

MALATHION

4. Mechanistic and Other Relevant Data

4.5 Other adverse effects

Malathion was tested in ten regulatory toxicity submissions which are included in ToxRefDB. This includes four chronic and or carcinogenicity studies, two developmental studies, one multigenerational reproductive study and three developmental neurotoxicity studies.

Liver Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), congestion and spongiosis hepatitis were observed in the high-mid and high dose groups and adenoma in the high dose females with accompanying liver weight increases (Daly 1996 and Hardisty 2000). In a mouse carcinogenicity study, tested at 17.4/20.8, 143/167, 1476/1707 and 2978/3448 mg/kg/day (M/F), hypertrophy, nodules, adenoma and combined adenoma/carcinoma incidence were increased along with liver weights in mid high and high dose males and females. Foci and increased liver mass were also observed grossly in the high dose animals (Slauter 1994).

Kidney Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), renal inflammation was observed for the low mid dose females and up in addition to high mid dose males and up. Congestion, nephropathy and irregular surface were observed in the top two doses for males and females in addition to increases in kidney weights (Daly 1996 and Hardisty 2000). In a mouse carcinogenicity study, tested at 17.4/20.8, 143/167, 1476/1707 and 2978/3448 mg/kg/day (M/F), decreased renal tubule vacuolation was observed in the top two male doses and increased mineralization was observed in the top two female doses (Slauter 1994).

Spleen Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), atrophy and depletion were noted in splenic lymphoid follicles at the top two doses for males and females. Increased spleen weight were observed in the top two male doses (Daly 1996 and Hardisty 2000).

Stomach Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), forestomach congestion, edema, hyperkeratosis, squamous and basal cell hyperplasia, inflammation and ulcers were observed in the top two doses in males and females (Daly 1996 and Hardisty 2000).

Thyroid Gland Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), congestion was observed in the two mid doses for males and at the top dose for males and females in addition to thyroid gland cysts at the high dose for males and females. Thyroid weights were increased at the top three males doses but decreased at the top two female doses (Daly 1996 and

Hardisty 2000).

Testicular Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), testicular atrophy, degeneration, oligospermia and arrested maturation were observed in high dose animals but only at the interim sacrifice (Daly 1996).

Adrenal Gland Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), increased adrenal gland vacuolization was observed at the top two male doses and the top two female groups experienced early disappearance of the x-zone of the adrenal cortex (Daly 1996 and Hardisty 2000).

Skin Toxicity:

No effects were observed related to skin.

Lung Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), increased lung congestion was observed at the top two male and female doses and collapsed alveoli were observed at the top two male doses (Daly 1996 and Hardisty 2000).

Brain Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), increased brain congestion was observed at the top three male doses and the highest female dose group (Daly 1996).

Other Organ Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), parathyroid hyperplasia was observed in all dose groups accompanied by increased parathyroid weights in the top two male dose groups. Nasal hyperplasia, cysts, degeneration, dilation and inflammation were observed in the top two male and female groups. Sternal and femoral bone marrow congestion was observed in low mid dose males and mid high and high dose males and females. Unspecified pharynx lesions were observed in the top two doses for males and females. Corneal mineralization and neutrophilic cellular infiltration were observed in the eyes of high mid dose males and high dose males and females. Lacrimal and Hardarian glands were congested for males and females at the top two doses. Heart congestion was observed in low mid dose males and in males and females at the top two doses. Pituitary glands were congested at the high mid dose males and high dose males and females. Depletion and atrophy of the mediastinal lymph nodes were observed at the top three male doses and in the mesenteric lymph nodes of high dose males (Daly 1996 and Hardisty 2000). In a mouse carcinogenicity study, tested at 17.4/20.8, 143/167, 1476/1707 and 2978/3448 mg/kg/day (M/F), fibrous osteodystrophy was observed in the femurs and sternums of the top two female doses (Slauter 1994).

