

Endosulfan; CASRN 115-29-7

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the [IRIS assessment development process](#). Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development 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](#).

STATUS OF DATA FOR Endosulfan

File First On-Line 09/30/1987

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	yes	10/01/1994
Inhalation RfC (I.B.)	not evaluated	
Carcinogenicity Assessment (II.)	not evaluated	

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Endosulfan

CASRN — 115-29-7

Last Revised — 10/01/1994

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of

information concerning the carcinogenicity of this 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.

I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
Reduced body weight gain in males and females; increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males	NOAEL: 15 ppm [0.6 mg/kg-day (male); 0.7 mg/kg-day (female)]	100	1	6E-3 mg/kg-day
	LOAEL: 75 ppm [2.9 mg/kg-day (male); 3.8 mg/kg-day (female)]			
2-Year Rat Feeding Study				
Hoechst Celanese Corp., 1989a				
Decreased weight gain in males and neurologic findings in both sexes	NOAEL: 10 ppm 0.57 mg/kg-day (female)			
1-Year Dog Feeding Study	LOAEL: 30 ppm [1.9 mg/kg-day (female); 2.1 mg/kg-day (male)]			
Hoechst Celanese Corp., 1989a				

*Conversion Factors and Assumptions — Actual dose tested

I.A.2. Principal and Supporting Studies (Oral RfD)

Hoechst Celanese Corporation. 1989a. MRID No. 40256502, 41099502. HED Doc. No. 007937. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Celanese Corporation. 1989b. MRID No. 41099501. HED Doc. No. 007937. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Groups of Sprague-Dawley rats (50/sex/dose) were administered endosulfan in the diet for 2 years at dietary concentrations of 0, 3, 7.5, 15 and 75 ppm (Male: 0, 0.1, 0.3, 0.6 and 2.9 mg/kg-day; Female: 0, 0.1, 0.4, 0.7 and 3.8 mg/kg-day) (Hoechst Celanese Corp., 1989a). A satellite group of 20 animals/sex/dose were used for toxicity evaluation and sampled at intervals for hematology and clinic chemistry; survivors were sacrificed after 104 weeks. An additional group of 10 rats/sex were used for pretest hematology and health check. Animals received food and water ad libitum.

No effects of dosing on clinical signs, mortality, food and water consumption, ophthalmological examinations and urinalysis were observed. Mean body weight gains tended to be decreased in both males and females receiving 15 and 75 ppm. Weight gains were significantly depressed ($p < 0.05$) during weeks 6-18 in males receiving 15 and 75 ppm when compared with controls; decreases did not achieve a level of significance in males or females receiving 15 ppm at other intervals. Between weeks 0-64, body weight gains were 9 and 13% lower in males and females of the 75 ppm group, respectively, than those of the controls. Overall weight gains (weeks 0-104) were 17% lower than the controls in both males and females receiving 75 ppm and 9% lower in rats receiving 15 ppm. The gains were significantly lower ($p < 0.01$) in both sexes at the 75 ppm dose. No weight gain effects were seen in males and females receiving 3 or 7.5 ppm when compared with controls.

No toxicologically important changes in hematology and clinical chemistry parameters were observed. The incidence of bilaterally enlarged kidneys was increased in females of both the satellite (2 and 8 for the control and 75 ppm dose groups, respectively) and main (8 and 18 for the control and 75 ppm dose groups, respectively) groups receiving 75 ppm when compared with controls. Other findings in the kidneys (paleness, irregular or uniform cortical scarring and cysts) occurred at similar frequencies in control and dosed groups. The incidence of progressive glomerulonephrosis was high in all dose groups including controls which is not an uncommon finding in studies with chlorinated hydrocarbon pesticides. The severity appeared to be dose-related. The incidence of severe (marked) glomerulonephrosis was increased in both males and females receiving 75 ppm. In males, the increased incidence at 75 ppm was accounted for by rats that died. In decedents, the incidence combining both males in the main and satellite groups was 10/41, 10/43, 17/46 and 14/46 and 20/46 at 0, 3, 7.5, 15 and 75 ppm. The incidence in high-dose males (30/70, 43%) was reported to be higher than normally observed for historical controls. The laboratory control incidence in six studies was 70/300 (19.7%) with a range of 10 to 38%.

