0418
Cis-1,2-Dichloroethylene; CASRN 156-59-2; 00/00/0000

Human health assessment information on a chemical substance is included in IRIS only after a comprehensive review of toxicity data by U.S. EPA health scientists from several program offices, regional offices, and the Office of Research and Development. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the positions that were reached during the review process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website at http://www.epa.gov/iris/backgrd.html.

STATUS OF DATA FOR cis-1,2-Dichloroethylene (cis-1,2-DCE)

File First On-Line 12/01/1990

Category (section)	Status	Last Revised
Chronic Oral RfD Assessment (I.A.)	on-line	00/00/0000
Chronic Inhalation RfC Assessment (I.B.)	discussion	00/00/0000
Carcinogenicity Assessment (II.)	on-line	00/00/0000

_I. HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE

Substance Name – cis-1,2-Dichloroethylene (cis-1,2-DCE)
CASRN – 156-59-2
Section I.A. Last Revised – 00/00/0000

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action. It is expressed in units of mg/kg-day. Please refer to the guidance documents at http://www.epa.gov/iris/backgrd.html for an elaboration of these concepts. Because RfDs can be derived for the noncarcinogenic health effects of substances that are also carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity

of this chemical substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

An oral RfD for cis-1,2-DCE was not previously available on IRIS.

___I.A.1. CHRONIC ORAL RfD SUMMARY

Critical Effect	Point of Departure*	UF	Chronic RfD
Increased relative kidney weight Subchronic oral rat study McCauley et al. (1995, 1990)	$BMDL_{10}$: 5.1 mg/kg-day	3,000	0.002 mg/kg-day

*Conversion Factors and Assumptions – The $BMDL_{10}$ is the lower confidence limit on the benchmark dose (BMD_{10}) corresponding to a 10% increase in relative kidney weight compared with controls.

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES

McCauley et al. (1995, 1990) administered 0, 32, 97, 291, or 872 mg/kg-day cis-1,2-DCE by corn oil gavage to male and female Sprague-Dawley rats (10 rats/sex/group) for 90 days. At the end of the 90-day exposure period, animals were sacrificed and the brain, gonads, heart, kidneys, adrenals, liver, spleen, and thymus were weighed and examined for gross pathology. Blood samples were collected for hematological and clinical chemistry examinations. Tissues from controls and the high-dose group animals were examined for histopathologic changes.

Clinical observations during the study were reported by the authors as minimal and not compound-related. Gavage deaths were present in both the treated and control groups (1/10 female rats at 32 mg/kg-day; 1/10 female rats at 97 mg/kg-day; 1/10 male controls; 3/10 male rats at 291 mg/kg-day; 4/10 male rats at 872 mg/kg-day). Terminal body weights in male rats at the two highest dose groups were lower than controls by 10–11%, but were not considered by the author as statistically significant; no treatment-related effects on body weight were reported in female rats.

Absolute liver weights were statistically significantly increased by 10, 15 and 24% in female rats at doses of 97, 291 and 872 mg/kg-day, respectively. The increases in absolute liver weight of 6, 13, 5 and 15% in male rats of the 32, 97, 291 and 872 mg/kg-day dose groups, respectively, were not statistically significant nor dose related. Relative liver weights were statistically significantly increased in a dose-related manner in males and females. The increases were 15, 17, and 32% for males and 14, 19, and 30% for females at 97, 291, and 872 mg/kg-day, respectively. Histopathological evaluation revealed no specific hepatic injury. The authors concluded that there was a consistent, dose-related increase in relative liver weight in both sexes and that this effect, in light of the negative histopathology findings, may reflect hypertrophy and hyperplasia.

Absolute kidney weights in female rats were increased by 3, 16, 17, and 17% compared to the

control at doses of 32, 97, 291 and 872 mg/kg-day, respectively, but were not statistically significant. In male rats increases in absolute kidney weight of 9, 17, 7 and 14% for the 32, 97, 291 and 872 mg/kg-day dose groups, respectively, were not statistically significantly elevated compared to the control nor dose related. Statistically significant increases in relative kidney weights were recorded in male rats in all dose groups (14, 19, 19, and 27% at 32, 97, 291, and 872 mg/kg-day, respectively). Female rats exhibited increased (although not statistically significant) relative kidney weights in the three highest doses (19, 23, and 23% at 97, 291, and 872 mg/kg-day, respectively). Relatively large variances in the female dose groups may explain why relative kidney weight increases in females were not statistically significant. Histopathological findings for kidney effects were negative, leading the authors to hypothesize that the increases in relative kidney weight may be due at least in part to decreased body weight gain.

