WORLD HEALTH ORGANIZATION

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans

Volume 30
Miscellaneous Pesticides

Summary of Data Reported and Evaluation

Insecticides (including acaricides)

- Chlordimeform
- Chlorobenzilate
- Dicofol
- Malathion
- Methyl parathion
- Parathion
- Piperonyl butoxide
- Tetrachlorvinphos
- Trichlorfon

Herbicides

- Diallate
- Fluometuron
- (4-Chloro-2-methylphenoxy)acetic acid (MCPA)
- Nitrofen
- Sulfallate

Fungicides

- Captan
- Chlorothalonil
- *ortho*-Phenylphenol and its sodium salt

Rodenticide

- 1-Naphthylthiourea (ANTU)

Last updated: 9 April 1998

CHLORDIMEFORM

VOL.: 30 (1983) (p. 61)

CAS No.: 6164-98-3
Chem. Abstr. Name: Methanimidamide, *N'*-(4-chloro-2-methylphenyl)-*N,N*-dimethyl-

5. Summary of Data Reported and Evaluation

5.1 Experimental data

No published study on the carcinogenicity of chlordimeform was available.

para-Chloro-*ortho*-toluidine, a metabolite of chlordimeform, was tested for carcinogenicity in two strains of mice and two strains of rats by oral administration in the diet. It was carcinogenic in both strains of mice, producing haemangiosarcomas. The studies in rats were not indicative of a carcinogenic effect, but certain limitations in their design were noted.

Chlordimeform is metabolized to a number of compounds, including *para*-chloro-*ortho*-toluidine, which has been identified in the urine of several animal species and of humans.

The available data were inadequate to evaluate the teratogenicity of chlordimeform to experimental animals.

Chlordimeform was negative in tests for DNA damage or mutagenicity in several cellular systems. No data were available, however, with regard to its mutagenicity in mammals, and no overall evaluation of the mutagenicity of chlordimeform could be made.

5.2 Human data

Chlordimeform was introduced in 1966. Its production, formulation and use as an insecticide on cotton are potential sources of occupational exposure.

No data were available to evaluate the teratogenic or chromosomal effects of chlordimeform in humans.

No case report or epidemiological study of the carcinogenicity of chlordimeform alone was available to the Working Group; however, it should be noted that the latent period since introduction of this compound may be too short for a carcinogenic effect to be detected in humans. (See also the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

No data were available on the carcinogenicity of chlordimeform to experimental animals. However, results of experiments in mice provide *sufficient evidence* that *para*-chloro-*ortho*-toluidine, a metabolite of chlordimeform, is carcinogenic to experimental animals. No relevant data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of chlordimeform to humans.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Suppl. 7 (1987) (p. 59: **Group 3**)

Synonyms

- Acaron
- Bermat
- C 8514
- Carzol
- Chlorfenamidine
- Chlorodimeform
- Chlorophenamidin
- Chlorophenamidine
- *N′*-(4-Chloro-ortho-tolyl)-*N,N*-dimethylformamidine
- Chlorphenamidine
- Ciba 8514
- Ciba - C85l4
- *N,N*-Dimethyl-*N′*-(2-methyl-4-chlorophenyl)formamidine
- ENT 27335
- ENT 27567
- EP-333
- Fundal
- Fundal 500
- Fundex
- Galecron
- *N′*-(2-Methyl-4-chlorophenyl)-*N,N*-dimethylformamidine
- RS 141
- Schering 36268
- SN 36268
- Spanon
- Spanone

Last updated: 16 April 1998

CHLOROBENZILATE

VOL.: 30 (1983) (p. 73)

CAS No.: 510-15-6
Chem. Abstr. Name: Benzeneacetic acid, 4-chloro-α-(4-chlorophenyl)- α-hydroxy-, ethyl ester

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Chlorobenzilate was tested for carcinogenicity by administration in the diet in three strains of mice and in two strains of rats. It induced hepatocellular carcinomas in both sexes of mice of one strain and in males of the two other strains. The data on rats were inadequate for evaluation.

Although no adverse effect was observed on reproduction in a three-generation study of rats, the teratogenic potential of chlorobenzilate has not been fully determined.

Chlorobenzilate was not mutagenic to *Salmonella typhimurium* with or without exogenous metabolic activation. Data from studies on other organisms were considered insufficient for evaluation, and no overall evaluation of the mutagenicity of chlorobenzilate could be made.

5.2 Human data

Chlorobenzilate was introduced in 1952. Its production, formulation and use as an acaricide on citrus and other crops are sources of potential exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of chlorobenzilate in humans.

No case report or epidemiological study of the carcinogenicity of chlorobenzilate alone was available to the Working Group. (See, however, the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

Results of the experiments in mice provide *limited evidence* that chlorobenzilate is carcinogenic to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of chlorobenzilate to humans.

For definition of the italicized terms, see Preamble Evaluation.

Previous evaluation: Vol. 5 (1974)

Subsequent evaluation: Suppl. 7 (1987) (p. 60: **Group 3**)

Synonyms

- Acar
- Acaraben

- Acaraben 4E, 250 EC, 500 EC, 25 WP
- Chlorbenzilat
- Chlorbenzilate
- Chlorbenzylate
- 4,4'-Dichlorobenzilate
- 4,4'-Dichlorobenzilic acid ethyl ester
- ENT 18596
- Ethyl 4-chloro-α-(4-chlorophenyl)-α-hydroxybenzeneacetate
- Ethyl *para,para'*-dichlorobenzilate
- Ethyl 4,4'-dichlorodiphenyl glycollate
- Ethyl 4,4'-dichlorophenyl glycollate
- Ethyl di(*para*-chlorophenyl)glycollate
- Ethyl 2-hydroxy-2,2-bis(3-chlorophenyl)acetate
- NCI-C00408

Last updated: 16 April 1998

DICOFOL

VOL.: 30 (1983) (p. 87)

CAS No.: 115-32-2
Chem. Abstr. Name: Benzenemethanol, 4-chloro-α-(4-chlorophenyl)-α-(trichloromethyl)-

4. Summary of Data Reported and Evaluation

4.1 Experimental data

Dicofol (technical-grade) was tested for carcinogenicity in mice and rats by administration in the diet. It induced hepatocellular carcinomas in male mice. The study in rats was considered to be inadequate for evaluation.