The incidence of aneurysms of the blood vessels was increased in the high-dose males of both the satellite (6/20) and the main study (13/50) when compared with controls (1/20 and 9/50). The percent incidence (27%) in the combined high-dose males was higher than normally found in historical controls (10%, range in five studies 4-18%). Other nonneoplastic findings were considered within the normal range of background.

Based on reduced body weight gain in males and females, and increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males, the LEL for systemic toxicity is 75 ppm (Male: 2.9 mg/kg-day; Female: 3.8 mg/kg-day). The NOEL for systemic toxicity is 15 ppm (Male: 0.6 mg/kg-day; Female: 0.7 mg/kg-day).

Endosulfan was fed in the diet to beagle dogs (6/sex/dose) for 1 year at dietary levels of 0, 3, 10 and 30 ppm (Male: 0, 0.2, 0.65 and 2.1 mg/kg-day; Female: 0, 0.18, 0.57 and 1.9 mg/kg-day) (Hoechst Celanese Corp., 1989b). An additional group of 6 dogs/sex received 30 ppm for 54 days, after which the dose was increased to 45 ppm (Male: 3.2 mg/kg-day; Female: 2.9 mg/kg-day) and continued at that level until a final increase to 60 ppm (Male: 4.1 mg/kg-day; Female: 3.8 mg/kg-day) was administered at 106 days. Animals received food and water ad libitum.

At the highest dose tested, severe nervous symptoms developed. A loss or weakening of placing and righting reactions was observed and substantial weight loss resulted (0.36 and 0.45 kg for males and females, respectively, between weeks 15 and 21). This group was sacrificed at 146-147 days owing to poor overall condition; one male had been sacrificed at 126 days. The overall weight gain in males receiving 30 ppm (to week 54) was 30% lower than the controls. Tonic contractions of the muscles of the abdomen and chaps a few hours after feeding was noted in both sexes receiving 30 ppm; one male was sacrificed at 39 weeks. The sacrificed dog at 30 ppm had gross and histologic changes in the lungs. The sacrificed dog receiving 60 ppm had pulmonary edema. The histologic findings in dosed and control groups for dogs sacrificed by design were generally unremarkable and incidental.

Based on decreased weight gain in males and neurologic findings in both sexes, the LEL for systemic toxicity is 30 ppm (Male: 2.1 mg/kg-day; Female: 1.9 mg/kg-day). The NOEL for systemic toxicity is 10 ppm (Male: 0.65 mg/kg-day; Female: 0.57 mg/kg-day).

I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — The uncertainty factor of 100 reflects 10 for intraspecies variability and 10 for interspecies extrapolation.

MF — None

I.A.4. Additional Studies/Comments (Oral RfD)

The observation of a yellowish discoloration of the kidneys was found in a 30-day rat feeding study (Hoechst Celanese Corp., 1985), a 90-day rat feeding study (Hoechst Aktiengesellschaft, 1985), and a 2-generation reproduction study in rats (Hoechst Aktiengesellschaft, 1984a). This observation was not found in the rat chronic feeding/oncogenicity study (Hoechst Celanese Corp., 1989a), the 1-year dog feeding study (Hoechst Celanese Corp., 1989b), or the mouse carcinogenicity study (Hoechst Celanese Corp., 1988). Hoechst Celanese Corporation argues that data from chronic toxicity studies, metabolism, and special studies indicate that this is not an adverse hematopoietic effect, but is indicative of the physical presence and harmless process of elimination of endosulfan and its metabolites via the kidney. Electron microscopy and tissue residue analysis of the kidneys from the 30-day study indicated that the alpha-endosulfan and to a lesser extent beta-endosulfan, endosulfan sulfate, and endosulfan lactone were stored temporarily in the kidneys. Additionally, negative results were obtained from the staining of the kidneys with Prussian Blue to detect the presence of ferritin (evidence of hemosiderosis).

