to *i* +1  $C_{1,i}$  and  $C_{1,i+1}$  = the concentrations in the use zone at times *i* and *i*+1, respectively  $C_{ROH,i}$  and  $C_{ROH,i+1}$  = the concentrations in the ROH zone at times *i* and *i*+1, respectively  $F_{i,i+1}$  = the fraction of time spent in the use zone during the time interval *i* to *i* +1

These calculations, illustrated in Figure H-11, were implemented in an Excel spreadsheet for each of the seven scenarios.

Figure H-11. Example of the Exposure Concentration Calculation as Defined in Equation H-11
--

A	L		-	: ×	√ f <sub>x</sub>	Mo						
	A	В	С	D	E	F	G	н	I	J	к	L
1	1 Model Results					Activity Patte	ern and Persor	hal Concen	trations			
2	Time	Time	Outdoors	Z1 (Workshop)	Z2 (ROH)	Time	Interval	Avg Z1 Conc (Workshop)	Avg Z2 Conc (ROH)	Fraction Spent Z1	User Exposure	Non-user Esposure
3	(days)	(hrs)	(mg/m²)	mg/m3,	(mg/m3)	(min)	(min)	(mg/m²)	(mg/m²)	opent 21	Conc (mg/m³)	Conc (mg/m³)
4	0	0	0	0	0	0.0	$\square$		(			
5	0.0007	0.0167	2.26E-59	Time	nterval	1.0	0.0 - 1.0	29.41855	0.0249428	_ 1	29.41855	0.02494285
6	0.0014	0.0333	1.72E-58	i iiiie i	litervar	2.0	1.0 - 2.0	139.41755	0.2143723	7 1	139.41755	0.21437235
7	0.0021	0.05	5.31E-58		1.10001	3.0	2.9 3.0	312.2195	0 77 1934	0	0.7719345	0.7719345
8	0.0028	0.0667	1.08E-57		D4) / 2	40	3.0-4.0	478.102	1.76231	0	1.762315	1.762315
9	0.0035	0.0833	1.78E-57	(03 +	D4) / 2	5.0	4.0-5.0	695.896	3,11755:	0	3.117595	3.117595
10	0.0042	0.1	2.59E-57	100.000	0.04012	6.0	5.0-6.0	713.1935		0	4.757845	4.757845
11	0.0049	0.1167	3.50E-57	827.068	7.5927	7.0	6.0-7.0	792.713	6.6164	0	6.61641	6.61641
12	0.0056	0.1333	4.48E-57	077 001	9 60796	8.0	79-8.0	852,4345		0	8.63783	8.63783
13	0.0063	0.15	5.51E-51	Fraction o	f Time	9.0	8.0-9.0	895.7135	10.7760	0	10.77608	10.77608
14	0.0069	0.1667	6.58E-5			10.0	9.0 - 10.0	925.3765	12.993	0	12.9931	12.9931
15	0.0076	0.1833	7.67E-5	Spent in U	se zone	11.0	10.0-1.0	943.8035	15.2575	0	15.25755	15.25755
16	0.0083	0.2	8.77E-57		10.0000	12.0	1.0 - 12.0	952.997	17.543	0	17.5437	17.5437
17	0.009	0.2167	9.88E-57		*/: 15)	13.0	12.0 - 13.0	954.6415	19.8304	0	19.83045	19.83045
18	0.0097	0.2333		H5*J5 + I5	·(I-J5)	14.0	13.0 - 14.0	950.151	22.100	0	22.1008	22.1008
19	0.0104	0.25	1.21E-56			15.0	14.0 - 15.0	940.7115	24.3411	0	24.34115	24.34115
20	0.0111	0.2667	1.32E-56	919.744	27.6287	16.0	15.0 - 16.0	927.317	26.540	0	26.5405	26.5405
21	0.0118	0.2833	1.42E-56	901.854	29.7518	17.0	16.0 - 17.0	910.799	28.6902	0	28.69025	28.69025
22	0.0125	0.3	1.53E-56	881.852	31.8158	18.0	17.0 - 18.0	891.853	30.783	1	891.853	30.7838
23	0.0132	0.3167	1.63E-56	860.263	33.8164	19.0	10.0 - 10.0	871.0575	32.8161		871.0575	32.8161
24	0.0139	0.3333	173E-56	837 524	35 7506	20.0	19.0 - 20.0	848 8935	34 7835	1	848 8935	34 7835

## H-4-2 TWA Concentrations

In addition to the maximum one-minute concentration and the 24-hr average concentration to which the user and non-user (bystander) were exposed, a maximum TWA exposure concentration was calculated for each of the following averaging periods: 10 minutes, 30 minutes, one hour, four hours, and eight hours. The maximum TWA concentration for any averaging period was defined as the highest value of the consecutive running averages for that averaging period. For any averaging period, there are (1,440 minus length of the averaging period) TWA concentration values within the 24-hr (1,440-minute) time series. For example, there are 1,430 10-minute averaging periods (1,440-10), the first of which is for time zero to 10 minutes, the second of which is for time one to 11 minutes, and so on, with the last for time 1,430 to 1,440 minutes. The running averages for each averaging period were computed in an Excel spreadsheet, from which the maximum value was determined.

## H-4-3 Modeling Results

The zone-specific and exposure concentrations predicted by MCCEM are presented in Figures H-12 though H-15<sup>50</sup>. Figure H-12 shows the zone-specific and the user's exposure-concentration results for Scenario 1 (brush application in the workshop with central parameter values, top two figures) and Scenario 4 (spray application) in the workshop using central parameter values. The dips in the user's exposure concentration during the application periods reflect temporary relocation to the ROH (Zone 2) during wait times between applying and scraping. The non-user's exposure concentrations are the same as those in the ROH.

As indicated in Figure H-12 the peak concentrations were higher for the spray-application scenario than those for the brush-application scenario, even though the mass of product applied was higher for brush application (900 g stripper applied for brush as compared to 680 g for spray). This difference is explained primarily by two factors: (1) the weight fraction of DCM in the stripper product (0.8 for the spray product as compared to 0.53 for the brush product); and (2) the higher fraction of the applied mass emitted, with the spray application double that of the brush application (*i.e.*, 66 percent as compared to 33 percent) based on analysis of the MRI chamber data (EPA, 1994a). As a result, the DCM mass emitted for the spray stripping was  $^22.25$  times as high as the mass released during the brush stripping (680 g × 0.8 × 0.66 = 359 g for the spray activity as compared to 900 g × 0.53 × 0.33 = 160 g for the brush activity).

Other than the mass of DCM emitted and some minor differences in application time, Scenarios 1 and 4 were identical, which resulted in a similar ratio in air concentrations. For example, for Scenario 4, the peak concentration in Zone 1 was 1,800 mg/m<sup>3</sup>, whereas the peak for Scenario 1 was 780, a ratio of 2.30. The similar ratios for applied mass and resultant peak air concentration apply when other model inputs, such as room volumes and air exchange rates, were kept at the same or similar values. The shape of the user's exposure concentration profile

<sup>&</sup>lt;sup>50</sup> Figures H-12 through H-15 are provided at the end of this section.

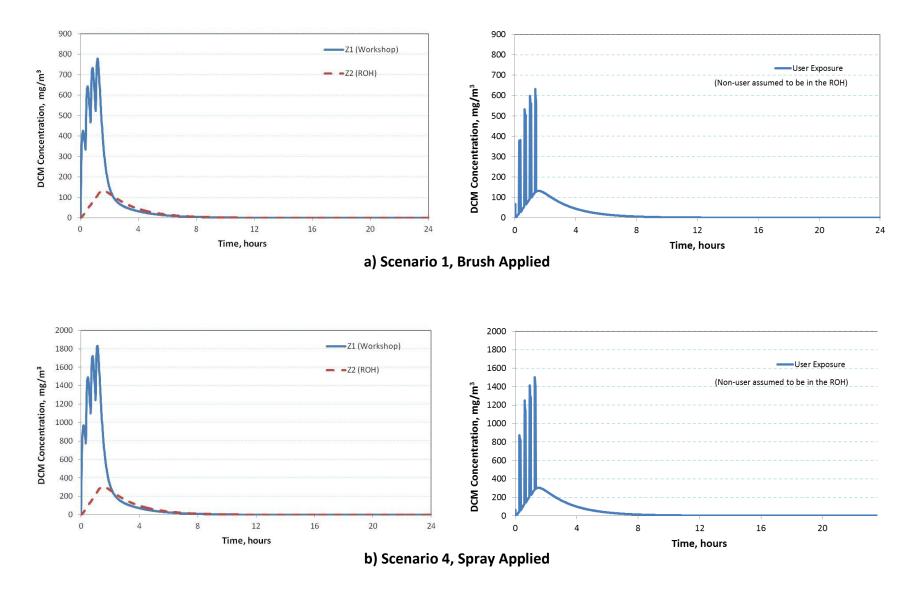
reflected the location of the user at various times during the stripping, waiting, and scraping activities.

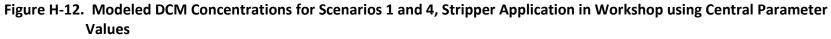
Figure H-13 shows the zone and user exposure concentration results for Scenarios 2 and 5 (brush and spray application, respectively) for the workshop with parameter values selected to estimate upper-end concentrations for the user. In this comparison, the emitted DCM mass was 390 g for the spray vs. 260 g for the brush application, a ratio of ~1.5. The peak concentrations (2,000 mg/m<sup>3</sup> for spray-on vs. 1,300 mg/m<sup>3</sup> for brush-on) again had a ratio (1.54) that was similar to that for the applied mass.

