# Pentachlorophenol (CASRN 87-86-5)

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Quantitative Estimate of

## 0086

**Pentachlorophenol; CASRN 87-86-5**

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Pentachlorophenol

**File First On-Line 01/31/1987**

| Category (section) | Status | Last Revised |
|---|---|---|
| Oral RfD Assessment (I.A.) | on-line | 02/01/1993 |
| Inhalation RfC Assessment (I.B.) | no data | |
| Carcinogenicity Assessment (II.) | on-line | 07/01/1993 |

## _I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

## _I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name -- Pentachlorophenol
CASRN -- 87-86-5
Last Revised -- 02/01/1993

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

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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.

Quantitative Estimate of Carcinogenic Risk from Oral Exposure

- Summary of Risk Estimates
- Dose-Response Data
- Additional Comments
- Discussion of Confidence

Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

- Summary of Risk Estimates
- Dose-Response Data
- Additional Comments
- Discussion of Confidence

EPA Documentation, Review and, Contacts

Bibliography
Revision History
Synonyms

### __I.A.1. Oral RfD Summary

| Critical Effect | Experimental Doses* | UF | MF | RfD |
|---|---|---|---|---|
| Liver and kidney pathology<br><br>Rat Oral Chronic Study<br><br>Schwetz et al., 1978 | NOAEL: 3 mg/kg/day<br><br>LOAEL: 10 mg/kg/day | 100 | 1 | 3E-2 mg/kg/day |

*Conversion Factors: none

### __I.A.2. Principal and Supporting Studies (Oral RfD)

Schwetz, B.A., J.F. Quast, P.A. Keelev, C.G. Humiston and R.J. Kociba. 1978. Results of 2-year toxicity and reproduction studies on pentachlorophenol in rats. In: Pentachlorophenol: Chemistry, Pharmacology and Environmental Toxicology, K.R. Rao, Ed. Plenum Press, NY. p. 301.

Only one chronic study regarding oral exposure (Schwetz et al., 1978) was located in the available literature. Twenty-five rats/sex were administered 1 of 3 doses in the diet. At the 30 mg/kg/day level of treatment, a reduced rate of body weight gain and increased specific gravity of the urine were observed in females. Pigmentation of the liver and kidneys was observed in females exposed at 10 mg/kg/day or higher levels and in males exposed to 30 mg/kg/day. The 3 mg/kg/day level of exposure was reported as a chronic NOAEL.

A number of studies that have investigated the teratogenicity of orally administered pentachlorophenol in rodents are available in the literature. Although these studies (Larsen et al., 1975; Schwetz and Gehring, 1973; Schwetz et al., 1978; Hinkle, 1973) did not reveal teratogenic effects, feto- maternal toxicity was seen at 30 mg/kg/day (Schwetz and Gehring, 1973). Since pentachlorophenol apparently does not cross the placental barrier, the observed fetotoxicity may be a reflection of maternal toxicity (Larsen et al., 1975). The NOAEL in these studies was concluded to be 3.0 mg/kg/day (U.S. EPA, 1984), which is the same as for the chronic study reported earlier.

### __I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF -- The 100-fold factor accounts for the expected intra- and inter- species variability to the toxicity of this chemical in lieu of specific data.

MF -- None

### __I.A.4. Additional Studies/Comments (Oral RfD)

None.

### __I.A.5. Confidence in the Oral RfD

Study -- High
Database -- Medium
RfD -- Medium

The confidence in the chosen study is rated high because a moderate number of animals/sex were used in each of three doses, a comprehensive analysis of parameters was conducted, and a reproductive study was also run. Confidence in the supporting data base is rated medium because only one chronic study is available. Other subchronic studies provide adequate but weaker supporting data. The confidence in the RfD is medium. More chronic/reproductive studies are needed to provide a higher confidence in the RfD.

