



Australian Government

Department of Health, Disability and Ageing

Australian Industrial Chemicals Introduction Scheme

Methane, dichloro- (Dichloromethane)

Evaluation statement (EVA00188)

30 September 2026

Draft



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AICIS evaluation statement (EVA00188)

Subject of the evaluation

Methane, dichloro- (Dichloromethane)

Chemical in this evaluation

CAS name	CAS number
Methane, dichloro-	75-09-2

Reason for the evaluation

New information is available about human health risks.

Parameters of evaluation

The chemical is listed on the Australian Inventory of Industrial Chemicals (the Inventory).

This evaluation statement includes a human health risk assessment for all identified industrial uses of dichloromethane (DCM).

The chemical was previously assessed, including all endpoints, under the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework, under the former National Industrial Chemicals Notification and Assessment Scheme (NICNAS). New information has become available regarding carcinogenicity, mutagenicity, exposure standards and public health and worker risks. The previous assessment of DCM should be read in conjunction with this evaluation statement (NICNAS 2014).

Therefore, this evaluation will:

- Review the weight of evidence regarding carcinogenicity and mutagenicity.
- Determine whether amendments to the classification of these endpoints are warranted to protect workers' health and safety.
- Assess whether current regulatory controls are adequate to prevent risks to the public and workers.

Summary of evaluation

Summary of introduction, use and end use

The chemical is introduced into Australia in high volumes with an annual introduction volume around 1,500 tonnes. The chemical is primarily used as a solvent.

Based on the available Australian and international use data, the chemical is used in several commercial and domestic applications including in:

- paint strippers/removers (with concentrations up to 90% w/w identified in Australia)
- paints and coatings (including welding anti spatter sprays with concentrations ranging from 60 to < 100% identified in Australia)
- adhesives and sealants
- degreasers
- cleaning and furniture care products (solvent-based)

and site-limited functional use as a solvent or an intermediate in the manufacture of:

- plastic and polymer products
- chemical products.

Despite being banned from use in cosmetics in multiple jurisdictions, the chemical has been reported internationally as present in a range of cosmetic products with high concentrations reported in some instances. Some cosmetics that contain DCM have been reported as not identifying it as an ingredient on the cosmetic product label.

Human health

Summary of health hazards

The identified health hazards are based on available data for the chemical. Based on the physicochemical properties and the available toxicity data, the chemical is readily absorbed following inhalation and oral exposure, and to a lesser extent via the dermal route. Metabolism is a critical factor in the toxicity of DCM.

Based on the existing and previously assessed data (NICNAS 2014), the chemical:

- has low acute oral, dermal toxicity
- is irritating to skin and eyes based on animal studies, although there is evidence of more severe effects following prolonged exposure in humans
- is not likely to be a skin sensitiser
- is not expected to cause specific adverse effects on fertility/sexual function and foetal development.

Based on the calculated median lethal concentration (LC50) in animals, the chemical has low acute inhalation toxicity. However, acute inhalation exposure to the chemical produced effects on the central nervous system (CNS) in animals and humans. Neurotoxicity, neurological effects, and human mortality following inhalation exposure to high concentrations of DCM have been reported.

In rats and mice, hepatotoxicity was the primary non-cancer effect observed following repeated oral exposure to DCM in short-term, subchronic, and chronic studies. However, the effects occurred mainly at high doses or were not severe enough to warrant GHS hazard classification.

Information on genotoxicity and carcinogenicity, including consideration of repeated dose toxicity, has been reviewed as part of this evaluation. DCM has genotoxic potential based on positive results in multiple *in vitro* and *in vivo* assays, including evidence from human cell

lines. DCM is expected to be carcinogenic, based on sufficient animal evidence, strong mechanistic evidence, and limited human evidence.

Although the available human data have not shown strong or consistent findings for carcinogenicity, several epidemiological studies have shown a positive association between DCM exposure and cancers of the biliary tract and non-Hodgkin lymphoma.

In animals, the chemical caused:

- hepatocellular carcinoma (a type of liver cancer) and/or adenoma (a non-cancerous liver tumour) and bronchioloalveolar carcinoma (a type of lung cancer) in male and female mice
- fibroma (a non-cancerous lump) or fibrosarcoma (cancerous tumour) of the subcutaneous tissues and salivary gland sarcomas in male rats and mammary gland adenoma in male and female rats.

The available data supports a genotoxic mode of action that is relevant to humans.

For further details of the health hazard information, see **Supporting Information**.

Hazard classifications relevant for worker health and safety

The chemical satisfies the criteria for classification according to the Globally Harmonised System of Classification and Labelling of Chemicals (GHS) for hazard categories relevant to workplace health and safety as follows. All classifications below are currently listed in the Hazardous Chemicals Information System (HCIS) except for the proposed amendment to carcinogenicity and a new classification for germ cell mutagenicity (refer to **Supporting Information**). It does not consider classification of physical hazards and environmental hazards.

Health hazards	Hazard category	Hazard statement
Skin corrosion/irritation	Skin Irrit. 2	H315: Causes skin irritation
Serious eye damage/eye irritation	Eye Irrit. 2A	H319: Causes serious eye irritation
Germ cell mutagenicity	Muta. 2	H341: Suspected of causing genetic defects
Carcinogenicity	Carc. 1B	H350: May cause cancer
Specific target organ toxicity (single exposure)	STOT Single Exp. 3	H336: May cause drowsiness or dizziness

Summary of health risk

Public

In Australia the chemical is widely available for use by the public. This includes being present in high concentrations in home maintenance products including paint strippers that can result in substantial inhalation and dermal exposure, particularly in confined spaces. Internationally the chemical has been identified in cosmetics both as a labelled and unlabelled ingredient.

The chemical is a highly volatile solvent with the vapour producing narcotic effects. These include drowsiness, headache, giddiness and, at high concentrations, unconsciousness and mortality. The chemical is a genotoxic carcinogen with genetic polymorphisms present in the human population. Vulnerable subpopulations have been identified for acute and chronic toxicity.

The chemical is listed on Schedule 5 of *Poisons Standard—The Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)* with exemptions relating to concentration and product type. A number of warning statements, first aid instructions and safety directions apply. However, the chemical has been prohibited for cosmetic use and other consumer products overseas.

There is no publicly available information regarding associated adverse events relating to the use of DCM in Australia. However, while labelling controls and use restrictions have been implemented overseas to reduce exposure, DCM-related adverse events, including mortalities, have been reported in association with consumer use of home maintenance products such as paint strippers, cleaning solvents, adhesives, and sealants (see **Supporting information**).

The current entry in the SUSMP would likely limit use of the chemical in cosmetics as an intentional ingredient. However, reports from overseas have identified instances where DCM was not declared on cosmetic product labels, and it is unknown whether similar instances may occur in Australia.

Overall, there is a risk associated with public use of the chemical that requires management (see **Proposed means for managing risk**).

Workers

The chemical is present in high concentrations in commercial and domestic products and can be used in relatively large volumes. Workers in small businesses are likely to use DCM-containing products in non-industrial settings, where the absence of engineering controls and inadequate ventilation can result in unsafe exposures.

The chemical is a highly volatile solvent with vapour producing narcotic effects. These include drowsiness, headache, giddiness and, at high concentrations, unconsciousness and mortalities. The chemical is a genotoxic carcinogen with genetic polymorphisms present in the human population. Vulnerable subpopulations have been identified for acute and chronic toxicity.

In Australia, the chemical has worker controls through hazard classification and workplace exposure standards. The available data support amendment to the hazard classifications. Since the review of the WES, additional information has become available supporting a genotoxic mode of action. In addition, lower exposure standards apply in some overseas jurisdictions.

International reports of DCM-related mortalities and other adverse events from worker use indicate that exposure standards, labelling and PPE alone could be insufficient to manage associated risks with the use of the chemical. Consequently, some overseas jurisdictions have banned DCM paint strippers or restricted their use to trained professionals in industrial settings (see **Supporting information**).

Therefore, a range of control measures is required to minimise dermal and inhalation exposure and mitigate risks to workers (see the Proposed Means for Managing Risk section).

(see **Proposed means for managing risk** section).

Proposed means for managing risk

Public health

Recommendation to Department of Health, Disability and Ageing

It is recommended that the delegate of the Secretary for Poisons Scheduling amend the entry for the chemical in the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP).

It is recommended that, to manage the potential risk associated with the use of these chemicals, the entry:

- Prohibits the use of the chemical in consumer products.

Consideration should be given to the following:

- The chemical is widely available for use by the public including at high concentrations.
- Adverse events including mortalities have been reported internationally from consumer use of the chemical despite labelling controls.
- Susceptible subpopulations at increased risk of mortality have been identified.
- The chemical is a genotoxic carcinogen with a mode of action relevant to humans.
- The chemical has been prohibited for consumer uses overseas with product types varying across jurisdictions.

Workers

Recommendation to Safe Work Australia

It is recommended that Safe Work Australia (SWA):

- Update the Hazardous Chemical Information System (HCIS) to include classifications relevant to work health and safety.
- Review the workplace exposure limit (WEL) for the chemical.
- Consider other mechanisms and controls to limit worker exposure.

Information relating to safe introduction and use

The information in this statement includes recommended hazard classifications, that should be used by a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) to determine the appropriate controls under the relevant jurisdiction Work Health and Safety laws.

Recommended control measures that could be implemented to manage the risk arising from dermal, ocular or inhalation exposure to the chemical include, but are not limited to:

- using closed systems or isolating operations
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker
- minimising manual processes and work tasks through automating processes
- adopting work procedures that minimise splashes and spills
- cleaning equipment and work areas regularly
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

These control measures may need to be supplemented with:

- conducting health monitoring for any worker who is at significant risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health
- conducting air monitoring to ensure control measures in place are working effectively and continue to do so.