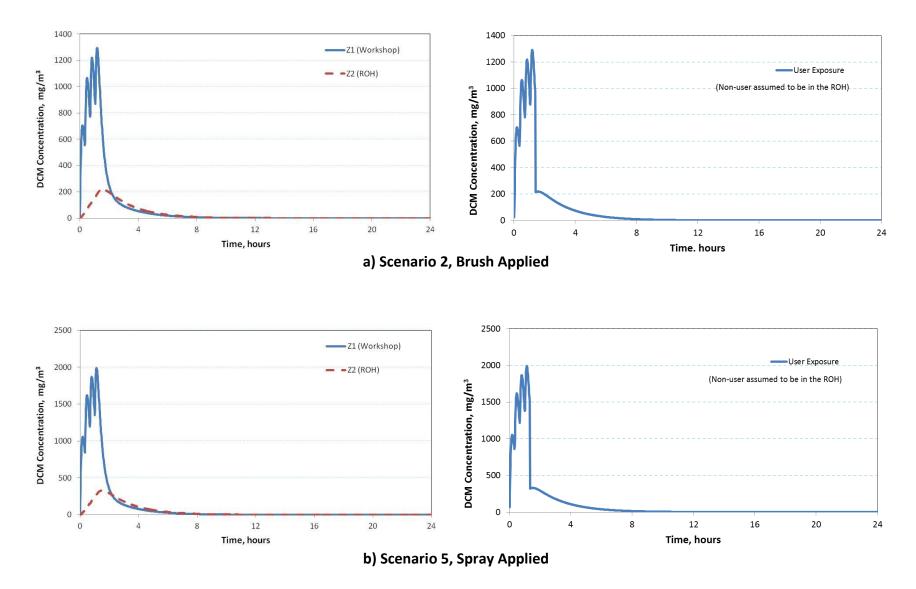
Figure H-14 shows the zone and user exposure concentration results for Scenarios 3 and 6 (brush and spray application, respectively) for the workshop with parameter values selected to estimate upper-end exposure concentrations for the user as well as the non-user (bystander). In this comparison, the spray-to-brush ratio of ~1.5 for applied mass translated directly to the 24-hr TWA concentration to which the non-user was exposed (180 mg/m<sup>3</sup> for spray application vs. 120 mg/m<sup>3</sup> for brush application). The more pronounced crossover of the Zone 1 and 2 concentration time series for these scenarios, shortly after the user finishes the last scraping, can be explained by the indoor-outdoor air exchange rates: 1.26/hr for the workshop vs. 0.18/hr for the ROH. Due to the higher dilution rate, the workshop concentrations fell more quickly after the stripping activity was completed than do the concentrations in the ROH.

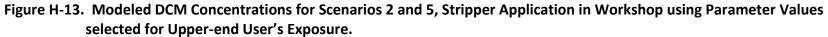
Figure H-15 shows the air concentrations for the simulation scenario, namely the bathtub stripping activity with a modeled peak concentration of ~3,000 mg/m<sup>3</sup>. The saw-tooth appearance of the concentration rise associated with the stripping activity—particularly pronounced in this scenario but evident in the other scenarios as well—was due to the eight application segments. Scenario 7 was intended, in part, to simulate the situation described in a CDC/NIOSH case fatality assessment (CDC, 2012; Chester et al., 2012).

The user's modeled maximum exposure concentration reported here for Scenario 7, on the order of 2,500 ppm, is substantially lower than that calculated (155,000 ppm) for the fatality assessment, but the value reported by CDC/NIOSH was a bounding estimate obtained by assuming that all DCM mass was released instantaneously. By comparison, monitoring of a bathtub application by Washington State Occupational Safety and Health staff, as described in the CDC/NIOSH report (CDC, 2012; Chester et al., 2012), indicated a 15-minute TWA exposure concentration for the applicator on the order of 2,000 ppm.









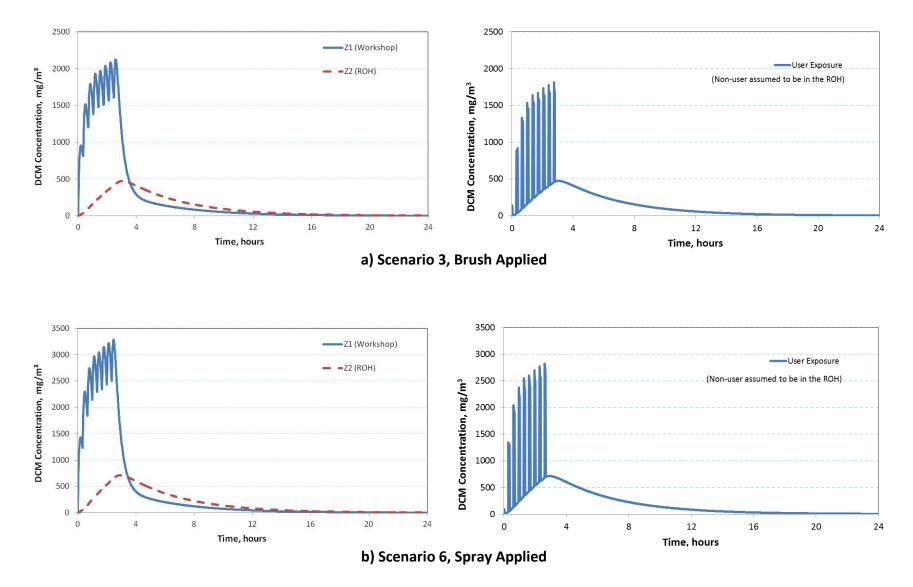


Figure H-14. Modeled DCM Concentrations for Scenarios 3 and 6, Stripper Application in Workshop using Parameter Values Selected for Upper-end-User's and Non-user (Bystander)'s Exposure

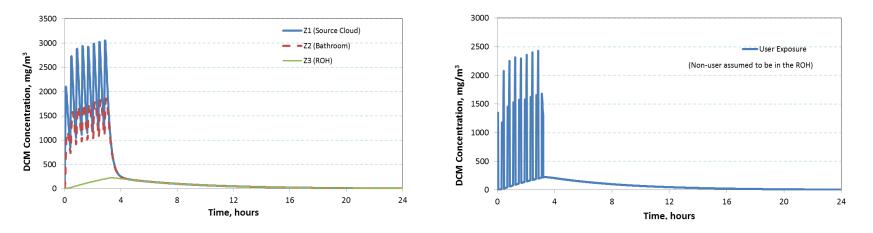


Figure H-15. Modeled DCM Concentrations for Scenario 7, Brush Application in Bathroom (Simulation)

# H-5 Comparison of Modeling-based and Monitoring-based Exposure Estimates

This section discusses similarities and differences between the DCM consumer modeling estimates from this assessment and the monitoring results from the Lawrence Berkeley Laboratory house study (<u>LBL, 1987</u>). It also discusses the exposure estimates for the most comparable scenarios.

## H-5-1 Scenario Similarities and Differences

The LBL study (<u>LBL, 1987</u>) and a related chamber study (<u>LBL, 1986</u>) were conducted by LBL for the U.S. Department of Energy (DOE) with support from the U.S. Consumer Product Safety Commission (CPSC).

The LBL (1987) study includes both monitored and modeled exposure results from consumeruse scenarios that share some similarities with those modeled in the EPA/OPPT risk assessment for DCM. However, the LBL results are expressed in ppm-hr exposure units, as opposed to TWA concentration units of mg/m<sup>3</sup> that were calculated for the EPA/OPPT assessment. With knowledge of the duration of each experiment (provided in the LBL report) and the molecular weight of DCM (84.9 g/mole), it was possible to recast the LBL exposure estimates in the same units as the EPA/OPPT modeling results. This was done by first dividing the ppm-hr estimates by the experiment's duration (in hours) to obtain TWA estimates in ppm. Then the estimates were converted to mass/volume units using the following relationship: 1 ppm DCM = 3.47 mg/m<sup>3</sup> DCM.

LBL conducted a total of 21 experiments, some outdoors and others indoors (i.e., in a garage, a basement workshop, and large and small rooms of a house). One of the study objectives was to identify practical ways to reduce exposures to DCM when using a paint remover. Consequently, the LBL base case for comparison was an upper-end exposure scenario with very conservative assumptions, especially the closed-room configuration with all windows as well as the interior door assumed to be closed. By comparison, all EPA/OPPT scenarios assumed an open interior door together with some form of ventilation, in accordance with both label instructions and predominant patterns of paint-stripper use based on household surveys (section H-3, *Airflow Rates and Volumes*).

For all 6 workshop cases in the EPA/OPPT assessment, the interior door to the room of application was assumed to be open, whereas the door was closed for 4 of the 5 LBL indoor cases (*i.e.*, bedroom, dining room, or basement). The ventilation rate for the workshop was greater than 1.0 ACH for all 6 cases in the EPA/OPPT assessment versus 2 of the 5 LBL indoor cases. At the opposite extreme, all LBL garage cases had high ventilation rates, ranging from ~ 2 to ~ 19 ACH.

There were greater similarities for the length of the work period, between 80 and 90 minutes for 4 of the 6 cases in the EPA/OPPT assessment and for 4 of the 5 indoor LBL cases. Three of the EPA/OPPT scenarios used a brush application and three used a spray application, whereas all LBL experiments used a brush application. The applied product mass was greater for the EPA/OPPT cases, whereas the fraction of DCM mass released to indoor air was greater for the LBL cases (somewhat greater than EPA/OPPT spray-on cases but much greater than EPA/OPPT brush-on cases).

## H-5-2 Comparison of Exposure Estimates

Table H-9 lists the exposure estimates and associated test conditions for EPA/OPPT workshop cases and LBL indoor cases. Despite the numerous differences, there were certain cases with a good number of similarities. For example, in terms of the room volume and ventilation rate, the EPA/OPPT workshop cases (volume = 54 m<sup>3</sup> and ventilation rate = 1.26 ACH) were quite similar to the LBL basement case.

Table H-9 highlights the most comparable cases for the user and non-user (bystander) exposures. In addition to similar room volumes and ventilation rates, these user cases had nearly identical theoretical estimates of DCM mass released to the indoor air, reflecting the combined effects of applied product mass, DCM weight fraction in the product, and fraction of applied DCM mass that is released to indoor air.

The most comparable cases for the non-user (bystander) exposure were similar with respect to room-of-use volume and DCM mass released, but not in terms of ventilation rate. The much lower ventilation rate for the LBL case (0.23 ACH) vs. the EPA/OPPT case (1.26 ACH) resulted in a substantially longer residence time for the airborne DCM mass and, hence, greater opportunity for transport to the rest of the house despite the closed-door configuration.

