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CHEMICAL SUMMARY FOR METHYLENE CHLORIDE (DICHLOROMETHANE)

prepared by
OFFICE OF POLLUTION PREVENTION AND TOXICS
U.S. ENVIRONMENTAL PROTECTION AGENCY
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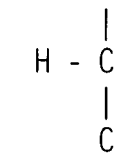
This summary is based on information retrieved from a systematic search limited to secondary sources (see Appendix A). These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials. No attempt has been made to verify information in these databases and secondary sources.

I. CHEMICAL IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

The chemical identity and physical/chemical properties of methylene chloride are summarized in Table 1.

TABLE 1. CHEMICAL IDENTITY AND CHEMICAL/PHYSICAL PROPERTIES OF METHYLENE CHLORIDE

Characteristic/Property	Data	Reference
CAS No.	75-09-2	
Common Synonyms	MC, dichloromethane, DCM, methylene bichloride, methylene dichloride	
Molecular Formula	CH2Cl2	
Chemical Structure		
Physical State	colorless liquid	Budavari et al. 1989
Molecular Weight	84.94	Budavari et al. 1989
Melting Point	-95øC	Budavari et al. 1989
Boiling Point	39.75øC at 760 mm Hg	Budavari et al. 1989
Water Solubility	1.32 x 104 mg/L at 20øC	U.S. Air Force 1989
Density	d20/4, 1.3255 g/mL	Budavari et al. 1989
Vapor Density (air = 1)	2.93	NIOSH 1986
KOC	25	ATSDR 1993
Log KOW	1.25	U.S. Air Force 1989
Vapor Pressure	400 mm Hg at 24.1øC	HSDB 1994

Reactivity		
Flash Point	Nonflammable	U.S. Air Force 1989
Henry's Law Constant	2.57 x 10-3 atm m3/mol	U.S. Air Force 1989
Fish Bioconcentration Factor	2 (estimated)	U.S. EPA 1984
Odor Threshold	214 ppm (in air)	U.S. Air Force 1989
Conversion Factors	1 ppm = 3.48 mg/m3;	
	1 mg/m3 = 0.288 ppm	U.S. Air Force 1989

II. PRODUCTION, USE, AND TRENDS

A. Production

There are 3 manufacturers with 5 plants producing methylene chloride in the United States: Dow Chemical, Occidental Chemical, and Vulcan Chemicals. Annual capacity is about 527 million pounds. In 1992, approximately 350 million pounds of methylene chloride were produced in the US. Since 1988, production of methylene chloride has fallen at an average rate of 8.6 percent per year. Imports do not account for much of U.S. methylene chloride consumption. In 1992, it was estimated that 13 million pounds of methylene chloride were imported into the US (Mannsville, 1993).

B. Use

Methylene chloride is used in many applications. Its largest use is as the principal active ingredient in organic-based paint strippers, accounting for approximately 125 million pounds (35 percent of all U.S. methylene chloride production in 1992). It is used in both consumer and industrial paint removers, where it is commonly present in 60-80% concentration. The second largest application (88 million pounds) of methylene chloride is in chemical processing. Table 2 presents the estimated 1992 end use pattern for methylene chloride.

C. Trends

Methylene chloride demand is expected to continue to decline by over 30% by 1995 because of environmental and occupational concerns. Users of methylene chloride have been under strong pressure to limit usage, restrict emissions, maximize recycling, and reduce worker exposure (Mannsville, 1993).