General Toxicity:

In a rat combined chronic/carcinogenicity study, tested at 4/5, 29/35, 359/415 and 739/868 mg/kg/day (M/F), decreased body weights were observed in males and females in the top two dose groups (Daly 1996 and Hardisty 2000). In a mouse carcinogenicity study, tested at 17.4/20.8, 143/167, 1476/1707 and 2978/3448 mg/kg/day (M/F), decreased body weights were observed in males and females in the

top two dose groups (Slauter 1994). In a rat developmental study dosed at 200, 400, 800 mg/kg/day, decreased body weights were observed in high dose females (Lochry 1989). In a rabbit developmental study dosed at 25, 50, 100 mg/kg/day, decreased body weights were observed in the top two maternal groups (Siglin 1985). In a two generation reproduction study dosed nominally at 27.5, 85, 250 and 375 mg/kg/day, high dose females in the original parental group and high dose male and female animals in the first offspring generation had decreased body weights (Schroeder 1990). In a range finding rat developmental neurotoxicity study dosed at 7.5, 750 and 1250 mg/kg/day, decreased body weight was observed at the top two maternal groups (Fulcher 2002b) In the rat combined chronic/carcinogenicity study increased mortality was noted at the two mid dose groups of males and at the top dose for males and females. Cholinesterase inhibition was observed at most dose levels in several of the different study types. This included decreases in general, plasma, erythrocyte and brain cholinesterase levels. The cholinesterase effects were accompanied by abnormal gait, tremors, and reduced activity by higher dose groups in the developmental neurotoxicity study.

Reproductive Toxicity:

In a two generation reproduction study dosed nominally at 27.5, 85, 250 and 375 mg/kg/day, offspring weights were reduced at the top two doses in males and females in multiple generations (Schroeder 1990). In a rabbit developmental study dosed at 25, 50, 100 mg/kg/day, increased resorptions were observed at the top two maternal groups (Siglin 1985).

Developmental Toxicity:

In a rat developmental neurotoxicity study dosed at 5, 50 and 150 mg/kg/day, renal dilation and vacuolation in addition to hydronephrosis were observed in high dose male offspring (Fulcher 2002a)

Developmental Neurotoxicity:

In a rat developmental neurotoxicity study dosed at 5, 50 and 150 mg/kg/day, increased thickness of the corpus callosum was observed in high dose males and females. Auditory reflexes were reduced at all does levels in males and females. Decreased vertical rearing and horizontal locomotion were observed in the top two females groups (Fulcher 2002a)

PARATHION

4. Mechanistic and Other Relevant Data

4.5 Other adverse effects

Parathion was tested in nine regulatory toxicity submissions and one open literature study which are included in ToxRefDB. This includes six chronic and or carcinogenicity studies, two subchronic studies, one multigenerational reproductive study and one developmental neurotoxicity study.

Liver Toxicity:

No effects were observed related to liver.

Kidney Toxicity:

No effects were observed related to kidney.

Spleen Toxicity:

No effects were observed related to spleen.

Stomach Toxicity:

No effects were observed related to stomach.

Thyroid Gland Toxicity:

No effects were observed related to thyroid gland.

Testicular Toxicity:

No effects were observed related to testes.

Adrenal Gland Toxicity:

No effects were observed related to adrenal gland.

Skin Toxicity:

No effects were observed related to skin.

Lung Toxicity:

No effects were observed related to lungs.

Brain Toxicity:

No effects were observed related to brain.

Other Organ Toxicity:

In a rat combined chronic/carcinogenicity study, tested at concentrations of 0.1/0.14, 0.42/0.53, 1.75/2.47 mg/kg/day (M/F), reduced ERG signals were observed in the eyes of mid and high dose males and females. Gross retinal abnormalities were observed in high dose males and females in addition to cataracts and turbid lenses in high dose females. A follow up review of this study revealed epithelium, optic nerve and ciliary body degeneration as well as retinal atrophy in high dose males. Stratification of the myelin sheath and structural alterations of axon in the optic nerve and alterations of the retina were observed in high dose females (Eiben 1986 and DER Memo 1986).

General Toxicity:

In a two generation reproductive study, dosed nominally at 0.05, 0.5 and 1 mg/kg/day, reduced body weights were observed in high dose males and females (Neeper-Bradley 1990). In a rat combined chronic/carcinogenicity study, tested at concentrations of 0.1/0.14, 0.42/0.53, 1.75/2.47 mg/kg/day (M/F), reduced body weights were observed for high dose males and females (Eiben 1986). In a subchronic rat study with only female animals, with doses of 0.04, 0.4 and 4 mg/kg/day, reduced body weight was observed in high dose animals (Atkinson 1991b) Cholinesterase inhibition was also observed in several studies including decreases in plasma, erythrocyte, brain and eye cholinesterase. Cholinesterase inhibition was observed at 4 mg/kg/day in the subchronic female rat study, from 0.0024 to 0.7937 mg/kg/day in a six month dog study, at 0.5 and 1 mg/kg/day in the rat reproduction study, from 0.01 to 0.1 mg/kg/day in a yearlong dog study, at 20 mg/kg/day in a mouse carcinogenicity study, and from 0.42 to 2.47 in chronic/carcinogenicity rat study (Atkinson 1991a) In the chronic/carcinogenicity rat study, the cholinesterase inhibition was accompanied by clinical signs like

tremors, abnormal gait, and poor general condition at the highest dose and increased mortality in females.