Data Considered for Establishing the RfD

- 1) 2-Year Feeding - rat: Principal study -- see previous description; Core grade minimum (Hoechst Celanese Corp., 1989a).
- 2) 1-Year Feeding - dog: Co-principal study -- see previous description; Core grade minimum (Hoechst Celanese Corp., 1989b).
- 3) 2-Generation Reproduction - rat: Core grade minimum (Hoechst Aktiengesellschaft, 1984a).

Four week old male and female rats of CrI:COBS CD(SD)BR strain were allowed to acclimate for 7 days and were then distributed randomly to groups of 32 of each sex for the F0 generation. F1b animals were distributed randomly to groups of 28 of each sex for the second generation. Animals were administered endosulfan in the diet at dose levels of 0, 3, 15 or 75 ppm (Male: 0, 0.2, 1.1 and 5.4 mg/kg-day; Female: 0, 0.25, 2.6 and 6.6 mg/kg-day).

Mortality, food/water consumption, and body weight gain were not affected in either generation, but a decrease in body weight gain ($p < 0.05$) was observed in the F0 females following the start of dosing. Pregnancy rate, gestation times, the ability to rear young to weaning, and precoital time were comparable among the groups at both matings in both generations. F0 males displayed increased heart weight at the mid- and high-dose levels (dose-related) and increased liver and kidney weights at the high-dose level. F0 females displayed increased brain and liver weights at the high-dose level. In the F1b adults, the high-dose males displayed increased kidney weights compared with the controls and females displayed increased liver weights at the mid- and high-dose levels. Although the changes in the heart and liver weights reported in the mid-dose F0 males and females were statistically significant, the U.S. EPA noted that these effects were slight and limited to one sex of one litter and occurred only in one generation. In view of this and in the absence of histopathological changes in the liver and heart, the U.S. EPA concluded that these statistically significant changes in organ weights should not be considered biologically significant. Therefore, based on a decrease in body weight gain in F0 females, the LEL for systemic toxicity is 75 ppm (Male: 5.4 mg/kg-day; Female: 6.6 mg/kg-day). The NOEL for systemic toxicity is 15 ppm (Male: 1.1 mg/kg-day; Female: 2.6 mg/kg-day).

No effect of treatment on litter size was observed throughout both matings of both generations. In the first mating of the F0 generation, an increase was noted in the cumulative litter loss (%) at the high-dose level. Litter and pup weights were comparable at birth among the groups in both generations, but a decrease in litter weight was observed during the lactation to weaning period in both matings in the F0 generation, which was significant at the high-dose level in the first mating and at the mid- and high-dose levels in the second mating (dose-related). Because there was no corroborative finding of a decrease in the number of pups per litter or in pup weight, this decrease in litter weight is not considered to be treatment-related. Increased pituitary weights (high-dose female pups of 1st mate of F0 generation) and increased uterine weights (high-dose female pup of 1st mate of F1b generation) were observed in the offspring. There were no histopathological findings observed in either the F1b adults of the selected pups from the second mate or the F1b generation that could be attributed to treatment. Based on increased pituitary and uterine weights, the LEL for offspring toxicity is 75 ppm (Male: 5.4 mg/kg-day; Female: 6.6 mg/kg-day). The NOEL for offspring toxicity is 15 ppm (Male: 1.1 mg/kg-day; Female: 2.6 mg/kg-day).

No evidence of reproductive toxicity was found at any of the dose levels tested. Therefore, the NOEL for reproductive toxicity is equal to or greater than 75 ppm (Male: 5.4 mg/kg-day; Female: 6.6 mg/kg-day).

4) Developmental toxicity - rat: Core grade supplementary (FMC Corp., 1980).

Groups of pregnant CD Sprague-Dawley rats were administered endosulfan by daily oral gavage on days 6 through 19 of gestation at dose levels of 0, 0.66, 2.0 or 6.0 mg/kg-day. Although the original protocol specified 25 animals per treatment group, 10 additional animals were added to the high-dose group (due to mortality among the original animals) and five additional animals were added to the control group (due to a loss of some tissues during the processing).