### __I.A.6. EPA Documentation and Review of the Oral RfD

U.S. EPA. 1984. Health Effects Assessment for Pentachlorophenol. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

Limited Peer Review and Agency-wide Internal Review, 1984.

U.S. EPA. 1985. Drinking Water Criteria Document for Pentachlorophenol. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

Two external peer reviews and an Agency internal review.

Agency Work Group Review -- 05/20/1985

Verification Date -- 05/20/1985

### __I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (301)345-2870 (phone), (301)345-2876 (FAX) or Hotline.IRIS@epamail.epa.gov (internet address).

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## _I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name -- Pentachlorophenol
CASRN -- 87-86-5

Not available at this time.

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## _II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name -- Pentachlorophenol
CASRN -- 87-86-5
Last Revised -- 07/01/1993

Section II 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 exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk

per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

## _II.A. Evidence for Human Carcinogenicity

### __II.A.1. Weight-of-Evidence Characterization

Classification -- B2; probable human carcinogen

Basis -- The classification is based on inadequate human data and sufficient evidence of carcinogenicity in animals: statistically significant increases in the incidences of multiple biologically significant tumor types (hepatocellular adenomas and carcinomas, adrenal medulla pheochromocytomas and malignant pheochromocytomas, and/or hemangiosarcomas and hemangiomas) in one or both sexes of B6C3F1 mice using two different preparations of pentachlorophenol (PeCP). In addition, a high incidence of two uncommon tumors (adrenal medulla pheochromocytomas and hemangiomas/hemangiosarcomas) was observed with both preparations. This classification is supported by mutagenicity data, which provides some indication that PeCP has clastogenic potential.

### __II.A.2. Human Carcinogenicity Data

Inadequate. Gilbert et al. (1990) attempted to study the effects of exposure to PeCP and other chemical preservatives among a cohort of 182 men employed in the wood treating industry in Hawaii. The study included both current and former workers who had experienced a minimum of 3 months of continuous employment treating wood between 1960 and November 1981. The first part of the study consisted of a cross-sectional clinical assessment of 88 workers (66 current, 22 former) and 58 nonexposed men employed in other occupations. Significantly elevated levels of urinary PeCP were found among the wood treaters but this was not related to any morbidity or mortality endpoint.

In part two of the study, the authors attempted to compare the mortality experience of the cohort with that expected in Hawaiian males of the same age. Only deaths that occurred in Hawaii were ascertained. Six deaths were observed compared with eight expected. Overall, the authors concluded that their results do not suggest any clinically significant adverse health effects nor any increased cancer morbidity or mortality from exposure to PeCP and other wood preserving chemicals. These conclusions must be seriously questioned based on the following: inadequate detail of selection for participation, particularly among the 58 unexposed "controls"; only 50% of eligible workers participated in the clinical portion which creates the potential for selection bias; employment eligibility criteria were different for current versus former workers; the clinical examiner was not blinded as to the exposure status of participants which raises questions about the presence of observation bias; the clinical data were presented and analyzed in a nonstandard way; no details are given about methods used to compute mortality "rates"; and, failure to ascertain deaths occurring outside of Hawaii. With over 30% of the original cohort apparently lost to follow-up, the study is of questionable validity. It cannot be used as evidence of no effect of the exposures but instead must be viewed as uninformative.

### __II.A.3. Animal Carcinogenicity Data

Sufficient. Two different 90% pure preparations of PeCP were tested in 2-year bioassays in B6C3F1 mice (NTP, 1989). Typical impurities present in both preparations included tri- and tetrachlorophenol, hexachlorobenzene, polychlorinated dibenzo-p-dioxins, and polychlorinated dibenzofurans. Technical grade PeCP (TG-PeCP) is a composite that consisted of equal proportions of product from Monsanto, Reichold and Vulcan. These specific products are no longer being produced. The second 90% pure preparation of PeCP, EC-7 PeCP, differed from