Room of Product Application	Application Method	Theoretical DCM Mass	Length of Work	Ventilation Rate for Work	User Location		sure during od, mg/m <sup>3</sup>						
(Volume)	Substrate	Released, g	Period, min	Area, ACH	during Wait Period	User	Non user						
OPP	OPPT Cases (modeled with door to room of application assumed to be open in all cases)												
Workshop (54 m <sup>3</sup> )	Brush-Table	157.4	84	1.26	ROH	174	25						
Workshop (54 m <sup>3</sup> )	Brush-Table	261.4	84	1.26	Workshop	917	42						
Workshop (54 m <sup>3</sup> )	Brush-Chest	653.4	168	1.26	ROH	563	186						
Workshop (54 m <sup>3</sup> )	Spray-Table	359.0	80	1.26	ROH	383	55						
Workshop (54 m <sup>3</sup> )	Spray-Table	390.5	80	1.26	Workshop	1,418	59						
Workshop (54 m <sup>3</sup> )	Spray-Chest	976.1	160	1.26	ROH	808	264						
LBL Cases (monitored with door to room of application closed in all cases except the 2nd)													
Bedroom (22.6 m <sup>3</sup> )	Brush-Panel	194.8	102	0.13	Bedroom	2,090							
Bedroom (22.6 m <sup>3</sup> )	Brush-Panel	223.4	89	a	Bedroom	771	143						
Bedroom (22.6 m <sup>3</sup> )	Brush-Panel	253.8	90	1.57	Bedroom	982							
Dining Room (73.2 m <sup>3</sup> )	Brush-Panel	231.4	88	0.23	Dining Room	574	67						
Basement (60.7 m <sup>3</sup> )	Brush-Panel	247.5	92	1.60 <sup>b</sup>	Basement	818							
Notes:													
<sup>a</sup> Not measured but likely s		-											

## Table H-9. Estimated Exposures and Associated Conditions for Selected OPPT and LBL Cases

<sup>b</sup> Measured in a separate experiment with the window closed and the stairway door open.

indicates the most comparable product user exposures and 🔄 indicates the most comparable non-user (bystander) exposures

Non-user= Residential bystander

# H-6 MCCEM Inhalation Modeling Scenario Summaries

Formula:	CH <sub>2</sub> Cl <sub>2</sub>
CAS Number:	75-09-2
Molecular Weight:	84.93 g/mol
Density:	1.33 g/cm <sup>2</sup> (liquid)
Appearance	colorless liquid
Melting Point:	-96.7 deg C = -142 deg F = 176 K
Boiling Point:	39.6 deg C = 103 deg F = 313 K
Solubility in Water:	13 g/L @ 20 deg C
Vapor Pressure:	47 kPa = 352.535 Torr = 0.4639 atm = 6.817 psi
Conversion units:	1 ppm = 3.4736 mg/m <sup>3</sup>
Saturation Concentration:	463,862 ppm = 1,611,281 mg/m <sup>3</sup>

# DCM Scenario 1. Coffee Table, Brush-On, Workshop, User in ROH during wait time, 0.45 ACH, 0.53 Weight Fraction

### MCCEM Input Summary

Application Method: Brush-on

## Volumes:

Workshop volume =  $54 \text{ m}^3$ ROH volume =  $492 - 54 = 438 \text{ m}^3$ 

### Airflows:

Workshop-outdoors	68 m³/h
ROH-outdoors	197.1 m <sup>3</sup> /h (0.45 ACH)
Workshop-ROH	107 m³/h

### DCM Mass Released:

Coffee table = 10 sq ft surface area

Applied product mass = 90 g/sq ft = 900 g

DCM mass = 900 g × 0.53 (wt fraction) × 0.33 (release fraction) = 157.4 g

### For each of the 4 application sections:

**k** = 10/hr

**Mass** = 157.4/4 = 39.35 g

**Eo** = Mass \* k = 393.5 g/hr (**NOTE:** only k and Mass are needed as MCCEM inputs) **Application Times by Section:** 

	Elapsed time from time zero, minutes					
Episode	Apply	Wait	Scrape			
first 1/2, 1st time	0-2 min	2-17 min	17-21 min			
second 1/2, 1st time	21-23 min	23-38 min	38-42 min			
first 1/2, 2nd time	42-44 min	44-59 min	59-63 min			
second 1/2, 2nd time	63-65 min	65-80 min	80-84 min			

## Model Run Time:

0-24 hrs

User takes out scrapings after 84 minutes

### **Activity Patterns:**

User in workshop during application and scrape periods, in ROH during wait periods User in ROH for the remainder of the run (22 hrs, 36 minutes)

## **MCCEM Results Summary**

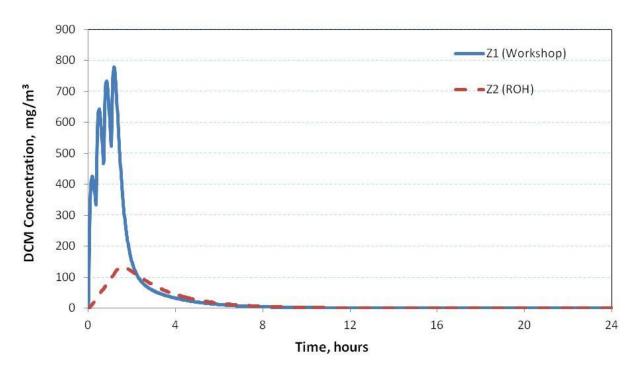
Exposure Concentrations (maximum values over first 24 hrs):

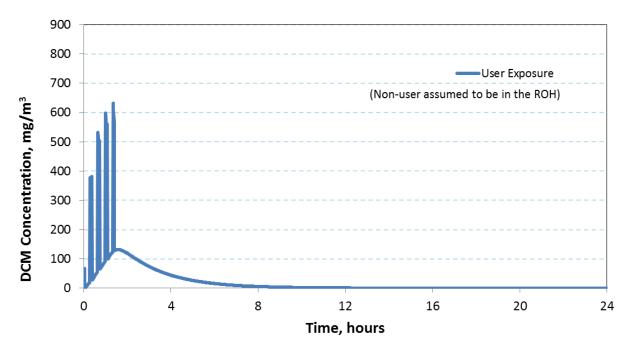
In mg/m³							-
Individual	1 min	10 min	30 min	1 hr	4 hr	8 hr	24 hr
User	632.4	376.1	269.4	224.0	120.5	68.6	23.3
Other	131.7	131.5	129.5	123.8	82.1	49.1	16.8

In ppm

Individual	1 min	10 min	30 min	1 hr	4 hr	8 hr	24 hr
User	182.1	108.3	77.6	64.5	34.7	19.7	6.7
Other	37.9	37.8	37.3	35.6	23.6	14.1	4.8

Plots:





Non-user= Residential bystander

# DCM Scenario 2. Coffee Table, Brush-On, Workshop, User in Workshop during wait time, 0.45 ACH, 0.88 Weight Fraction

## MCCEM Input Summary

## Application Method: Brush-on

Volumes:

Workshop volume =  $54 \text{ m}^3$ 

ROH volume =  $492 - 54 = 438 \text{ m}^3$ 

### Airflows:

Workshop-outdoors	68 m³/h
ROH-outdoors	197.1 m <sup>3</sup> /h (0.45 ACH)
Workshop-ROH	107 m³/h

### DCM Mass Released:

Coffee table = 10 sq ft surface area

Applied product mass = 90 g/sq ft = 900 g

DCM mass = 900 g × 0.88 (wt fraction) × 0.33 (release fraction) = 261.4 g

## For each of the 4 application sections:

**k** = 10/hr

**Mass** = 261.4/4 = 65.35 g

**Eo** = Mass \* k = 653.5 g/hr (**NOTE:** only k and Mass are needed as MCCEM inputs) **Application Times by Section:** 

	Elapsed time from time zero, minutes					
Episode	Apply	Wait	Scrape			
first 1/2, 1st time	0-2 min	2-17 min	17-21 min			
second 1/2, 1st time	21-23 min	23-38 min	38-42 min			
first 1/2, 2nd time	42-44 min	44-59 min	59-63 min			
second 1/2, 2nd time	63-65 min	65-80 min	80-84 min			

### Model Run Time:

0-24 hrs

User takes out scrapings after 84 minutes

### **Activity Patterns:**

User in workshop during application, wait and scrape periods User in ROH for the remainder of the run (22 hrs, 36 minutes)

## MCCEM Results Summary

Exposure Concentrations (maximum values over first 24 hrs):

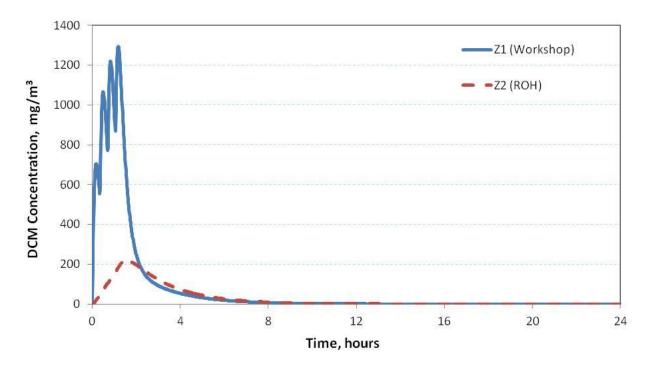
## In mg/m<sup>3</sup>

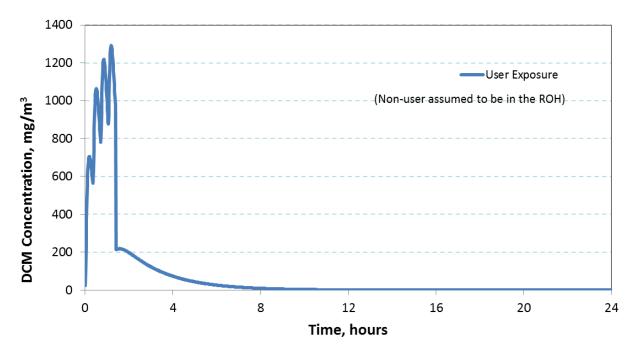
Individua							24 hrs
I	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	
User	1,292.4	1,251.7	1,136.7	1,058.0	416.7	223.8	75.3
Other	218.7	218.3	215.1	205.6	136.3	81.5	27.9

## In ppm

Individual	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	24 hrs
User	372.0	360.4	327.2	304.6	120.0	64.4	21.7
Other	63.0	62.9	61.9	59.2	39.2	23.5	8.0

## Plots:





Non-user= Residential bystander

## DCM Scenario 3. Chest, Brush-On, Workshop, User in ROH during wait time, 0.18 ACH, 0.88 Weight Fraction

### MCCEM Input Summary

# Application Method: Brush-on Volumes:

Workshop volume =  $54 \text{ m}^3$ 

ROH volume = 492 – 54 = 438 m<sup>3</sup>

## Airflows:

Workshop-outdoors	68 m <sup>3</sup> /h		
ROH-outdoors	78.8 m <sup>3</sup> /h (0.18		
	ACH)		
Workshop-ROH	65.8 m <sup>3</sup> /h		

## DCM Mass Released:

Chest = 25 sq ft surface area

Applied product mass = 90 g/sq ft = 2,250 g

DCM mass = 2,250 g × 0.88 (wt fraction) × 0.33 (release fraction) = 653.4 g

## For each of the 4 application sections:

**k** = 10/hr

**Mass** = 653.4/8 = 81.675 g

**Eo** = Mass \* k = 816.75 g/hr (**NOTE:** only k and Mass are needed as MCCEM inputs) **Application Times by Section:** 

	Elapsed time from time zero, minutes					
Episode	Apply	Wait	Scrape			
first 1/4, 1st time	0-2 min	2-17 min	17-21 min			
second 1/4, 1st time	21-23 min	23-38 min	38-42 min			
third 1/4, 1st time	42-44 min	44-59 min	59-63 min			
fourth 1/4, 1st time	63-65 min	65-80 min	80-84 min			
first 1/4, 2nd time	84-86 min	86-101 min	101-105 min			
second 1/4, 2nd time	105-107 min	107-122 min	122-126 min			
third 1/4, 2nd time	126-128 min	128-143 min	143-147 min			
fourth 1/4, 2nd time	147-149 min	149-164 min	164-168 min			