Dicofol (technical-grade), even when given at high doses, had no effect on reproduction or foetal development in mice; however, high doses in rats appeared to have an adverse effect on preimplantation stages of embryonal development.

Dicofol was negative in bacterial tests for mutagenicity and for DNA damage, with or without exogenous metabolic activation. The experimental protocols and results of studies with eukaryotes were not presented in adequate detail for an evaluation to be made. No overall evaluation of the mutagenicity of dicofol could be made.

4.2 Human data

Dicofol was introduced in 1955. Its production, formulation and widespread use as an acaricide on cotton and edible crops are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of dicofol in humans.

No case report or epidemiological study of the carcinogenicity of dicofol alone was available to the Working Group. (See, however, the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

4.3 Evaluation

Results of the experiment in mice provide *limited evidence* that dicofol is carcinogenic to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of dicofol to humans.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Suppl. 7 (1987) (p. 62: **Group 3**)

Synonyms

- Acarin
- 1,1-Bis(chlorophenyl)-2,2,2-trichloroethanol
- 1,1-Bis(*para*-chlorophenyl)-2,2,2-trichloroethanol

- 1,1-Bis(4-chlorophenyl)-2,2,2-trichloroethanol
- Carbax
- Cekudifol
- Childion
- CPCA
- Decofol
- Dichlorokelthane
- Di-(*para*-chlorophenyl)trichloromethylcarbinol
- 4,4'-Dichloro-α-(trichloromethyl)benzhydrol
- DTMC
- ENT 23,648
- FW 293
- Hilfol
- Hilfol 18.5 EC
- Keltane
- Kelthane
- *para,para'*-Kelthane
- Kelthane A, AP, EC, MF, & 35
- Kelthane Dust Base
- Kelthanethanol
- Milbol
- Mitigan
- NCI CO0486
- 2,2,2-Trichloro-1,1-bis(*para*-chlorophenyl)ethanol
- 2,2,2-Trichloro-1,1-di(4-chlorophenyl)ethanol

Last updated: 16 April 1998

MALATHION

VOL.: 30 (1983) (p. 103)

CAS No.: 121-75-5
Chem. Abstr. Name: Butanedioic acid,[(dimethoxyphosphinothioyl)thio]-, diethyl ester

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Malathion and its metabolite malaoxon were tested for carcinogenicity in mice and rats by administration in the diet. No evidence of carcinogenicity was found.

There was no evidence of teratogenicity or embryotoxicity in rats given maternally tolerated doses of malathion during pregnancy. In animals fed very high doses for two generations, malathion had no effect on reproduction other than to decrease survival in the first generation and to reduce growth in the second generation of rats.

Malathion was not mutagenic in most studies of bacteria or in two studies of yeast. No mutagenic action was observed in *Drosophila melanogaster*. Malathion increased sister chromatid exchange frequency in cultured mammalian cells but did not induce unscheduled DNA synthesis. Chromosomal aberrations were increased in certain types of cultured mammalian cells and in mice treated *in vivo* with malathion. Dominant lethal tests in mice were reported to be negative. There is thus limited evidence for the mutagenicity of malathion.

5.2 Human data

Malathion was introduced in 1950. Its production, formulation and widespread use as an insecticide in a variety of agricultural, vector control and household applications are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic effects of malathion in humans. The available data were insufficient to evaluate the chromosomal effects of malathion in humans.

No case report or epidemiological study of the carcinogenicity of malathion alone was available to the Working Group. (See, however, the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

The available data do not provide evidence that malathion or its metabolite malaoxon is carcinogenic to experimental animals. No data on humans were available.

The available data provide no evidence that malathion is likely to present a carcinogenic risk to humans.

Subsequent evaluation: Suppl. 7 (1987) (p. 62: **Group 3**)

Synonyms

- American Cyanamid 4,049
- *S*-[1,2-Bis(carbethoxy)ethyl] *O,O*-dimethyl dithiophosphate
- *S*-1,2-Bis(ethoxycarbonyl)ethyl-*O,O*-dimethylthiophosphate

- Calmathion
- Carbetovur
- Carbetox
- Carbofos
- Carbophos
- Celthion
- Chemathion
- Cimexan
- Compound 4049
- Cythion
- Detmol MA
- *S*-(1,2-Dicarbethoxyethyl)-*O,O*-dimethyldithiophosphate
- Dicarbethoxyethyl *O,O*-dimethyl phosphorodithioate
- *S*-[1,2-Di(ethoxycarbonyl)ethyl] dimethylphosphorothiolothionate
- Diethyl (dimethoxyphosphinothioylthio) butanedioate
- Diethyl(dimethoxyphosphinothioylthio)succinate
- Diethyl mercaptosuccinate, *O,O*-dimethyldithiophosphate, *S*-ester
- Diethyl mercaptosuccinate, *O,O*-dimethylphosphorodithioate, diethyl mercaptosuccinate, *O,O*-dimethyl thiophosphate
- Diethyl mercaptosuccinate *S*-ester with *O,O*-dimethylphosphorodithioate
- Diethyl mercaptosuccinic acid *O,O*-dimethylphosphorodithioate
- *O,O*-Dimethyl *S*-[1,2-bis-(ethoxycarbonyl)ethyl]dithiophosphate
- *O,O*-Dimethyl *S*-(1,2-dicarbethoxyethyl) dithiophosphate
- *O,O*-Dimethyl *S*-(1,2-dicarbethoxyethyl)phosphorodithioate
- *O,O*-Dimethyl *S*-(1,2-dicarbethoxyethyl)thiothionophosphate
- *O,O*-Dimethyl *S*-1,2-di(ethoxycarbamyl)ethylphosphorodithioate
- *O,O*-Dimethyldithiophosphate diethylmercaptosuccinate
- *O,O*-Dimethyl phosphorodithioate of diethylmercaptosuccinate
- EI 4049
- Emmatos
- Emmatos Extra
- ENT 17,034
- Ethiolacar
- Etiol
- Extermathion
- Fog 3
- Formal
- For-Mal
- Forthion
- Fosfothion
- Fosfotion
- Four Thousand Forty Nine
- Fyfanon
- 8059 HC
- Hilthion
- Hilthion 25WDP
- Insecticide No. 4049
- Karbofos
- Kop-thion
- Kypfos
- Malacide
- Malafor
- Malagran
- Malakill
- Malamar
- Malamar 50
- Malaphele
- Malasol
- Malaspray