TG-PeCP in the level and nature of impurities present (e.g., EC-7 PeCP contained lower levels of dioxins and dibenzofurans). TG-PeCP was administered daily in the feed at dose levels of 0, 100, and 200 ppm to groups of 50 male and 50 female B6C3F1 mice for 2 years. The average doses of TG-PeCP were approximately 17-18 or 35 mg/kg for males and females, respectively. Two groups of control mice (35/sex) were fed basal diets. Survival of the mice did not appear to be affected by exposure to TG-PeCP at any dose level tested. However, it should be noted that survival of the male control mice (12/35) was low compared with historical control values. The early deaths were found to be due to urinary tract infections resulting from injuries sustained during fighting among the group-housed control male mice. After month 16 of the study, the male mice were singly housed to reduce the incidence of fighting and consequent high mortality. The incidences of hepatocellular adenomas and/or carcinomas were significantly increased in male mice exposed to TG-PeCP when compared with controls; the incidences were 7/32, 26/47 and 37/48 in control, low-dose and high-dose male mice, respectively. The incidences of benign and malignant pheochromocytomas of the adrenal medulla were also significantly greater in dosed male mice than in controls; the incidences were 0/31 in controls, 10/45 in low-dose animals and 23/45 in the high-dose animals. There was no significant increase in the numbers of liver tumors or pheochromocytomas in female mice exposed to TG-PeCP. However, the nonsignificant increase in liver tumors in TG-PeCP exposed females was considered biologically significant. TG-PeCP- and EC-7 PeCP-exposed females showed comparable responses in the 100- and 200-ppm dose groups with a marked increase observed only at 600 ppm in the EC-7 PeCP females. The liver tumor incidences for TG-PeCP exposed females was 3/33, 9/49 and 9/50, respectively. Vascular tumors (hemangiomas and/or hemangiosarcomas) were observed in female mice but not in male mice. Incidences of the hemangiosarcoma tumors were statistically significantly increased when compared to controls and all were malignant (0/30, 3/48, 6/46 in the control, low-dose and high-dose females, respectively).

EC-7 PeCP was administered daily in the feed at dose levels of 0, 100, 200, and 600 ppm (NTP, 1989). The average daily doses of EC-7 PeCP were approximately 17-18, 34-37, and 114-118 mg/kg, for the low-, mid-, and high- dose groups, respectively. Two groups of control mice (35/sex) were fed basal diets. Survival did not appear to be affected by exposure to EC-7 PeCP at any of the dose levels tested. The incidences of hepatocellular adenomas and/or carcinomas were significantly increased in dosed male mice exposed to EC-7 PeCP when compared with controls (6/35, 19/48, 21/48, 34/49 in the control, low-, mid-, and high-dose males, respectively). The incidences of benign and malignant pheochromocytomas of the adrenal medulla in males were also significantly greater in treated males than in the controls (1/34, 4/48, 21/48, 45/49 in the control, low-, mid-, and high-dose males, respectively). There was a significant increase in liver tumors (adenomas and/or carcinomas) (1/34, 4/50, 6/49 and 31/48 in the control, low-, mid-, and high-dose females, respectively) and benign and malignant pheochromocytomas in female mice exposed to EC-7 PeCP at the high-dose only (0/35, 2/49, 2/46, 38/49 in the control, low-, mid-, and high-dose females, respectively). Vascular tumors (hemangiomas and/or hemangiosarcomas) were observed in female mice but not in male mice. The incidence of these latter tumors was statistically significantly elevated in the high-dose group when compared with controls and all but one of the tumors was malignant (0/34, 1/50, 3/48, 9/47 in the control, low-, mid-, and high-dose females, respectively).