Sporadic changes (although noted as statistically significant) in some clinical chemistry parameters were observed. Blood urea nitrogen (BUN) levels were significantly decreased (40%) at the highest dose in males but not in females. Serum calcium levels were significantly elevated by 8 and 10% in males at the 32 and 97 mg/kg-day doses, respectively, and serum phosphorus was significantly decreased by 14% in males exposed to 32 mg/kg-day. In females, serum phosphorus was significantly increased by 34 and 25% in the groups dosed with 97 and 291 mg/kg-day, respectively. No significant changes were reported in AST activity. Hemoglobin and hematocrit level, and red blood cell (RBC) count were significantly decreased in female rats dosed at 291 mg/kg-day, while only hematocrit was significantly decreased in females dosed with 872 mg/kg-day. In males, similar decreases (ranging from 6 to 10% compared with the control) occurred in hemoglobin in the 291 and 872 mg/kg-day groups and in hematocrit in the 97, 291, and 872 mg/kg-day groups. Overall the changes in clinical chemistry and hematology parameters were considered by the authors to be marginal and of questionable biological significance. No noteworthy compound-related histopathological changes were observed in any dose group.

Method of Analysis. Increased relative kidney weight in male and female rats (McCauley et al. (1995, 1990) was identified as the critical effect. Benchmark dose (BMD) modeling methodology (U.S. EPA, 2000) was used to determine the point of departure (POD) by estimating the effective dose at a specified level of response (BMDx) and its 95% lower confidence limit (BMDLx). A 10% change in relative kidney weight compared with the control was selected as the benchmark response (BMR) level. A BMR of 10% change in relative kidney weight was selected by analogy to body weight, for which a 10% change is generally recognized as a minimally biologically significant change (U.S. EPA, 2000).

All of the models for continuous data (i.e., linear, polynominal, power, and Hill models) in U.S. EPA's BMDS (version 2.1) were fit to relative kidney weight data. For the male rat, BMDS modeling of relative kidney weight data showed that only the Hill model adequately fit the data (test 4 χ^2 $p > 0.1$). The other continuous models fit to these data, the polynomial (linear and degree>2) and power models, exhibited significant lack of fit. The Hill model predicted a BMD_{10} and $BMDL_{10}$ of 19.8 and 5.1 mg/kg-day, respectively. For the female rat, the Hill model provided the best fit of the relative kidney weight data (based on the model with the lowest Akaike Information Criteria (AIC) value and adequate visual fit of the data). The Hill model predicted a BMD_{10} and $BMDL_{10}$ of 55.2 and 10.4 mg/kg-day, respectively. The POD for the RfD for cis-1,2-DCE based on kidney weight changes was chosen as 5.1 mg/kg-day, the lower of the male and female $BMDL_{10}$ values.

___I.A.3. UNCERTAINTY FACTORS

UF = 3,000

An intraspecies UF (UF_H) of 10 was applied to account for potentially sensitive human subpopulations in the absence of quantitative information on the variability of response to cis-1,2-DCE in the human population.

An interspecies UF (UF_A) of 10 was applied to account for the variability in extrapolating from laboratory animals to humans. No information was available to characterize the toxicokinetic or toxicodynamic differences between experimental animals and humans for cis-1,2-DCE.

An UF of 1 was used for extrapolation from a LOAEL to a NOAEL (UF_L) because the current approach is to address this factor as one of the considerations in selecting a BMR for BMD modeling. In this case, a BMR of a 10% change in relative kidney weight compared with the control was selected under an assumption that it represents a minimal biologically significant change.

An UF of 10 was used to account for extrapolating from a POD for a subchronic exposure duration to estimate chronic exposure conditions (UF_S).