Measures required to eliminate, or manage risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk.

Model codes of practice, available from the Safe Work Australia website, provide information on how to manage the risks of hazardous chemicals in the workplace, prepare an SDS and label containers of hazardous chemicals. Your Work Health and Safety regulator should be contacted for information on Work Health and Safety laws and relevant Codes of Practice in your jurisdiction.

Consumer products

Recommendation to Australian Competition and Consumer Commission

It is recommended that the ACCC consider reviewing whether there are any compliance concerns with DCM-containing cosmetics relating to the Competition and Consumer Act 2010 and the Australian Consumer Law, and if necessary, consider any support measures to prevent recurrence.

Conclusions

The Executive Director proposes to be satisfied that the identified risks to human health and the environment from the introduction and use of the industrial chemical can be managed.

Note:

1. Obligations to report additional information about hazards under section 100 of the *Industrial Chemicals Act 2019* apply.
2. You should be aware of your obligations under environmental, workplace health and safety and poisons legislation as adopted by the relevant state or territory.

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Supporting information

Chemical identity

CAS number	75-09-2
CAS name	Methane, dichloro-
Molecular formula	CH ₂ Cl ₂
Associated names	Dichloromethane Methylene chloride
Molecular weight (g/mol)	84.93
SMILES (canonical)	ClCCl
Structural formula	

Relevant physical and chemical properties

The following measured physical and chemical property data for DCM were retrieved from the ECHA (European Chemical Agency) (ECHA RAC 2025a).

Physical form	liquid
Melting point/freezing point	−95.15 °C at 101.325 kPa
Boiling point	39.85 °C at 101.325 kPa
Vapour pressure	58.4 kPa at 25°C (352 mm Hg)
Water solubility	13.2 g/L at 25°C and pH 7
log K_{ow}	1.25 at 20°C and pH 7

Introduction and use

Australian

Based on available data, DCM is introduced into Australia in high volumes. The chemical is listed on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported volume of 1,000–9,999 tonnes (NICNAS 2006). According to the World Integrated Trade Solution (WITS) import data, the following quantities of DCM were imported into Australia:

- 2020: 1,649 tonnes
- 2021: 1,522 tonnes
- 2022: 1,392 tonnes
- 2023: 1,475 tonnes
- 2024: 1,533 tonnes.

The following site-limited industrial uses of DCM in Australia have been identified through National Pollutant Inventory reporting (DCCEEW n.d.) or information previously reported to NICNAS (NICNAS 2014):

- solvent used in chemical manufacturing
- use in the production of polymer foams.

In addition, an online search identified that DCM is publicly available in Australia in paint strippers at concentrations ranging from 60% to 90% (w/w) and welding anti splatter sprays (60–100%).

Information obtained through a voluntary call for information also indicated that DCM is marketed in Australia and is present in adhesive aerosols at a concentration of 0.0007%.

International

The following international uses have been identified through the:

- European Union Registration, Evaluation, Authorisation and Restriction of Chemicals (EU REACH) dossier (REACH n.d.)
- OECD Screening Information Dataset Initial Assessment Report (SIAR) (OECD 2011)
- Galleria Chemica (Chemwatch n.d.)
- United States Environmental Protection Agency (US EPA) assessments (US EPA 2014; US EPA 2020)
- IARC Monograph (IARC 2017)
- DCCEEW website (DCCEEW n.d.)
- ATSDR (Agency for Toxic Substances and Disease Registry report) (ATSDR 2000)
- Performance Measurement Evaluation for the Risk Management of Dichloromethane (DCM) / Methylene Chloride (Government of Canada 2022).

DCM is used in domestic and commercial applications including:

- adhesives and sealants
- paint, adhesive and coating removers
- automotive products including brake and engine cleaners
- furniture finishing
- aerosol propellants
- metal cleaning and degreasing
- shoe cleaner
- removal of photo-resistant coatings in the production of printed circuit boards
- welding anti-splatter sprays.

A review of 155 relevant Canadian Safety Data Sheets (SDSs) for products containing DCM, retrieved between 2004 and 2018, reported the following concentration ranges (Government of Canada 2022):

- shoe cleaners, furniture strippers, paint removers, and adhesive removers had average midpoint dichloromethane concentrations of approximately 75% to 85%
- lubricants, cleaners, coatings, and sealants had average midpoint dichloromethane concentrations ranging from 12% to 40%.

According to the Consumer Product Information Database (CPID), DCM is present in at least 84 brands (67 of which inactive or discontinued). These include:

- 3 commercial/institutional products (mark and stain remover sprays, 40–60% concentration)
- 67 home maintenance products (paint stripper, varnish, stain and mark remover, liquid adhesive remover, spray and liquid, mostly 25–100%)
- 13 automotive products (carburettor and choke and valve cleaner, brake parts cleaner, degreaser, paint, varnish and adhesive remover, spray and liquid, mostly >40%)
- 1 agricultural pesticide (herbicide).

The chemical is not listed in the International Nomenclature of Cosmetic Ingredients (INCI) database (Personal Care Products Council n.d.). DCM was historically used in cosmetic products such as hair sprays and nail polish removers. A 1994 study (Rastogi SC, 1994) confirmed its use in hair-care cosmetics, noting that up to 35% DCM was permitted under the European Union's cosmetic directive at the time. However, due to growing evidence of its carcinogenicity and neurotoxicity, regulatory agencies including the United States Food and Drug Administration (US FDA) and the US EPA have since banned its use in cosmetics (see **International regulatory status**). Despite this ban, the US FDA recently found several cosmetic products marketed as gel nail polish remover that contain DCM with concentrations, as high as 92.7%. Reports indicate that cosmetics containing DCM may not always be identified as an ingredient on the cosmetic product label (National Archives n.d.). Several products containing the chemical were reported to the California Safe Cosmetics Program. A total of 55 products were reported between 2009 and 2025 with 14 of those products having either been discontinued or had the chemical removed since the initial reports. The types of products containing this chemical include hair care products and leave-on skin care products (CSPC n.d.). Online product searches indicate that the chemical is present in a few makeup products including lipsticks (EWG n.d.).

DCM is reported to have the following site-limited uses:

- in the manufacture of chemicals including hydrofluorocarbon (HFC) refrigerants
- in the manufacture of textiles, cellulose triacetate fibres and photographic films
- as a blowing and cleaning agent in the production of urethane foam
- in electronics manufacturing.

Additionally, DCM has non-industrial use in the manufacture of pharmaceuticals and in food processing.

Existing Australian regulatory controls

Public

Dichloromethane is listed in the Poisons Standard—*The Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) in Schedule 5* as follows (TGA 2026):

'DICHLOROMETHANE (methylene chloride) except:

- a) in preparations in pressurised spray packs labelled as degreasers, decarbonisers or paint strippers and containing 10% or less of dichloromethane;
- b) in other preparations in pressurised spray packs;
- c) in paints and tinters containing 5% or less of dichloromethane; or
- d) in preparations for human therapeutic use.

Schedule 5 chemicals are labelled with 'Caution'. These are 'substances with a low potential for causing harm, the extent of which can be reduced through the use of appropriate packaging with simple warnings and safety directions on the label' (TGA 2026).'

Workers

Dichloromethane (CAS No. 75-09-2) is listed on the HCIS with the following hazard category and statements for human health (SWA n.d.):

Health hazards	Hazard category	Hazard statement
Skin corrosion/irritation	Skin Irrit. 2	H315: Causes skin irritation
Serious eye damage/eye irritation	Eye Irrit. 2A	H319: Causes serious eye irritation
Carcinogenicity	Carc. 2	H351: Suspected of causing cancer
Specific target organ toxicity (single exposure)	STOT Single Exp. 3	H336: May cause drowsiness or dizziness

The chemical is listed on the HCIS with a workplace exposure standard of 174 mg/m³ (50 ppm) as an 8 hr time-weighted average (TWA) with a skin absorption notation (Sk). Safe Work Australia has published the Workplace Exposure Limits for airborne contaminants (WEL list). From 1 December 2026 and following implementation into the work health and safety laws of the Commonwealth, states and territories, new Workplace Exposure Limits (WEL) for airborne contaminants (WEL list) will be adopted throughout Australia (SWA 2025). Part 7.1 to the model WHS Regulations governs the use, handling, storage and generation of hazardous chemicals in the workplace. In addition, there are other WHS requirements that are applicable to hazardous chemicals, such as the primary duty of care, hierarchy of controls, and health and air monitoring requirements. The WEL for this chemical is the same as the WES.

International regulatory status

Exposure standards

The following occupational exposure limits (OELs) are identified (Galleria Chemica n.d.):

The chemical has an exposure limit of 50 ppm (174 mg/m³) in Austria, Belgium, Canada, Germany, Malaysia, New Zealand and Singapore and 100 ppm (348 mg/m³) in Bulgaria, the EU and the UK and 200 ppm (696 mg/m³) in Argentina (Chemwatch n.d.). In the USA, the

American Conference of Governmental Industrial Hygienists (ACGIH) recommends a TWA of 50 ppm (174 mg/m³). The Occupational Safety and Health Administration (OSHA) has also established a Permissible Exposure Limit (PEL) of 25 ppm (87 mg/m³) as an 8 hr time-weighted average (TWA) with an action level of 12.5 ppm. These limits are set based on central nervous system effects (OSHA n.d.).

The chemical also has a short-term exposure limit (STEL) of 125 ppm (434 mg/m³) in the USA (set by OSHA), 200 ppm (696 mg/m³) in Austria, Belgium, Denmark, EU and UK and 250 ppm (869 mg/m³) in Argentina (Chemwatch n.d.).