In a study reported by BRL (1968) and Innes et al. (1969), 18 male and 18 female crossbred mice were administered 46.4 mg/kg EC-7 PeCP in gelatin by gavage on days 7 through 28 after birth, followed by administration of 130 ppm (17 mg/kg/day) EC-7 PeCP in the diet for 18 months. It is not possible to ascertain whether the EC-7 PeCP used in this study is the same as the EC-7 used in the NTP (1989) study, since the level and nature of the impurities present in the preparation were not reported by Innes or BRL. Groups of mice from each strain served as negative or vehicle controls. Results indicated that there was no difference between the incidence of tumors in the PeCP- treated group and the control groups. Only tumor incidences were reported, so it is not known what other toxic effects (if any) may have occurred. This study is limited for drawing conclusions concerning the carcinogenicity of PeCP, however, because only one dose level was used. Furthermore, an insufficient number of animals (according to current guidelines) was studied.

In a chronic oral study on a different species conducted by Schwetz et al. (1978), groups of 25 Sprague-Dawley rats/sex were fed diets of 0, 8, 23, 77, or 231 ppm PeCP for 22 (for male) or 24

(for female) months (equivalent to 1, 3, 10, or 30 mg PeCP/kg/day). The PeCP preparation used in this study was reported to be 90% pure, and representative of the commercially available Dowicide EC-7 PeCP used in the NTP (1989) study. Results from the experiment indicated that in the high-dose group a reduced rate of body weight gain (i.e., a 12% lower mean monthly body weight during the last 12 months of the study) and an increased specific gravity of the urine were observed in females. Pigmentation of the liver and kidneys was observed in females exposed at 10 mg/kg/day or higher levels and in males exposed to 30 mg/kg/day. There was no significant increase in tumor incidence as compared with controls. A slight increase in pheochromocytomas of the adrenal medulla was noted at the lower dose levels. Survival was reported to be unaffected by treatment. Since the high dose (30 mg/kg/day) elicited signs of only mild toxicity, NTP suggested that the MTD had been reached but not exceeded in this study.

Catilina et al. (1981) also found no evidence of carcinogenicity in Wistar rats following subcutaneous administration of purified and technical grades of PeCP (6 mg/kg/dose). Test compounds were administered 3 times/week for 40 weeks followed by a 3-month post-treatment observation period. The use of only one dose, the use of an inappropriate route of administration, the relatively short exposure time, and excessive mortality limit the usefulness of this study for drawing conclusions concerning the carcinogenicity of PeCP.

In another study, Boutwell and Bosch (1959) applied a 20% solution of commercial grade PeCP in benzene to the shaved skin of Sutter mice twice weekly for 13 weeks following an initial exposure with 0.3% DMBA in benzene. Because of the dose level, frequency and duration of exposure in this study, only limited conclusions concerning the effectiveness of PeCP as a complete carcinogen can be made; these results, however, are sufficient to conclude that PeCP was not a tumor promoter in this assay.

### __II.A.4. Supporting Data for Carcinogenicity

Results from cytogenetic studies provide evidence for the clastogenic potential of PeCP. In cytogenicity studies with cultured CHO cells, TG-PeCP produced an increase in chromosomal aberrations in the presence but not the absence of S9 hepatic homogenate activation. Conversely, SCEs were induced only in the absence of S9 hepatic homogenate (NTP, 1989).

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## _II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

### __II.B.1. Summary of Risk Estimates

Oral Slope Factor -- 1.2E-1 per (mg/kg)/day

Drinking Water Unit Risk -- 3E-6 per (ug/L)

Extrapolation Method -- Linearized multistage procedure

Drinking Water Concentrations at Specified Risk Levels:

| Risk Level | Concentration |
|---|---|
| E-4 (1 in 10,000) | 3E+1 ug/L |
| E-5 (1 in 100,000) | 3E+0 ug/L |
| E-6 (1 in 1,000,000) | 3E-1 ug/L |