Maternal toxicity was apparent in the high-dose group in the form of significantly reduced body weights and body weight gain during gestation ($p < 0.01$). Toxic signs observed in the high-dose group included face rubbing (20/35 animals), brown exudate (4/35), rough coat (5/35), flaccidity (8/35) and hyperactivity (11/35). Face rubbing was reported in 6/25 mid-dose animals and alopecia was reported in 2/25. No face rubbing was reported in low dose animals or controls. Mean fetal weight and crown-rump length were significantly reduced ($p < 0.05$ and $p < 0.01$, respectively) in the 6 mg/kg-day group. An increase in misaligned sternebrae was observed in all treated groups compared with concurrent controls. The increase in litters and fetuses affected at each dose level was above that reported in the historical control database (18% of litters and 1.85% of fetuses based upon the examination of 65 litters and 863 fetuses). However, the variability between studies was not reported. An increased incidence of litters with extra ribs and poorly ossified and unossified sternebrae was observed at the high-dose level. More detailed historical control data may be useful in determining whether the apparent increase in misaligned sternebrae is due to an unusually low incidence in the concurrent control group and within the variability observed between studies. In the absence of such information, it is recommended that this finding be considered to be compound-related. An additional review of this study by the U.S. EPA concluded that replacement of animals during or after the study made it difficult to interpret the data and derive a NOEL and LEL for this study. The U.S. EPA has recommended a repeat of this study.

5) Developmental toxicity - rabbit: Core grade minimum (FMC Corp., 1981).

Groups of 20 pregnant New Zealand white rabbits were administered endosulfan by oral gavage on days 6 through 28 of gestation at dose levels of 0, 0.3, 0.7 or 1.8 mg/kg-day. When mortality was observed at the highest dose level, six more mated rabbits were added to this group.

Two animals in the control and one in the middle dose level showed nasal congestion. In the highest dose level, four animals showed a noisy and rapid breathing, hyperactivity and convulsions. Body weight gains during the days 19-29 and corrected for gravid uterine weights at sacrifice were less in the high-dose group than in controls. The former was also less than the control for the mid-dose group. However, these differences were not statistically significant. Based on these effects, the NOEL and LEL for maternal toxicity are 0.7 and 1.8 mg/kg-day,

respectively. No developmental effects were observed at any dose tested. Therefore, the NOEL for developmental toxicity is equal to or greater than 1.8 mg/kg-day, the highest dose tested.

Other Data Reviewed:

6) 2-Year Feeding - mouse: Core grade minimum (Hoechst Celanese Corp., 1988).

Groups of Hoe:NMRKf mice (60/sex/dose) were fed endosulfan in the diet at dose levels of 0, 2, 6, or 18 ppm (Male: 0, 0.28, 0.84, and 2.51 mg/kg-day; Female: 0, 0.32, 0.97, and 2.86 mg/kg-day) for 2 years. A satellite group of 20 mice/sex/group was used for interim sacrifices at 12 and 18 months. Animals were individually housed and received food and water ad libitum.

No overt signs of toxicity or dose-related effects were noted on clinical observations, food consumption, hematology, clinical chemistry, urinalysis, organ weights, macroscopic pathology, or microscopic pathology. Decreased survival ($p < 0.05$) in high-dose females and body weight reduction ($p < 0.05$) in high-dose males throughout the study were considered to be compound-related effects. Based on these findings, the NOEL and LEL for systemic toxicity are 6 ppm (Male: 0.84 mg/kg-day; Female: 0.97 mg/kg-day) and 18 ppm (Male: 2.51 mg/kg-day; Female: 2.86 mg/kg-day), respectively.

7) 90-Day Feeding - rat: Core grade minimum (Hoechst Aktiengesellschaft, 1985).

Groups of CD Sprague-Dawley rats (25/sex/dose) were fed endosulfan in the diet at dose levels of 0, 10, 30, 60 or 360 ppm (0, 0.5, 1.5, 3 and 18 mg/kg-day) for 13 weeks. Twenty of each group were sacrificed at 13 weeks and five were sacrificed after an additional 4-week recovery period.