Reproductive Toxicity:

In a two generation reproductive study, dosed nominally at 0.05, 0.5 and 1 mg/kg/day, reduced pup weights were observed in high dose male and female offspring (Neeper-Bradley 1990). In a developmental neurotoxicity study dosed at 1.3 and 1.9 mg/kg/day, reduced pup weights were observed in male animals at both dose levels (Stamper 1988).

Developmental Toxicity:

No developmental effects were observed.

Developmental Neurotoxicity:

In a developmental neurotoxicity study dosed at 1.3 and 1.9 mg/kg/day, motor activity was reduced in addition to the density of muscarinic binding sites in males at both doses (Stamper 1988). High dose animals also saw impaired reflexes. In another developmental neurotoxicity study dosed at 0.1 and 0.2 mg/kg/day, high dose males and females saw learning and memory impairment when tested with a maze and decreased reflexes (Timofeeva 2008).

DIAZINON

4. Mechanistic and Other Relevant Data

4.5 Other adverse effects

Diazinon was tested in thirteen regulatory toxicity submissions which are included in ToxRefDB. This includes five chronic and or carcinogenicity studies, two subchronic studies, two developmental studies, two multigenerational reproductive studies and two developmental neurotoxicity studies.

Liver Toxicity:

In a subchronic rat study, tested at concentrations of 0.03/0.04, 0.3/0.4, 15/19 and 168/212 mg/kg/day (M/F), liver hypertrophy was observed in high dose females along with increases in liver weight (Singh 1988).

Kidney Toxicity:

No effects were observed related to kidney.

Spleen Toxicity:

No effects were observed related to spleen.

Stomach Toxicity:

Though not specifically attributed to the stomach, gastrointestinal tract issues were observed in the high dose animals in a rabbit developmental toxicity study dosed at 7, 25 and 100 mg/kg/day. Congestion, erosion and hemorrhages were observed in the gastrointestinal tract of animals that died (Harris 1981).

Thyroid Gland Toxicity:

No effects were observed related to thyroid glands.

Testicular Toxicity:

No effects were observed related to testes.

Adrenal Gland Toxicity:

No effects were observed related to adrenal glands.

Skin Toxicity:

No effects were observed related to skin.

Lung Toxicity:

In a chronic dog study, lung weights were decreased at all dose levels for females ranging from 0.0037 to 9.1 mg/kg/day (Rudzki 1991).

Brain Toxicity:

No effects were observed related to brain.

Other Organ Toxicity:

In a chronic dog study, mandibular salivary gland weights were decreased at the top two of four dose levels for females, which were dosed at 4.5 and 9.1 mg/kg/day (Rudzki 1991).

General Toxicity:

Decreased body weights were observed in high dose male and female rats in the subchronic study (Singh 1988). In the chronic dog study, reduced body weight was observed in mid-high dose males and high dose males and females (Rudzki 1991). Decreased body weight was also observed at the highest dose in rat and rabbit developmental studies and the rat reproduction study. Increased mortality was observed in the high dose females in the reproduction study. Cholinesterase inhibition was observed at most dose levels in several of the different study types. This included decreases in general, plasma, erythrocyte and brain cholinesterase levels.

Reproductive Toxicity:

In a two generation reproductive study, rats were dose at 0.67/0.77, 6.69/7.63, and 35.15/41.43 mg/kg/day (M/F). Reduced mating, litter size, viability index were observed in high dose animals. Fertility and gestational interval were reduced in high dose females.

Developmental Toxicity:

In a rat developmental study dosed at 10, 20 and 100 mg/kg/day, increased rudimentary T-14 ribs and decreased fetal weights were observed in high dose animals (Infurna 1985). In a rat developmental neurotoxicity study, dosed at 0.026, 2.36 and 24.2 mg/kg/day, decreased pup weight was observed in high dose male and female animals. In this same study, delayed vaginal opening and preputial separation were observed in high dose animals (Mandella 2003). In another developmental neurotoxicity study, which served as a range-finding study for the first, rats were dosed at 0.0125, 0.063, 6.56, and 38.06 mg/kg/day. Decreased pup weight was observed in high dose males and females (Mandella 2002).