The US EPA recently established an Existing Chemical exposure Limit (ECEL) for DCM of 2 ppm TWA and 16 ppm STEL (US FR 2024). The approach to managing potential risks associated with the use of DCM is continuing to evolve in the USA. The point of departure for developing this standard is a no observed adverse effect level (NOAEL) of 200 ppm based on liver vacuolation in female rats in a well-conducted 2 year combined chronic toxicity and carcinogenicity inhalation study. The US EPA concluded that hepatic vacuolation associated with DCM exposure is toxicologically relevant as an adverse effect. This conclusion was based on evidence demonstrating that:

- It correlates with fatty change or as a vacuolation of lipids in the hepatocyte.
- The increased incidence follows a dose-response relationship in rats and mice exposed to DCM through both inhalation and oral pathways.
- Vacuole content was consistently identified as lipid.
- Accumulation of lipids in the hepatocyte may lead to the more serious liver effects observed following DCM exposure, such as hepatic steatosis (fatty liver) reported in dogs and rats (US EPA 2011).

Similar to OELs, Biological Exposure Indices (BEIs), Biological Limit Values (BLVs) and Biological Monitoring Guidance Values (BMGVs) for DCM also vary across countries and organisations.

In the USA, the American Conference of Governmental Industrial Hygienists (ACGIH) recommends a BEI of 0.3 mg/L of DCM in urine at the end of a work shift (ACGIH, 2026).

In the European Union (EU), the Scientific Committee on Occupational Exposure Limit Values (SCOEL) has recommended the following BLVs: 4% carboxyhaemoglobin in blood, 0.3 mg/L of DCM in urine, and 1 mg/L of DCM in blood (SCOEL 2009).

In the UK, 30 ppm of carbon monoxide in end-tidal breath has been proposed as the BMGV for DCM exposure (HSE n.d.).

Canada

Dichloromethane was assessed by the government of Canada, and a Priority Substances List (PSL) Assessment Report was published in 1993. The report concluded that DCM is present at concentrations that may be harmful to the environment, and that may constitute a danger in Canada to human life or health. It concluded that DCM occurs at concentrations that do not constitute a danger to the environment on which human life depends. The following three risk management actions were undertaken by the Government of Canada to address both the human health and environmental concerns that were identified in the PSL:

- preventing deliberate use of dichloromethane in aerosol cosmetic products by describing the substance as a restricted ingredient on the Health Canada Hotlist since 1995
- reducing aggregate dichloromethane releases from 5 targeted industry sectors (adhesives formulation, aircraft paint stripping, flexible polyurethane foam blowing, industrial cleaning and pharmaceuticals and chemical intermediates manufacturing and tablet coating) by 85% from 1995 base year levels by January 1, 2007
- and implementing a code of practice (2003), under which the commercial uses of dichloromethane could be reduced by 20% (Government of Canada 1993).

Additionally, a 2022 report by the Canadian government concluded that based on the information available, risk management actions taken by the Government of Canada have been successful in reducing the exposures of Canadians to DCM through outdoor air and when using aerosol cosmetic products. The effectiveness of risk management actions on long-term indoor air exposures cannot be conclusively evaluated due to lack of comparable exposure information. Exposures to DCM in the occupational setting, and from products containing DCM available to consumers were not considered in the PSL assessment. Since such exposures have recently been deemed to be of concern in other jurisdictions and there is evidence suggesting that products containing DCM are available to consumers in Canada, there is a need to further characterise the possible risk from these exposures to dichloromethane (Government of Canada 2022).

European Union

The chemical is listed in Annex XVII of the REACH Regulation (EC) No 1907/2006. Paint strippers containing DCM in concentrations greater than or equal to 0.1 percent by weight are banned for sale to the general public. This ban includes both placing the chemical on the market and chemical use. Professional use in paint strippers is also banned after 6 June 2012, except under tightly controlled conditions outlined in Entry 59 of Annex XVII to the REACH Regulation (EC No 1907/2006) where a Member State grants a derogation for specifically trained professionals (ECHA n.d.). Use in industrial installations is allowed only under strict conditions (EC 2010).

The use of DCM is also prohibited in cosmetics, as it is listed in Annex II of the EU Cosmetics Regulation No. 1223/2009, which includes substances banned from cosmetic use due to safety concerns (EC n.d.).

New Zealand

In New Zealand, DCM is listed in Schedule 4 of the Cosmetic Products Group Standard, which includes ingredients that are prohibited in cosmetic products (New Zealand, 2020).

United States of America

In April 2024, US EPA issued a final rule regulating DCM under the Toxic Substances Control Act (TSCA) to protect human health from risks such as neurotoxicity and cancer resulting from inhalation or dermal exposures (US EPA Factsheet 2024).

- A Workplace Chemical Protection Program (WCPP) is required to continue 13 conditions of use of methylene chloride. The WCPP requires that owners and operators of facilities using methylene chloride take appropriate measures to meet new inhalation exposure limits (including 2 ppm as an 8 hr time-weighted average) and develop and implement an exposure control plan, among other requirements.

- The distribution of methylene chloride for consumer use has been prohibited after May 5, 2025.
- Most of its commercial uses have been prohibited after April 28, 2026.
- Methylene chloride may be used only for very specific furniture refinishing until May 8, 2029, with workplace protections. After this date, this use is prohibited.

The approach to managing potential risks associated with the use of DCM is continuing to evolve in the USA.

In the USA, DCM is also on the list of prohibited & restricted ingredients in cosmetics (US FDA- Prohibited & Restricted Ingredients in Cosmetics), and any cosmetic product that contains DCM as an ingredient is deemed adulterated and is subject to regulatory action under sections 301 and 601(a) of the Federal Food, Drug, and Cosmetic Act, US FDA- Code of Federal Regulations (National Archives n.d.).

Association of Southeast Asian Nations (ASEAN)

In ASEAN countries, the use of DCM in cosmetics is prohibited under the ASEAN Cosmetic Directive and methylene chloride (dichloromethane; CAS No. 75-09-2) is included in Annex II Part 1, which lists substances that must not form part of the composition of cosmetic products (HSA 2026).

China

In 2023, the Chinese Ministry of Ecology and Environment (MEE) published a list of Key Controlled New Pollutants. This list includes DCM as a key controlled pollutant. The following restrictions apply:

- Use in cosmetics is banned under the Safety and Technical Standards for Cosmetics and is included in Annex II of the ASEAN Cosmetic Directive (HSA 2026).
- Paint removers containing DCM must not be produced.
- The following limits on DCM concentrations in cleaning products are defined under GB 38508 – Limits for Volatile Organic Compounds Content in Cleaning Agents (Envilience Asia 2023):
 - water-based: $\leq 0.5\%$
 - semi-water-based: $\leq 2\%$
 - organic solvent-based: $\leq 20\%$.

Human exposure

Workers

The chemical is used principally as a solvent, in paint removers, degreasers and aerosol products, and in the manufacture of foam polymers. Widespread exposure occurs during the production and industrial use of DCM.

According to the 1990–93 CAREX database for 15 countries of the European Union and the 1981–83 United States National Occupational Exposure Survey, approximately 250,000 workers in Europe and as many as 1.4 million workers in the USA were potentially exposed to DCM. Concentrations of DCM measured in 1968–73 in the general work area of a plant producing plastic films in the USA were 458–2060 mg/m³ in the casting area, 583–3350

mg/m³ in the filtration area, 625–659 mg/m³ in the winding area, and 160–1130 mg/m³ in offices. Personal occupational exposures (mg/m³) measured in a pharmaceutical industry, a flexible polyurethane manufacturing plant, furniture stripping shops, a polyester industry, and plastic and synthetic fibres industries were up to 201, 260, 663, 742, 1600 and, 1950, respectively. These levels varied widely by operation and within operations. Concentrations exceeding 1000 mg/m³ have been measured, e.g., in paint stripping, in the printing industry, and in the manufacture of plastics and synthetic fibres.

Exposure of workers undertaking full time shifts to levels above 100 mg/m³ of dichloromethane is possible, e.g., in furniture stripping shops and in certain jobs in aeronautical, pharmaceutical, plastic and footwear industries (IARC 1999). A range of worker exposure estimates are included in the US EPA risk evaluation (US EPA 2020). Several occupational scenarios were estimated to result in 8 hr TWA inhalation concentrations above 50 ppm. These included:

- adhesive and caulk remover applications
- cellulose triacetate film manufacturing
- conveyerised vapour degreasing
- flexible PU foam manufacturing scenarios.

In addition, occupational non-users working in the vicinity of conveyerised vapour degreasing operations were also estimated to be exposed to inhalation concentrations exceeding 50 ppm.

Separate assessment of commercial paint and coating remover uses also identified high occupational exposures in several professional paint-stripping scenarios, including (US EPA 2014):

- professional contractors
- automotive and furniture refinishing
- aircraft paint stripping
- graffiti removal activities.

Public

There is minimal data on exposure levels to DCM of the general population using do-it-yourself household products. Online websites for DCM-containing paint strippers available in Australia indicate application rates of 1-2 L per m².

A 1990 US EPA investigation of the use of household liquid products in the USA estimated consumer exposure levels ranging from less than 35 mg/m³ (10 ppm) to a few short-term exposures of 14,100–21,200 mg/m³ (4059–6103 ppm). Most of the concentration estimates were below 1,770 mg/m³ (510 ppm) (IARC 1999). Modelled air concentrations for consumer use of DCM as a paint stripper ranged from 69–580 mg/m³ (20-170 ppm) as an 8-hr average and 380–1,900 mg/m³ (110–548 ppm) as a 10 min average (US EPA 2014). A range of consumer exposure estimates are included in the US EPA risk assessment (US EPA 2020). The highest estimated consumer inhalation exposures were associated with the use of DCM in the following products:

- coil cleaners
- engine cleaners
- carbon remover
- weld spatter protectants
- gasket removers

- carburettor cleaners
- brake cleaners
- cold pipe insulation sprays
- sealants

with estimated 8 hr TWA concentrations ranging from 74–597 ppm. Consumer paint and coating remover uses were not included in the consumer exposure assessment because they had already been addressed through separate US EPA rulemaking and were outside the scope of the risk evaluation.