An UF of 3 was used to account for database deficiencies (UF_D). The study used in this RfD derivation, McCauley et al. (1995, 1990), is the only study of repeat-dose toxicity available for cis-1,2-DCE. The database for this isomer is missing studies of reproductive toxicity, including a two-generation reproductive toxicity study, and developmental toxicity; however, the developmental toxicity potential for cis-1,2-DCE is informed by a series of range-finding studies of the developmental toxicity of a mixture of cis-1,2-DCE isomers (composition of isomers unknown) (NTP, 1991a, b, c). No evidence of developmental toxicity was observed in mice or rats based on the parameters evaluated in these range-finding studies (gravid uterus weight, fetal body weight, number of fetuses [live/dead], implantation sites, and resorptions).

___I.A.4. ADDITIONAL STUDIES/COMMENTS

No studies of the effects of oral exposure to cis-1,2-DCE in humans were identified, and the experimental toxicity database for this isomer is limited. The only investigation of repeat-dose toxicity of cis-1,2-DCE by the oral route is McCauley et al. (1995, 1990).

___I.A.5. CONFIDENCE IN THE CHRONIC ORAL RfD

Study – Medium
Data Base – Low-medium
RfD – Low

The overall confidence in this RfD assessment is low. Confidence in the principal study (McCauley et al. 1995, 1990) is medium. The 90-day oral gavage study (McCauley et al. 1995, 1990) used four dose groups plus a control and measured multiple parameters, including body weight, liver weight, kidney weight, clinical chemistry, and hematology parameters. There are no oral studies of chronic, reproductive, or developmental toxicity of cis-1,2-DCE. The

McCauley et al. (1995, 1990) study is the only available subchronic study of cis-1,2-DCE and was used as the basis for the oral RfD. However, the developmental toxicity potential is informed by several range-finding studies for a mixture of cis-1,2-DCE isomers (NTP, 1991a, b, c) that showed no evidence of developmental toxicity. Thus, the confidence in the database is low to medium.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE CHRONIC ORAL RfD

Source Document – U.S. EPA, 2010

This document has been provided for review to EPA scientists, interagency reviewers from other federal agencies and White House offices, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of cis-1,2-Dichloroethylene and trans-1,2-Dichloroethylene* (U.S. EPA, 2010).

Agency Completion Date -- __/__/__ *[note: leave this BLANK until completion is reached*]

___I.A.7. EPA CONTACTS

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

__I.B. REFERENCE CONCENTRATION (RfC) FOR CHRONIC INHALATION EXPOSURE

Substance Name – cis-1,2-Dichloroethylene (cis-1,2-DCE)
CASRN – 156-59-2
Section I.B. Last Revised – 00/00/0000

The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). The inhalation RfC (generally expressed in units of mg/m^3) is analogous to the oral RfD and is similarly intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action.

Inhalation RfCs are derived according to *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994). Because RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity of this chemical substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

An inhalation assessment for cis-1,2-DCE was not previously developed for the IRIS database.

___I.B.1. CHRONIC INHALATION RfC SUMMARY

The inhalation toxicity database for cis-1,2-DCE does not support derivation of an RfC. No studies of the effects of cis-1,2-DCE by inhalation exposure in humans were identified. In experimental animals, investigation of the inhalation toxicity of cis-1,2-DCE is limited to an acute 4-hour inhalation LC_{50} study in rats (DuPont, 1999). There are no inhalation studies of subchronic, chronic, reproductive, or developmental toxicity of cis-1,2-DCE. Therefore, an inhalation RfC was not derived for cis-1,2-DCE.

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES

Not applicable.

___I.B.3. UNCERTAINTY FACTORS

Not applicable.

___I.B.4. ADDITIONAL STUDIES/COMMENTS

Not applicable.

___I.B.5. CONFIDENCE IN THE CHRONIC INHALATION RfC

Not applicable.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE CHRONIC INHALATION RfC

Source Document – U.S. EPA, 2010

This document has been provided for review to EPA scientists, interagency reviewers from other federal agencies and White House offices, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of cis-1,2-Dichloroethylene and trans-1,2-Dichloroethylene* (U.S. EPA, 2010).

Agency Completion Date -- __/__/__ *[note: Leave this BLANK until completion is reached]*

___I.B.7. EPA CONTACTS

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name – cis-1,2-Dichloroethylene (cis-1,2-DCE)
CASRN – 156-59-2
Section I.B. Last Revised – 00/00/0000

This section provides information on three aspects of the carcinogenic assessment for the substance in question: the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral and inhalation exposure. Users are referred to Section I of this file for information on long-term toxic effects other than carcinogenicity.