## Model Run Time:

0-24 hrs

User takes out scrapings after 168 minutes

### **Activity Patterns:**

User in workshop during application and scrape periods, in ROH during wait periods User in ROH for the remainder of the run (21 hrs, 12 minutes)

## MCCEM Results Summary

Exposure Concentrations (maximum values over first 24 hrs):

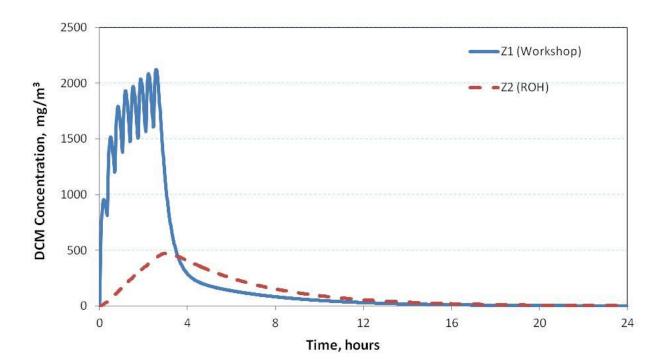
In mg/m<sup>3</sup>

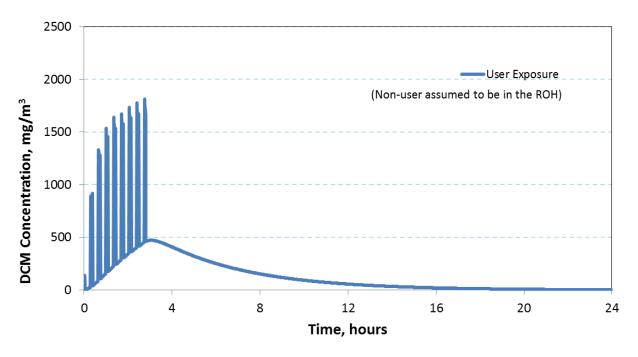
Individua							24 hrs
I	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	
User	1,810.4	1,178.1	898.3	762.4	562.8	400.1	157.1
Other	472.7	472.3	469.7	461.4	383.0	287.9	118.2

In ppm

Individual	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	24 hrs
User	521.2	339.2	258.6	219.5	162.0	115.2	45.2
Other	136.1	136.0	135.2	132.8	110.2	82.9	34.0

Plots:





Non-user= Residential bystander

# DCM Scenario 4. Coffee Table, Spray-On, Workshop, User in ROH during wait time, 0.45 ACH, 0.8 Weight Fraction

#### MCCEM Input Summary

#### Application Method: Spray-on

Volumes:

Workshop volume =  $54 \text{ m}^3$ 

ROH volume =  $492 - 54 = 438 \text{ m}^3$ 

#### Airflows:

Workshop-outdoors	68 m³/h
ROH-outdoors	197.1 m <sup>3</sup> /h (0.45 ACH)
Workshop-ROH	107 m <sup>3</sup> /h

#### DCM Mass Released:

Coffee table = 10 sq ft surface area

Applied product mass = 68 g/sq ft = 680 g

DCM mass = 680 g × 0.8 (wt fraction) × 0.66 (release fraction) = 359 g

#### For each of the 4 application sections:

**k** = 10/hr

**Mass** = 359/4 = 89.75 g

**Eo** = Mass \* k = 897.5 g/hr (**NOTE:** only k and Mass are needed as MCCEM inputs) **Application Times by Section:** 

	Elapsed time from time zero, minutes			
Episode	Apply	Wait	Scrape	
first 1/2, 1st time	0-1 min	1-16 min	16-20 min	
second 1/2, 1st time	20-21 min	21-36 min	36-40 min	
first 1/2, 2nd time	40-41 min	41-56 min	56-60 min	
second 1/2, 2nd time	60-61 min	61-76 min	76-80 min	

## Model Run Time:

0-24 hrs

User takes out scrapings after 80 minutes

## **Activity Patterns:**

User in workshop during application and scrape periods, in ROH during wait periods User in ROH for the remainder of the run (22 hrs, 40 minutes)

## **MCCEM Results Summary**

Exposure Concentrations (maximum values over first 24 hrs):

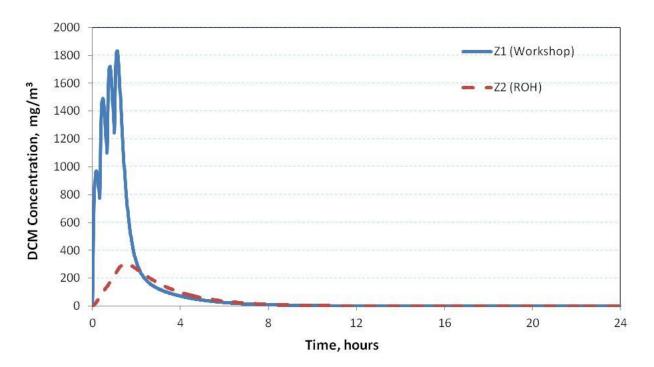
In	mg/m³
	mg/m

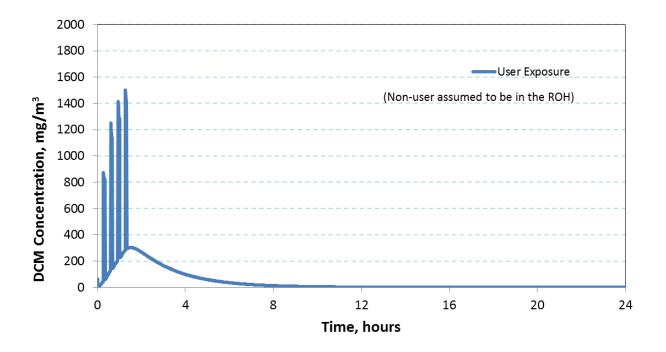
<u></u>							
Individual	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	24 hrs
User	1,502.0	781.0	598.2	491.7	266.9	152.1	51.7
Other	303.1	302.5	298.0	284.8	187.8	112.0	38.3

#### In ppm

Individual	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	24 hrs
User	432.4	224.8	172.2	141.6	76.8	43.8	14.9
Other	87.2	87.1	85.8	82.0	54.1	32.3	11.0

## Plots:





Non-user= Residential bystander

# DCM Scenario 5. Coffee Table, Spray-On, Workshop, User in workshop during wait time, 0.45 ACH, 0.87 Weight Fraction

#### MCCEM Input Summary

## Application Method: Spray-on

Volumes:

Workshop volume =  $54 \text{ m}^3$ 

ROH volume =  $492 - 54 = 438 \text{ m}^3$ 

#### Airflows:

Workshop-outdoors	68 m³/h
ROH-outdoors	197.1 m <sup>3</sup> /h (0.45 ACH)
Workshop-ROH	107 m³/h

#### DCM Mass Released:

Coffee table = 10 sq ft surface area

Applied product mass = 68 g/sq ft = 680 g

DCM mass = 680 g × 0.87 (wt fraction) × 0.66 (release fraction) = 390.5 g

#### For each of the 4 application sections:

**k** = 10/hr

**Mass** = 390.5/4 = 97.625 g

**Eo** = Mass \* k = 976.25 g/hr (**NOTE:** only k and Mass are needed as MCCEM inputs) **Application Times by Section:** 

	Elapsed time from time zero, minutes				
Episode	Apply	Wait	Scrape		
first 1/2, 1st time	0-1 min	1-16 min	16-20 min		
second 1/2, 1st time	20-21 min	21-36 min	36-40 min		
first 1/2, 2nd time	40-41 min	41-56 min	56-60 min		
second 1/2, 2nd time	60-61 min	61-76 min	76-80 min		

## Model Run Time:

0-24 hrs

User takes out scrapings after 80 minutes

## **Activity Patterns:**

User in workshop during application, wait and scrape periods User in ROH for the remainder of the run (22 hrs, 40 minutes)

# MCCEM Results Summary

Exposure Concentrations (maximum values over first 24 hrs):

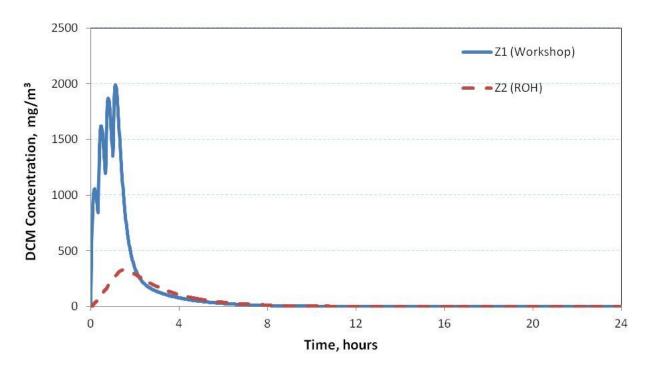
In mg/m<sup>3</sup>

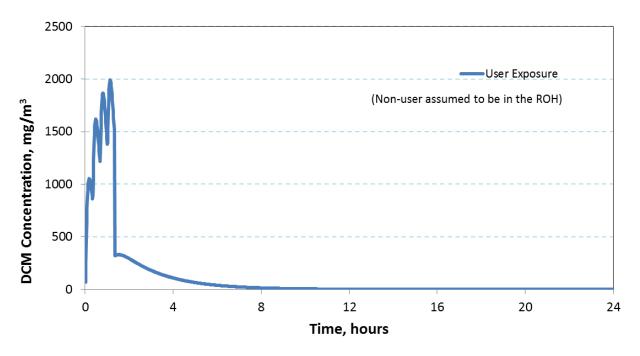
Individua							24 hrs
I	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	
User	1,991.0	1,926.4	1,760.8	1,609.4	619.4	332.4	111.9
Other	329.6	329.0	324.1	309.8	204.2	121.9	41.7

In ppm

Individual	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	24 hrs
User	573.2	554.6	506.9	463.3	178.3	95.7	32.2
Other	94.9	94.7	93.3	89.2	58.8	35.1	12.0

Plots:





Non-user= Residential bystander

## DCM Scenario 6. Chest, Spray-On, Workshop, User in ROH during wait time, 0.18 ACH, 0.87 Weight Fraction

#### MCCEM Input Summary

#### Application Method: Spray-on

Volumes:

Workshop volume =  $54 \text{ m}^3$ 

ROH volume =  $492 - 54 = 438 \text{ m}^3$ 

#### Airflows:

Workshop-outdoors	68 m³/h
ROH-outdoors	78.8 m <sup>3</sup> /h (0.18 ACH)
Workshop-ROH	65.8 m³/h