Health hazard information

This evaluation reviews available data for DCM relating to genotoxicity and carcinogenicity. In addition, toxicological data that have been used for risk assessment, including information on toxicokinetics, repeat dose toxicity and observations of adverse effects in humans have been reviewed.

More information on other endpoints not considered in this evaluation are available in the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework report conducted under the former scheme, NICNAS (NICNAS 2014).

Toxicokinetics

Absorption

Due to its lipophilicity and to its low relative molecular mass, DCM can readily cross biological membranes. DCM is readily absorbed through inhalation exposure in humans and animals. Pulmonary uptake is rapid, approaching steady state within hours for exposures up to 200 ppm in humans, and ranges between 40 and 60%, but may be up to 70% during the first minutes of exposure (ECHA 2025a; IARC 2017; US EPA 2020).

Data on oral absorption in humans are limited to case reports of accidental ingestion and suggest that DCM is also readily absorbed by this route of exposure, although quantitative estimates of oral bioavailability in humans are not available (IARC 2017; ECHA 2025a). In animals, absorption from the gut after oral doses is rapid and nearly complete (on average 97%). The permeability of human skin to DCM is 24 g/m² per hr (ECHA 2025a).

Distribution

After absorption, DCM enters the blood circulation and undergoes rapid systemic distribution to tissues, with the highest concentrations expected in adipose tissue and other fatty tissues. DCM is distributed to many organs, including liver, kidneys, lungs, brain, muscle and adipose tissue, epididymal fat and testes after respiratory and oral exposure (ECHA 2025a). Experiments in animals show that the chemical can cross the blood-brain barrier and the placenta. Low levels have been detected in human breast milk. The chemical was found in foetal and maternal blood in pregnant rats exposed to the chemical vapour at 500 ppm for 1 hr (IARC 2017; NICNAS 2014; US EPA 2020).

Metabolism

Metabolism occurs predominantly in the liver, with additional transformation reported in the lungs and kidneys (US EPA 2020).

The chemical is metabolised through two competitive pathways in experimental animals and humans (ECHA 2025a; IARC 2017; US EPA 2020):

- i) cytochrome P450 2E1 (CYP2E1)-mediated reductive dehalogenation (referred to as CYP-oxidation)
- ii) glutathione S-transferase (GST)-mediated conjugation to glutathione (GSH) (referred to as GST-mediated conjugation).

Biotransformation of DCM by CYP-oxidation (mainly by the CYP2E1 enzyme) results in dichloromethanol as the initial product of biotransformation. Dichloromethanol is not chemically stable and spontaneously rearranges to form formyl chloride which can subsequently generate CO or react with nucleophiles such as glutathione (GSH) to form formylglutathione. This subsequently rearranges to release CO₂. A significant part of the CO formed from DCM binds to haemoglobin in the blood to form carboxyhaemoglobin (CO-Hb). CoHb serves as a biomarker for the oxidation of metabolism of DCM by CYP-oxidation and has been detected in humans following DCM exposure (Dekant et al. 2021). CYP-oxidation may serve as a detoxification mechanism by diverting it from pathways that could transform it into genotoxic compounds.

The second pathway of DCM biotransformation, GST-mediated conjugation, (conjugation of DCM with glutathione) is catalysed by a specific glutathione S-transferase designated GSTT1. The reaction of DCM with glutathione initially results in the formation of S-chloromethyl GSH. Due to the high reactivity of S-chloromethyl GSH in biological media, this compound is rapidly hydrolysed to hydroxymethyl GSH, which can either decompose to release formaldehyde or be oxidised by formaldehyde dehydrogenase to form S-formyl GSH. The latter is subsequently hydrolysed to release formic acid and GSH. Formic acid further decomposes to release CO₂. Alternatively, S-chloromethyl GSH may bind to nucleophilic sites in DNA, potentially leading to genotoxicity and carcinogenicity.

Some researchers proposed an alternative explanation for DCM metabolism in which only CYP-oxidation is involved (Evans and Caldwell 2010). IARC concluded there were no data supporting biotransformation reactions of DCM that exclude GSH conjugation, particularly at higher DCM concentrations (IARC 2017).

Although these metabolic pathways operate in both humans and animals there are quantitative differences which vary according to species, the location of metabolism, exposure levels and genetic makeup. This variability is likely to explain the pattern of genotoxic and carcinogenic effects (see the **Genotoxicity** and **Carcinogenicity** sections for further discussion). Key aspects of DCM metabolism are outlined below (ECHA RAC 2025a; IARC 2017; US EPA 2011).

1) The predominant pathway depends on DCM exposures

The chemical binds to the CYP receptors more readily compared to GST receptors. Therefore, CYP oxidation predominates at lower exposure levels. When the available CYP receptors become saturated, more DCM is available to bind to the lower-affinity GST metabolic sites, and the proportion of DCM metabolised by GST increases. Saturation of the CYP oxidation pathway has been reported in humans at levels of exposure which are readily observed in occupational settings, including continued exposure at low levels. Saturation can also occur due to genetic factors that decrease CYP2E1 activity (see point 2 below). Regardless of the predominant pathway, both pathways are expected to operate even at low exposures.

2) Enzyme activity varies according to location, species and genetic makeup

In rats and mice, GSTT1 activity has been found in the liver, lungs, and kidneys, and in hamsters in the liver and kidneys. In humans, particularly high levels of the mRNA for GSTT1 were found in the liver, kidneys, skeletal muscle and pancreas. Unlike rodents, high levels of GSTT1 have been detected in human erythrocytes depending on genetic polymorphism.

Mice have the highest specific liver GSTT1 activity followed by rats, human high conjugators, human low conjugators, hamsters and human non-conjugators. A polymorphism of the GSTT1 gene has been demonstrated in humans with some high conjugators and other non-conjugators.

Subcellular localisation and the absolute level of the expressed GSTT1 enzyme is thought to play a role in carcinogenesis with nuclear production of S-(chloromethyl) glutathione more likely to lead to DNA alkylation than cytoplasmic production. In mouse liver tissue, GSTT1 appears to be predominantly localised in the nuclei. In liver slices obtained from two human subjects (Sherratt et al. 2002), the most intense nuclear staining was associated with bile duct epithelial cells, and some, but not all, hepatocytes that expressed the enzyme.

Several genetic polymorphisms for the human CYP2E1 gene have been described, but clear and consistent correlations with associated enzyme activities have not been identified. Individuals with decreased CYP2E1 activity may experience decreased generation of CO and an increased level of GST-related metabolites following exposure to dichloromethane. One study has reported an association between a CYP2E1 polymorphism and Non-Hodgkins Lymphoma although the significance of this is unknown (see **Carcinogenicity section**).

Excretion

Following inhalation, the chemical is rapidly excreted unchanged in exhaled air and in urine in humans. In rats, the chemical is excreted in exhaled air, urine and faeces. Small amounts are excreted in milk.

After oral exposure, DCM is also quite rapidly excreted, mostly via the lungs in the exhaled air. Exhalation of DCM after inhalation exposure increases when exposed to higher concentrations. The remainder is metabolised to carbon monoxide, carbon dioxide and inorganic chloride.

Exhalation is the main route of excretion of DCM in humans. The primary metabolites are CO₂ and CO, with lesser amounts of DCM excreted in the urine. Only 5% of absorbed DCM is exhaled unchanged, 25–34% excreted converted as CO, and the balance excreted as CO₂. After cessation of exposure, the half-life of DCM in the blood has been estimated to be about 40 min, with concentrations of parent and metabolites returning to the pre-exposure levels within a few days. Urinary excretion occurs mostly during and/or within the first hr after cessation of exposure, and in total accounts for less than 0.1% of uptake (IARC 2017).

Acute toxicity

Based on the available data, the chemical has low acute toxicity via oral, inhalation and dermal routes. The oral and dermal LD₅₀ values are >2000 mg/kg bw in rats (NICNAS 2014). The 6 hr inhalation LC₅₀ values across all tested species range from 40,200 to 55,870 mg/m³ (IPCS 1996).

Neurotoxicity and other neurological effects were the most frequently observed outcomes in acute and short-term studies.

Animal studies also primarily identified CNS effects following acute exposure, such as decreased motor activity, impaired memory, and altered responses to sensory stimuli (US EPA, 2020).

Observation in humans

In humans, the main cause of death from high levels of DCM inhalation is related to CNS effects. These include loss of consciousness and respiratory depression leading to irreversible coma, hypoxia and mortality. The organ most often affected in fatal accidents is the brain, followed by the lungs and heart. Changes in these organs include congestion and oedema. The lungs and heart also showed petechiae (tiny spots on skin or mucous membranes caused by bleeding capillaries) in a few cases. Cardiotoxic effects were observed in a few cases (US EPA 2020). From 1980 to 2018, 85 DCM related mortalities were identified in the US, including 74 (87%) in occupational settings. Paint strippers were the most common products involved in DCM related mortalities (n = 60) (Hoang et al, 2021).

Vulnerable subpopulations have been identified for acute and chronic toxicity (see **Human health risk characterisation**). For example, of the 85 DCM related mortalities in the study described above, 11 (52%) of the 21 workers whose hearts were autopsied, had considerable atherosclerotic coronary artery disease (Hoang et al. 2021). Concerns about enhanced risks associated with the use of DCM by people with underlying cardiovascular disease have been raised by regulatory bodies in the EU and USA (ATSDR 2000; EC 2004; US EPA 2020).

Acute exposure to the chemical at concentrations >200 ppm for 4–8 hrs resulted in neurobehavioural deficits (measured in psychomotor tasks). In a well-conducted study of 12 volunteers decreased visual peripheral performance was reported after 1.5 hrs of exposure to 195 ppm (200 ppm nominal) (US EPA 2011; US EPA 2020).