III. ENVIRONMENTAL FATE

A. Environmental Release

Of the total methylene chloride released to the environment, about

86% is released to the atmosphere, 12% to land, and 2% to water (ATSDR 1993). The highly volatile methylene chloride [vapor pressure, 400 mm Hg at 24.1øC (HSDB 1994)] is released into the atmosphere from industrial and consumer uses (ATSDR 1993). The principal releases of methylene chloride to land are from the disposal of methylene chloride products and containers in landfills (ATSDR 1993). The principal releases to surface water and potentially to groundwater are via industrial effluents and underground injections, respectively (ATSDR 1993). In 1992, releases of methylene chloride to environmental media, as reported to the Toxic Chemical Release Inventory by certain types of U.S. industries, totaled about 74 million pounds to the atmosphere, 221 thousand pounds to surface water, 1 million pounds to underground injection, and 79 thousand pounds to land (TRI88 1990). In addition, an estimated 243 million pounds of methylene chloride were released to the atmosphere in 1988 from consumer products and other sources, such as hazardous waste sites (ATSDR 1993). Maximum levels of methylene chloride measured in environmental media are 6.7 micrograms/m3 (urban U.S. air), 39 micrograms/m3 (air around hazardous waste sites), 743 æg/L (New Jersey surface water), 3600 micrograms/L (groundwater), and 13 micrograms/L (sediment) (ATSDR 1993).

TABLE 2. ESTIMATED 1992 U.S. END USE PATTERN OF METHYLENE CHLORIDE

Use of Methylene Chloride [typical Standard Industrial [Classification (SIC) code] (see end note 1)	1992 US Consumption (in millions of pounds)	Percentage of US Methylene Chloride Use
Paint Removers (production, SIC 2851; use, various industries)	125	35
Chemical Processing (SIC 2821, 2823)	88	25
Foam Blowing (various industries)	49	14
Metal Cleaning & Finishing (various industries)	28	8
Aerosols (various industries)	25	7
Adhesives and Coatings (production, SIC 2851; use, various industries)	14	4
Electronics (SIC 3672)	11	3

Miscellaneous (see end note 2)	14	4
TOTAL	354	100

Source: Mannsville 1993

B. Transport

Methylene chloride tends to volatilize to the atmosphere from the water and soil (ATSDR 1993; U.S. Air Force 1989). With an organic carbon partitioning coefficient (KOC) of 25, methylene chloride is expected to be highly mobile in soils, and leaching of the chemical into groundwater is likely (ATSDR 1993). In deep, saturated soils that contain no soil air and negligible soil organic carbon, as much as 96% of environmental methylene chloride may be present in the soil-water phase and transported with flowing groundwater (U.S. Air Force 1989).

C. Transformation/Persistence

1. Air - The main degradation pathway for methylene chloride in the atmosphere is reaction with photochemically produced hydroxyl radicals (ATSDR 1993). The estimated lifetime for the chemical in the atmosphere is 130 days. The small amount of methylene chloride reaching the stratosphere (about 1%) would probably undergo direct photolysis (ATSDR 1993).

2. Soil - In surface soil, volatilization to air is an important fate process for methylene chloride (U.S. Air Force 1989; ATSDR 1993). The biodegradation of methylene chloride has been demonstrated in the laboratory and may occur in subsurface soils (ATSDR 1993); however, biodegradation (except, perhaps in landfills with active microbial populations) is probably not a significant degradation pathway in the soil-groundwater system (U.S. Air Force 1989). It has been suggested that microorganisms used in biological sewage treatment can degrade the chemical with suitable acclimatization (U.S. Air Force 1989).

3. Water - An important fate process for methylene chloride is volatilization; in the laboratory, the volatility half-life was estimated to be 21 minutes (ATSDR 1993). Methylene chloride has been shown to hydrolyze slowly (experimental half-life in water at 25øC, ~18 months) (ATSDR 1993).

4. Biota - The estimated fish bioconcentration factor (BCF) for methylene chloride of 2 (U.S. EPA 1984) suggests that biomagnification of the chemical in the aquatic and terrestrial

food chains is not likely.

IV. HEALTH EFFECTS

A. Pharmacokinetics

1. Absorption - Studies in humans and animals have demonstrated that methylene chloride is readily absorbed via the lungs and the gastrointestinal tract (U.S. EPA 1984; ATSDR 1993). Limited animal data indicate some skin absorption (ATSDR 1993).

2. Distribution - In studies with animals exposed to radiolabeled methylene chloride, radioactivity was detected in the liver, kidneys, lungs, brain, muscle, adipose tissues, and adrenals about 1 hour after inhalation exposure; and in the liver, kidneys, lungs, brain, epididymal fat and testes 48 hours after single oral doses (ATSDR 1993).