Developmental Neurotoxicity:

In a rat developmental neurotoxicity study, dosed at 0.026, 2.36 and 24.2 mg/kg/day, an increased number of errors and latent period were observed in high dose male animals tested in a maze (Mandella 2003). In another developmental neurotoxicity study, which served as a range-finding study for the first, rats were dosed at 0.0125, 0.063, 6.56, and 38.06 mg/kg/day. Decreased surface righting reflex was observed in high dose females (Mandella 2002).

TETRACHLORVINPHOS

4. Mechanistic and Other Relevant Data

4.5 Other adverse effects

Tetrachlorovinphos was tested in two regulatory toxicity submissions which are included in ToxRefDB. This includes two carcinogenicity studies and one developmental neurotoxicity study.

Liver Toxicity:

In a rat carcinogenicity study dosed at 212.5 and 425 mg/kg/day, liver granuloma was observed for both female doses and in high dose males. In a mouse carcinogenicity study tested at 1200 and 2400 mg/kg/day, liver granuloma, hepatocellular carcinoma and neoplastic nodules were observed in male and female groups at both doses (1978 Bioassay)

Kidney Toxicity:

No effects were observed related to kidney.

Spleen Toxicity:

No effects were observed related to spleen.

Stomach Toxicity:

No effects were observed related to stomach.

Thyroid Gland Toxicity:

In a rat carcinogenicity study dosed at 212.5 and 425 mg/kg/day, c-cell and follicular cell hypertrophy were observed in the thyroids of male and female animals at both dose levels (1978 Bioassay)

Testicular Toxicity:

No effects were observed related to testes.

Adrenal Gland Toxicity:

No effects were observed related to adrenal gland.

Skin Toxicity:

No effects were observed related to skin.

Lung Toxicity:

No effects were observed related to lungs.

Brain Toxicity:

No effects were observed related to brain.

Other Organ Toxicity:

No effects were observed related to any other organs.

General Toxicity:

Decreased body weights were observed in rats and mice at both dose levels in the carcinogenicity studies. There was increased mortality in the high dose male rats (1978 Bioassay)

Reproductive Toxicity:

No reproductive effects were observed.

Developmental Toxicity:

In a rat developmental neurotoxicity study tested at 10, 50, and 200 mg/kg/day decreased pup weight was observed at the high dose (Barnet 2005).

Developmental Neurotoxicity:

In a rat developmental neurotoxicity study tested at 10, 50, and 200 mg/kg/day decreased thickness of the striatum, corpus callosum, and hippocampus were observed in high dose males and females and decreased thickness of the cerebellum was observed in high dose males (Barnet 2005).

GLYPHOSATE

4. Mechanistic and Other Relevant Data

4.5 Other adverse effects

Glyphosate was tested in nine regulatory toxicity submissions which are included in ToxRefDB. This includes four chronic and or carcinogenicity studies, one subchronic study, two multigenerational reproductive studies and two developmental studies.

Liver Toxicity:

In a rat combined chronic/carcinogenicity study, tested at nominal concentrations of 100, 400, and 1000 mg/kg/day, liver weight increased in high dose males (Stout 1990). In a mouse carcinogenicity study tested at 150, 1500 and 4500 mg/kg/day, liver hypertrophy and necrosis were observed in high dose males (Knezevich 1983).

Kidney Toxicity:

Spleen Toxicity:

No effects were observed related to spleen.

Stomach Toxicity:

In a rat combined chronic/carcinogenicity study, tested at nominal concentrations of 100, 400, and 1000 mg/kg/day, inflammation was observed in the stomach mucosa or mid and high dose females (Stout 1990).

Thyroid Gland Toxicity:

No effects were observed related to thyroid gland.

Testicular Toxicity:

In a mouse carcinogenicity study tested at 150, 1500 and 4500 mg/kg/day, testes weight was increased in high dose males (Knezevich 1983).

Adrenal Gland Toxicity:

No effects were observed related to adrenal gland.

Skin Toxicity:

No effects were observed related to skin.

Lung Toxicity:

No effects were observed related to lungs.