Repeat dose toxicity

Oral

Cancer is identified as the critical toxicological endpoint following repeated oral exposure to DCM. Details on the chemical's carcinogenic effects are provided in the **Carcinogenicity Section**. Studies in rats and mice identified hepatotoxicity as the primary non-cancer effect following repeated oral exposure to DCM in short-term, subchronic and chronic studies (US EPA 2011). However, the effects occurred mainly at high doses or were not severe enough to warrant GHS hazard classification.

Oral studies in rats and mice with exposures ranging from 2 to 13 weeks found liver effects including centrilobular necrosis, granulomatous foci, ceroid/lipofuscin accumulation, hepatocyte vacuolation at doses starting from 333 mg/kg bw/day (NICNAS 2014, IPCS 1996).

In a combined chronic toxicity/carcinogenicity study conducted similarly to OECD TG 453, F344 rats (50–85/sex/dose) received the chemical in drinking water at doses of 0, 5, 50, 125, 250 mg/kg bw/day (with mean intake at 0, 6, 52, 125 and 235 mg/kg bw/day in males and 0, 6, 58, 136 and 263 mg/kg bw/day in females) for 104 weeks. No treatment-related histopathological effects were observed in these rats except for the liver.

Statistically significant increases in the incidences of foci/areas of cellular alteration were observed at all dose levels except the lowest dose. Fatty changes were also observed. A NOAEL of 5 mg/kg bw/day and a lowest observed adverse effect level (LOAEL) of 50 mg/kg bw/day was established for males and females based on liver changes (US EPA 2011).

In a 2 year drinking water study similar to the previously described study in F344 rats, B6C3F1 mice (100–200 males/group and 50–100 females/group) received the chemical in drinking water at doses of 0, 0, 60, 125, 185 and 250 mg/kg bw/day for 104 weeks. The study design included 2 control groups. Treatment-related nonproliferative histopathologic effects were only found in the liver of the rats and consisted of marginal increases of fat. A NOAEL of 185 mg/kg bw/day and a LOAEL of 250 mg/kg bw/day was established for males and females based on these changes (US EPA 2011).

Dermal

No data are available in either humans or experimental animals regarding the effects of repeated dermal exposure to DCM.

Inhalation

A large number of repeated dose subacute, subchronic and chronic inhalation toxicity studies in rats, mice, hamsters, guinea pigs, dogs and monkeys at mostly high concentrations found a number of non-neoplastic liver effects including (IARC 2017; IPCS 1996; NICNAS 2014; REACH):

- histopathological changes (cytoplasmic vacuolisation multinucleated hepatocytes, hypertrophic hepatocytes, increased lipid peroxidation of hepatic tissues, necrosis of centrilobular hepatocytes, fatty changes in the liver, swelling of hepatocyte rough endoplasmic reticulum, increased mitotic activity in hepatocytes, haemosiderosis, bile duct fibrosis)
- increased absolute and relative liver weights
- increased activity of liver enzymes.

Increased incidences of nonneoplastic liver lesions were reported when rats (two species) and mice were exposed to high doses (≥ 500 , $\geq 1,000$ or ≥ 2000 ppm) in 2 year inhalation studies (US EPA 2011).

In a combined chronic toxicity/carcinogenicity study conducted similarly to OECD TG 453, Sprague Dawley (SD) rats (90 males, 108 females) were exposed (whole body) to the chemical vapour at doses of 0, 50, 200 or 500 ppm (0, 177, 710 or 1770 mg/m³), six hrs/d, five days/week for 20 and 24 months respectively. A no observed adverse effect concentration (NOAEC) of 710 mg/m³ was determined based on histopathological changes (increased incidences of hepatocellular vacuolisation) in the liver at 1770 mg/m³. Multinucleated hepatocytes were observed in the females at the highest dose level. A dose-dependent increase in COHb levels in blood was observed from 710 mg/m³ and above (NICNAS 2014). This study was used as the point of departure for quantitative risk assessments conducted by the US EPA and developing a new ECEL for DCM of 2 ppm (US FR 2024).

No signs of liver or lung cell proliferation preceded tumours in chronic studies (US EPA 2011).

Genotoxicity

The chemical has potential genotoxicity, warranting classification as genotoxic Category 2.

Although mixed results are available from *in vivo* genotoxicity studies with the chemical, mutagenicity and genotoxicity has been observed in somatic cells. Effects were mainly observed in mice although DNA damage was reported in a study in rats in rats. In mice the chemical:

- induced micronucleus in peripheral blood erythrocytes and lung cells
- caused a small increase in the frequency of chromosomal aberration bone marrow and lung cells
- caused a significant increase in frequencies of SCEs in lung cells and peripheral blood erythrocytes
- consistently induced DNA damage in the lung and liver (cancer target tissues).

The genotoxicity observed *in vivo* is supported by positive results from *in vitro* mutagenicity assays.

No data are available for germ cell mutagenicity.

In vitro studies

Mostly positive results were reported in bacterial reverse mutation assays with and without metabolic activation with mutations in *Salmonella typhimurium* strains TA100 and TA98. This was supported by positive results in yeast and fungal mutagenesis assays (US EPA 2011).

Mixed results were reported in human, rat, mouse and hamster cells reflecting the varying levels of metabolic activity in both the CYP-oxidation and GST-conjugation pathways. Positive results were reported in some gene mutation, chromosome aberration, micronucleus and DNA damage assays. The linkage between GST activity and genotoxicity was further supported by results from these studies. For example, mostly negative results were reported following rat and hamster cell lines exposure to DCM, which have significantly lower GST activity compared to mouse cells. However, genotoxic effects were reported when mouse liver cytosol or transfected mouse GST were added to the same lines that previously found no genotoxicity. Positive results were reported in mouse cell lines (Clara cells). *In vitro* studies using human cells reported genotoxic effects on the frequency of micronuclei, DNA damage (comet assay), and sister chromatid exchanges but no effects on unscheduled DNA synthesis, DNA single strand breaks or DNA-protein cross-links (ECHA 2025b; IARC 2017 US EPA 2011).

In vivo studies

In a mammalian DNA damage study B6C3F1 mice and rats were exposed to DCM via inhalation for 3 or 6 h at 2000 or 4000 ppm (nominal doses). DNA single strand breaks were only detectable in hepatocyte DNA from mice (US EPA 2011).

Exposure of B6C3F1 mice to DCM by inhalation caused DNA damage, detected as increased DNA single strand breaks, in liver and lung tissue immediately following 3 hr exposures (up to 8000 ppm) suggesting the presence of an active DNA repair process. Pretreatment of the mice with a glutathione depletor, buthionine sulfoximine (BSO), reduced the extent of DNA damage to control levels, suggesting the involvement of a GST-mediated mechanism. DCM did not induce DNA damage in a similar study in rats (IARC 2017; US EPA 2011).

Exposure of SD rats to DCM caused DNA damage, detected as single-strand following oral administration at 1275 mg/kg (US EPA 2011).

No DNA damage or increased gene mutations were observed in the livers of Delta mice after 4 weeks of inhalation exposure to 800 ppm. This was a lower exposure concentration compared with the levels inducing DNA strand breaks (≥ 2000 ppm) or increased tumour incidences. One possible explanation is that CYP2E1 metabolism was not saturated at the lower concentration, thereby limiting the formation of DNA-reactive GST metabolites (US EPA 2020).

DCM did not induce DNA-protein cross links in liver or lung cells in hamsters exposed to 4000 ppm via inhalation for 3 days (US EPA 2020).

In a non-guideline sister chromatid exchange (SCE) assay, 8–9 week-old female B6C3F1 mice (8 animals/dose) were subcutaneously administered single non-cytotoxic doses of 0, 2500 or 5000 mg/kg of DCM in corn oil or exposed to concentrations of 0, 4000 or 8000 ppm of DCM (5 animals/dose) via inhalation route for 6 h/day, 5 days/week for 10 days or 2000 ppm for 3 months. No increase in the frequency of SCEs in bone marrow cells was observed after a single subcutaneous injection of 2500 or 5000 mg/kg DCM. Inhalation exposure to DCM for 10 days at concentrations of 4000 or 8000 ppm resulted in significant increases in frequencies of SCEs in lung cells and peripheral blood lymphocytes. DCM inhalation at 2000 ppm for 3 months resulted in a slight increase of SCEs in lung cells (Allen et al. 1990).

No increase in the frequency of sister chromatid exchanges was observed in the bone marrow cells of C57BL/6J mice following a single subcutaneous injection of 100, 1000, 1500, or 2000 mg/kg of DCM (IARC 2017; US EPA 2020). The negative genotoxic effects in bone marrow (a tissue with minimal GST activity) is not surprising given that the tumours observed in mice are generally found in the liver (due to high GST activity) and the lung (due to increased availability of GST-mediated metabolism (US EPA 2011).

In several studies, DCM did not induce micronucleus formation in the bone marrow of mice treated by gavage or intraperitoneal (IP) administration. This is consistent with the minimal GST activity in this tissue and evidence that halogenated hydrocarbons are not very effective in inducing micronucleus formation in mouse bone marrow (ECHA 2025a; US EPA 2020).

In a mouse micronucleus assay, similar to OECD TG 474, 8–9 week-old female B6C3F1 mice (5 animals/dose) were exposed to DCM concentrations of 0, 4000 or 8000 ppm via inhalation route for 6 h/day, 5 days/week for 10 days or 2000 ppm for 3 months. A positive dose-dependent response was noted in the incidence of MPEs in the peripheral blood erythrocytes which was statistically significant at 8000 ppm. Statistically significant increases in the incidence of micronucleated normochromatic erythrocytes (NCEs) over the control group were also noted both at 4000 and 8000 ppm. The highest dose of DCM was found to be cytotoxic as it resulted in a significant reduction in the percentage of PCEs. DCM inhalation at 2000 ppm for 3 months resulted in a significant increase in the frequency of micronucleated NCEs and PCEs in peripheral blood erythrocytes compared with controls. (Allen et al. 1990).