- Malathion E50
- Malathiozol
- Malathiozoo
- Malathon
- Malathyl LV Concentrate & ULV Concentrate
- Malatol
- Malatox
- Maldison
- Malmed
- Malphos
- Maltox MLT
- Mercaptosuccinic acid diethyl ester, *S*-ester with *O,O*-dimethyl phosphorodithioate
- Mercaptothion; mercaptotion
- Moscarda
- NCI COO215
- Oleophosphothion
- Ortho Malathion
- Phosphorodithioic acid *O,O*-dimethyl ester, *S*-ester with diethyl mercaptosuccinate
- Phosphothion
- Prioderm
- Sadofos
- Sadophos
- SF60
- Siptox 1
- Siptox I
- Sumitox
- TAK
- TM-4049
- Vegfru Malatox
- Vetiol
- Zithiol

Last updated: 16 April 1998

METHYL PARATHION

VOL.: 30 (1983) (p. 131)

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Methyl parathion was tested for carcinogenicity in mice and rats by administration in the diet. No evidence of carcinogenicity was found.

Methyl parathion, administered intraperitoneally at maternally lethal doses, was teratogenic to mice. In rats, decreased viability and body weight were seen in the progeny of treated animals, but no teratogenicity was observed.

Methyl parathion was weakly or not mutagenic in bacterial systems and in *Drosophila melanogaster*, but it was mutagenic in yeasts. In mammalian cells, sister chromatid exchange and presumed gene mutations were induced, but neither chromosomal aberration nor unscheduled DNA synthesis was elicited. Chromosomal aberrations and dominant lethal mutations were not increased in mice treated with methyl parathion. There is sufficient evidence that methyl parathion is mutagenic in a variety of cellular systems, but insufficient evidence that it is mutagenic in mammals.

5.2 Human data

Methyl parathion was introduced in 1949. Its production, formulation and use as an insecticide on cotton and other crops are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic effects of methyl parathion in humans. The available data were insufficient to evaluate the chromosomal effects of methyl parathion in humans.

No case report or epidemiological study of the carcinogenicity of methyl parathion alone was available to the Working Group. (See, however, the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

The available data do not provide evidence that methyl parathion is carcinogenic to experimental animals. No data on humans were available.

The available data provide no evidence that methyl parathion is likely to present a carcinogenic risk to humans.

Subsequent evaluation: Suppl. 7 (1987)

Last updated: 16 April 1998

PARATHION

VOL.: 30 (1983) (p. 153)

CAS No.: 56-38-2
Chem. Abstr. Name: *O,O*-Diethyl *O*-4-nitrophenyl phosphorothioate

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Parathion was tested for carcinogenicity in one strain of mice and in three strains of rats by administration in the diet. Although a dose-related increase in the incidence of adrenal cortical adenomas was observed in male and female rats of one strain, the significance of the occurrence of those lesions in aged rats is not well understood. A low incidence of carcinomas at this site was observed in each of the treated groups in animals of both sexes. The other experiments in rats and the experiment in mice were considered to be inadequate for evaluation.

Parathion produced embryocidal effects and foetal growth reduction but no malformation in mice and rats at doses that were generally below the level that is toxic for the maternal organism.

Parathion was not mutagenic to a wide range of microorganisms in the presence or absence of rat liver microsomal preparations. It did not induce unscheduled DNA synthesis in cultured mammalian cells, recessive lethal mutations in *Drosophila melanogaster* or dominant lethal mutations in mice. Thus, no evidence has been found that parathion is mutagenic.

5.2 Human data

Parathion was introduced in 1947. Its production, formulation and use as an insecticide in a variety of agricultural applications are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of parathion in humans.

No case report or epidemiological study of the carcinogenicity of parathion alone was available to the Working Group. (See, however, the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

There is *inadequate evidence* to evaluate the carcinogenicity of parathion to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of parathion to humans.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Suppl. 7 (1987) (p. 69: **Group 3**)

Synonyms

- AAT
- AATP
- AC 3422
- ACC 3422
- Alkron
- Alleron
- American Cyanamid 3422
- Aphamite
- Aralo
- Bayer E-605
- Bladan
- Bladan F
- Corothion
- Danthion
- Diethyl *para*-nitrophenol thiophosphate
- Diethyl *para*-nitrophenyl phosphorothionate
- Diethyl 4-nitrophenyl phosphorothionate
- *O,O*-Diethyl *O*-(*para*-nitrophenyl) phosphorothioate
- Diethyl-*para*-nitrophenyl monothiophosphate
- Diethyl *para*-nitrophenyl thionophosphate
- *O,O*-Diethyl *O*-(*para*-nitrophenyl) thionophosphate
- *O,O*-Diethyl *O*-*para*-nitrophenyl thiophosphate
- *O,O*-Diethyl *0*-(4-nitrophenyl) thiophosphate
- Diethyl parathion
- DNTP
- Drexel Parathion 8E
- E 605
- E 605f
- Ekatin WF & WF ULV
- Ekatox
- ENT 15,108
- Ethlon
- Ethyl parathion
- Etilon
- Folidol
- Folidol E & E 605
- Folidol oil
- Fosfermo
- Fosferno
- Fosferno 50
- Fostox
- Gearphos
- Genithion
- Lirothion
- NCI CO0226
- Niran
- Niran E-4
- Nitrostygmine
- NIU1F 100
- Nourithion
- Oleofos 20
- Oleoparaphene
- Oleoparathion
- Orthophos
- Pacol
- Panthion
- Paradust
- Paraflow
- Paramar