#### DCM Mass Released:

Chest = 25 sq ft surface area

Applied product mass = 68 g/sq ft = 1,700 g

DCM mass = 1,700 g × 0.87 (wt fraction) × 0.66 (release fraction) = 976.1 g

#### For each of the 8 application sections:

**k** = 10/hr

**Mass** = 976.1/8 = 122.0 g

**Eo** = Mass \* k = 1,220 g/hr (**NOTE:** only k and Mass are needed as MCCEM inputs) **Application Times by Section:** 

	Elapsed time from time zero, minutes				
Episode	Apply	Wait	Scrape		
first 1/4, 1st time	0-1 min	1-16 min	16-20 min		
second 1/4, 1st time	20-21 min	21-36 min	36-40 min		
third 1/4, 1st time	40-41 min	41-56 min	56-60 min		
fourth 1/4, 1st time	60-61 min	61-76 min	76-80 min		
first 1/4, 2nd time	80-81 min	81-96 min	96-100 min		
second 1/4, 2nd time	100-101 min	101-116 min	116-120 min		
third 1/4, 2nd time	120-121 min	121-136 min	136-140 min		
fourth 1/4, 2nd time	140-141 min	141-156 min	156-160 min		

## Model Run Time:

0-24 hrs

User takes out scrapings after 160 minutes

#### **Activity Patterns:**

User in workshop during application and scrape periods, in ROH during wait periods User in ROH for the remainder of the run (21 hrs, 20 minutes)

# MCCEM Results Summary

Exposure Concentrations (maximum values over first 24 hrs):

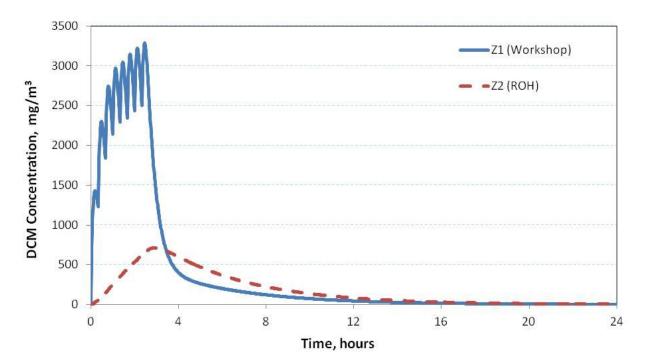
In	ma	/m³
	my,	/ ***

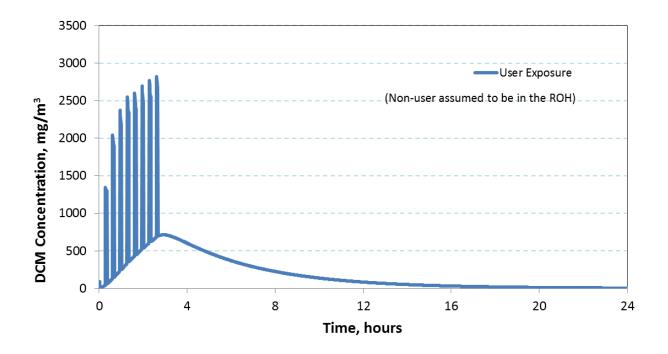
Individua							24 hrs
I	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	
User	2,821.3	1,632.3	1,266.5	1,108.6	813.6	580.2	228.2
Other	713.5	713.0	709.0	696.5	575.9	431.3	176.6

## In ppm

Individual	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	24 hrs
User	812.2	469.9	364.6	319.1	234.2	167.0	65.7
Other	205.4	205.3	204.1	200.5	165.8	124.2	50.8

## Plots:





Non-user= Residential bystander

## DCM Scenario 7. Bathtub, Brush-On, Bathroom + Source Cloud, User in ROH During Wait Time, 0.18 ACH, 1.0 Weight Fraction

#### MCCEM Input Summary

# Application Method: Brush-on Volumes:

Source Cloud =  $1.0 \text{ m}^3$ Rest of Bathroom volume =  $9 - 1 = 8 \text{ m}^3$ ROH volume =  $492 - 9 = 483 \text{ m}^3$ 

#### Airflows:

Source Cloud-Bathroom	80 m <sup>3</sup> /h (0 to outdoors/ROH)
Bathroom-outdoors	1.6 m³/h
ROH-outdoors	86.9 m³/h (0.18 ACH)
Bathroom-ROH	35 m³/h

#### DCM Mass Released:

Tub = 36 sq ft surface area

Applied product mass = 90 g/sq ft = 3,240 g

DCM mass = 477 g × 1.0 (wt fraction) × 0.33 (release fraction) = 157.4 g

#### For each of the 8 application sections:

**k** = 10/hr

**Mass** = 157.4/8 = 19.7 g

Eo = Mass \* k = 197 g/hr (NOTE: only k and Mass are needed as MCCEM inputs)

#### Application Times by Section:

	Elapsed time from time zero, minutes			
Episode	Apply	Wait	Scrape	
first 1/4, 1st time	0-3 min	3-18 min	18-24 min	
second 1/4, 1st time	24-27 min	27-42 min	42-48 min	
third 1/4, 1st time	48-51 min	51-66 min	66-72 min	
fourth 1/4, 1st time	72-75 min	75-90 min	90-96 min	
first 1/4, 2nd time	96-99 min	99-114 min	114-120 min	
second 1/4, 2nd time	120-123 min	123-138 min	138-144 min	
third 1/4, 2nd time	144-147 min	147-162 min	162-168 min	
fourth 1/4, 2nd time	168-171 min	171-186 min	186-192 min	

#### Model Run Time:

0-24 hrs

User takes out scrapings after 192 minutes

#### **Activity Patterns:**

User in source cloud during application and scrape periods, in ROH during wait periods User in ROH for the remainder of the run (20 hrs, 48 minutes)

#### **MCCEM Results Summary**

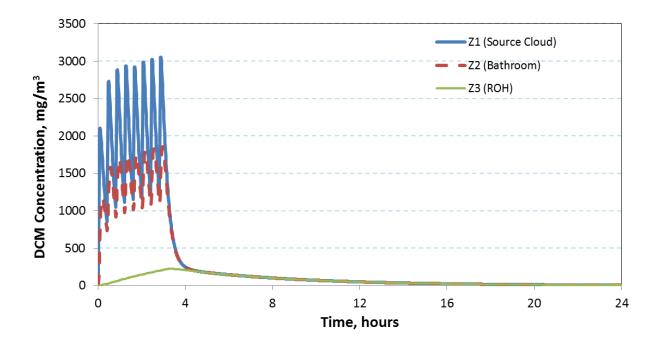
Exposure Concentrations (maximum values over first 24 hrs):

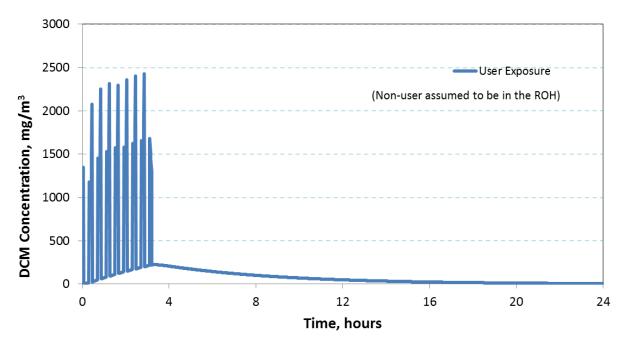
Individua I	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	24 hrs
User	2428.0	1455.3	886.6	798.9	536.4	339.6	135.3
Other	223.8	223.6	222.2	218.2	186.9	149.5	69.5

In ppm

Individual	1 min	10 min	30 min	1 hr	4 hrs	8 hrs	24 hrs
User	699.0	419.0	255.3	230.0	154.4	97.8	39.0
Other	64.4	64.4	64.0	62.8	53.8	43.0	20.0

Plots:





Non-user= Residential bystander

# Appendix I RISK ASSESSMENT GUIDELINES, LITERATURE SEARCH STRATEGY AND DATA QUALITY CRITERIA USED IN THE HAZARD/DOSE-RESPONSE ASSESSMENTS OF METHYLENE CHLORIDE

EPA/OPPT's work plan risk assessment for dichloromethane (DCM; methylene chloride) is based on the peer-reviewed hazard and dose-response information published in the following reports:

- *Toxicological Review of Methylene Chloride* published in 2011 by the EPA's Integrated Risk Information System (IRIS) (EPA, 2011c);
- Interim Acute Exposure Guideline Levels (AEGL) for methylene chloride (NAC, 2008);
- Spacecraft Maximum Allowable Concentrations (SMAC) for Selected Airborne Contaminants: Methylene chloride (Volume 2) published by the U.S. National Academies (NRC, 1996);
- Acute Reference Exposure Level (REL) and Toxicity Summary for Methylene Chloride published by the Office of Environmental Health Hazard Assessment (OEHHA, 2008).

The sections below contain a summary of the risk assessment guidelines, literature search strategy and data quality criteria used in these assessments.

# I-1 EPA/IRIS Toxicological Review

## *I-1-1 Risk Assessment Guidelines*

The description below was extracted from the DCM IRIS assessment published in November 2011 (<u>EPA, 2011c, pages 1-2</u>).

Development of these hazard identification and dose-response assessments for DCM has followed the general guidelines for risk assessment as set forth by the National Research Council (NRC) (<u>NRC, 1983</u>). EPA's Guidelines and Risk Assessment Forum Technical Panel Reports that may have been used in the development of the DCM IRIS assessment include the following:

- **1.** Guidelines for the Health Risk Assessment of Chemical Mixtures (<u>EPA, 1986b</u>);
- 2. Guidelines for Mutagenicity Risk Assessment (EPA, 1986a);
- **3.** Recommendations for and Documentation of Biological Values for Use in Risk Assessment (<u>EPA, 1988</u>);
- **4.** Guidelines for Developmental Toxicity Risk Assessment (<u>EPA, 1991</u>);
- **5.** Interim Policy for Particle Size and Limit Concentration Issues in Inhalation Toxicity (<u>EPA, 1994b</u>);
- **6.** Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (<u>EPA, 1994c</u>);
- 7. Use of the Benchmark Dose Approach in Health Risk Assessment (EPA, 1995b);

- 8. Guidelines for Reproductive Toxicity Risk Assessment (EPA, 1996b);
- 9. Guidelines for Neurotoxicity Risk Assessment (EPA, 1998a);
- **10.** Science Policy Council Handbook: Risk Characterization (EPA, 2000c);
- **11.** Benchmark Dose Technical Guidance Document (<u>EPA, 2000b</u>, <u>2012a</u>);
- **12.** Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures (EPA, 2000d);
- 13. A Review of the Reference Dose and Reference Concentration Processes (EPA, 2002)
- 14. Guidelines for Carcinogen Risk Assessment (EPA, 2005a);
- **15.** Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens (<u>EPA, 2005b</u>);
- **16.** Science Policy Council Handbook: Peer Review (EPA, 2006b);
- **17.** A Framework for Assessing Health Risks of Environmental Exposures to Children (<u>EPA,</u> <u>2006a</u>).