Chromosomal aberrations were not induced in the bone marrow of mice or rats exposed to DCM via the IP or inhalation routes respectively (IARC 2017).

In a mammalian chromosomal aberration (CA) test, similar to OECD TG 475, 8–9 week-old female B6C3F1 mice (8 animals/dose) were subcutaneously administered single non-cytotoxic doses of 0, 2500 or 5000 mg/kg of DCM in corn oil or exposed to concentrations of 0, 4000 or 8000 ppm of DCM (5 animals/dose) via inhalation route for 6 h/day, 5 days/week for 10 days. Subcutaneous administration of DCM did not result in a significant increase in the mean percentage of bone marrow aberrant cells. Inhalation exposure to DCM was associated with a dose-related increase in CA in lung cells at both

doses which was statistically significant at 8000 ppm and a significant dose-related increase in CA in bone marrow at 8000 ppm (Allen et al. 1990).

Unscheduled DNA synthesis was not induced in hepatocytes from mice or rats. It is generally recognised that this assay is not sensitive for detecting genotoxic chemicals (US EPA 2011).

Human data

Using comet and micronucleus assays, genomic damage was investigated in 16 workers occupationally exposed to a mixture of four confirmed or suspected carcinogens, ethylene oxide, 1,2-dichloroethane, phenylhydrazine, and DCM, in a pharmaceutical facility (Zeljezic et al., 2016). The investigation was conducted at two time points: initially, when the use of PPE was irregular, and again 8 months after the enforcement of strict PPE requirements. After 8 months of consistent PPE use, both micronuclei frequency and comet assay parameters in lymphocytes of the exposed workers significantly decreased compared with levels observed during the period of irregular PPE use. While this study provides some evidence supporting a positive association between exposure to multiple chemicals, including DCM, and genomic damage, the absence of quantitative exposure assessment data and the confounding effects of simultaneous exposure to several chemicals limits the ability to draw conclusions regarding the effects of DCM.

Carcinogenicity

The chemical is classified as 'Carcinogenicity — Category 2 (H351): 'Suspected of causing cancer' in the HCIS (SWA n.d.). The following evidence supports amending this classification to the hazard category 'Carcinogenicity — Category 1B' and the hazard statement 'H350 — May cause cancer'.

DCM is expected to be carcinogenic based on sufficient evidence from animal studies, strong mechanistic evidence and limited human evidence.

In animals, the chemical caused benign and malignant tumours:

- in multiple organs
- in males and females
- in rats and mice.

The available human data have not shown strong or consistent findings for carcinogenicity (IARC 2017). However, several epidemiological studies have shown a positive association between DCM exposure and cancers of the biliary tract and non-Hodgkin lymphoma.

The mode of action for the carcinogenicity of DCM is considered relevant to humans

The carcinogenicity data has been extensively reviewed internationally (ECHA 2025a: ECHA 2025b, IARC 2017, US EPA 2020). A summary of the key animal, human and mechanistic data relevant to GHS hazard classification is provided below.

Animal data

There are 14 carcinogenicity studies in rats, mice, and hamsters using inhalation, oral and IP exposure routes.

The strongest evidence of carcinogenic effects is from the inhalation studies in mice and rats. Benign and malignant tumours were observed at multiple sites in both sexes.

Mice

In mice, there are a total of 3 inhalation studies available for 2 strains (Crj:BDF1 and B6C3F1). In all three studies, there were carcinogenic effects in the lung and liver.

In a 2 year inhalation carcinogenicity study (similar to OECD TG 451) groups of Crj:BDF1 mice (50/sex/concentration), were exposed to dichloromethane (purity 99%) via whole-body inhalation at concentrations of 0, 1000, 2000, or 4000 ppm for 104 weeks (6 h/day, 5 days/week). DCM produced dose-related and statistically significant increases in bronchiolar-alveolar carcinoma and adenoma or carcinoma (combined) and hepatocellular carcinomas and adenomas or carcinomas (combined) in both sexes. The incidences of haemangioma of the liver were significantly increased at the highest dose in males while the incidence of liver haemangioma or haemangiosarcoma (combined) was increased in females (Aiso et al. 2014; ECHA 2025b IARC 2017).

In a 2 year inhalation carcinogenicity study (similar to OECD TG 451) groups of B6C3F1 mice (50/sex/concentration), were exposed to dichloromethane (purity 99%) via whole-body inhalation at concentrations of 0, 2000, or 4000 ppm for 102 weeks (6 h/day, 5 days/week). Dose-related and statistically significant increases in bronchioalveolar adenomas, bronchioalveolar carcinomas, hepatocellular carcinomas and hepatocellular adenoma or carcinoma (combined) were observed in both sexes (NTP 1986).

In a 2 year whole body inhalation study (similar to OECD TG 451) groups of female B6C3F1 mice (68 mice/group), were exposed to 0 or 2000 ppm of DCM (6 h/day, 5 days/week) and only the lungs and liver were evaluated histopathologically. Additionally, a series of stop-exposure treatments (26, 52 or 78 weeks) was conducted to evaluate the role of different DCM exposure durations on the induction of hepatic and pulmonary neoplasia. Continuous exposure to 2000 ppm caused marked increases in incidences of bronchiolo-alveolar adenoma and carcinoma and hepatocellular adenoma and carcinomas. Statistical analysis was applied to combined incidence only. The incidences of bronchiolo-alveolar adenoma or carcinoma (combined), and the incidences of hepatocellular adenoma or carcinoma (combined) were significantly increased in all groups in which exposure occurred during the first 26 weeks of the study. A reduced tumour latency was reported for lung (52 weeks compared to 75 weeks in controls) and liver (52 weeks compared to 83 weeks in controls) tumours (ECHA 2025a; IARC 2017; Kari et al. 1993).

Rats

There are a total of 5 inhalation studies in rats available for 2 strains (F344 and SD). Benign tumours of the mammary gland were observed in 4 of the studies, with effects in both males and females observed in the 3 studies that had exposure doses > 500 ppm. In studies in F344 rats, an increased incidence of fibrosarcoma of subcutaneous tissues and salivary gland sarcoma were observed in males.

In a 2 year inhalation carcinogenicity study (similar to OECD TG 451), groups of F344/DuCrj rats (50/sex/concentration) were exposed to DCM (purity 99.9%) via whole-body inhalation at concentrations of 0, 1000, 2000, or 4000 ppm for 104 weeks (6 h/day, 5 days/week). The incidence of fibroadenoma of the mammary gland was significantly increased in males at the highest dose and with a significant positive trend in females. In male rats, the incidences of fibromas in the subcutis were significantly increased in the 2000 and 4000 ppm exposed groups, with incidences at 4000 ppm exceeding historical control ranges. Subcutis fibromas

in male rats were observed in the mammary chain area but there was no histological evidence that these tumours had developed from the mammary gland. The incidence of mesothelioma of the peritoneum was significantly increased with a positive trend. The incidences at 4000 ppm exceeded the historical control ranges (Aiso et al. 2014, ECHA, 2025b; IARC, 2017; JBRC 2000).

In a 2 year inhalation carcinogenicity study, groups of SD rats (92–97 rats/sex/concentration), aged 8 weeks, were exposed to DCM (purity 99%) via whole-body inhalation at concentrations of 0, 500, 1500, or 3500 ppm for 104 weeks (6 h/day, 5 days/week). Male rats showed increased salivary gland sarcomas, which were statistically significant at the highest concentration. However, the affected animals were infected with the sialodacryoadenitis virus, and the influence of this virus on carcinogenesis remains unknown. A dose-related but not significant increases in benign mammary gland tumours (type not specified) were observed primarily in females with a slight increase noted in males (Burek et al. 1984; ECHA RAC 2025a IARC 2017).

In a 2 year inhalation carcinogenicity study, groups of F344 rats (50 rats/sex/concentration), aged 7–8 weeks, were exposed to DCM (purity 99%) via whole-body inhalation at concentrations of 0, 1000, 2000, or 4000 ppm for 102 weeks (6 h/day, 5 days/week). Exposure to 4000 ppm significantly increased mammary gland adenoma or fibroadenoma (combined) in both sexes. There was a significant positive trend in the incidences of subcutis fibroma or sarcoma (combined) located in the mammary area in males. When the subcutis tumours and the mammary gland tumours were combined for comparative purposes, the incidence in the high dose group was significantly greater than that in the controls (ECHA RAC 2025a; IARC 2017; NTP 1986; Mennear et al. 1988).

In a 2 year inhalation study female SS rats, aged 13 weeks (breeders) or male and female rats at day 12 of gestation (embryos), were exposed to concentrations of 0, 60, or 100 ppm of DCM via whole-body inhalation for 7 hrs/d, 5 days/week for 104 weeks. There was no treatment-related increase in tumour incidence in either breeders or offspring (Maltoni et al. 1988). The IARC Working Group noted the low concentration of exposure in this study (up to 100 ppm) (IARC, 2017).

In an inhalation study SD rats (90 males and 108 females/group) were exposed to concentrations of 0, 50, 200, 500 ppm) of DCM via whole-body inhalation for 6 hrs/d, 5 d/week for 20 (males) or 24 (females) months. Additionally, a series of stop-exposure treatments were conducted in additional groups of 30 females. No significant differences in tumour incidence between control and treated male rats were noted. In females, the incidences of benign tumours of the mammary gland (adenomas and fibroadenomas, combined) were slightly increased compared to controls but the difference was statistically significant only at 200 ppm. The number of benign mammary tumours per rat was slightly increased compared to controls in the 500 ppm group and stop exposure group with exposure to 500 ppm for first 12 months (IARC 2017; Nitschke 1988).