- Paramar 50
- Paraphos
- Parathene
- Parathion-ethyl
- Parawet
- Pencap E
- Penncap E
- Penphos
- Phoskil
- Phos-Kil
- Phosphenol
- Phosphorothioic acid *O,O*-diethyl *O*-(4-nitrophenyl) ester
- Phosphorothioic acid *O,O*-diethyl *O*-(*para*-nitrophenyl) ester
- RB
- Rhodiasol
- Rhodiatox
- Rhodiatrox
- Selephos
- Sixty-Three Special E.C. Insecticide
- SNP
- Soprathion
- Stathion
- Sulphos
- Super Rodiatox
- Thiophos
- Thiophos 3422
- Vapophos
- Vitrex

Last updated: 9 April 1998

PIPERONYL BUTOXIDE

VOL.: 30 (1983) (p. 183)

CAS No.: 51-03-6
Chem. Abstr. Name: 1,3-Benzodioxole, 5[(2-[2-butoxyethoxy]ethoxy)methyl]-6-propyl

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Piperonyl butoxide was tested for carcinogenicity in two studies in rats and in three studies in mice by oral administration. It was also tested in mice of three strains by subcutaneous administration (in one, neonatally). No statistically significant incidence of tumours was observed in mice or rats.

Treatment of rats with high doses of piperonyl butoxide during organogenesis did not affect foetal development; however, its continued administration at high dose levels for three generations caused reduction in the number of pregnancies and offspring.

Piperonyl butoxide has been reported to be nonmutagenic to bacteria, silkworms and cultured mammalian cells. A dominant lethal study in mice was inconclusive. Lack of availability to the Group of much of the detailed data prevented overall evaluation of the mutagenicity of piperonyl butoxide, but all cited results have been negative.

5.2 Human data

Piperonyl butoxide was developed in 1947. Its production, formulation and use as an insecticide synergist in a variety of nonagricultural and agricultural applications are potential sources of exposure, both of workers and of the general population.

The available data were insufficient to evaluate the teratogenicity of piperonyl butoxide to humans. No data were available to evaluate its chromosomal effects in humans.

No case report or epidemiological study of the carcinogenicity of piperonyl butoxide alone was available to the Working Group. (See also the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

The available data do not provide evidence that piperonyl butoxide is carcinogenic to experimental animals. No data on humans were available.

The available data provide no evidence that piperonyl butoxide is likely to present a carcinogenic risk to humans.

Subsequent evaluation: Suppl. 7 (1987) (p. 70: **Group 3**)

Synonyms

- Butacide
- Butocide

- Butoxide
- α-[2-(2-Butoxyethoxy)ethoxy]-4,5-(methylenedioxy)-2-propyltoluene
- α-[2-(2-*n*-Butoxyethoxy)ethoxy]-4,5-methylene dioxy-2-propyltoluene
- Butyl carbitol-6-propylpiperonyl ether
- Butyl carbityl(3-propylpiperonyl)ether
- ENT 14,250
- FAC 5273
- NIA 5273
- Nusyn-Noxfish
- Prentox
- 6-Propylbenzyl)(butyl)diethylene glycol ether
- 3,4-Methylenedioxy-6-propylbenzyl-*n*-butyl diethyleneglycol ether
- NCI CO28l3
- 6-Propylpiperonyl butyl diethylene glycol ether
- Pybuthrin
- Pyrenone 606
- Synpren-Fish

Last updated: 9 April 1998

TETRACHLORVINPHOS

VOL.: 30 (1983) (p. 197)

CAS No.: 22248-79-9
Chem. Abstr. Name: Phosphoric acid, 2-chloro-1-(2,4,5-trichlorophenyl)ethenyl dimethyl ester, (Z)-

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Tetrachlorvinphos was tested for carcinogenicity in mice and rats by administration in the diet. It produced hepatocellular carcinomas in male mice and benign and malignant liver-cell tumours in female mice. In rats, it increased the incidences of C-cell adenoma of the thyroid and of cortical adenoma of the adrenal in females.

No data were available to evaluate the teratogenicity of tetrachlorvinphos to experimental animals.

Tetrachlorvinphos was not mutagenic to bacteria in the systems studied. It was a weak inducer of chromosomal aberrations in human lymphocytes *in vitro*. No overall evaluation of the mutagenicity of tetrachlorvinphos could be made.

5.2 Human data

Tetrachlorvinphos was introduced in 1966. Its production, formulation and use as an insecticide on livestock and poultry are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of tetrachlorvinphos in humans.

No case report or epidemiological study of the carcinogenicity of tetrachlorvinphos alone was available to the Working Group; however, it should be noted that the period since introduction of this compound may be too short for a carcinogenic effect to be detected in humans. (See also the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

Results of experiments in mice and rats provide *limited evidence* that tetrachlorvinphos is carcinogenic to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of tetrachlorvinphos to humans. likely to present a carcinogenic risk to humans.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Suppl. 7 (1987) (p. 72: **Group 3**)

Synonyms

- Appex
- 2-Chloro-1-(2,4,5-trichlorophenyl)vinyl dimethyl phosphate
- 2-Chloro-1-(2,4,5-trichlorophenyl)vinyl phosphoric acid dimethyl ester

- CVMP
- Dimethyl 2,4,5-trichloro-α-(chloromethylene)benzyl phosphate
- Dimethyl-1-(2,4,5-trichlorophenyl)-2-chlorovinyl phosphate
- Dust M
- ENT 2541
- Gardicide
- Gardona
- *cis*-Gardona
- NCI CO0168
- Rabon
- Rabon Oral Larvicide (ROL)
- Rabond Ravap
- ROL
- SD 8447
- Stirofos
- Stirophos
- 2,4,5-Trichloro-α-(chloromethylene)benzyl alcohol, dimethyl phosphate
- Vinfos

Last updated: 16 April 1998

TRICHLORFON

VOL.: 30 (1983) (p. 207)

CAS No.: 52-68-6
Chem. Abstr. Name: Phosphoric acid, (2,2,2-trichloro-1-hydroxyethyl)-, dimethyl ester

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Trichlorfon was tested for carcinogenicity by oral administration in mice and rats, by intraperitoneal administration in mice, rats and hamsters, by dermal application in mice and by intramuscular administration in rats. The studies in mice and two of the studies in rats were considered inadequate for evaluation. The other studies in rats and that in hamsters were not indicative of a carcinogenic effect.

Trichlorfon was teratogenic to mice, rats and hamsters when given at dose levels which also caused reductions in maternal food intake and maternal body weight gain.