The rationale and methods used to develop the carcinogenicity information in IRIS are described in the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a) and the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA, 2005b). The quantitative risk estimates are derived from the application of a low-dose extrapolation procedure, and are presented in two ways to better facilitate their use. First, route-specific risk values are presented. The "oral slope factor" is a plausible upper bound on the estimate of risk per mg/kg-day of oral exposure. Similarly, a "unit risk" is a plausible upper bound on the estimate of risk per unit of concentration, either per μg/L drinking water (see Section II.B.1.) or per $\mu g/m^3$ air breathed (see Section II.C.1.). Second, the estimated concentration of the chemical substance in drinking water or air when associated with cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000 is also provided.

__II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

Under the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), there is "inadequate information to assess the carcinogenic potential" of cis-1,2-DCE. This cancer descriptor is based on the absence of epidemiological studies in humans and lack of animal studies designed to evaluate the carcinogenic potential of cis-1,2-DCE.

___II.A.2. HUMAN CARCINOGENICITY DATA

No epidemiologic studies evaluating possible long-term health effects of cis-1,2-DCE or a mixture of cis- and trans-1,2-DCE were identified.

___II.A.3. ANIMAL CARCINOGENICITY DATA

No cancer bioassays of cis-1,2-DCE are available.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Evidence from genotoxicity and mutagenicity studies is inconclusive. cis-1,2-DCE and mixtures of the cis- and trans-isomers were mostly nonpositive in bacterial genotoxicity assays for gene reversion or DNA damage but gave positive results in some bacterial assays for mitotic recombination or aneuploidy, frequently in the absence of metabolic activation by S9. Results for chromosomal aberrations or sister chromatid exchanges in mammalian cells in culture were mixed, providing positive findings in the presence or absence of metabolic activation. Some in

vivo assays gave positive results (host-mediated assay, chromosomal aberrations) for cis-1,2-DCE.

cis-1,2-DCE is converted into reactive epoxides (oxiranes) by CYP450 enzymes. It is likely that epoxides are responsible for the inactivation of CYP2E1 by binding to its heme moiety, and protein adduct formation via sulfhydryl groups of amino acids has been shown to occur with 1,2-DCE (Maiorino et al., 1982; Sipes and Gandolfi, 1980). However, DNA adduct formation has not been demonstrated. DNA binding of 1,2-DCE was negative in an in vitro assay where other chlorinated hydrocarbons gave positive results (Sipes and Gandolfi, 1980).

Positive results have been obtained with cis-1,2-DCE in several genotoxicity assays in the absence of metabolic activation, suggesting that the C=C double bond positioned next to two chlorine substituents might be reactive on its own. However, Henschler (1977), in an evaluation of the mutagenicity of halogenated olefins, pointed out that asymmetric distribution of chlorine substituents across the C–C bond, such as exists in 1,1-DCE, was far more likely to give rise to mutagenic events because the resulting epoxides are unstable, as compared with a symmetric distribution of the chlorines as exists in cis-1,2-DCE. Evidence for other effects that could potentially lead to tumor formation, such as redox cycling, GSH depletion, or lipid peroxidation, has not been shown for cis-1,2-DCE.

Carcinogenic activity of a metabolite of cis-1,2-DCE, dichloroacetic acid, has been demonstrated in several animal bioassays but not in humans (U.S. EPA, 2003).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

Not applicable.

___II.B.1. SUMMARY OF RISK ESTIMATES

Not applicable.

___II.B.2. DOSE-RESPONSE DATA

Not applicable.

___II.B.3. ADDITIONAL COMMENTS

Not applicable.

___II.B.4. DISCUSSION OF CONFIDENCE

Not applicable.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM

INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

Not applicable.

___II.C.2. DOSE-RESPONSE DATA

Not applicable.

___II.C.3. ADDITIONAL COMMENTS

Not applicable.

___II.C.4. DISCUSSION OF CONFIDENCE

Not applicable.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document – U.S. EPA, 2010

This document has been provided for review to EPA scientists, interagency reviewers from other federal agencies and White House offices, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of cis- and trans-1,2-Dichloroethylene* (U.S. EPA, 2010).