Following evaluation of this data, the IARC concluded that ‘there is *sufficient* evidence for the carcinogenicity of dichloromethane in experimental animals.’ (IARC 2017).

Human studies

Numerous epidemiological cohorts, case-control, and case series studies have investigated the risk of cancer associated with occupational exposure to DCM. Quantitative exposure data for DCM are available only from cohort studies conducted in cellulose triacetate production facilities. While the inclusion of detailed exposure information is a major strength of these studies, the relatively small number of exposed workers represents an important limitation.

Based on a positive association between DCM exposure and cancer of the biliary tract and non-Hodgkin lymphoma (NHL), IARC classified the evidence in humans for the carcinogenicity of DCM as limited (IARC 2017). The European Chemicals Agency (ECHA) Risk Assessment Committee (RAC) also supports the previous evaluation of IARC that “there is limited evidence in humans for the carcinogenicity of dichloromethane.” (ECHA 2025b). This conclusion is mainly based on the confirmed positive association between DCM exposure and cancer of the biliary tract and, to a lesser extent, on evidence concerning non-Hodgkin lymphoma. The observation that the GST-T1 enzyme is expressed at a high level in the nuclei of bile duct epithelial cells in human samples supports the biological plausibility of the association between DCM and liver and biliary tract cancer (EC 2004; US EPA 2011).

Cancers of the liver and biliary tract

Two cohort studies of workers employed in the production of cellulose triacetate fibre exposed to DCM (as well as acetone and methanol, but not 1,2-dichloropropane) in the USA reported findings for cancers of the liver and biliary tract. The study by Lanes et al. 1993 (n=1,271) reported an increased mortality risk for liver and biliary tract cancers with a standardised mortality ratio (SMR) of 2.98 (95% CI: 0.81–7.63) based on four observed cases. All cases occurred among workers with ≥ 10 years of employment and ≥ 20 years since first exposure, resulting in an SMR of 5.83 (95% CI: 1.59–14.92) for this subgroup. Based on a combination of personal and area sampling, median exposure levels (8 hr TWA) in 1977 were reported as 140, 280, and 475 ppm across three main work areas; however, no dose–response analysis was conducted. In their previous report (Lanes et al., 1990), the authors noted that three of the four deaths were biliary tract cancers, for which the SMR was markedly elevated (SMR 20; 95% CI: 5.2–56). The increased cancer risk could not be attributed to co-exposure to methanol and acetone, given their lack of carcinogenic potential. This conclusion aligns with the IARC’s position, which states that these co-exposures are unlikely to explain the observed cancer risk, as they are not associated with liver carcinogenicity. In another study by Gibbs et al 1996 (n=3211) there were 2 deaths attributed to cancer of the biliary tract. The SMR for cancers of liver and biliary tract was not increased (SMR, 0.78; 95 % CI, 0.09–2.81). However, an SMR for cancer of biliary tract alone was not reported and this may be higher than that reported for liver and biliary tract combined (IARC 2017). In addition, the cohort in the Gibbs et al study was employed between 1970 and 1981 and only followed until 1989. In the Lane et. al study, all cases occurred ≥ 20 years since first exposure.

Several investigations evaluated biliary tract cancers among workers in the printing industry in Japan (see Table 1). These exposures took place decades ago (primarily in the early 1990s) and involved workers mainly exposed to 1,2-dichloropropane (1,2-DCP), alongside DCM and multiple other chemicals, making retrospective attribution of risk to a single agent highly complex. These studies recorded from 2 to 17 cases of the very rare cancer, cholangiocarcinoma, and calculated a standard incidence ratio (SIR) value above 1000. Where comparative data were available, risk estimates for biliary tract cancer tended to be higher among workers co-exposed to 1,2-DCP and DCM than among those exposed only to 1,2-DCP. Although, overlapping confidence intervals rendered these differences statistically inconclusive.

Table 1. Cholangiocarcinoma (bile duct cancer) in workers of different printing plants

No of cases	Co-exposures	Comment	Conclusions	Source
11	1,2-DCP (11) and DCM (10).	-	SMR = 2,900.	Kumagai 2013
2	DCM, 1,1,1-trichloroethane and kerosene (1); 1,2-DCP and white gasoline (1).	-	-	Kumagai 2014
17	1,2-DCP (17); also exposed to DCM (11); also exposed to 1,1,1-trichloroethane (8).	111 current or former workers.	Minimal/brief exposure to other chemicals.	Kubo 2014
7	1,2-DCP and DCM (4); DCM only (3).	Estimated maximum exposure concentrations up to 420 ppm 1,2-DCP and up to 720 ppm DCM. DCM only exposures up to 1300 ppm.	DCM may play a role in cholangiocarcinoma development in humans.	Yamada 2015a
6	1,2-DCP and DCM (5) 1,2-DCP only (1).	Estimated shift TWA up to 230 ppm 1,2-DCP and up to 470 ppm DCM. 5 were printing workers, 1 was a coating worker (only exposed to 1,2-DCP).	Supports role of 1,2-DCP in development of cholangiocarcinoma in humans + DCM may have a contributing role.	Yamada 2015b
17	1,2-DCP (6), 1,2-DCP and DCM (11).	106 workers. Age at diagnosis ranged from 20 to 49 years.	Exceptionally high risk of cholangiocarcinoma among workers. SIR = 1132.5. Elevated risk associated with cumulative exposure to 1,2-DCP, while noting that other unidentified substances may have contributed.	Sobue 2015
17	1,2-DCP (6), 1,2-DCP and DCM (11).	Study examining the association between cumulative exposure (ppm-years) to 1,2-DCP and the risk of cholangiocarcinoma among 95 workers. 33 workers were co-exposed to both 1,2-DCP and DCM.	SIR was higher when exposed to both solvents compared to those only exposed to 1,2-DCP (1275 vs 1019). However, because of overlapping confidence intervals, the difference in SIR was not conclusive.	Kumagai 2016

Non-Hodgkin Lymphoma (NHL)

The risk of NHL associated with occupational exposure to DCM has been investigated in a number of studies. All studies, except one cohort study (Hearne and Pifer (1999), reported elevated risks among exposed workers. The cohort mortality study by Radican et al. (2008) involved workers at a U.S. military aircraft maintenance facility. The cohort comprised civilian employees hired between 1952 and 1956 and were followed up until 2000. Workers were exposed to multiple chemicals. Quantitative exposure assessment was conducted for trichloroethylene, while exposure to other agents, including DCM, was assessed qualitatively (ever/never). A total of 1,222 workers were exposed to DCM. Exposure was linked to an increased risk of NHL (hazard ratio [HR] 2.02; 95% CI: 0.76–5.42; 8 exposed cases). A positive association between DCM exposure and NHL occurrence was reported in three case-control studies (Miligi et al. 2006; Seidler et al. 2007; Wang et al. 2009). However, participants were also exposed to various other organic solvents. In the report's summary of human carcinogenicity data, IARC (2017) noted that while positive associations for NHL were consistent across studies of different designs and countries, most subjects had multiple solvent exposures (some previously linked to NHL), and risk estimates were based on small numbers. This association was also suggested in two additional studies (Park et al. 2017; Talibov et al. 2017;) not included in the IARC 2017 evaluation. In Talibov et al. (2017), a significantly increased risk was observed for cumulative DCM exposure ≤ 12.5 ppm-years (OR 1.19; 95% CI: 1.01–1.41) and 12.5–74.8 ppm-years (OR 1.23; 95% CI: 1.01–1.51) among men in an analysis with a 5 year lag, though no dose-response trend was evident. In Park (2017), possible excess risks were noted based on very small numbers, when analysing childhood chronic lymphocytic leukemia (CLL) and DCM exposure within a 3 km radius of emitting facilities. These findings lacked statistical significance.

An association between a CYP2E1 polymorphism and NHL in dichloromethane-exposed individuals has been reported. However, it is uncertain whether this is because of differences in the formation of CYP2E1-mediated metabolites, which may also be reactive, or to a shift in the proportion of GST-mediated reactive metabolites (RAC 2025b; IARC 2017).

Other Cancer Sites

The association between exposure to DCM and various other cancer sites has been investigated in several case-control studies. These included a study on cancer at oesophagus, liver, kidney, colon, rectum, prostate, stomach, pancreas, bladder, melanoma, and NHL (Christensen et al. 2013), as well as studies on breast cancer (Cantor et al., 1995), pancreatic cancer (Kernan et al. 1999), kidney cancer (Dosemeci et al. 1999), and lung cancer (Vizcaya et al. 2012). However, these studies did not report any significant excess risk of cancer, and the evidence for these cancer sites was considered inadequate by the IARC working group (IARC 2017).

Among these, breast cancer entails some specific concern as this neoplasm has been observed in rats exposed to DCM. Two studies (Niehoff et al. 2019; Radican et al. 2008) did report increased risk of breast cancer associated with exposure to DCM, with relatively low HR ratios of 1.21 and 2.35, respectively. However, the caveat of simultaneous exposure to other solvents does not allow a conclusive causal association between exposure to DCM and breast cancer to be established.

Different studies reported association between brain and CNS cancers and exposure to chlorinated solvents. The specific association between these cancers and DCM was in general non-significant or of low significance except one study by Makris 2018 (RAC 2025b). In a retrospective comparative population study conducted by Makris et al. (2018) in an area around a plant using DCM in shoe sole production in Cyprus, after adjusting for age at

diagnosis, the authors reported a standardised incidence ratio for brain/CNS cancer of 6.5 (95% CI, 3.02–12.3). This was based on 8 observed cases versus 1 or 2 expected, among people living or working in the area within a radius of 500 meters from the plant.