There is sufficient evidence that trichlorfon or its degradation products is mutagenic in bacteria, mammalian cells and mice. Inconsistent results have been reported with many systems, however, and the instability of trichlorfon may be an important consideration in interpreting these data.

5.2 Human data

Trichlorfon was introduced in 1952. Its use as an insecticide in agricultural applications is a potential source of exposure, both of workers and of the general population. Its use in human medicine as an anthelmintic is another possible source of human exposure.

No data were available to evaluate the teratogenic effects of trichlorfon in humans. The available data were insufficient to evaluate the chromosomal effects of trichlorfon in humans.

No case report or epidemiological study of the carcinogenicity of trichlorfon alone was available to the Working Group. (See, however, the section 'Cancer Epidemiology of Pesticide Manufacturers, Users and Formulators', in this volume.)

5.3 Evaluation

The available data, while providing no evidence that trichlorfon is carcinogenic to mice or rats, were considered inadequate to evaluate the carcinogenicity of this compound to experimental animals. In view of its mutagenicity, confirmed in several experimental systems, further studies on the carcinogenicity of this compound are warranted. The Working Group was aware that a long-term bioassay was in progress.

No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of trichlorfon to humans.

Subsequent evaluation: Suppl. 7 (1987) (p. 73: **Group 3**)

Synonyms

- Aerol 1
- Agroforotox
- Anthon
- Bay 15922
- Bayer 15922
- Bayer L l3/59
- Bilarcil
- Bovinox
- Briten
- Briton
- Cekufon
- Chlorak
- Chlorfos
- Chlorofos
- Chloroftalm
- Chlorophos
- Chlorophthalm
- Chloroxyphos
- Ciclosom
- Clorfos
- Clorofos
- Combot
- Crimex
- Danex
- DEP
- Depthon
- DETF
- Dicontal
- Dimethoxy-2,2,2-trichloro-1-hydroxyethyl phosphine oxide
- Dimethyl ester of 2,2,2-trichloro-1-hydroxyethyl phosphonate
- Dimethyl 1-hydroxy-2,2,2-trichloroethylphosphonate
- *O,O*-Dimethyl(1-hydroxy-2,2,2-trichloroethyl)phosphonate
- Dimethyltrichlorohydroxyethyl phosphonate
- *O,O*-Dimethyl(2,2,2-trichloro-1-hydroxyethyl)phosphonate
- Dimetox
- Dipterax
- Dipterex
- Diptevur
- Ditrifon
- Dylox
- Dylox-Metasystox-R
- Dyrex
- Dyvon
- ENT 19,763
- Equino-Aid
- Flibol E
- Fliegenteller
- Forotox
- Foschlor
- Foschlor R & R50
- Foschlorem
- 1-Hydroxyethyl) dimethylphosphonate
- 1-Hydroxy-2,2,2-trichloroethylphosphonic acid dimethyl ester
- Hypodermacid
- Loisol
- Masoten
- Mazoten
- Metifonate
- Methyl chlorophos

- Metrifonate
- Metriphonate
- NCI-C54831
- Neguvon
- Neguvon A
- Phoschlor
- Phoschlor R50
- Polfoschlor
- Proxol
- Ricifon
- Ritsifon
- Satox 20WSC
- Soldep
- Sotipox
- Trichlorofon
- (2,2,2-Trichloro-1-hydroxyethyl) phosphonic acid dimethyl ester
- Trichlorophon
- Trichlorphene
- Trichlorphon
- Trichlorphone
- Trinex
- Tugon
- Tugon Stable Spray
- Tugon Fly Bait
- Vermicide Bayer 2349
- Volfartol
- Votexit
- WEC 50
- Wotexit

Last updated: 16 April 1998

DIALLATE

VOL.: 30 (1983) (p. 235)

CAS No.: 2303-16-4
Chem. Abstr. Name: Carbamothioic acid, bis(1-methylethyl)-, *S*-(2,3-dichloro-2-propenyl)ester

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Diallate was tested for carcinogenicity in two strains of mice by oral and subcutaneous administration and in one strain of rats by oral administration. Following its administration in the diet, the incidence of hepatomas was increased in male mice of both strains, and the incidence of lung adenomas was increased in male mice of one strain. The study in rats was considered to be inadequate for evaluation.

No data were available to evaluate the teratogenicity of diallate to experimental animals.

Diallate was mutagenic in bacteria and yeasts. It induced mutations in *Drosophila melanogaster*, and unscheduled DNA synthesis, sister chromatid exchange, gene mutations and chromosomal aberrations in cultured mammalian cells. Exogenous metabolic activation was usually required to produce or enhance mutagenic activity. Diallate was ineffective in a dominant lethal test in mice. There is sufficient evidence that diallate is mutagenic in cellular systems, but insufficient evidence for its mutagenicity in mammals.

5.2 Human data

Diallate was introduced in 1960. Its production, formulation and use as a herbicide in agricultural applications are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of diallate in humans.

No case report or epidemiological study of the carcinogenicity of diallate alone was available to the Working Group. (See, however, the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

Results of experiments in mice provide *limited evidence* that diallate is carcinogenic to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of diallate to humans.

For definition of the italicized terms, see Preamble Evaluation.

Previous evaluation: Vol. 12 (1976)

Subsequent evaluation: Suppl. 7 (1987) (p. 61: **Group 3**)

Synonyms

- Avadex
- Bis(1-methylethyl)carbamothioic acid *S*-(2,3-dichloro-2-propenyl) ester
- CP 15336
- DATC
- 2,3-DCDT
- Diallate
- Dichloroallyl diisopropylthiocarbamate
- *S*-(2,3-Dichloroallyl)-*N,N*-diisopropyl-monothiocarbamate
- 2,3-Dichloroallyl *N,N*-diisopropylthiocarbamate
- *S*-(2,3-Dichloroallyl)-*N,N*-diisopropylthiocarbamate
- *S*-(2,3-Dichloroallyl)diisopropylthiolcarbamate
- 2,3-Dichloro-2-propene-1-thiol diisopropylcarbamate
- Diisopropylthiocarbamic acid *S*-(2,3-dichloroallyl) ester
- Pyradex

Last updated: 16 April 1998

FLUOMETURON

VOL.: 30 (1983) (p. 245)

CAS No.: 2164-17-2
Chem. Abstr. Name: Urea, *N,N*-dimethyl-*N'*-[3-(trifluoromethyl)phenyl]-

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Fluometuron was tested for carcinogenicity in mice and rats by administration in the diet. In male mice, an increased incidence of liver-cell tumours was found. In female mice and in rats of both sexes, the results were not indicative of a carcinogenic effect.