No significant mortality during the test period was reported. Body weight was marginally lowered in males and females at 360 ppm. Depressed RBC parameters were observed in the 60 and 360 ppm groups (6 and 13 weeks, both sexes) and in males at 30 ppm (6 weeks). Several other statistically significant decreases from control were observed but these were not dose-related. In general, the magnitude of the differences observed at each time point is small ($< 10\%$). Kidney weight (relative) was increased in both sexes at the high-dose level (360 ppm) and in males at 60 ppm. Two types of histopathological findings in the kidney were noted at 13 weeks: (1) occasional cells of proximal tubules showing yellowish discoloration of the cytoplasm (all dose levels), and (2) darker and more particulate granular and/or clumped pigmentation, predominantly in cells of the straight portions and, to a lesser extent in the proximal convoluted tubules (both sexes at 360 ppm/males at 60 ppm). From the data following the 4-week recovery period, the discoloration and/or pigmentation was not persistent after withdrawal of treatment, which Hoechst Aktiengesellschaft states is indicative of the ongoing process of excretion of test

material via the kidneys. Hoechst Aktiengesellschaft concludes that, although this process can be observed (due to the coloration of the kidney tissues and the observation of "dark urine"), it is not indicative of an adverse effect on the kidney, as confirmed by the histopathological findings.

8) 13-Week Feeding - mouse: Core grade minimum (Hoechst Aktiengesellschaft, 1984b).

Groups of CD-1 mice (20/sex/group) were fed endosulfan in the diet at dose levels of 0, 2, 6, 18 or 54 ppm (Male: 0, 0.24, 0.74, 2.13 and 7.3 mg/kg-day; Female: 0, 0.27, 0.8, 2.39 and 7.52 mg/kg-day) for 13 weeks. Ten animals of each sex were sacrificed after an approximately 20 day observation period prior to the test period and examined microscopically.

Increased mortality (12/20 males and 10/20 females) was observed at 54 ppm. Glucose levels in females were significantly ($p < 0.01$) lowered at 6, 18 and 54 ppm. Hemoglobin levels were significantly ($p < 0.05$) elevated at 2, 6 and 18 ppm in females and appeared elevated at 54 ppm; however, this value was not analyzed due to the few survivors. Mean corpuscular hemoglobin concentration was significantly ($p < 0.05$) lowered at 2, 6, and 18 ppm in females and appeared lowered at 54 ppm, however this value was not analyzed for the reason cited above. Based on the effects observed in females at the lowest dose tested, the LEL for systemic toxicity was 2 ppm (0.27 mg/kg-day). A NOEL for systemic toxicity was not established.

9) 30-Day Feeding - rat: Core grade supplementary (Hoechst Celanese Corp., 1985).

Groups of male Wistar rats (10 animals for control, 50 animals for each test dose) were fed endosulfan in the diet at dose levels of 0, 360 and 720 ppm (0, 34, and 67.8 mg/kg-day) for 30 days.

No overt signs of toxicity or dose-related effects were observed on body weight, food or water consumption, clinical observations, or ophthalmology. Two dosed animals died during the study with no discernible signs of toxicity. Absolute and relative liver weights of males receiving doses of 360 and 760 ppm and kidney weights of males receiving 720 ppm were increased ($p < 0.05$) following the dosing period; organ weights of dosed males were similar to controls following a 30-day recovery period. Macroscopic examination revealed discoloration of the kidneys following the dosing period; histopathologically, the number and size of the lysosomes of the proximal convoluted tubules of the kidneys were increased following the dosing period with this finding exhibited to a greater extent in high-dose males. The renal changes were found to be reversible following the recovery period without evidence of renal lesions. No evidence of comparable lysosomal activity in the brain or liver was reported. Electron microscopy and tissue analysis confirmed that alpha- endosulfan and to a lesser extent beta-endosulfan, endosulfan sulfate and endosulfan-lactone were stored temporarily in the kidneys during the dosing period; only negligible amounts of endosulfan metabolites were found in the liver. Based on kidney

changes during the dosing period, the LEL for systemic toxicity was 360 ppm (34 mg/kg-day), the lowest dose tested. A NOEL for systemic toxicity was not established.