Brain Toxicity:

No effects were observed related to brain.

Other Organ Toxicity:

In a rat combined chronic/carcinogenicity study, tested at nominal concentrations of 100, 400, and 1000 mg/kg/day, cataracts and lens degeneration were observed in the eyes of high dose males. In the same study pancreatic acinar cell atrophy was observed in high dose males (Stout 1990). Pancreatic inflammation was also observed in high dose male rats in a subchronic study where animals were dosed nominally at 50, 250 and 1000 mg/kg/day (Stout 1987). In a mouse carcinogenicity study tested at 150, 1500 and 4500 mg/kg/day, ovary weights were increased in high dose females (Knezevich 1983).

General Toxicity:

Decreased body weights were observed in the high dose male rats in the combined chronic/carcinogenicity study and high dose male and female mice in the carcinogenicity study (Stout 1990 and Knezevich 1983). The decreased body weight was also similarly observed at the high doses in a reproductive and developmental studies.

Reproductive Toxicity:

In a developmental study dosing rats at 300, 1000 and 3500 mg/kg/day, reduced implantations and fewer live fetuses were observed in the high dose maternal animals (Rodwell 1980).

Developmental Toxicity:

In a developmental study dosing rats at 300, 1000 and 3500 mg/kg/day, unossified sternebra were observed in the high dose fetal animals. Reduced fetal weight was also observed in the high dose group (Rodwell 1980).

Developmental Neurotoxicity:
No developmental neurotoxicity effects were observed.

REFERENCES

Atkinson, J (1991a) A Six Month Oral Study of Ethyl Parathion in Dogs with Specific Emphasis on Ocular Effects: Final Report: Lab Project Number: 89-3439. Unpublished study prepared by Bio/ Dynamics, Inc. 504 p. MRID 41836601.

Atkinson, J (1991b) A Three Month Oral Toxicity Study in Rats via the Diet with Ethyl Parathion to Investigate Ocular Effects and Cholinesterase Activity: Lab Project Number: 89-3469. Unpublished study prepared by Bio/dynamics, Inc. 261 p. MRID 41834501.

Barnet, J.F. (2005). Oral (gavage) developmental neurotoxicity study of Tetrachlorvinphos in CrI:CD[®] (SD)IGS BR VAF/Plus[®] rats. CR-DDS Argus Division, Horsham, PA. Laboratory Project ID: CR-DDS Argus Protocol Number: 1608-003, September 9, 2005. MRID 46660601.

(1978). BIOASSAY OF TETRACHLORVINPHOS FOR POSSIBLE CARCINOGENICITY (CAS No. 961-11-5)..
Study No. NCI-CG-TR-33.

Daly, I (1996). A 24-Month Oral Toxicity/Oncogenicity Study of Malathion in the Rat via Dietary Administration: Final Report: Lab Project Number: 90-3641: J-11 90-3641. Unpublished study prepared by Huntingdon Life Sciences. 5666 p. MRID 43942901.

DER Memo, Katz to Zendzian re Parathion; Chronic Feeding Study in Rats; Light and Electron Microscopic Examination. MRID 4088701.

Eiben, R (1986). Parathion: Study for Chronic Toxicity and Cancerogenicity in Wistar Rats (Administration in Diet for Twenty-six Months): Report No. 16305. Unpublished study prepared by Bayer Ag. 1753 p. MRID 40644704.

Fulcher, S. M (2002a) Malathion. Developmental neurotoxicity study in the CD rat by oral gavage administration. Huntingdon Life Sciences, Ltd., Woolley Road, Alconbury, Huntingdon, Cambridgeshire, PE28 4HS, England. Laboratory report # CHV/066:013331. MRID 45646401.

Fulcher, S (2002b) Malathion: Dose Finding Study in CD Rats by Oral Gavage Administration Preliminary to Developmental Neurotoxicity Study: Lab Project Number: CHV/062. Unpublished study prepared by Huntingdon Life Sciences, Ltd. 450 p. MRID 45627001.

Hardisty, J (2000). Pathology Working Group (PWG) Peer Review of Proliferative Lesions of the Liver in Female Rats in a 24-Month Oral Toxicity/Oncogenicity Study of Malathion: Lab Project Number: 297-006. Unpublished study prepared by Experimental Pathology. MRID 45069401.