No data were available to evaluate the teratogenicity of fluometuron to experimental animals.

Fluometuron was weakly or nonmutagenic to *Salmonella typhimurium*. It was not active in inducing mitotic gene conversion in yeast. Mitotic chromosomal aberrations were observed in treated *Vicia faba* root tips and cotton seeds. In all studies that gave positive results, commercial formulations were known or suspected to have been used, suggesting that the observed activity may be attributable to other ingredients. There are insufficient data to evaluate the mutagenicity of pure fluometuron, but there is limited evidence that commercial formulations of this compound are mutagenic in experimental animals.

5.2 Human data

Fluometuron was introduced in 1960. Its production, formulation and use as a herbicide on cotton are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenicity of fluometuron to humans. The available data were insufficient to evaluate its chromosomal effects in humans.

No case report or epidemiological study of the carcinogenicity of fluometuron alone was available to the Working Group. (See also the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

The available data are *inadequate* to evaluate the carcinogenicity of fluometuron to experimental animals. No data on humans were available.

No evaluation of the carcinogenicity of fluometuron to humans can be made.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Suppl. 7 (1987) (p. 63: **Group 3**)

Synonyms

- C 2059

- Ciba 2059
- Cotoran
- Cotoran Multi 50WP
- Cottonex
- 1,1-Dimethyl-3-(3-trifluoromethylphenyl)urea
- Herbicide C-2059
- Lanex
- NCI CO8695
- Pakhtaran
- *N*-(*meta*-Trifluoromethylphenyl)-*N,N'*-dimethylurea
- *N*-(3-Trifluoromethylphenyl)-*N,N'*-dimethylurea
- 3-(*meta*-Trifluoromethylphenyl)-1,1-dimethylurea
- 3-(3-Trifluoromethylphenyl)-1,1-dimethylurea

Last updated: 16 April 1998

(4-CHLORO-2-METHYLPHENOXY)ACETIC ACID (MCPA)

VOL.: 30 (1983) (p. 255)

5. Summary of Data Reported and Evaluation

5.1 Experimental data

No adequate study of the carcinogenicity of MCPA to animals was available to the Working Group.

MCPA and maternally toxic doses of its ethyl ester were teratogenic and embryotoxic to rats.

MCPA was not mutagenic in bacterial systems in plate or host-mediated assays. A questionable result was obtained with MCPA in yeast, but its methyl ester was mutagenic. Recessive lethal mutations, but no chromosome loss, non-disjunction or X-Y chromosome rearrangement, were induced in *Drosophila melanogaster*. No overall evaluation of the mutagenicity of MCPA could be made.

5.2 Human data

MCPA was introduced in 1945. The production and formulation of this compound and of its derivatives and their use as herbicides in agricultural applications are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of MCPA in humans.

Exposure to MCPA alone was reported for some cases in one of three case-control studies and in one case report. The case-control studies are also discussed in detail in the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.

5.3 Evaluation

No adequate data were available to evaluate the carcinogenicity of MCPA to experimental animals. The data on humans were also considered to be *inadequate*.

The available data are insufficent to evaluate the carcinogenicity to humans of MCPA alone.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluations: Suppl. 7 (1987); see also Vol. 41 (1986) (Chlorophenoxy herbicides, occupational exposure to)

Last updated: 16 April 1998

NITROFEN

VOL.: 30 (1983) (p. 271)

CAS No.: 1836-75-5
Chem. Abstr. Name: Benzene, 2,4-dichloro-1-(4-nitrophenoxy)-

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Nitrofen (technical grade) was tested for carcinogenicity in one strain of mice and in two strains of rats by administration in the diet. In two studies in mice, it significantly increased the incidence of hepatocellular carcinomas. In one study, haemangiosarcomas at various anatomical sites occurred in a significantly higher incidence among male mice given a higher dose. In rats of one strain, adenocarcinomas of the pancreas were produced in females.

Nitrofen was teratogenic to mice and rats when given at very high doses. It decreased neonatal and postnatal viability in three-generation reproduction studies in rats.

Nitrofen was mutagenic to *Salmonella typhimurium*. In two studies, there was no evidence for chromosomal damage in mammals. The data are insufficient to evaluate the mutagenicity of nitrofen in mammals.

5.2 Human data

Nitrofen was introduced in about 1964. Its production, formulation and use as a herbicide in agricultural applications are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of nitrofen in humans.

No case report or epidemiological study of the carcinogenicity of nitrofen alone was available to the Working Group; however, it should be noted that the period since introduction of this compound may be too short for a carcinogenic effect to be detected in humans. (See also the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

No data on humans were available.

There is *sufficient evidence* that technical-grade nitrofen is carcinogenic to experimental animals.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Suppl. 7 (1987) (p. 67: **Group 2B**)

Synonyms

- 2,4-Dichloro-4'-nitrodiphenyl ether
- 2,4-Dichloro-4'-nitrophenyl ether
- 4-(2,4-Dichlorophenoxy)nitrobenzene

- 2,4-Dichlorophenyl-*para*-nitrophenyl ether
- FW 925
- Mezotox
- NCI CO0420
- Niclofen
- Nip
- Nitrafen
- Nitraphen
- Nitrochlor
- 4'-Nitro-2,4-dichlorodiphenyl ether
- 4-Nitro-2',4'-dichlorophenyl ether
- Nitrofene
- Nitrophen
- Nitrophene
- Tok
- Tok 2, E, E25, & WP50
- Tokkorn
- Trizilin

Last updated: 16 April 1998

SULFALLATE

VOL.: 30 (1983) (p. 283)

CAS No.: 95-06-7
Chem. Abstr. Name: Carbamodithioic acid, diethyl-, 2-chloro-2-propenyl ester

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Sulfallate (technical grade) was tested for carcinogenicity in one strain of mice and in one strain of rats by administration in the diet. It was carcinogenic in mice, inducing mammary gland tumours in females and lung tumours in males. It was also carcinogenic in rats, inducing mammary gland tumours in females and benign and malignant tumours of the forestomach in males.