Mode of action

DCM is genotoxic in both *in vitro* and *in vivo* studies (see the **Genotoxicity section**).

The observed *in vitro* mutagenicity of DCM cannot be univocally attributed to a specific metabolic pathway. Genotoxicity occurred in bacterial, human, hamster, rat, and mouse cells, with and without the exogenous metabolic activation.

The available information indicates the observed carcinogenicity is linked with the GST-metabolic pathway (reaction with glutathione, initially resulting in the formation of S-chloromethylglutathione, an electrophilic compound that may bind to nucleophilic sites present in DNA constituents). In *in vivo* genotoxicity studies in mice, the strongest responses were observed in sites where DCM is highly metabolised by GSTT-1 (lung and liver tissues), and these sites align with observations of malignant tumours in carcinogenicity studies.

Other mode of actions for the carcinogenicity have been proposed such as effects being caused by persistent hypoxia resulting from DCM oxidation to CO (Andersen 2017). However, the weight of evidence supports a genotoxic mode of action with little evidence for non-genotoxic mechanisms of carcinogenesis with DCM (IARC 2017; RAC 2025b; US EPA 2020).

The genotoxic mode of action for the carcinogenicity of DCM is relevant to humans for the following reasons (ECHA RAC 2025b; IARC 2017; US EPA 2011):

- The relevant enzymes implicated in the carcinogenic pathway exist in humans. Although there are metabolic differences between species, these are considered quantitative not qualitative.
- There are positive *in vitro* genotoxicity studies in human cells without exogenous GSTT1.
- Bile duct epithelial cells in human tissue samples have shown nuclear localisation of GSTT1. This supports the relevance of the MOA to humans given the observation of three biliary tract cancers (a very rare cancer) in a small cohort of DCM exposed workers.
- Unlike rodents, high levels of GSTT1 have been detected in human erythrocytes depending on genetic polymorphism.
- There are polymorphisms in the human population which may lead to differential susceptibility to DCM-related carcinogenesis.
- The CYP2E1 metabolic pathway readily becomes saturated at concentrations of DCM observed in occupational continued exposure to dichloromethane, even at relatively low concentrations.

Human health risk characterisation

Critical health effects are acute lethality and cancer. In addition, the US EPA has based regulatory action on additional chronic non-cancer endpoints including liver, cardiovascular, immune, reproductive and developmental effects. The US EPA considers liver effects to be the critical noncancer effect from chronic DCM inhalation in animals and that preventing chronic adverse liver effects would protect against potential adverse effects where the evidence raises concerns but is incomplete (US FR 2024; US EPA 2022).

Vulnerable subpopulations relating to the risks of DCM have been identified (ATSDR 2000; USEPA; EC 2004; IARC 2017; USEPA 2011; 2020). These include:

- fetuses, babies, young children (differences in CYP2E activity in the developing brain and liver compared to adults, potential for higher exposure due to higher ventilation rate than adults and DCM vapour tends to be higher at floor level)
- people with certain polymorphisms for CYP2E or GSTT1 enzymes
- people with certain co-morbidities (cardiovascular disease and diabetes)
- smokers
- people co-exposed to other chemicals that may induce or inhibit the CYP2E activity (including alcohol, various solvents or medications)
- people who are overweight or obese (may absorb and store more DCM).

Commercial and domestic products

The chemical is present in high concentrations in commercial and domestic products and can be used in relatively large volumes. Products containing DCM are likely to be used by consumers and numerous workers in small businesses. As such, products are often used by people with fewer resources, knowledge or experience in non-industrial settings such as in homes without adequate ventilation or engineering controls, leading to unsafe exposures (US EPA 2014).

The chemical is a highly volatile solvent and can rapidly reach levels that cause lethality, especially in enclosed spaces. Thus, the US EPA evaluated risks to bystanders and non-occupational users in addition to workers and consumers (US EPA 2022). DCM vapour is heavier than air and will therefore tend to accumulate at lower levels in spaces where it is used (EC 2004; Park et al. 2017;).

The use of exposure standards, labelling and PPE requirements internationally have proven to be insufficient to manage risks relating this chemical. Adverse events including DCM-related mortalities have been reported despite the regulatory requirements including classification, labelling and exposure standards (EC 2004; EC 2009; Hoag 2021; US EPA 2022; US FR 2024). SCHER indicates that dermal exposures are important to note given that currently there is no glove material that can completely prevent exposures. Breakthrough times of different gloves were reported to vary considerably ranging from 2 to 150 min for latex and fluoropolymer gloves, respectively. However, the more protective gloves were reported not to be usually used during paint stripping because they were considered to be expensive.

Commonly available vapour cartridge respirators do not provide sufficient protection given the chemical's rapid saturation of filter components. Government authorities from Germany, the UK and the USA recommend the use of independent air supply respirators, although the UK Health and Safety Executive does refer to vapour filter cartridges. These were considered to be the most effective form of respiratory protection equipment when ventilation is not adequate. However, it was reported that consumers and self-employed workers are unlikely to have access to these types of respirators (Hoang et al. 2021; RPA 2007; Safe Work NSW n.d.).

No publicly available reports of adverse events associated with the use of DCM in Australia were identified.

A study analysing all available data regarding DCM-related adverse events in the USA reported 85 fatalities from 1980 to 2018 and 37,201 non-fatalities incidents from 1980 to 2017 (see Table 2 below). However, the authors note that these numbers are likely to be

undercounted because there is no unified data repository in the USA. Not all workers are covered by the national worker protection agency. Consumer fatalities (including fatal cardiovascular events) associated with DCM exposure have been reported in the media. In some cases, these events may not be recognised as being attributable to chemical exposure.

Consumer and worker fatalities were reported for uses of paint strippers, cleaning and degreasing solvents and adhesives and sealants. Fatalities were reported despite the use of respiratory equipment. Three of the fatalities involved people who did not use the product but entered a room where vapours persisted after DCM had been used. The following trends were reported despite the decrease in the national exposure standard (8 hr TWA) from 500 to 25 ppm in 1997 (Hoag et al. 2021):

- There has been no linear trend indicating a decrease in fatalities during that time period (US FR 2024).
- The proportion of occupational fatalities related to paint stripping increased from 22 (55%) before 2000 to 30 (88%) after 2000.
- The proportion of occupational fatalities associated with bathtub or paint stripping in bathrooms increased from 2 (5%) before 2000 to 21 (62%) after 2000.
- The annual number of cases of non-fatalities decreased starting in the late 1990s.

Table 2. DCM-related fatalities identified in the USA, 1980-2018*

Characteristics	Occupational fatalities (n = 74)	Consumer fatalities (n = 11)	Fatalities with autopsy (n = 23)
Year of fatality	-	-	-
Total No. reported	74	11	23
1980-1989	32 (43)	2 (18)	NA
1990-1999	8 (11)	3 (27)	NA
2000-2009	14 (19)	1 (9)	8 (35)
2010-2018	20 (27)	5 (46)	15 (65)
Product used	-	-	-
Total No. reported	67	11	23
Paint stripper	52 (78)	8 (73)	23 (100)
Cleaning/degreasing solvent	12 (18)	1 (9)	NA
Adhesive/sealant	3 (4)	2 (18)	NA
Setting of incidents	-	-	-
Total No. reported	67	10	23
Bathroom	23 (34)	3 (30)	19 (82)
Industrial equipment	20 (30)	NA	2 (9)
Bystander onsite	3 (4)	NA	NA
Other	21 (32)	7 (70)	2 (9)
Respirator use	-	-	-
Total No. reported	36	4	21
No respirator used	20 (56)	2 (50)	13 (62)
Respirator used but not NIOSH** approved	16 (44)	2 (50)	8 (38)

*Adopted from Hoang et al. (2021)

**National Institute for Occupational Safety and Health (NIOSH)

Occupational mortalities linked to DCM have been recorded as recently as 2020, 2021 and 2023 (US FR 2024).

In the UK, DCM is reported to be implicated in a number of prosecutions each year, including some mortalities (HSENI, n.d.). The number of cases and related end uses were not reported.

The USA and EU reported on difficulties getting DCM-related mortality and poisoning data (Hoang et al. 2021; RPA 2007). The EU noted that there is no evidence to suggest that the risk profile of DCM would differ from the USA.

In Australia the chemical is widely available for use by the public and workers including at high concentrations (see **Introduction and use** section). The chemical is listed on Schedule 5 of the SUSMP. For preparations containing more than 10 % of the chemical, a number of warning statements, first aid instructions and safety directions apply. Internationally including in Europe, USA, China and Canada the chemical has been prohibited or restricted for use by public and workers (see **International regulatory controls**). A large focus of the international action relates to use in paint stripper products, with use by the public prohibited and use by workers either prohibited or restricted to workers with adequate training.

The USA completed a risk evaluation for the remaining uses of the chemical and determined that 52 out of 53 conditions of use of the chemical presented unreasonable risks (US EPA 2020). As a result, the USA expanded restrictions to cover a broader range of professional uses. It addressed this risk by prohibiting most uses of DCM apart from highly restricted circumstances where users can demonstrate compliance with a number of requirements including an ECEL of 2ppm (8 hr TWA) and a WCPP. The approach to managing potential risks associated with the use of DCM is continuing to evolve in the USA.

Cosmetics

DCM has historical use in cosmetics. There is some evidence of continued use of DCM as an ingredient. In addition, in the USA, high concentrations of DCM were identified in nail products that did not list the chemical on the label. The use of the chemical in cosmetics is prohibited in several jurisdictions (see **International Regulatory Controls**). Cosmetics containing DCM in Australia are required to be labelled with caution (see Existing Australian regulatory controls), which is likely to limit use of the chemical as an intentional ingredient. However, reports from overseas have identified instances where DCM was not declared on cosmetic product labels, and it is unknown whether similar instances may occur in Australia.) Overall, there is a potential risk to the public that requires management.

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