# I-1-2 Literature Search Strategy

When developing the DCM IRIS assessment, the literature search strategy was based on the chemical name, Chemical Abstracts Service Registry Number (CASRN), and multiple common synonyms (<u>EPA, 2011c</u>). Any pertinent scientific information submitted by the public to the IRIS Submission Desk was also considered in the development of this document.

Primary, peer-reviewed literature identified through September 2011 was included where that literature was determined to be critical to the assessment. The relevant literature included publications on DCM which were identified through Toxicology Literature Online (TOXLINE), the U.S. National Library of Medicine's MEDLINE, the Toxic Substance Control Act Test Submission Database (TSCATS), the Registry of Toxic Effects of Chemical Substances (RTECS), the Chemical Carcinogenesis Research Information System (CCRIS), the Developmental and Reproductive Toxicology/Environmental Teratology Information Center (DART/ETIC), the Hazardous Substances Data Bank (HSDB), the Genetic Toxicology Data Bank (GENE-TOX), Chemical abstracts, and Current Contents. Other peer-reviewed information, including health assessments developed by other organizations, review articles, and independent analyses of the health effects data were retrieved and included in the assessment when appropriate (EPA, 2011c).

# I-1-3 Study Selection and Data Quality Criteria

The following study selection and data quality criteria were used by the EPA's IRIS program when developing the DCM IRIS assessment (<u>EPA, 2011c</u>). In addition, EPA/OPPT uses these criteria when evaluating hazard/dose-response studies for chemical assessments.

• *Epidemiology data*: Study quality evaluation criteria include a review of factors such as the study selection criteria, study power, potential bias in data collection, selection bias, measurement biases associated with exposure and outcome, and consideration of potential confounding and effect modification (Figure I-1).

- Animal toxicology data: Study quality evaluation criteria for animal studies (i.e., *in vivo* and *in vitro*) include a review of factors such as the following:
  - the adequacy of study design,
  - test animals (e.g., species, strain, source, sex, age/lifestage/embryonic stage),
  - environment (e.g., husbandry, culture medium),
  - test substance (e.g., identification, purity, analytical confirmation of stability and concentration),
  - treatment (e.g., dose levels, controls, vehicle, group sizes, duration, route of administration),
  - endpoints evaluated (e.g., schedule of evaluation, randomization and blinding procedures, assessment methods), and
  - reporting (quality and completeness) (Figure I-2).

#### Figure I-1. Study Quality Considerations for Epidemiological Studies

F	eature	Example Questions
	Participants	<ul> <li>Were inclusion and exclusion criteria applied consistently across study groups?</li> </ul>
Selection		• Are baseline characteristics similar between groups? If not, did the analysis control for differences?
	Comparability	<ul> <li>Is the comparison group appropriate, including having both exposed and non-exposed subjects drawn from the same population:</li> </ul>
	Attrition Rate	Was the attrition rate uniformly low?
		<ul> <li>In cohort studies: Dose the length of follow-up differ between groups?</li> </ul>
Attrition	Length of Follow-Up	<ul> <li>In case-control studies: Is the time period between exposure between exposure/intervention and outcome the same for cases and controls?</li> </ul>
		<ul> <li>Was follow-up long enough to assess the outcome of interest?</li> </ul>
		<ul> <li>What is the level of exposure misclassification?</li> </ul>
	Exposure Characteristics	<ul> <li>Is there an adequate level of exposure variability to detect an effect?</li> </ul>
		<ul> <li>Are there adequate numbers of persons exposed at various exposure levels to detect a dose-response effect?</li> </ul>
		• What is the extent of reliance on imputed exposure levels?
Detection	Outcome	<ul> <li>Were the outcome assessors blinded to the exposure or intervention status of participants?</li> </ul>
	Assessment	<ul> <li>Is there confidence that the outcome of interest preceded exposure?</li> </ul>
	Confounding Variables or	<ul> <li>Are confounding variables assessed using reliable and consistent measures?</li> </ul>
	Exposure	<ul> <li>Did researchers adjust or control for other exposures or interventions that are anticipated to bias results?</li> </ul>
	Statistical Tests	<ul> <li>Are statistical analyses performed with reliable tests and implemented consistently?</li> </ul>

Feature	Example Questions							
Feature	Were the exposures well designed and tightly controlled?	<ul> <li>Inhalation exposure: Was the chamber type appropriate?</li> </ul>						
	<ul> <li>Was the test article/formulation adequately identified and characterized? Are co-exposures expected as a result of test article composition?</li> </ul>	<ul> <li>Dynamic chambers should be used; static chambers are not recommended.</li> <li>Inhalation exposure: Were appropriate methods used to generate the test article and measure the analytical concentration?</li> </ul>						
Exposure Quality	<ul> <li>Is the administration route relevant to human exposure?</li> <li>Are the exposure levels relevant?</li> </ul>	• Diet/Water Exposure: Was consumption measured to allow for accurate dose determinations? Were stability and homogeneity of the test substance maintained? Was palatability an issue?						
	<ul> <li>Inhalation exposure: Were analytical concentrations in the test animals' breathing zone measured and reported (i.e., not just target or nominal concentrations)?</li> <li>Inhalation exposure: For aerosol studies, were the mass median aerodynamic diameter and geometric standard dividual concentration</li> </ul>	• Gavage Exposure: Was an appropriate vehicle used? Are there any toxicokinetic differences due to bolus dosing? Consider relevance to human exposures.						
	<ul><li>deviation reported?</li><li>Were the test animals appropriate for evaluation of the specified effect(s)?</li></ul>	•Were an appropriate number of animals examined, based on what is known about the particular endpoint(s) in question?						
Test Animals	<ul> <li>Were the species, strain, sex, and/or age of the test animals appropriate for the effect(s) measured?</li> </ul>	•Were there any notable issues regarding animal housing or food and water consumption?						
	• Were the control and exposed populations matched in all aspects other than exposure?							
	<ul> <li>Is the study design appropriate for the effect(s) and chemical analyzed?</li> <li>Were exposure frequency and duration appropriate for</li> </ul>	<ul> <li>Was it designed according to established guidelines (e.g., EPA, OECD)? Was it designed to specifically test the endpoint(s) in question?</li> </ul>						
Study Design	<ul> <li>Were exposure inequency and duration appropriate for the effect(s) measured?</li> <li>Were anticipated confounding factors caused by selection bias controlled for in the study design (e.g.,</li> </ul>	• Did the study design include other experimental procedures (e.g surgery) that may influence the results of the toxicity endpoint(s in question? Were they controlled for?						
	correction for potential litter bias; randomization of treatment groups)?	• Was the study design able to detect the most sensitive effects in the most sensitive population(s)?						
	<ul> <li>Was the timing of the endpoint evaluation (e.g., latency from exposure) appropriate?</li> <li>Was it a Good Laboratory Practices (GLP) study?</li> </ul>	•Were multiple exposure groups tested? Was justification for exposure group spacing given? Was recovery or adaptation tested?						
	<ul> <li>Are the protocols used for evaluating a specific endpoint reliable and the study endpoints chosen</li> </ul>	•Were all necessary control experiments performed to allow for selective examination of the endpoint in question?						
Toxicity	relevant to humans? •Are the endpoints measured relevant to humans? Do the endpoints evaluate an adverse effect on the health	<ul> <li>As appropriate, were steps taken to minimize experimenter bias (e.g., blinding)?</li> <li>Does the methodology employed represent the most appropriate</li> </ul>						
Endpoints	<ul> <li>outcome in question?</li> <li>Were the outcomes evaluated according to established protocols? If not work the approaches historically.</li> </ul>	and discriminating option for the chosen endpoint?						
	protocols? If not, were the approaches biologically sound? Were any key protocol details omitted?							
Data	<ul> <li>Were statistical methods and presentation of data sufficient to accurately define the direction and magnitude of the observed effect(s)?</li> </ul>	• Does the data present pooled groups that should be displayed separately (e.g., pooled exposure groups; pooled sexes) and/or analyzed separately?						
Data Presentation and Analysis	<ul> <li>Are the statistical methods and comparisons appropriate?</li> </ul>	•Was an unexpectedly high/low level of within-study variability and/or variation from historical measures reported or explained?						
	•Was sufficient sampling performed to detect a biologically relevant effect (e.g., appropriate number of slides examined)?	<ul> <li>As appropriate, were issues such as systemic and maternal toxicity (e.g., body weight) considered?</li> </ul>						
	•Are descriptions of study methods and results for all endpoints sufficient to allow for study quality	• Are the statistical methods applied for data analysis provided and applied in a transparent manner? Was variability reported?						
	<ul><li>•Were the details of the exposure protocols and</li></ul>	• Did the study evaluate a unique cohort of animals (i.e., are multiple studies linked)?						
Reporting	equipment provided? •Were test animal specifics adequately presented?	• Are group sizes and results reported quantitatively for each						
	<ul> <li>Were test animal specifics adequately presented?</li> <li>Are the protocols for all study endpoints clearly described? Is sufficient detail provided to reproduce the experiment(s)?</li> </ul>	exposure group, time-point, and endpoint examined?						

# Figure I-2. Study Quality Considerations for Animal Studies

# I-2 Acute Exposure Guideline Levels (AEGLs)

AEGLs are emergency response guideline levels for once-in-a-lifetime short-term exposures to airborne chemicals (*see Appendix K for more details*). AEGL are developed based on the procedures, methods and criteria documented in the *Standing Operating Procedures (SOP) for Developing Acute Exposure Guideline Levels for Hazardous Chemicals* (NRC, 2001). Specifically, the AEGL SOP contains the following information supporting the derivations of AEGLs, including those for DCM (NRC, 2001):

- Empirical toxicological endpoints and methods for determining exposure concentrations used to derive AEGLs 1,2, and 3 (*Chapter 2.2 of the AEGL SOP*);
- Guidelines and criteria for the search strategy, evaluation, selection, and documentation of key data and supporting data used for the derivation of AEGL values (*Chapter 2.3 of the AEGL SOP*);
- Dosimetry corrections from animal to human exposures (*Chapter 2.4 of the AEGL SOP*)
- Guidelines and criteria for selection of uncertainty factors to address the variability between animals and humans and within the human population (*Chapter 2.5 of the AEGL SOP*);
- Guidelines and criteria for time scaling (*Chapter 2.7 of the AEGL SOP*).

# I-3 Spacecraft Maximum Allowable Concentrations (SMACs)

SMACs are guideline levels intended for spacecraft chemical exposures and developed following the criteria and methods described in *Guidelines for Developing Spacecraft Maximum Allowable Concentrations for Space Station Contaminants* (<u>NRC, 1992</u>). Chapter 6 of the *SMAC Guidelines* contains information about the derivation of the SMAC values, including the following (<u>NRC, 1992</u>):

- Sources of data for developing SMACs (i.e., chemical-physical properties, in vitro studies, animal toxicity studies, epidemiological data),
- Types of data used in recommending SMACs (i.e., dosimetry, pharmacokinetics and metabolism, biological markers and toxicity endpoints in humans and animals),
- Risk assessment (e.g., issues about animal to human extrapolation), and
- General approach to establishing SMACs.