Data Gap(s): Rat Developmental Toxicity Study

I.A.5. Confidence in the Oral RfD

Study — Medium

Database — Medium

RfD — Medium

The principal studies are of adequate quality and therefore the given a medium confidence rating. Additional studies are supportive of the principal studies. However, due to the replacement of test animals during the available developmental toxicity study (FMC Corp., 1980), the data are considered inadequate to address the requirement for testing in a second species. Due to the lack of these developmental data in a second species, the database is given a medium-to-high confidence rating. Medium-to-high confidence in the RfD follows.

I.A.6. EPA Documentation and Review of the Oral RfD

Source Document — This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation — None

Agency Work Group Review — 05/20/1985, 05/31/1985, 11/21/1985, 02/05/1986, 06/11/1986, 03/18/1987, 11/04/1992, 03/31/1993

Verification Date — 03/31/1993

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfD for Endosulfan conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

I.A.7. EPA Contacts (Oral RfD)

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 (internet address).

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Endosulfan
CASRN — 115-29-7

Not available at this time.

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Endosulfan
CASRN — 115-29-7

Not available at this time.

III. [reserved]

IV. [reserved]

V. [reserved]

VI. Bibliography

Substance Name — Endosulfan
CASRN — 115-29-7

VI.A. Oral RfD References

FMC Corporation. 1980. MRID No. 00055544. HED Doc. No. 000416. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

FMC Corporation. 1981. MRID No. 00094837. HED Doc. No. 001488. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Aktiengesellschaft. 1984a. MRID No. 00148264. HED Doc. No. 004881, 008868, 009552. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Aktiengesellschaft. 1984b. MRID No. 00147182. HED Doc. No. 004733. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Aktiengesellschaft. 1985. MRID No. 00145668. HED Doc. No. 005115, 008868. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

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Hoechst Celanese Corporation. 1989a. MRID No. 40256502, 41099502. HED Doc. No. 007937. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Celanese Corporation. 1989b. MRID No. 41099501. HED Doc. No. 007937. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

VI.B. Inhalation RfC References

None

VI.C. Carcinogenicity Assessment References

None

VII. Revision History

Substance Name — Endosulfan
CASRN — 115-29-7

Date	Section	Description
12/01/1992	I.A.	Withdrawn; new Oral RfD verified (in preparation)
10/01/1994	I.A.	Oral RfD summary replaced; new RfD
12/03/2002	I.A.6.	Screening-Level Literature Review Findings message has been added.

VIII. Synonyms

Substance Name — Endosulfan
CASRN — 115-29-7
Last Revised — 09/30/1987

- 115-29-7
- BENZOEPIN
- BEOSIT
- BIO 5,462
- CHLORTHIEPIN
- CRISULFAN
- CYCLODAN
- DEVISULPHAN
- ENDOCEL
- ENDOSOL
- Endosulfan
- ENDOSULPHAN
- ENSURE
- ENT 23,979
- FMC 5462
- 1,2,3,4,7,7-HEXACHLOROBI-CYCLO(2.2.1)HEPTEN-5,6-BIOXYMETHYLENESULFITE

- alpha,beta-1,2,3,4,7,7-HEXACHLOROBICYCLO(2.2.1)-2-HEPTENE-5,6-BISOXYMETHYLENE SULFITE
- HEXACHLOROHEXAHYDROMETHANO 2,4,3-BENZODIOXATHIEPIN-3-OXIDE
- 6,7,8,9,10,10-HEXACHLORO-1,5,5a,6,9,9a-HEXAHYDRO-6,9-METHANO-2,4,3-BENZODIOXATHIEPIN-3-OXIDE
- 1,4,5,6,7,7-HEXACHLORO-5-NORBORNENE-2,3-DIMETHANOL cyclic SULFITE
- HILDAN
- HOE 2,671
- INSECTOPHENE
- KOP-THIODAN
- MALIX
- NA 2761
- NCI-C00566
- NIA 5462
- NIAGARA 5,462
- 5-NORBORNENE-2,3-DIMETHANOL, 1,4,5,6,7,7-HEXACHLORO-,CYCLIC SULFITE
- OMS 570
- RCRA WASTE NUMBER P050
- THIFOR
- THIMUL
- THIODAN
- THIOFOR
- THIOMUL
- THIONEX
- THIOSULFAN
- THIOSULFAN TIONEL
- TIOVEL