3. Metabolism - The major metabolites of inhaled and ingested methylene chloride are carbon dioxide probably via glutathione transferase and carbon monoxide probably via mixed function oxidases (ATSDR 1993). Elevated levels of carboxyhemoglobin (COHb) have been observed in exposed human subjects and animals (ATSDR 1993).

4. Excretion - Methylene chloride and its metabolites are excreted primarily in expired air. Small amounts are also eliminated in the urine and feces. Following inhalation exposure, exhaled air contained 58 to 79% of a dose of radiolabeled methylene chloride.

B. Acute Effects

Humans acutely exposed to methylene chloride experience adverse effects of the central nervous system and the heart. Animal studies indicate acute exposures to high levels of methylene chloride can adversely affect the liver and the kidney.

1. Humans - Direct contact with methylene chloride causes corneal burns and erythema and burning of the skin (ATSDR 1993). The lowest lethal ingested dose of methylene chloride reported for humans is 357 mg/kg (RTECS 1993). Occupational overexposure to methylene chloride (concentrations were not reported) has resulted in worker deaths (ATSDR 1993). Exposure to 500 ppm methylene chloride for 8 hours produced euphoria in humans (RTECS 1994). The concentration of 500 ppm is roughly equivalent to a total of 248 mg/kg over an 8-hour period (see end note 3). Methylene chloride is metabolized to carbon monoxide in humans, resulting in the formation of carboxyhemoglobin (COHb) and subsequent oxygen

deprivation (U.S. Air Force 1989). The formation of COHb is concentration-dependent and saturable. Human subjects exposed to 100 ppm for 7.5 hours developed COHb levels of >5%, subjects exposed to 500 ppm for 1 hour had COHb levels of 1-4%, and subjects exposed to 1000 ppm for 2 hours had COHb levels of 10% (saturation) (ATSDR 1993). A concentration of 2.5% COHb is associated with impairment of time-interval discrimination, whereas levels of ò5% COHb are associated with other psychomotor effects and cardiovascular changes (Amdur et al. 1991). The cardiovascular changes include increased cardiac output, A-V oxygen difference, and coronary blood flow in patients without coronary disease. Patients with coronary heart disease and elevated COHb levels may experience decreased coronary sinus blood PO2 and impaired oxidative metabolism of the myocardium, producing an added burden on the patient (Amdur et al. 1991).

2. Animals - LD50/LC50 values for rats, reported in the literature searched, are as follows for the various routes of exposure: 2100 mg/kg (oral) (ATSDR 1993), 25,287 ppm for 30 min (respiratory), and 916 mg/kg [intraperitoneal (i.p.) injection] (RTECS 1994). LD50/LC50 values for mice are slightly lower. Signs and symptoms of the acute toxicity of methylene chloride include (a) liver damage in mice exposed orally to 133 to 665 mg/kg (IARC 1986), in rats exposed by inhalation to 552 ppm, 6 hours/day for 5 days, and in guinea pigs exposed by inhalation to 5200 ppm for 6 hours; and (b) kidney damage in dogs and mice injected i.p. with "near-lethal doses" and in rats injected i.p. with 1330 mg/kg (IARC 1986). In animals, as in humans, methylene chloride is metabolized to carbon monoxide, resulting in the formation of carboxyhemoglobin (COHb) and subsequent oxygen deprivation (U.S. Air Force 1989). The formation of COHb is concentration-dependent and saturable. COHb levels reached 13% in rats exposed to 500 ppm methylene chloride for 6 hours, but no further increase in COHb occurred after 6 hours' exposure to higher concentrations up to 1500 ppm (U.S. Air Force 1989).

C. Subchronic/Chronic Effects

Humans chronically exposed to methylene chloride experience adverse effects of the central nervous system and the heart. Animal studies indicate chronic exposures to high levels of methylene chloride adversely affects the liver and the kidney. EPA has derived an oral RfD (reference dose) (see end note 4) of 0.06 mg/kg/day for methylene chloride.

1. Humans - Available information suggests that the central nervous system and the cardiovascular system are affected by subchronic or chronic exposure to methylene chloride. Two workers were exposed intermittently for 13 to 20 years; one reported leg and arm pain, dizziness, fatigue, loss of appetite, and poor night vision; and the other reported drowsiness, headache, and tingling of hands and feet (U.S. Air Force 1989). Deaths occurring following chronic inhalation exposure have been attributed to cardiac injury and heart failure (U.S. EPA 1984). No other target organs were identified.