# I-4 California's Acute Reference Exposure Levels (RELs)

An acute REL is defined as the concentration level at or below which no adverse health effects are anticipated (*i.e.*, 1 or 8 hrs) in a human population, including sensitive subgroups, exposed on an intermittent basis (<u>OEHHA</u>, <u>1999</u>). The Office of Environmental Health Hazard Assessment (OEHHA) from the State of California has developed guidance on how to develop acute RELs including guidance on the appropriate exposure durations and patterns for acute exposure, hazard identification and dose-response, criteria for selecting key studies and identifying adverse health effects, time extrapolation and characterization of uncertainties (<u>OEHHA</u>, <u>1999</u>).

# Appendix J SUMMARY OF THE DERIVATIONS OF THE EPA IRIS CANCER INHALATION UNIT RISK AND NON-CANCER HUMAN EQUIVALENT CONCENTRATION FOR CHRONIC EXPOSURES

The reader is referred to the DCM IRIS assessment for detailed explanations of the toxicological studies and the derivation approaches supporting the cancer inhalation unit risk and the non-cancer hazard value associated with chronic exposures to DCM (<u>EPA, 2011c</u>).

# J-1 Cancer Inhalation Unit Risk

DCM's cancer inhalation unit risk (IUR) of  $4 \times 10^{-5}$  per ppm  $(1 \times 10^{-5} \text{ per mg/m}^3)^{51}$  was derived from mouse liver and lung tumor incidence data (<u>Mennear et al., 1988</u>; <u>NTP, 1986</u>). Figure J-1 describes the steps that the EPA's IRIS program used to derive the DCM IUR using physiologically-based pharmacokinetic (PBPK) modeling. Refer to the DCM IRIS assessment for a full discussion of the IUR derivation (<u>EPA, 2011c</u>).

The derivation steps are the following:

- Dose conversion: A deterministic mouse PBPK model was used to convert the mouse inhalation exposures to long-term daily average internal doses in the liver or lung. The selected internal dose-metric was long-term average daily mass of DCM metabolized *via* the GST pathway per unit volume of liver or lung tissue. The choice of the dose metric was based on evidence related to the involvement of the GST metabolites in DCM-induced carcinogenicity (EPA, 2011c).
- Dose-response modeling and extrapolation: The multistage dose-response model (Benchmark Dose Software [BMDS] version 2.0) was used to fit the mouse liver tumor incidence and PBPK-derived internal doses and derive a mouse internal BMD<sub>10</sub> and BMDL<sub>10</sub><sup>52</sup> associated with 10% extra risk (EPA, 2011c).

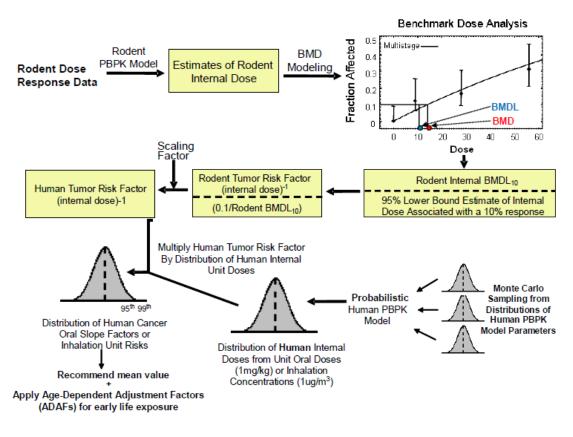
The mouse internal BMDL<sub>10</sub> (0.1/BMDL10) were used to derive inhalation risk factors for lung and liver tumors by linear extrapolation. Consistent with EPA *Guidelines for Carcinogen Risk Assessment* (EPA, 2005a), a linear low-dose extrapolation approach is used for chemicals with DNA-reactive and mutagenic properties (EPA, 2011c).

<sup>&</sup>lt;sup>51</sup> The inhalation unit risk for dichloromethane should not be used with exposures exceeding the point of departure (BMDL<sub>10</sub> = 7,700 mg/m<sup>3</sup> or 2,200 ppm), because above this level the fitted dose-response model better characterizes what is known about the carcinogenicity of dichloromethane.

<sup>&</sup>lt;sup>52</sup> The benchmark dose (BMD) is a dose or concentration that produces a predetermined change in response rate of an adverse effect (called the benchmark response or BMR) compared to background (<u>EPA, 2011b</u>). BMD<sub>10</sub>= benchmark dose at the 10% response

 $<sup>\</sup>mathsf{BMDL}_{10}$ =lower confidence limit of the benchmark dose at the 10% response





Source: EPA (2011c, p. 212)

3. Application of allometric scaling factor: The chosen dose metrics is a rate of metabolism rather than the concentration of putative toxic metabolites. Currently, there are no data pertaining to the reactivity or clearance rate of the relevant metabolite(s). A scaling factor was used to address the possibility that the rate of clearance for the metabolite is limited by processes that are known to scale allometrically. The human BMDL<sub>10</sub> was derived by applying a mouse:human dose-rate scaling factor of 7 [i.e., (Body Weight human/Body Weight mouse)<sup>0.25</sup> = 7] to adjust the mouse-based BMDL<sub>10</sub> values downward based on the potential slower clearance per volume tissue in the human compared with the (EPA, 2011c).

A linear extrapolation approach using the internal human BMDL<sub>10</sub> for liver and lung tumors was used to calculate human tumor risk factors by dividing the benchmark response (BMR) of 0.1 by the human BMDL for each tumor type, given a 70-year lifetime exposure (EPA, 2011c). Currently, there are no data from chronic inhalation cancer bioassays in mice or rats providing support for a nonlinear dose-response relationship (EPA, 2011c).

- 4. Calculation of the inhalation unit risk and consideration of sensitive human
  - **subpopulations:** A probabilistic human PBPK model with Monte Carlo sampling was used to determine a distribution of human internal doses lung, liver, or blood doses associated with chronic unit inhalation  $(1 \ \mu g/m^3)$  exposures. The distribution of IURs was derived by multiplying the human inhalation tumor risk factors by the respective distributions of human average daily internal doses resulting from chronic, unit inhalation exposures of one  $\mu g/m^3$  DCM. The mean of the distribution of candidate IUR values from the most sensitive (GST-T1<sup>+/+</sup>) genotype (*i.e.*, the group that would be expected to be most sensitive to the carcinogenic effects of DCM) was chosen as the IUR for liver and lung tumors. A procedure for combining risks for liver and lung tumors was used to derive DCM's IUR.

The slope of the linear extrapolation from the lower 95 percent bound estimate  $BMDL_{10}$  is  $1 \times 10^{-8}$  per  $\mu$ g/m<sup>3</sup> (4 x 10<sup>-5</sup> per ppm ), which represents an upper-bound estimate for continuous lifetime exposure (70 years) without consideration of increased early-life susceptibility due to DCM's mutagenic mode of action.

# J-2 Non-Cancer Hazard Value

The EPA's IRIS program based the non-cancer hazard value for DCM on liver effects. These effects were reported in female rats exposed to DCM for 6 hrs/day, 5 days/week for 2 years (<u>Nitschke et al., 1988a</u>). The rat data were suitable for non-cancer dose-response analysis in the DCM IRIS assessment.

Since the study was suitable for dose-response analysis, the EPA's IRIS program used a PBPK model to estimate rat internal doses from the <u>Nitschke et al. (1988a)</u> study. Benchmark dose modeling used the rat internal doses and their corresponding incidence data (i.e., hepatic vacuolation) to estimate the rat internal BMDL<sub>10</sub> for hepatic effects. In other words, the BMDL<sub>10</sub> is the lower 95% confidence limit of the benchmark dose at the 10% benchmark response (BMR) (<u>EPA, 2011c</u>). A BMR of 10% was selected because, in the absence of information regarding the magnitude of change in a response that is thought to be minimally biologically significant, a BMR of 10% is generally recommended since it provides a consistent basis of comparison across assessments. Moreover, there were no additional data to suggest that the severity of the critical effect or the power of the study would warrant a lower BMR (<u>EPA, 2011c</u>).

The rat internal BMDL<sub>10</sub> was allometrically adjusted because the dose-metric is a rate of metabolism and the clearance of these metabolites may be slower per volume tissue in the human compared with the rat. This adjustment consisted of dividing the rat internal BMDL<sub>10</sub> by 4.09 [(BW<sub>human</sub>)/(BW<sub>rat</sub>)<sup>0.25</sup>  $\approx$  4.09)]<sup>53</sup> to obtain a human equivalent internal BMDL<sub>10</sub> of 130.03 mg DCM metabolized via CYP<sup>54</sup> pathway/litter liver tissue/day (EPA, 2011c).

<sup>&</sup>lt;sup>53</sup> BW=body weight

<sup>&</sup>lt;sup>54</sup> CYP=cytochrome P450

A probabilistic PBPK model for dichloromethane in humans was then used with Monte Carlo sampling to calculate distributions of chronic human equivalent concentrations (HEC) (in units of mg/m<sup>3</sup>) associated with the internal BMDL<sub>10</sub> based on the responses in female Sprague-Dawley rats. Estimated HECs corresponding to the mean, 1<sup>st</sup>, and 5<sup>th</sup> percentiles of the distribution were 48.5, 17.2 and 21.3 mg/m<sup>3</sup>, respectively. The 1<sup>st</sup> percentile of the distribution of HECs i.e. the HEC<sub>99</sub> the concentration at which there is 99% likelihood an individual would have an internal dose less than or equal to the internal dose of hazard, 17.2 mg/m<sup>3</sup>, was chosen as the point of departure (POD)<sup>55</sup> for the non-cancer hazard value because it would protect toxicokinetically sensitive individuals (EPA, 2011c).

<sup>&</sup>lt;sup>55</sup> A point of departure (POD) is a dose or concentration that can be considered to be in the range of observed responses, without significant extrapolation. A POD is used to mark the beginning of extrapolation to determine risk associated with lower environmentally relevant human exposures (EPA, 2011b).

# Appendix K THE DERIVATIONS OF THE ACUTE HAZARD VALUES USED IN THE DCM RISK ASSESSMENT OF PAINT STRIPPERS

The reader is referred to the U.S. National Academies' *Spacecraft Maximum Allowable Concentrations for Selected Airborne Contaminants* (Volumes 2 and 5; <u>NRC, 2008, 2010</u>), the *Acute Reference Exposure Level (REL) and Toxicity Summary for Methylene Chloride* from the State of California, and the *Interim Acute Exposure Guideline Levels* technical support document (<u>NAC, 2008</u>) for detailed explanations of toxicological studies and derivation approaches for these values.