No data were available to evaluate the teratogenicity of sulfallate to experimental animals.

Sulfallate was mutagenic to *Salmonella typhimurium* in the presence of exogenous metabolic activation and to *Aspergillus* and *Streptomyces* but not to *Saccharomyces cerevisiae* with or without metabolic activation. There is sufficient evidence for the mutagenicity of sulfallate in bacterial systems but insufficient evidence for its mutagenicity in mammalian cells or in mammals.

5.2 Human data

Sulfallate was introduced in 1954. Its production and use as a herbicide in agricultural applications are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of sulfallate in humans.

No case report or epidemiological study of the carcinogenicity of sulfallate alone was available to the Working Group. (See also the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

No data on humans were available.

There is *sufficient evidence* that sulfallate is carcinogenic to experimental animals.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Suppl. 7 (1987) (p. 72: **Group 2B**)

Synonyms

- CDEC
- Chloroallyl diethyldithiocarbamate
- 2-Chloroallyl *N,N*-diethyldithiocarbamate
- 2-Chloro-2-propene-1-thiol diethyldithiocarbamate

- CP 4742
- CP 4572
- Diethylcarbamodithioic acid 2-chloro-2-propenyl ester
- Diethyldithiocarbamic acid 2-chloroallyl ester
- NCI CO0453
- Thioallate
- Vegadex
- Vegdex Super

Last updated: 16 April 1998

CAPTAN

VOL.: 30 (1983) (p. 295)

CAS No.: l33-06-2
Chem. Abstr. Name: 1*H*-Isoindole-l,3(2H)-dione, 3a,4,7,7a-tetrahydro-2-[(trichloromethyl)thio]-

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Captan was tested for carcinogenicity in mice and rats by administration in the diet. It was carcinogenic to one strain of mice, inducing duodenal tumours (adenocarcinoma and adenomatous polyp). No evidence of carcinogenicity was found in rats.

Captan has shown little, if any, embryotoxic or teratogenic potential in mice or rats at maternally tolerated doses. Results obtained in tests with rabbits and hamsters were considered inconclusive.

Captan was mutagenic to bacteria and yeast. Both positive and negative results were obtained in the host-mediated assay in mice. Weak or negative effects were observed in *Drosophila melanogaster*. Captan induced chromosomal aberrations, sister chromatid exchange and mutations at several loci, but not unscheduled DNA synthesis, in cultured mammalian cells. No increase in micronucleated erythrocytes or chromosomal aberrations was detected in treated mice or rats; positive results obtained in dominant lethal tests in mice and rats were not confirmed by other studies. Thus, there is sufficient evidence to establish the mutagenicity of captan in cellular systems, but the data were insufficient to establish its mutagenicity in mammals.

5.2 Human data

Captan was introduced commercially in 1951. Its production, formulation and use as a fungicide in agricultural and industrial applications and in cosmetics are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of captan in humans.

No case report or epidemiological study of the carcinogenicity of captan alone was available to the Working Group. (See, however, the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

Results of the experiments in mice provide *limited evidence* that captan is carcinogenic to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of captan to humans.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Suppl. 7 (1987) (p. 59: **Group 3**)

Synonyms

- Aacaptan
- Agrosol S
- Agrox 2-Way and 3-Way
- Amercide
- Bangton
- Bean Seed Protectant
- Captaf, Captan 50W
- Captan-Streptomycin 7.5-0.l Potato Seed Piece Protectant
- Captane
- Captex
- ENT 26538
- Esso Fungicide 406
- Flit 406
- Fungus Ban Type II
- Glyodex 37-22
- Granox PFM
- Gustafson Captan 30-DD
- Hexacap
- Isotox Seed Treater "D" and "F"
- Kaptan
- Malipur
- Merpan
- Micro-Check 12
- Neracid
- Orthocide
- Orthocide 7.5, 50, 75 & 83
- Osocide
- SR 406
- Stauffer Captan
- 3a,4,7,7a-Tetrahydro-*N*-(trichloromethanesulphenyl) phthalimide
- *N*-Trichloromethylmercapto-4-cyclohexenel,2-dicarboximide
- *N*-Trichloromethylmercapto-tetrahydrophthalimide
- *N*-(Trichloromethylmercapto)-Δ^4-tetrahydrophthalimide
- *cis*-*N*-[(Trichloromethyl)thio]-4-cyclohexene-l,2-dicarboximide
- *N*-[(Trichloromethyl)thio]-4-cyclohexene-1,2- dicarboximide
- *N*-(Trichloromethylthio)-cyclohex-4-ene-1,2- dicarboximide
- *N*-Trichloromethylthiocyclohex-4-ene-1,2-dicarboxyimide
- *N*-[(Trichloromethyl)thio]tetrahydrophthalimide
- *N*-[(Trichloromethyl)thio]-Δ^4-tetrahydrophthalimide
- *N*-Trichloromethylthio-3a,4,7,7a-tetrahydrophthalimide
- Vancide P-75, 89 and 89 RE
- Vangard K
- Vanguard K
- Vanicide
- Vondcaptan

Last updated: 16 April 1998

CHLOROTHALONIL

VOL.: 30 (1983) (p. 319)

CAS No.: 1897-45-6
Chem. Abstr. Name: 1,3-Benzenedicarbonitrile, 2,4,5,6-tetrachloro-

5. Summary of Data Reported and Evaluation

5.1 Experimental data

Chlorothalonil was tested for carcinogenicity in mice and rats by administration in the diet; it produced adenomas and adenocarcinomas of the kidney in rats. No evidence of carcinogenicity was found in mice.

No published data were available to evaluate the teratogenicity of this compound to experimental animals.

Chlorothalonil was not mutagenic in two fungal systems and was reported to be nonmutagenic in *Salmonella typhimurium*. Available data on other systems are inadequate, and no overall evaluation of the mutagenicity of chlorothalonil could be made.