2. Animals - The major target organs for the subchronic/chronic toxicity of methylene chloride are the liver and kidney. Methylene chloride, administered to F344 male and female rats in their drinking water for 2 years, induced histological alterations of the liver at doses ò50 mg/kg/day; the no-observed-adverse effect level (NOAEL) for the study was 5 mg/kg/day (U.S. EPA 1994). Based on these data, the U.S. EPA (1994) calculated an oral RfD of 0.06 mg/kg/day for methylene chloride. In inhalation studies with methylene chloride, cytoplasmic vacuolization and fatty infiltration of the liver and tubular degeneration and regenerative changes of the kidneys were observed in rats exposed continuously to 25 or 100 ppm of the chemical for 100 days (ATSDR 1993). Rats exposed to 500 ppm methylene chloride, 6 hours/day, 5 days/week for 2 years developed multinucleated hepatocytes (the liver was not affected at 200 ppm), and increased hemosiderosis, cytomegaly, and cytoplasmic vacuolization of the liver after exposure to 1000 ppm, 6 hours/day, 5 days/week for 2 years (ATSDR 1993). IARC (1986) reported that Sprague-Dawley rats exposed to ò500 ppm methylene chloride for 2 years exhibited pathological changes in the liver and kidney more frequently than did control animals.

D. Carcinogenicity

There is inadequate evidence of carcinogenicity of methylene chloride in humans. There is sufficient evidence of its carcinogenicity in animals. EPA has classified methylene chloride as a probable human carcinogen.

1. Humans - Epidemiology studies revealed no increased risk for cancer among workers who were exposed to methylene chloride for up to 22 years (IARC 1986; HSDB 1994). Exposure levels of the chemical ranged (for several studies) from 26 to 475 ppm.

2. Animals - Various inhalation bioassays conducted in rodents, exposed to methylene chloride concentrations up to 3500 or 4000 ppm 6 hours/day, 5 days/week for approximately 2 years, showed

statistically significant increases in the following types of tumors: benign mammary tumors and sarcomas in rats; alveolar/bronchiolar adenomas, alveolar/bronchiolar carcinomas, and hepatocellular adenomas in mice; and lymphosarcomas in hamsters (IARC 1986). Hepatocellular carcinoma and neoplastic nodules were observed in female rats and male mice in the 2-year drinking water study described in the Suchronic/Chronic Section. Based on inadequate human data and sufficient evidence for carcinogenicity in animals, the U.S. EPA classification for methylene chloride is B2, probable human carcinogen (U.S. EPA 1994). The oral slope factor (see end note 5) for methylene chloride is 0.0075 per (mg/kg)/day (U.S. EPA 1994). The inhalation unit risk (see end note 6) for methylene chloride is 4.7 x 10-7 per (micrograms/m3) (U.S. EPA 1994). IARC (1987) classifies the chemical as 2B, possibly carcinogenic to humans, and the National Toxicology Program (NTP 1994) concluded that methylene chloride shows some evidence of carcinogenicity in male rats and clear evidence of carcinogenicity in female rats and in male and female mice.

E. Genotoxicity

In the EPA GENETOX Program, methylene chloride was positive for cell transformation in rat embryo cells, mitotic recombination or gene conversion in Saccharomyces cerevisiae, reverse gene mutation in S. cerevisiae, and histidine reversion in the Ames assay; the chemical was negative in the micronucleus test and the sex-linked recessive lethality assay in D. melanogaster (GENETOX 1994). USEPA (1994) reports methylene chloride as mutagenic in Salmonella and negative in gene mutation and chromosome aberration tests in mammalian cells.

F. Developmental/Reproductive Toxicity

There is no information on the developmental toxicity or reproductive system effects of methylene chloride in humans. Information on these effects in animals is limited; no conclusions on the can be made on the developmental toxicity or reproductive system effects of methylene chloride from this information.