### __II.B.2. Dose-Response Data (Carcinogenicity, Oral Exposure)

Tumor Type -- hepatocellular adenoma/carcinoma, pheochromocytoma/malignant

pheochromocytoma, hemangiosarcoma/hemangioma (pooled incidence)
Test Animals -- mouse/B6C3F1, female
Route -- diet
Reference -- NTP, 1989

| Administered Dose ppm | Administered Dose (mg/kg)/day | Human Equivalent Dose (mg/kg)/day | Pooled Hepatocellular and Hemangiosarcoma Tumor Incidence |
|---|---|---|---|
| Technical grade pentachlorophenol | | | |
| 0 | 0 | 0 | 5/31 |
| 100 | 17 | 1.4 | 12/48 |
| 200 | 35 | 2.7 | 15/46 |
| Dowicide EC-7 pentachlorophenol | | | |
| 0 | 0 | 0 | 1/34 |
| 100 | 17 | 1.3 | 6/49 |
| 200 | 34 | 2.7 | 9/46 |
| 600 | 114 | 8.7 | 42/49 |

### __II.B.3. Additional Comments (Carcinogenicity, Oral Exposure)

Two different pentachlorophenol preparations induced liver tumors, pheochromocytomas and hemangiosarcomas in female mice and liver tumors and pheochromocytomas in male mice. All three tumor types are considered related to the administration of pentachlorophenol. The hemangiosarcomas, however, are considered to be the tumor of greatest concern; the EPA Science Advisory Board found that "these tumors were related to the administration of the pentachlorophenol formulations tested, occurred in a dose-response manner in the treated animals, and are morphologically related to known fatal human cancers that are induced by xenobiotics." Hemangiosarcomas were found only in female mice. To give preference to the data on hemangiosarcomas and because some male groups experienced significant early loss, only the female mice are used in the quantitative risk assessment.

In developing these estimates, benign and malignant tumors are combined; the liver tumors and pheochromocytomas were mostly benign. The pooled incidence counts animals with any of the three tumor types. Animals dying before the first tumor was observed are not considered to be at risk and are not included in the totals. Equivalent human doses are calculated using a surface-area adjustment. There are no pharmacokinetic data on pentachlorophenol. The slope factor is calculated as the geometric mean of the slope factors for each pentachlorophenol preparation.

### __II.B.4. Discussion of Confidence (Carcinogenicity, Oral Exposure)

For purposes of comparison, a slope factor of 0.05 can be derived from the incidence of hemangiosarcomas alone. Also for comparison, a slope factor of 0.5 can be derived from the pooled incidence of liver tumors and pheochromocytomas in male B6C3F1 mice.

The carcinogenicity assessment is based on results in a single animal species.

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### _II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

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## _II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

### __II.D.1. EPA Documentation

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

### __II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review -- 11/10/1987, 09/22/1988, 10/19/1988, 12/06/1989, 02/08/1990, 08/02/1990

Verification Date -- 08/02/1990

### __II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (301)345-2870 (phone), (301)345-2876 (FAX) or Hotline.IRIS@epamail.epa.gov (internet address).

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

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## _VI. Bibliography

Substance Name -- Pentachlorophenol
CASRN -- 87-86-5
Last Revised -- 03/01/1991

### _VI.A. Oral RfD References

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Larsen, R.V., G.S. Born, W.V. Kessler, S.M. Shaw and D.C. Van Sickle. 1975. Placental transfer and teratology of pentachlorophenol in rats. Environ. Lett. 10: 121-128.

Schwetz, B.A. and P.J. Gehring. 1973. The effect of tetrachlorophenol and pentachlorophenol on rat embryonal and fetal development. Toxicol. Appl. Pharmacol. 25: 455.

Schwetz, B.A., J.F. Quast, P.A. Keelev, C.G. Humiston and R.J. Kociba. 1978. Results of 2-year toxicity and reproduction studies on pentachlorophenol in rats. In: Pentachlorophenol: Chemistry, Pharmacology and Environmental Toxicology, K.R. Rao, Ed. Plenum Press, NY. p. 301.

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Cincinnati, OH for the Office of Drinking Water, Washington, DC.