# K-1 Spacecraft Maximum Allowable Concentrations (SMAC)

SMACs are developed by the U.S. NAS to provide guidance on chemical exposures that may occur during normal operations of spacecraft as well as emergency situations (<u>NRC, 1996</u>). EPA/OPPT used the SMAC's dose-response assessment as the starting point for deriving protective air concentrations for residential users of DCM-based paint strippers as well as other residential occupants that may be indirectly exposed (*e.g.*, children).

The 1-hr SMAC is the concentration of DCM which it is not expected to compromise the performance of specific tasks by healthy astronauts during emergency conditions or cause serious or permanent toxic effects. SMACs are designed for healthy individuals, and reversible effects might occur but they are not expected to impair the astronauts' judgment or interfere with proper responses to emergencies. By definition, the SMACs are not safe levels and are not meant to protect the general population, including children and the elderly.

The following paragraphs were extracted from SMAC technical support document for DCM and explain the dose-response evaluation leading to the selection of 100 ppm (350 mg/m<sup>3</sup>) as the point of departure (POD) for the 1-hr SMAC (<u>NRC, 1996</u>):

"...one of the major acute effects of methylene chloride is CNS depression, which appears to be due to carbon monoxide (CO) formed from methylene chloride's metabolism. A 4-hr exposure to methylene chloride at 200 ppm, which yields 5% carboxyhemoglobin (COHb) in blood, impairs the hand-eye coordination and auditory vigilance (<u>Peterson, 1978</u>), but there are no data on the no-observed-adverse-effect level (NOAEL) of methylene chloride. It makes sense to adopt the NOAEL of COHb used in setting the 1-h and 24-h SMACs of CO as a potential basis for setting the 1-h and 24-h SMACs of methylene chloride.

Three percent COHb is the target COHb concentration used to set both the 1-h and 24-h SMACs for carbon monoxide (<u>Wong, 1990</u>). The task here is to determine the methylene

chloride concentrations that produce about 3% COHb in 1 and 24 hr. Assuming a baseline COHb concentration of 0.6% due to endogenous CO production, the task is to determine the methylene chloride concentrations that would increase the COHb concentration by 2.4%.

To derive the 1-hr SMAC based on CO formation, a linear regression line was fitted through the data of percent COHb increase versus concentration x time (C x T) by forcing the fitted line through the origin. All the data in the above table [table not included in Appendix I-see NRC (1996)] were used except the data points at 3750 and 1972 ppm-hr because their corresponding responses of 10% and 9.3% increases in COHb were too far away from the region of interest, 2.4%. The linear regression yielded a line with a slope of 0.0038, r2 of 0.74, and a 95 % confidence limit of 100 ppm-hr at a 2.4% increase in COHb. Accordingly, 100 ppm is selected as the 1-hr SMAC based on CO formation (NRC, 1996)."

The value of 100 ppm (350 mg/m<sup>3</sup>) was considered a NOAEL<sup>56</sup> for CNS effects associated with COHb formation and is used as the POD for acute inhalation risk estimates (<u>NRC, 2008</u>). The application of UFs was not included in the derivation of the 1-hr SMAC value, which is consistent with the intended purpose of the SMAC values.

# K-2 California's Acute Reference Exposure Level (REL)

Acute RELs are developed by the Office of Environmental Health Hazard Assessment (OEHHA) from the State of California. The acute REL is defined as the concentration level at or below which no adverse health effects are anticipated (*i.e.*, one or eight hrs) in a human population, including sensitive subgroups, exposed on an intermittent basis (OEHHA, 1999). Since safety factors are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact (OEHHA, 1999).

OEHHA developed a 1-hr acute REL based on (Putz et al., 1979). The study reported significant performance decrements on dual-task and auditory vigilance tests in volunteers (n = 12) exposed to 195 ppm DCM (696 mg/m<sup>3</sup>) for 1.5 hrs (Putz et al., 1979). The blood COHb levels increased from 1.35 percent pre-exposure to 5.1 percent post-exposure. The 1.5-hr exposure to 195 ppm was considered the LOAEL in the REL derivations.

A UF of 6 was applied to the LOAEL<sup>57</sup> to develop a NOAEL and an intraspecies UF of 10 was applied to account for variability in the human population. An equivalent 1-hr exposure was estimated from the 1.5-hr exposure using the ten Berge equation ( $C^n * T = k, n = 2$ )<sup>58</sup> resulting in

<sup>&</sup>lt;sup>56</sup> NOAEL= No-observed-adverse-effect level

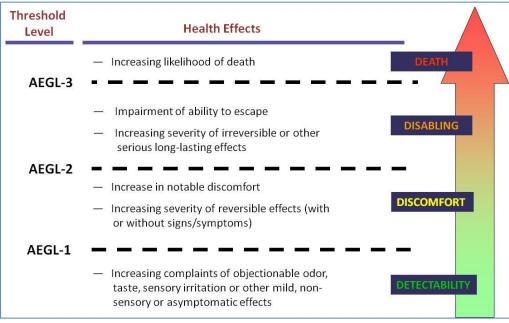
<sup>&</sup>lt;sup>57</sup> The acute REL documentation does not provide the basis for the selection of a LOAEL-to-NOAEL UF of 6.

<sup>&</sup>lt;sup>58</sup> In the ten Berge equation ( $C^n * T = k$ , n = 2), C = concentration of the chemical of interest, n=chemical-specific exponent, t=time, and k=constant (NRC, 2001).

a 1-hr acute REL of 4 ppm (14 mg/m<sup>3</sup>). The rationale for the choice of n = 2 was not documented in the OEHHA document.

# K-3 Acute Exposure Level Guidelines (AEGL)

AEGLs represent threshold exposure limits for the general public, including infants and children as well as other individuals who may be sensitive or susceptible. They are applicable for oncein-a-lifetime acute exposures (*i.e.*, ≤24 hrs) to airborne concentrations of toxic chemicals typically occurring during emergency response situations. AEGL values are developed for three different health effect end point tiers (discomfort = AEGL-1 threshold; disability = AEGL-2 threshold; and death = AEGL-3 threshold) at different durations of exposure (10 minutes; 30 minutes; 1 hr; 4hrs; and 8 hrs). Figure K-1 depicts the three AEGL tiers and the associated health effects.



# Figure K- 1. Illustration of the Different AEGL Threshold Levels



Two toxic endpoints were of importance for setting the AEGL-values for DCM: (1) CNS depression caused by the concentration of the parent compound in brain and (2) the formation of carboxyhemoglobin (COHb) from the carbon monoxide (CO) metabolite. A GST isozyme is responsible for the metabolic pathway yielding CO. It is estimated that 20 percent of the U.S. population lack this enzyme resulting in higher COHb levels in enzyme-deficient individuals (NAC, 2008).

CNS effects are expected to occur soon after the onset of exposure, while peak levels of COHb can be reached hours after cessation of exposure. Likewise, it was expected that the toxic

endpoint of interest would change over an exposure range of 10 minutes to 8 hrs. The AEGLvalues for the shorter exposure durations would be triggered by the CNS effects, whereas the formation of COHb would determine the AEGL values for longer exposure durations (<u>NAC</u>, <u>2008</u>).

The following derivation summaries were extracted from the AEGL document for DCM (<u>NAC</u>, <u>2008</u>).

## AEGL-1 values

The AEGL-1 values were based on the observation in humans that exposure concentrations of 868 and 986 ppm (3,100 and 3,521 mg/m<sup>3</sup>) may lead to light-headedness and difficulties in enunciation (Stewart et al., 1972). These effects were absent at a 1-hr exposure to 514 or 515 ppm (1,836 or 1,839 mg/m<sup>3</sup>). The concentration of 514 ppm was used as the POD for the AEGL-1 derivations. These effects could be attributed to the DCM concentration in the brain rather than to CO (NAC, 2008). A PBPK model estimated that 0.063 mM was the human brain concentration of DCM following a 1-hr exposure to 514 ppm. Since susceptibility for gross CNS-depressing effects do not vary by more than a factor of two- to three-fold in humans, an intraspecies UF of three was applied, resulting in a maximum target concentration to estimate the AEGL values for the different time durations (*i.e.*, 10 minutes to 8 hrs). Because the calculated AEGL-1 values at 4- and 8- hrs (160 and 140 ppm, respectively) were at or above the corresponding AEGL-2 values, no AEGL-1 values for these time periods were recommended (NAC, 2008).

## AEGL-2 values

AEGL-2 derivations were estimated for CNS effects based on a human study reporting the absence of AEGL-2 related CNS effects<sup>59</sup> during a DCM exposure to 751 ppm (2,682 mg/m<sup>3</sup>) for 230 minutes (Winneke, 1974). A PBPK model estimated that 0.137 mM was the human brain concentration of DCM. AEGL-2 values were also estimated for the formation of COHb formation, assuming a maximum COHb level of 4% in patients with coronary artery disease humans (NAC, 2008; NRC, 2010). PBPK modeling was used to calculate exposure concentrations for both types of effects (*i.e.*, CNS effects and COHb formation). The lowest value was selected as the AEGL-2 value for each time period. An intraspecies UF of 1 for the CNS effects was considered sufficient since the toxic effects studied were less severe than those defined for AEGL-2 and the application of a greater value would result in values that were inconsistent with the available human data. Similarly, an intraspecies UF of 1 was applied for the effects associated with COHb formation because the POD was based on experimental data on the most susceptible individuals (*i.e.*, coronary artery disease patients), which is also protective for other human subpopulations (NAC, 2008; NRC, 2010).

<sup>&</sup>lt;sup>59</sup> This is an effect level and should not be considered as a NOAEL.

#### AEGL-3 values

AEGL-3 derivations were based on an animal study reporting no CNS-related mortality in rats exposed to 11,000 ppm (39,286 mg/m<sup>3</sup>) DCM for 4-hrs (DuPont, 1982). A rat PBPK model estimated that 3.01 mM was the maximum target DCM concentration in rat brain. AEGL-2 values were also estimated for the formation of COHb formation, assuming a maximum COHb level of approximately 15 percent in humans (NRC, 2010). PBPK modeling was used to calculate exposure concentrations for both types of effects. The lowest value was selected as the AEGL-2 value for each time period. An interspecies UF of 1 for the CNS effects was considered to be sufficient since the differences in susceptibility regarding mortality between species appear to be very small and because a human PBPK model is used to calculate the external human exposure (NAC, 2008). An intraspecies UF of 3 was applied since the susceptibility for CNSdepressing effects is not expected to vary by more than a factor of 2- to 3-fold in the human population (NAC, 2008). Application of an overall UF of 3 results in a maximum target DCM concentration in human brain of 1.0 mM. Similarly, an intraspecies factor of 3 was applied for the effects associated with COHb formation because the POD was supported by information on effects, such as myocardial infarction and stillbirths, reported in more susceptible subpopulations (NRC, 2010).