___II.D.2. EPA REVIEW

Agency Completion Date -- __/__/__ *[note: Leave BLANK until completion is reached]*

___II.D.3. EPA CONTACTS

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

_III. [reserved]
_IV. [reserved]
_V. [reserved]

_VI. BIBLIOGRAPHY

Substance Name – cis-1,2-Dichloroethylene (cis-1,2-DCE)
CASRN – 156-59-2
Section I.B. Last Revised – 00/00/0000

__VI.A. ORAL RfD REFERENCES

McCauley, PT; Robinson, M; Daniel, FB; et al. (1990) The effects of subacute and subchronic oral exposure to cis-1,2-dichloroethylene in rats. Health Effects Research Laboratory, U.S. Environmental Protection Agency, Cincinnati, OH and Toxic Hazards Division, Air Force Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, OH; unpublished report.

McCauley, PT; Robinson, M; Daniel, FB; et al. (1995) The effects of subacute and subchronic oral exposure to cis-1,2-dichloroethylene in Sprague-Dawley rats. Drug Chem Toxicol 18:171–184.

NTP (National Toxicology Program). (1991a) Range finding studies: developmental toxicity 1,2-dichloroethylene when administered via feed in Swiss CD-1 mice. Public Health Service, U.S. Department of Health and Human Services; NTP TRP 91022. Available from the National Institute of Environmental Health Sciences, Research Triangle Park, NC.

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NTP. (1991c) Range finding studies: developmental toxicity 1,2-dichloroethylene (repeat) when administered via feed in CD Sprague-Dawley rats. Public Health Service, U.S. Department of Health and Human Services; NTP TRP 91033. Available from the National Institute of Environmental Health Sciences, Research Triangle Park, NC.

U.S. EPA (U.S. Environmental Protection Agency). (2000) Benchmark dose technical guidance document [external review draft]. Risk Assessment Forum, Washington, DC; EPA/630/R-00/001. Available online at http://cfpub.epa.gov/ncea/cfm/nceapublication.cfm?ActType=PublicationTopics&detype=DOCUMENT&subject=BENCHMARK+DOSE&subjtype=TITLE&excCol=Archive.

U.S. EPA. (2010) Toxicological Review of cis-1,2-Dichloroethylene and trans-1,2-Dichloroethylene in Support of Summary Information on Integrated Risk Information System (IRIS), National Center for Environmental Assessment, Washington, DC. Available online at http://www.epa.gov/iris.

__VI.B. INHALATION RfC REFERENCES

DuPont. (1999) Initial submission: letter from DuPont Haskell Laboratory to U.S. EPA re results of 4-hour inhalation median lethality study (LC50) in rats w/cis-1,2-dichloroethylene, dated 8/26/99. E.I. DuPont de Nemours and Company, Wilmington, DE. Submitted under TSCA Section 8E; EPA Document No. 88990000257; NTIS No. OTS0559785.

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__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

U.S. EPA. (2003) Toxicological Review of Dichloroacetic Acid in Support of Summary Information on Integrated Risk Information System (IRIS), National Center for Environmental Assessment, Washington, DC. Available online at http://www.epa.gov/iris.

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_VII. REVISION HISTORY

Substance Name – cis-1,2-Dichloroethylene (cis-1,2-DCE)
CASRN – 156-59-2
Section I.B. Last Revised – 00/00/0000

Date	Section	Description
12/01/1990	II.	Carcinogen assessment on-line
12/01/1990	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line

02/01/1995	II.D.3.	Primary contact changed
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
12/03/2002	II.D.2.	Screening-Level Literature Review Findings message has been added.
00/00/0000	I., II., VI.	RfD assessment added; RfC and cancer assessment sections revised.

_VIII. SYNONYMS

Substance Name – cis-1,2-Dichloroethylene (cis-1,2-DCE)
CASRN – 156-59-2
Section I.B. Last Revised – 00/00/0000
156-59-2
ethene, 1,2-dichloro-, (Z)-
(Z)-1,2-dichloroethene
(Z)-1,2-dichloroethylene
cis-dichloroethylene
cis-1,2-dichloroethene
cis-1,2-dichloroethylene
ethene, 1,2-dichloro-, (Z)-
ethylene, 1,2-dichloro-, (Z)-
HSDB 5656
NSC 6149
1,2-cis-dichloroethylene