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## _VI.B. Inhalation RfC References

None

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## _VI.C. Carcinogenicity Assessment References

Boutwell, R.K. and D.K. Bosch. 1959. The tumor-promoting action of phenol and related compounds for mouse skin. Cancer Res. 19: 413-424.

BRL (Bionetics Research Laboratories). 1968. Evaluation of the carcinogenic, teratogenic and mutagenic activities of selected pesticides and industrial chemicals, Vol. 1. Carcinogenic Study. Prepared for the National Cancer Institute, Bethesda, MD. NTIS PB-223-159. p. 393.

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Innes, J.R.M., B.M. Ulland, M.G. Valerio, et al. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice. A preliminary note. J. Natl. Cancer Inst. 42: 1101-1114.

NTP (National Toxicology Program). 1989. Technical Report on the Toxicology and Carcinogenesis Studies of Pentachlorophenol (CAS No. 87-86-5) in B6C3F1 mice (Feed Studies). NTP Tech. Report No. 349. NIH Publ. No. 89-2804.

Schwetz, B.A., J.F. Quast, P.A. Keeler, C.G. Humiston and R.J Kociba. 1978. Results of two-year toxicity and reproduction studies on pentachlorophenol in rats. In: Pentachlorophenol: Chemistry, Pharmacology and Environmental Toxicology, K.R. Rao, Ed. Plenum Press, NY. p. 301-309.

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## _VII. Revision History

Substance Name -- Pentachlorophenol
CASRN -- 87-86-5

| Date | Section | Description |
|---|---|---|
| 03/01/1988 | III.A. | Health Advisory added |
| 06/30/1988 | I.A.6. | Documentation year corrected |
| 01/01/1990 | II. | Carcinogen assessment now under review |
| 01/01/1990 | VI. | Bibliography on-line |
| 04/01/1990 | I.A.2. | NOEL corrected to NOAEL in last sentence, 1st paragraph |
| 07/01/1990 | I.B. | Inhalation RfC now under review |
| 07/01/1990 | IV.F.1. | EPA contact changed |
| 08/01/1990 | III.A.10 | Primary contact changed |
| 03/01/1991 | II. | Carcinogenicity assessment on-line |
| 03/01/1991 | VI.C. | Carcinogenicity references added |
| 01/01/1992 | I.A.7. | Primary contact changed |
| 01/01/1992 | IV. | Regulatory actions updated |
| 02/01/1993 | I.A.7. | Minor text change |
| 07/01/1993 | II.D.3. | Primary contact's phone number changed |
| 08/01/1995 | I.B. | EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the workgroup functions beginning in September, 1995. |
| 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. |
| 01/02/1998 | I., II. | This chemical is being reassessed under the IRIS Program. |

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## _VIII. Synonyms

Substance Name -- Pentachlorophenol
CASRN -- 87-86-5
Last Revised -- 01/31/1987

87-86-5
Chem-Tol
Chlorophen
Cryptogil OL
Dowcide 7
Dowicide EC-7
DP-2, technical
Durotox
EP 30
Fungifen
Glazd penta
Grundier arbezol
1-Hydroxy- 2,3,4,5,6-pentachlorobenzene
Lauxtol
Lauxtol A
Liroprem
NCI-C54933
NCI-C55378
NCI-C55389

NCI-C56655
PCP
Penchlorol
Penta
Pentachloorfenol
Pentachlorofenol
Pentachlorofenolo
Pentachlorophenate
Pentachlorophenol
2,3,4,5,6-Pentachlorophenol.
Pentachlorphenol
Pentaclorofenolo
Pentacon
Penta-Kil
Pentasol
Penwar
Peratox
Permacide
Permagard
Permasan
Permatox
Permatox dp-2
Permatox penta
Permite
Phenol, pentachloro-
Preventol P
Priltox
Santobrite
Santophen
Santophen 20
Sinituho
Term-i-trol
WLN: QR BG CG DG EG FG

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Last updated on Thursday, September 19th, 2002
URL: http://www.epa.gov/iris/subst/0086.htm