5.2 Human data

Chlorothalonil was introduced commercially in 1969. Its production, formulation and use as a fungicide in agricultural and industrial applications are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of chlorothalonil in humans.

No case report or epidemiological study of the carcinogenicity of chlorothalonil alone was available to the Working Group; however, it should be noted that the period since introduction of this compound may be too short for a carcinogenic effect to be detected in humans. (See also the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulations and Users', in this volume).

5.3 Evaluation

Results of one experiment in rats provide *limited evidence* that chlorothalonil is carcinogenic to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of chlorothalonil to humans.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluations: Suppl. 7 (1987) (p. 60); Vol. 73 (1999)

Synonyms

- Bravo
- Bravo 6F, 500 & W-75
- Chloroalonil
- DAC 2787

- Daconil
- Daconil 2787, 2787 W-75, 2787 WP
- Daconil 2787 Flowable Fungicide
- Daconil 2787 Fungicide
- Dacosoil
- 1,3-Dicyanotetrachlorobenzene
- Exotherm
- Exotherm Termil
- Forturf
- NCI CO0102
- Nopcocide
- Nopcocide N40D & N96
- Sweep
- TCIN
- *meta*-TCPN
- Termil
- Tetrachlorisophthalonitrile
- 2,4,5,6-Tetrachloro-3-cyanobenzonitrile
- 2,4,5,6-Tetrachloroisophthalonitrile
- *meta*-Tetrachlorophthalodinitrile
- Tetrachloro-*meta*-phthalodinitrile
- TPN[pesticide]

Last updated: 30 September 1999

ortho-PHENYLPHENOL AND ITS SODIUM SALT

VOL.: 30 (1983) (p. 329)

***ortho*-Phenylphenol**
CAS No.: 90-43-7
Chem. Abstr. Name: (1,1'-Biphenyl)-2-ol

Sodium *ortho*-phenylphenate
CAS No.: 132-27-4
Chem. Abstr. Name: (1,1'-Biphenyl)-2-ol, sodium salt

5. Summary of Data Reported and Evaluation

5.1 Experimental data

ortho-Phenylphenol was tested for carcinogenicity in mice and rats by administration in the diet. Sodium *ortho*-phenylphenate was tested in rats by administration in the diet. No evidence of carcinogenicity of *ortho*-phenylphenol was found in mice or rats, but both studies had some limitations. In rats, sodium *ortho*-phenyphenate is carcinogenic to the urinary tract, producing both benign and malignant tumours.

The available data were insufficient to evaluate the teratogenicity of sodium *ortho*-phenylphenate to experimental animals.

ortho-Phenylphenol and sodium *ortho*-phenylphenate have been subjected to a variety of mutagenicity tests, including those using rodents, without demonstrating any unambiguous mutagenic or DNA damaging effect. Commercial preparations have been reported to contain uncharacterized impurities capable of inducing chromosomal aberrations in cultured mammalian cells.

5.2 Human data

ortho-Phenylphenol was first made in commercial quantities in 1934, and sodium *ortho*-phenylphenate was introduced commercially in 1941-1943. Their production, formulation and use as fungicides and as antibacterial agents in agricultural applications and in a wide variety of industrial and household products are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of ortho-phenylphenol or sodium *ortho*-phenylphenate in humans.

No case report or epidemiological study of the carcinogenicity of ortho-phenylphenol or sodium ortho-phenylphenate alone was available to the Working Group. (See also the section 'Cancer Epidemiology of Pesticide Manufacturers, Formulators and Users', in this volume.)

5.3 Evaluation

The available data are *inadequate* to evaluate the carcinogenicity of *ortho*-phenylphenol to experimental animals. Results of the experiments in rats provide *limited evidence* that sodium *ortho*-phenylphenate is carcinogenic to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of *ortho*-phenylphenol or sodium *ortho*-phenylphenate to humans.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluations: Suppl. 7 (1987) (p. 70: *ortho*-phenylphenol - **Group 3**; p. 392: sodium *ortho*-phenylphenate - **Group 2B**); Vol. 73 (1999)

Synonyms for *ortho*-Phenylphenol

- *ortho*-Biphenylol
- *ortho*-Diphenylol
- Dowicide 1
- *ortho*-Hydroxybiphenyl
- 2-Hydroxybiphenyl
- *ortho*-Hydroxydiphenyl
- 2-Hydroxydiphenyl
- Orthoxenol
- 2-Phenylphenol
- Preventol O Extra
- Remol TRF
- Tetrosin OE
- Tumescal OPE
- *ortho*-Xenol

Last updated: 30 September 1999

1-NAPHTHYLTHIOUREA (ANTU)

VOL.: 30 (1983) (p. 347)

5. Summary of Data Reported and Evaluation

5.1 Experimental data

1-Naphthylthiourea was tested for carcinogenicity in mice and rats by administration in the diet. The studies were considered to be inadequate for evaluation.

No data were available to evaluate the teratogenicity of 1-naphthylthiourea to experimental animals.

1-Naphthylthiourea was mutagenic to *Salmonella typhimurium* in the presence of an exogenous metabolic activation system. It induced a transformed phenotype in Syrian hamster embryo cells *in vitro*. There is *limited evidence* that 1-naphthylthiourea is mutagenic in cellular systems. No data were available to evaluate its mutagenicity to mammals.

5.2 Human data

1-Naphthylthiourea was introduced in 1946. Its production, formulation and use as a rodenticide are potential sources of exposure, both of workers and of the general population.

No data were available to evaluate the teratogenic or chromosomal effects of 1-naphthylthiourea in humans.

No case report or epidemiological study of the carcinogenicity of 1-naphthylthiourea was available to the Working Group.

5.3 Evaluation

The available data were inadequate to evaluate the carcinogenicity of 1-naphthylthiourea to experimental animals. No data on humans were available.

The available data are insufficient to evaluate the carcinogenicity of 1-naphthylthiourea to humans.

N.B. - Subsequent to the meeting, the Working Group became aware of a study by Davies *et al.* (1982) in which 12 cases of urothelial tumour were reported among rodent operatives in England who used 1-naphthylthiourea which contained up to 0.2% 2-naphthylamine.

Subsequent evaluation: Suppl. 7 (1987)

Last updated: 16 April 1998