1. Humans - No information was found in the secondary sources searched to indicate that methylene chloride is a developmental/reproductive toxicant in humans.

2. Animals - No compound-related effects were observed in Charles River CD male and female rats or their offspring, given oral doses of up to 225 mg methylene chloride/kg/day in a two-generation reproductive toxicity study (HSDB 1994). In inhalation studies, the offspring of pregnant mice and rats exposed to

1250 ppm (7 hours/day) or 4500 ppm (exposure details not given) of methylene chloride during gestation exhibited treatment-related reductions in fetal weight, skeletal anomalies and/or alterations in spontaneous locomotor activities (IARC 1986; ATSDR 1993). There was no indication of fetal malformations in these studies, even at maternally toxic concentrations. No compound-related effects were observed in Fischer 344 male and female rats or their offspring exposed to methylene chloride concentrations of up to 1500 ppm (exposure details not given) in a two-generation reproductive toxicity study (HSDB 1994).

G. Neurotoxicity

Human and animal studies indicate that methylene chloride adversely affects the central nervous system.

1. Humans - Inhalation of 300 to 800 ppm for 4 hours has caused impairment of visual, auditory, and psychomotor functions; the effects were reversible at lower concentrations (ATSDR 1993). Inhalation of 515 ppm for 1 to 2 hours and 1000 ppm for 1 to 2 hours resulted in decreased visual evoked response; exposure to <500 ppm for 1-2 hours had no effect on visual evoked response (ATSDR 1993). In an additional case study of longer-term exposure, a chemist who breathed 660 to 3600 ppm (mean, 900 ppm in the breathing zone) for 5 years experienced forgetfulness, insomnia, and auditory and visual hallucinations (U.S. EPA 1984).

2. Animals - Various inhalation studies with methylene chloride in cats have demonstrated various effects ranging from slight narcosis (6000 ppm for 3 to 4 hours) to deep CNS depression (10,000 ppm for 293 min) (HSDB 1994). Gerbils exposed to 210 ppm for 7 to 16 weeks (exposure details not given) exhibited decreased hippocampal DNA concentration and alterations in brain amino acids (ATSDR 1993).

V. ENVIRONMENTAL EFFECTS

A. Toxicity to Aquatic Organisms

Methylene chloride has low acute toxicity to aquatic organisms; lethal concentrations are generally greater than 100 mg/L. Ninety-six-hour LC50 values for fish are 193 mg/L for Pimephales promelas (fathead minnow; flowthrough conditions) and 220 mg/L for Lepomis macrochirus (bluegill; static conditions) (AQUIRE 1993). The 14-day LC50 for Poecilia reticulata (guppy) is 294 mg/L (AQUIRE 1993). In Scenedesmus quadricauda (green algae), the toxicity threshold for cell multiplication inhibition test (TT) and for mortality (static conditions) is 1,450 mg/L (AQUIRE 1993).

B. Toxicity to Terrestrial Organisms

No information was found in the secondary sources searched for terrestrial organism toxicity. The oral LD50 in the rat, 2100 mg/kg (ATSDR 1993), suggests that the chemical would not be acutely toxic to terrestrial animals unless present in very high concentrations. Studies in laboratory animals also suggest that methylene chloride would not cause developmental/reproductive effects in terrestrial species at expected environmental levels (see section IV.F).

C. Abiotic Effects

The reaction of methylene chloride with ozone in the upper atmosphere is not expected to be significant. Most methylene chloride in the lower atmosphere is removed by reaction with hydroxyl radicals (ATSDR 1993).

VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

Voluntary reduction of methylene chloride environmental releases has occurred since 1991, as a result of a joint industry/EPA pollution prevention initiative known as the 33/50 program. The 1990 Clean Air Act Amendments list methylene chloride as a hazardous air pollutants.

Workplace exposure to methylene chloride is primarily regulated by the Occupational Safety and Health Administration (OSHA); it has proposed a reduction of the permissible exposure limit (PEL) to 25 ppm. Other federal agencies and groups (listed in Table 4) have developed recommendations to assist in controlling workplace exposure.