Harris, S.B.; Holson, J.F.; Fite, K.R.; et al (1981). A Teratology Study of Diazinon (CAS Number 333-41-5). in New Zealand White Rabbits: CGA/SAI 281005. (Unpublished study, including submit- ter summary, received Aug 27, 1981 under 100-524). MRID 00079017.

Infurna, R (1985). A Teratology Study of Diazinon Technical in Charles River Rats: Report No. 52-83. Unpublished report prepared by Ciba-Geigy Corp. 264 p. MRID 00153017.

Knezevich, A.; Hogan, G (1983). A Chronic Feeding Study Of Glyphosate (Roundup Technical) In Mice: Project No. 77-2061: Bdn-77- 420. Final Rept (Unpublished Study Received Aug 17, 1983 Un- Der 524-308; Prepared By Bio/Dynamics, Inc. MRID 00130406.

Lochry, E (1989). A Development Toxicity Study with AC 6,601 in Rats: Argus Research Laboratories Protocol 101-005. Unpublished study prepared by Argus Research Laboratories, Inc. 222 p. Study No. 971-88-142. MRID 41160901.

Mandella, R.C (2003). Diazinon: a developmental neurotoxicity study in rats. Huntingdon Life Sciences, Mettlers Road, East Millstone, New Jersey. Laboratory study no. 01-4532; November 17, 2003. MRID 46195601. Unpublished.

Mandella, R.C (2002). Diazinon: a dietary range-finding developmental neurotoxicity study in rats. Huntingdon Life Sciences, Mettlers Road, East Millstone, New Jersey. Laboratory study no. 01-4530; November 13, 2002. MRID 45842601. Unpublished.

Neeper-Bradley, T (1990). Two Generation Reproduction Study of Ethyl Parathion Technical Administered in the Diet to CD (Sprague-Dawley) Rats: Lab Project Nos. 52-630: 88-88-42001; 88- 88-42002. Unpublished study prepared by Bushy Run Research Center. MRID 41418501.

Rodwell, D.E.; Tasker, E.J.; Blair, A.M.; Et Al (1980). Teratology Study In Rats: Irdc No. 401-054 (Unpublished Study Including Irdc No. 999-021; Received May 23, 1980 Under 524-308; Prepared By International Research And Development Corp. MRID 00046362.

Rudzki, M.; McCormick, G.; Arthur, A (1991). Diazinon (MG-8).: 52- Week Oral Toxicity Study in Dogs: Lab Project Number: 882014. Unpublished study prepared by Ciba-Geigy. 621 p. MRID 41942001.

Schroeder, R (1990). A Two-generation (Two-litters) Reproduction Study with AC 6,601 to Rats: Laboratory Report No.: 87-3243. Unpublished study prepared by Bio/dynamics, Inc. 2025 p. MRID 41583401.

Siglin, J., et al (1985). A Teratology Study with AC 6,601 in Rabbits: FDRL Study No. 8171. Unpublished study prepared by Food and Drug Research Laboratories. 204 p. MRID 00152569.

Singh, A (1988). Diazinon (MG-8).: 90-Day Oral Toxicity Study in Rats: Project ID 882011. Unpublished study prepared by Ciba-Geigy Corp. 641 p. MRID 40815003.

Slauter, R (1994). 18-Month Oral (Dietary) Oncogenicity Study in Mice: Malathion: Lab Project Number: 668-001. Unpublished study prepared by International Research and Development Corp. 1454 p. MRID 43407201.

Stamper, CR, W Balduini, SD Murphy & LG Costa. 1988. Behavioral and Biochemical Effects of Postnatal Parathion Exposure in the Rat. *Neurotoxicology and Teratology*, 10: 261-266.

Stout, L.; Johnson, C (1987). 90-day Study of Glyphosate Adminis- tered in Feed to Sprague/Dawley Rats:

Proj. ID ML-86-351/EHL 86128. Unpublished study prepared by Monsanto Agricultural Co. 267 p. MRID 40559401.

Stout, L.; Ruecker, F (1990). Chronic Study Of Glyphosate Administered In Feed To Albino Rats: Lab Project Number: Msl-10495: R.D. 1014. Unpublished Study Prepared By Monsanto Agricultural Co. 2175 P. MRID 41643801.

Timofeeva, OA et al. 2008. Persistent behavioral alterations in rats neonatally exposed to low doses of the organophosphate pesticide, parathion. Brain Res Bull., 77 (6).: 404-411.