Regarding consumer exposure, the Consumer Product Safety Commision (CPSC) requires that all consumer products containing more than 1% methylene chloride carry a label warning that the contents may cause cancer. Also, the Food and Drug Administration (FDA) in 1989 banned the use of the chemical in hairspray and other cosmetic products.

Federal agencies and other groups that can provide additional information on methylene chloride are listed in Tables 3 and 4.

TABLE 3. EPA OFFICES AND CONTACT NUMBERS
FOR INFORMATION ON METHYLENE CHLORIDE

EPA OFFICE	LAW	PHONE NUMBER

Pollution Prevention & Toxics	Toxic Substances Control Act (Sec. 8A/8D/8E)	(202) 554-1404
	Emergency Planning and Community Right-to-Know Act (EPCRA) Regulations (Sec. 313)	(800) 535-0202
	Toxics Release Inventory data	(202) 260-1531
Air	Clean Air Act	(919) 541-0888
Solid Waste & Emergency Response	Comprehensive Environmental Response, Compensation, and Liability Act (Superfund)/ Resource Conservation and Recovery Act / EPCRA (Sec. 304/311/312)	(800) 535-0202
Water	Clean Water Act	(202) 260-7588
	Safe Drinking Water Act (Drinking Water Standard: 0.005 mg/L)	(800) 426-4791

Agency for Toxic Substances and Disease Registry	(404) 639-6000
American Conference of Governmental Industrial Hygienists	(513) 742-2020
Consumer Product Safety Commission	(301) 504-0994
Food and Drug Administration	(301) 443-3170
National Institute for Occupational Safety and Health (NIOSH)	(800) 356-4674
Occupational Safety and Health Administration (Check your local phone book under U.S. Department of Labor)	

TABLE 4. OTHER FEDERAL OFFICE/OTHER GROUP CONTACT NUMBERS FOR INFORMATION ON METHYLENE CHLORIDE

Other Agency/Department/Group	Contact Number
Agency of Toxic Substances & Disease Registry	(404) 639-6000
American Conference of Governmental Industrial Hygienists (Recommended Exposure Limit (see end note 7): 50 ppm)	(513) 742-2020
Consumer Product Safety Commission	(301) 504-0994
Food & Drug Administration	(301) 443-3170
National Institute for Environmental Health Sciences (EnviroHealth Clearinghouse)	(800) 643-4794
National Institute for Occupational Safety & Health	

(Recommended Exposure Limit (see end note 8): Lowest Feasible Concentration)	(800) 356-4674
Occupational Safety & Health Administration (Proposed Permissible Exposure Limit (see end note 9): 25 ppm)	Check local phone book for phone number under Department of Labor

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VII. END NOTES

1. The Standard Industrial Classification (SIC) code is the statistical classification standard for all Federal economic statistics. The code provides a convenient way to reference economic data on industries of interest to the researcher. SIC codes presented here are not intended to be an exhaustive listing; rather, the codes listed should provide an indication of where a chemical may be most likely to be found in commerce.

2.Miscellaneous uses of methylene chloride include use as an extraction solvent (including the extraction of heat-sensitive substances such as caffeine, cocoa, and edible fats); and processing of cellulose triacetate fiber.

3. Calculated using the factor, 3.48 (U.S. Air Force 1989), to convert 500 ppm to 1740 mg/m3 which is multiplied by 0.143 (the standard occupational 8-hour breathing rate, 10 m3, divided by the assumed adult body weight, 70 kg) to obtain the dose in mg/kg (U.S. EPA 1988).

4. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during the time period of concern.

5. The slope factor is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The slope factor is used in risk assessments to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen.

6. The unit risk is a quantitative estimate in terms of risk per unit intake of a chemical. The unit risk for methylene chloride incorporates information on pharmacokinetics and metabolism.

7. The ACGIH exposure limit is a time-weighted average (TWA) concentration for an 8-hour workday during a 40-hour workweek.

8. Exposure should be reduced to the lowest feasible limit; use of only the

most reliable and protective respirators is recommended.

9. The OSHA exposure limit is a time-weighted average (TWA) concentration that must not be exceeded during any 8-hour workshift during a 40-hour workweek.

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APPENDIX A. SOURCES SEARCHED FOR FACT SHEET PREPARATION

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